2019 Fall
CURRENT CONCEPTS IN DERMATOLOGY

Omni Nashville Hotel
Nashville, TN
September 26-28, 2019

Amy Spizuoco, D.O., FAOCD
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Continuing Medical Education Statements

This activity will change your practice and improve patient outcomes!

Content included in AOCD’s Educational conferences will not include individually identifiable health information, in accordance with the Health Insurance Portability and Accountability Act (HIPAA), as amended.

The American Osteopathic College of Dermatology (AOCD) is accredited by the Accreditation Council for Continuing Medical Education (ACCME) to provide continuing medical education for physicians.

The American Osteopathic College of Dermatology AOCD designates this live activity for a maximum of 21 AMA PRA Category 1 Credit(s)™. Physicians should claim only the credit commensurate with the extent of their participation in the activity.

The American Osteopathic College of Dermatology is accredited by the American Osteopathic Association to provide osteopathic continuing medical education for physicians.

The American Osteopathic College of Dermatology designates this program for a maximum of 21 AOA Category 1-A credits and will report CME and specialty credits commensurate with the extent of the physician’s participation in this activity.

This meeting will provide a diversified CME presentation focusing on the art and science of dermatology. Information will be presented through lectures and scientific paper presentations. The activity actively encourages members to develop enduring materials as an evolving tool for continuing education. The College is committed to exploring the development of its capacity to expand resources in other educational techniques, including Web-based activities and point-of-care technologies.
American Osteopathic College of Dermatology
Mission Statement &
Continuing Medical Education Needs Assessment

The Continuing Medical Education Program of the American Osteopathic College of Dermatology will support, enhance and advance new models of academic excellence and community health care.

Vision, Mission & Values
The vision of the American Osteopathic College of Dermatology is to advocate for our members and patients.
The mission of the American Osteopathic College of Dermatology is to create innovative education, support, and opportunities in dermatology that promote excellence in patient care and community health through advocacy, consciousness, inclusivity, and osteopathy.
The American Osteopathic College of Dermatology will instill the following values in all of our activities:
1. Inclusivity for all members
2. Consciousness of dermatologic issues
3. Excellent patient outcomes
4. Promotion of life-long learning

Purpose
The purpose of the CME program is to provide AOA-accredited continuing medical education activities to inform the dermatologist physician. The program will provide a mechanism by which its constituents can improve competency, maintain board certification and cultivate lifelong learning. CME will provide physicians with the opportunity to further develop their knowledge through individual and group learning activities. The Continuing Medical Education Committee will monitor the quality of all programs conducted by the AOCD.

Accreditation:
Content included in AOCD’s Educational conferences will not include individually identifiable health information, in accordance with the Health Insurance Portability and Accountability Act (HIPAA), as amended.
The American Osteopathic College of Dermatology (AOCD) is accredited by the Accreditation Council for Continuing Medical Education (ACCME) to provide continuing medical education for physicians.
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Commercial Support Disclosure
AOCD CME will identify relevant financial relationships prior to awarding AOA Category 1A and/or AMA PRA Category 1 Credit™ for CME activities. All persons in a position to influence or control CME content (course
directors, program planning committee members, speakers, authors and staff) will complete a standardized disclosure form. Information about funding will be requested to identify CME activities at higher risk for commercial bias.

All AOCD CME activities will be evaluated by learners and possibly peer reviewers to determine if the content was free of commercial bias. All those identified as having influence and/or control of CME content perceived as either manifesting conflicts of interest or being biased may be disqualified from consideration as resources (planning group member, authors, faculty, etc) in subsequent CME activities.

Learners will be provided with information on identified COI from any of the above categories of persons that affect the content of CME, and that information will be positioned in course materials such that it is read by learners prior to the execution of the CME activity. Speakers for the AOCD will be required to provide disclosure information to meeting attendees during their introduction of their topic. Additionally, disclosure statements are provided in the program schedule given to each meeting attendee and is available online at www.aocd.org.

In accordance with the ACCME’s Standards for Commercial Support of Continuing Medical Education, the Policy on Collection of Financial Relationships and Resolution of Conflicts of Interest (COI) exists to provide guidance for staff, instructors, planners, reviewers and managers of CME activities sponsored by The American Osteopathic College of Dermatology, (AOCD). This policy addresses the underlying philosophy of disclosure to learners, mechanisms to collect disclosure information and the parties from whom financial disclosure shall be collected, the mechanisms to resolve COI, and requirements to make disclosure to learners prior to the start of an activity.

Professional Practice Gap Statement:
Physicians need to understand, update and manage changes in dermatology in order to provide optimal patient care. Dermatologists in private practice may not have immediate access to new updates in therapies and treatments. This activity will help to close gaps in physician’s areas of surgical dermatology, oral dermatology, psoriasis treatment updates, dermoscopy, pediatric dermatology, allergies, practice management and physician burnout.

Expected Outcomes:
As a result of participation in the AOCD/CME activity, practicing clinicians will improve competency; maintain specialty board certification; and cultivate lifelong learning. It is expected that attendees of this meeting will improve their diagnostic competence regarding a wide range of dermatologic conditions. In addition to increased diagnostic competence, enhanced concepts of therapy and treatment in dermatologic care will be gained for implementation in everyday practice.

• Attendees will learn a new and novel way to treat resistant melasma with compounded hydroquinone, learn to use oral tranexamic acid in the treatment of melasma, and view a case series of active patients that have been treated with high percentage hydroquinone and/or tranexamic acid.
• Attendees will learn the importance of reflectance confocal microscopy in practice and why physicians should consider the use of reflectance confocal microscopy in your practice.
• Attendees will better understand the various considerations when choosing the order of products in a regimen, learn which ingredients affect the efficacy and side effects of products in a regimen, and learn the order in which to apply products in a regimen to improve efficacy.
• Attendees will be able to better recognize bacterial infections and better recognize and treat fungal infections.
• Attendees will learn how to order labs effectively, treat psoriasis effectively, and treat hidradenitis effectively.
• Attendees will be able to identify the potential sources of burnout that exist with the system that allows patients to review medical practices like consumers, recognize behavioral patterns and other warning signs of patient dissatisfaction that could lead to compromising a practice’s reputation, and maximize strategies to optimize the relationship with patients that lead to satisfaction in addition to improved outcomes.
• Attendees will learn about new developments of chronic inflammatory skin disorders, modes of action related to specific newer therapies that may contribute to therapeutic benefit and/or adverse effects, and newer therapeutic agents and their potential advantages as part of the therapeutic armamentarium.
• Attendees will learn about homeopathic sodium chloride, a readily available over-the-counter medicine, which can be very helpful in the treatment of acne, warts, molluscum, atopic eczema and hyperhidrosis for best results. Attendees will be able to better recognize the patient’s signs and symptoms which match the clinical criteria for the “sodium chloride” patient type and the sodium chloride clinical criteria. Successfully treated patient cases will be presented.
• Attendees will learn to better recognize when is further evaluation of urticarial necessary, when is allergy testing important and what kind of testing is necessary, and what treatments exist for urticarial and to know when to use them.
• Attendees will review evidence-based, PubMed sources focused upon dermatology, misleading advertisement and social media, and side effects of cannabis oil.
• Attendees will learn about 30 years of research proving antioxidants can be quite harmful, new studies which show antioxidants promote metastatic melanoma, and learn how antioxidants may promote skin cancer more in women than men.
• Attendees will learn evidence for and against popular dermatological uses of oils, gain a better awareness of side effects, and develop broad-based knowledge to communicate with patients that prefer oils to standard treatment.
• Attendees will be able to formulate management plans for patients with pigmented facial lesions on sun damaged skin, describe how the dermatoscope can be incorporated into daily practice, and identify the benefits of utilizing the dermatoscope to aid in diagnosis.
• Attendees will explore state rules and regulations, review compliance mechanisms, and review medico-legal issues.
• Attendees will gain a better understanding of basic HIPAA requirements and identify potential business risks associated with HIPAA, how to minimize HIPAA liability, including an overview of the severe government fines, and proper HIPAA breach response.
• Attendees will better understand the pathogenesis of atopic dermatitis and various treatment options for the disease, gain increased comfort in the treatment options for pediatric psoriasis and learn about options for common issues in kids like molluscum and warts.

The overall result being improved physician/provider performance and increased positive patient outcomes. These objectives will be achieved in a setting which is evidence-based, culturally sensitive and free of commercial bias. The AOCD is committed to the practice of continuing program improvement. The AOCD will actively explore new educational technologies, develop collaborative relationships with other CME providers and seek to build the capacity to evaluate competency-based outcomes among the clinicians we serve. CME will provide physicians with the opportunity to further develop their knowledge through individual and group learning activities.

**Needs Assessments:**
The activity was developed based upon the needs of physicians within the association identified through:
• An evaluation/survey provided to meeting participants at both our annual and midyear meeting
• Consensus of faculty members within a department or service area
• New advances in dermatologic treatment identified in major publications or research studies
• New methods of diagnosis or treatment
• Availability of new medication(s) or indication(s)
• Development of new technology
• Acquisition of new facilities or equipment
• Input from experts regarding advances in medical knowledge
• Legislative, regulatory, or organizational changes effecting patient care
• Epidemiological data
• Quality assurance/audit data
• Statistics infection control data
• Surgical procedures statistics
• Journal articles/literature citations

The AOCD Continuing Medical Education Committee works to assure the inclusion of appropriate Osteopathic content in the Continuing Medical Education activities presented by AOCD, and to assure that the Continuing Medical Education Programs of the AOCD will achieve the stated objectives of each meeting in a setting which is evidence-based, culturally sensitive and free of commercial bias.

The Continuing Medical Education Committee of the AOCD will monitor the quality of all activities conducted.
Content Areas:
The AOCD approves the CME activities based upon needs assessment data to ensure that all offerings present current, up to date and cutting edge information. Specific areas of emphasis include, new advances in dermatologic treatment, new methods of diagnosis or treatment, availability of new medication(s) or indication(s), development of new technology, advances in medical knowledge and legislative, regulatory, or organizational changes effecting patient care. The Osteopathic Core Competencies of Osteopathic Philosophy, Principles, Practice and Manipulative Medicine, Medical Knowledge, Patient Care, Interpersonal and Communication Skills, Professionalism, Practice-Based Learning and Improvement and System-Based Practice will also be incorporated into all CME activities.

Target Audience:
The primary target audience of the CME activities conducted by the AOCD are the dermatologist physician members. The College also serves community physicians, volunteer clinical faculty, academic clinicians and students affiliated with the AOCD. The activity will also actively seek to broaden its audience through developing affiliations with CME providers on the national level.

Faculty Disclosure:
As a sponsor accredited by the AOA, it is the policy of the AOCD to require the disclosure of anyone who is in a position to control the content of an educational activity. All relevant financial relationships with any commercial interests and/or manufacturers must be disclosed.

AOCD Anti-Trust Statement
Members participating in meetings, events or activities conducted or sponsored by the American Osteopathic College of Dermatology or the Foundation for Osteopathic Dermatology, have an obligation to review and follow the AOCD’s Antitrust Compliance Policy. They should particularly refrain from making statements or distributing materials at AOCD, Foundation meetings or events that would violate the policy, such as suggesting minimum fees for particular services, urging AOCD members to boycott third party payers based on reimbursement levels or other terms of contracting with such entities, or recommending that AOCD members avoid competing with each other in certain geographic areas or markets or across specialties.

Disclosure of Commercial Support of CME:
As you undoubtedly know from the national media, there has been much discussion concerning the relationships between CME sponsors, faculty and commercial companies providing support of CME.

Both the American Osteopathic Association and the Committee on Continuing Medical Education have adopted regulations for ethical actions in this area which the American Osteopathic College of Dermatology endorse and have adopted for all our educational activities.

Please be assured that having an affiliation with a company does not imply in any way that something is wrong or improper; however, we want to inform attendees that such a relationship exists.

Should you have any questions regarding the facilities, handouts, activity content, or concerns about CME compliance with the AOA “Uniform Guidelines,” feel free to contact the AOCD representative:

Marsha A. Wise, BS
Executive Director
P.O. Box 7525
Kirksville, MO 63501
660-665-2184
800-449-2623

Unresolved issues regarding compliance with the AOA “Uniform Guidelines” can be brought to the attention of the AOA Division of CME by calling: 800-621-1773, or by writing:

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142 East Ontario Street
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Disclosures: Physician Training: Sensus Healthcare; Medical Officer/Minority Shareholder: SkinCure Oncology; Director, Officer or Employee of: Shade Project; Spouse is Director, Officer or Employee of: Shade Project

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Disclosures: No relevant financial relationships to disclose
Meeting Faculty & Needs Assessments

Amy Spizuoco, DO, FAOCD – Program Chair
Dr. Amy Spizuoco is a board-certified dermatologist and dermatopathologist. She received her Bachelor of Arts at SUNY Binghamton with a double major in Italian and Biology. She earned her medical degree at New York College of Osteopathic Medicine. She completed a medical internship at Lutheran Medical Center. She then went on to Alta Dermatology Residency Program in Mesa, Arizona where she spent a year researching reflectance confocal microscopy, and subsequently, completed her dermatology residency. During residency, she received training at the Mayo Clinic Scottsdale, as well as Phoenix Children’s Hospital. She was named chief resident in her last year of residency. After residency, Dr. Spizuoco completed a fellowship in dermatopathology.

Currently Dr. Spizuoco is a member of the American Academy of Dermatology, the American Osteopathic College of Dermatology, the American Society for Dermatopathology, the American Society of Mohs Surgery, the American Society for Dermatologic Surgery, the New York State Osteopathic Medical Society, the Women's Dermatologic Society and the Dermatologic Society of Greater New York.

Derrick Adams, DO, FAOCD
Growing up in Oklahoma, he earned his Bachelor of Science in Physiology at Oklahoma State University. After graduation he was commissioned into the U.S. Air Force and completed his internship in internal medicine at Wilford Hall Medical Center in San Antonio, TX. Dr. Adams was stationed at Travis Air Force Base where he served as a Captain and General Medical Officer at David Grant Medical Center. After his tour of duty, Dr. Adams spent three intensive years at Michigan State University, where he completed his residency in dermatology with a special focus in dermatological surgery.

Dr. Adams has a special interest in skin cancer and medical dermatology, authoring a wide range of articles across dermatology, pharmacology and dermatological surgery. He treats all diseases of the skin to include: psoriasis, eczema and autoimmune diseases. As the field of dermatology arose from within internal medicine and Dr. Adams’ approach to the skin takes into account the global health and well-being of the patient.

A particular interest for Dr. Adams is evaluating the cost/benefit ratio and science behind treatments. He prides himself on being the “Consumer Reports” of dermatology and not over-treating patients.

Hey, Doctor, What About CBD Oil?

Objectives:
1. Evidence-based, PubMed review focused upon dermatology
2. Misleading advertising and social media
3. Side effects of cannabis oil

Needs:
1. New advances in dermatologic treatment

References:

Core Competencies: 6

Antioxidants: The Enemy of My Enemy?

Objectives:
1. 30 years of research proves antioxidants can be quite harmful
2. New studies show antioxidants promote metastatic melanoma
3. Antioxidants may promote skin cancer more in women than men
 Needs:
1. Advances in medical knowledge

References:

Core Competencies: 2, 3

**Essential Oils: Is There a Scent of Evidence?**

Objectives:
1. Learn evidence for and against popular dermatological uses of oils
2. Raise awareness of side effects
3. Raise broad based knowledge to communicate with patients that prefer oils to standard treatment

Needs:
1. New advances in dermatologic treatment
2. Advances in medical knowledge
3. Legislative, regulatory, or organizational changes effecting patient care

References:

Core Competencies: 2, 3, 6

Disclosures: No relevant financial relationships to disclose.

**Leslie Baumann, MD**

Dr. Baumann authored the first textbook about cosmetic dermatology in 2002, and the second edition of *Cosmetic Dermatology: Principles and Practice* (McGraw Hill) now ranks as the bestselling dermatology textbook in the world and has been translated into 14 languages. In 2005, Dr. Baumann authored *The Skin Type Solution*, a *New York Times* bestseller that has been published in many languages. The latest edition of *The Skin Type Solution* was published in December 2010 to coincide with the PBS special, “Skin Type Solutions with Dr. Leslie Baumann,” which began airing in late 2010. Dr. Baumann's latest textbook, *Cosmeceuticals and Cosmetic Ingredients* (McGraw Hill) was released in November 2014. Dr. Baumann also pens a bi-monthly column in *The Miami Herald* and regularly contributes to magazines, trade publications and medical journals.

**Cosmeceuticals: The Science of Designing a Skincare Regimen**

Objectives:
1. Understand the various considerations when choosing the order of products in a regimen
2. Learn which ingredients affect the efficacy and side effects of products in a regimen
3. Learn what order to apply products in a regimen to improve efficacy

Needs:
1. Availability of new medication(s) or indication(s)
2. Development of new technology
3. Advances in medical knowledge

References:
Do You Know JAK? New Applications for Janus Kinas Inhibitors in Dermatology

Objectives:
1. Identify the mechanisms of action of Janus Kinas Inhibitors and the potential applications for dermatology
2. Recognize the balance between efficacy and safety of JAK Inhibitors including routine lab monitoring and assessments
3. Discuss new developments in clinical research

Needs:
1. New advances in dermatologic treatment
2. Availability of new medication(s) or indication(s)
3. Development of new technology

References:

Core Competencies: 2, 3

Patients vs. Doctors: One-Star Reviews, Yelp and Social Media

Objectives:
1. Identify the potential sources of burnout that exist with the system that allows patients to review medical practices like consumers
2. Recognize behavioral patterns and other warning signs of patient dissatisfaction that could lead to compromise of a practice's reputation
3. Maximize strategies to optimize the relationship with patients that lead to satisfaction in addition to improved outcomes

Needs:
1. Advances in medical knowledge
2. Legislative, regulatory, or organizational changes effecting patient care

References:

Core Competencies: 2, 3, 4, 5, 6, 7

Disclosures: Advisor, Consultant, Investigator, Speaker: Abbvie, Activis, Allergan, Aqua, Bayer, Biofrontera, BiopharmX, Castle, Cipher, Dermira, Encore, Exeltis, Ferndale, Foamix, Galderma, Intraderm, ISDIN, LaRoche-Posay, Leo, Novan, Novartis, PharmaDerm, Pfizer, Promius, Regeneron, Sanofi, SunPharma, Valeant

Lloyd Cleaver, DO, FAOCD
Dr. Lloyd Cleaver, DO founded the Cleaver Dermatology Clinic in 1986. Dr. Cleaver completed his internship and residency at the Navy Regional Medical Center in San Diego, CA. He is a graduate of Kirksville College of Osteopathic Medicine. He is also a board-certified dermatologist, Fellow of American Osteopathic College of Dermatology and board-certified in Mohs micrographic surgery.

A leader in medical education, Dr. Cleaver is a Professor of Dermatology at the Kirksville College of Osteopathic Medicine/A.T. Still University and Assistant Dean of Continuing Medical Education at the Kirksville Osteopathic Medical Center/A.T. Still University. He serves as Vice Chair for the Certification Committee of American Osteopathic Association and has been Vice Chair and is currently Secretary to the American Osteopathic Board of Dermatology. He is a Past President of the Kirksville Osteopathic Alumni Association and a Past President of American Osteopathic College of Dermatology.

Osteopathic Continuous Certification Update

Objectives:
1. Describe the objectives of maintenance of Osteopathic Continuous Certification
2. Review the requirements of OCC
3. Describe changes in OCC

Needs:
1. Legislative, regulatory, or organizational changes effecting patient care

References:

Core competencies: 1, 3, 5, 6

Disclosures: No relevant financial relationships to disclose.
James Del Rosso, DO, FAOCD
Dr. James Q. Del Rosso, DO, FAOCD, FAAD, was born in Brooklyn, New York, and attended pharmacy school at St. John's University in Jamaica, New York. He completed a hospital pharmacy residency at Temple University in Philadelphia, PA. Dr. Del Rosso graduated from medical school at Ohio University with honors and then completed his approved dermatology residency at the Atlantic Skin Disease Association in Fort Lauderdale, Florida.

He additionally completed a fellowship approved by the American College of Mohs Surgery in Mohs Micrographic Surgery and Cutaneous Oncology at Ohio State. Dr. Del Rosso is board certified in Dermatology and Mohs Micrographic Surgery and is fully licensed in the State of Nevada.

Dr. Del Rosso is an internationally renowned educator and speaker with several publications in recognized dermatology journals. He is a frequently invited presenter at major dermatology meetings in both the United States and globally, often talking about what is new in therapeutics and drug development. Additionally, he has written and published several articles on a variety of skin diseases including proper care of the skin barrier, acne, rosacea, psoriasis, atopic dermatitis, and eczemas, actinic keratosis, and skin cancers. He is also the co-editor of the textbook, *Acne Vulgaris*.

He is a past president of the American Acne & Rosacea Society, the American Society for Mohs Surgery, and the American Osteopathic College of Dermatology. He is editor-in-chief of the *Journal of Clinical Aesthetic Dermatology* since its inception in 2005. He has also authored several recognized publications on guidelines for management of acne, rosacea, atopic dermatitis, psoriasis, and skin cancer.

He founded the Scientific Panel for Antibiotic Use in Dermatology in 2005, with three meetings and scientific publications authored by the group under his direction to guide clinicians on optimal antibiotic use.

Dr. Del Rosso is research director and principal investigator of JDR Dermatology Research, in addition to having his dermatology clinic that is fully dedicated to the care of people with skin disorders or have concerns about their skin. The research center conducts studies for a wide variety of skin conditions, such as acne, rosacea, psoriasis, eczemas, urticaria, actinic keratosis, and skin cancer. Dr. Del Rosso has over 25 years of experience in dermatology research and related publications, and he employs a highly experienced and skilled staff. Dr. Del Rosso is in dermatology practice at Thomas Dermatology in Las Vegas, Nevada. He has been practicing dermatology in the Las Vegas area for 20 years.

Dr. Del Rosso was the recipient of a lifetime achievement award by the American Academy of Dermatology (AAD) in March 2016 for his lifelong commitment to dermatology and his contributions to the field. He now has the distinguished title of Honorary Member with the Academy.

Outside of his professional activities, Dr. Del Rosso is an avid lover of music and sports. He is the proud owner of what can be considered to be a “museum” of sports memorabilia and rock and roll music memorabilia, including a collection of limited edition guitars and vintage guitars. He also attends many concerts, usually sitting in the first row middle section. His motto about attending concerts is admittedly a selfish one: “The concert is being put on specifically for me…I am just nice enough to let everyone else in”. In 2015, Dr. Del Rosso recorded a CD entitled “My Royal Dream by Dr. D and the LL-7”, at the legendary Royal Studios in Memphis Tennessee. The producer and recording engineer of this CD was Lawrence “Boo” Mitchell, who has recorded several major artists, and won a Grammy Award for being the recording engineer for “Uptown Funk” by Bruno Mars, which was also recorded at Royal Studios. Dr. Del Rosso is proud to say he is a good father and a “very cool” grandfather. The loves of his life are his wife Karyn, his daughter Jaclyn, his sister Marilyn and brother-in-law Pat, his stepchildren Chrsytyna and Ron, his son-in-law David, and his grandchildren Allyson, Blake, Emily, and Asher. He is also thankful for having had great parents and loves his inner circle of great loyal friends. Dr. Del Rosso is proud and happy to say, “I have no bucket list. I have done everything I have wanted to do and I believe it has always been done well. It is time for the icing on the cake at this point in my life. I do what I enjoy and get great pleasure in giving back through personal generosity and the professional work that I do. My hope is to record another CD with Boo Mitchell. That was the greatest week of my life”.

*What's New in the Medicine Chest? A Therapeutic Update*

**Objectives:**
1. Discuss new developments in management of chronic inflammatory skin disorders such as atopic dermatitis, psoriasis, acne, and rosacea and also infectious disorders (i.e. warts, molluscum)
2. Explain modes of action related to specific newer therapies that may contribute to therapeutic benefit and/or adverse effects
3. List specific newer therapeutic agents and their potential advantages as part of the therapeutic armamentarium.

**Needs:**
1. New advances in dermatologic treatment
2. New methods of diagnosis or treatment
3. Availability of new medication(s) or indication(s)
4. Development of new technology
5. Advances in medical knowledge

**References:**

**Core Competencies:** 2, 3, 6, 7

**What Laboratory Tests Are Important to Order?**

**Objectives:**
1. Discuss new developments in management of chronic inflammatory skin disorders such as atopic dermatitis, psoriasis, acne, and rosacea and also infectious disorders (i.e. warts, molluscum)
2. Explain modes of action related to specific newer therapies that may contribute to therapeutic benefit and/or adverse effects
3. List specific newer therapeutic agents and their potential advantages as part of the therapeutic armamentarium

**Needs:**
1. New advances in dermatologic treatment
2. New methods of diagnosis or treatment
3. Availability of new medication(s) or indication(s)
4. Development of new technology
5. Advances in medical knowledge

**References:**

**Core Competencies:** 2, 3, 6, 7

**Disclosures:** Aqua/Almirall, Celgene, Encore, Epi Health, Ferndale, Galderma, Genentech, Leo Pharma, Ortho, Pfizer, Regeneron, Sanofi-Genzyme, Sun Pharma, Taro. Athenex, BioPharma, Botanix, Celgene, Dermira, Epi Health, Foamix, Novan, Dermira, La Roche Posay, Sonoma (Intraderm)

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**Dirk Elston, MD**

Dr. Elston is Professor and Chairman of the Department of Dermatology and Dermatologic Surgery at the Medical University of South Carolina, in Charleston. He is a past-president of the American Academy of Dermatology and the American Society of Dermatopathology, has served on the board of directors of the International Society of Dermatopathology and the American Board of Dermatology, and serves as the Editor of the Journal of the American Academy of Dermatology. He is an Honorary Professor at China Medical University in Shenyang China and Guest Professor at the Xiangya School of Medicine, Central South University in Changsha, China, and is an honorary member of the German Society of Dermatology.

Dr. Elston is a graduate of Jefferson Medical College, did his dermatology residency at Walter Reed Medical Center and a dermatopathology fellowship at the Cleveland Clinic. He is the author of over 400 peer reviewed publications, is one of 3 authors of *Andrews Diseases of the Skin*, Associate Editor in Chief of *eMedicine* dermatology, and Editor in Chief of the *Requisites in Dermatology* series of textbooks. He received the 2008 Walter Nickel Award for Excellence in Dermatopathology Education and the 2013 Founder’s Award of the American Society of Dermatopathology. The first edition of his dermatopathology textbook, written together with Tammie Ferringer, received the Highly Commended Award in the British Medical Association Medical Book competition. The third edition is now in press.

**JAAD Update**

**Objectives:**
1. Order labs effectively
2. Treat psoriasis effectively
3. Treat hidradenitis effectively

**Needs:**
1. New advances in dermatologic treatment
2. New methods of diagnosis or treatment
3. Advances in medical knowledge

**References:**

**Core Competencies:** 2, 3

**Cutaneous Infections**

**Objectives:**
1. Recognize bacterial infections
2. Recognize fungal infections
3. Treat fungal infections
Jill Fichtel, MD

Jill Fichtel, MD is an American Board of Dermatology certified physician with clinical expertise and specialization in dermatologic surgeries and cosmetic dermatology. Dr. Fichtel is a graduate of the University of Tennessee. She went on to receive her Doctor of Medicine in 2001 from the University of Tennessee College of Medicine. She completed her dermatology residency at the Medical College of Georgia in 2005.

Early on, Dr. Fichtel realized she became fulfilled as she embraced the challenges of cosmetic dermatology and has a talent that must be shared. Her true passion lies in restoring patient's natural beauty and seeing the transformation they make visually and mentally along their beauty journey.

She started her own practice in Nashville, TN called Transformative Dermatology in 2019. She is also a Top Doctor for RealSelf. Dr. Fichtel is married to Dr. Matthew Zirwas.


In Dr. Fichtel's free time she enjoys spending time with her family, reading a good book, and traveling to fun destinations with her husband, Dr. Matthew Zirwas.

**Cosmetic Procedures and Contact Dermatitis**

**Objectives:**
1. New and cutting edge cosmetic procedures
2. On label and off label cosmetic procedures

**Needs:**
1. New advances in dermatologic treatment
2. New methods of diagnosis or treatment
3. Advances in medical knowledge

**References:**
1. Andrews Diseases of the Skin. 11 Edition

**Core Competencies:** 2, 3, 4, 5, 6, 7

**Disclosures:** No relevant financial relationships to disclose.
Michael H. Gold, MD
Dr. Michael H. Gold is the founder and medical director of Gold Skin Care Center, Advanced Aesthetics Medical Spa, The Laser & Rejuvenation Center, and Tennessee Clinical Research Center in Nashville, TN. He is a board-certified dermatologist and dermatologic surgeon and oversees the various facets of the center’s operations — a combination of medical and surgical dermatology, cosmetic dermatology, aesthetic services and research endeavors, which began in 1990.

Dr. Gold has earned worldwide recognition for providing patients with leading-edge technological advances in dermatology and aesthetic skin care. He plays an integral role in the development of new pharmaceutical products and medical devices through his clinical research. He presents the results regularly at national and international dermatology and cosmetic meetings.

Dr. Gold has authored over 300 published scientific articles, 35 textbook chapters, and has edited two textbooks on Photodynamic Therapy. He serves on most major dermatology journal boards and is the current Editor-in-Chief of the Journal of Cosmetic Dermatology. In addition, Dr. Gold helped establish the Tennessee Society for Laser Medicine and Surgery (TSLMS), a group of health care providers interested in the distribution of information and proper training for those in the cosmetic arena. The TSLMS puts on an annual meeting known as SCALE, or Symposium for Cosmetic Advances & Laser Education. It is one of the leading U.S. dermatologic and aesthetic meetings.

Dr. Gold also helped start two international groups: the Dermatologic Aesthetic Surgery International League (DASIL), which aims to create a global community for the open exchange of knowledge and innovation by physicians specializing in Dermatologic and Aesthetic Surgery. It has become one of the most prominent and important international dermatology groups. It showcases meetings all over the world; and 5-Continent-Congress (5CC), is one of the world’s leading conferences on Dermatologic and Aesthetic Surgery, where he is the current President of the Congress.

What’s New in the Treatment of Hypertrophic Scars & Keloids – 2019

Objectives:
1. To understand the use of silicone gel in the treatment of hypertrophic scars and keloids
2. To understand the use of lasers in the treatment of hypertrophic scars and keloids
3. To understand the use of superficial radiation therapy in the treatment of hypertrophic scars and keloids

Needs:
1. New advances in dermatologic treatment
2. New methods of diagnosis or treatment
3. Availability of new medication(s) or indication(s)
4. Development of new technology
5. Advances in medical knowledge

References: Pending

Core Competencies: 2, 3, 7

Ujwala Kaza, MD
Ujwala Kaza is a board certified allergist that has been treating adults and children in New York City for over 10 years. She is a Clinical Assistant Professor at NYU’s School of Medicine where she teaches residents and medical students. She is also on staff at NYU Langone. Dr. Kaza is a Diplomate of the American Board of Allergy and Immunology. In addition, she is a fellow of both the American College of Allergy, Asthma and Immunology (ACAAI) and the American Academy of Allergy, Asthma and Immunology (AAAAI). She is a member of the New York Allergy and Asthma Society as well as the Joint Council of Allergy and Immunology.

Allergy for the Dermatologist, Parts 1 & 2

Objectives:
1. When is further evaluation of urticarial necessary?
2. When is allergy testing important and what kind of testing is necessary?
3. What treatment exist for urticarial and when do I know to use them?

Needs:
1. New methods of diagnosis or treatment
2. Availability of new medication(s) or indication(s)
3. Advances in medical knowledge

References:
2. Middle's Principles of Allergy and Immunology Textbook. 8th ed, Vol 1, Ch 36, pages 575-587.

Core Competencies: 2, 6

Disclosures: Pfizer, Astra Zeneca, Regeneron Sanofi Genzyme

Stephen Kessler, DO, FAOCD
Dr. Kessler completed his undergraduate education at Miami University in Oxford, Ohio. He then attended medical school at the Michigan State University College of Osteopathic Medicine. He became board-certified in dermatology in 1983 and established Alta Dermatology the same year. He is a frequent speaker at dermatology conferences and is the program director for the KCU-GMEC Mesa Program dermatology residency program.

Dermatologic Surgery: Lessons Learned Along the Way

Objectives:
1. Experiences that influence everyday practice
2. Developing a safer, more efficient surgery experience

Needs:
1. New advances in dermatologic treatment
2. Legislative, regulatory, or organizational changes effecting patient care

References:

Core Competencies: 2, 3, 4, 5, 6, 7

Disclosures: No relevant financial relationships to disclose.
Will Kirby, DO, FAOCD

Board-certified dermatologist, Dr. Will Kirby, has a degree in biology from Emory University. He received his medical degree from Nova Southeastern University and completed his first year of postgraduate training in internal medicine at Mount Sinai Medical Center. His dermatology residency training took place in association with Western University/Pacific Hospital where he was honored by being selected to serve as chief resident in the Department of Dermatology. Academically, Dr. Kirby proudly serves as a Clinical Assistant Professor of Dermatology at Western University of Health Science and as a Clinical Assistant Professor in the Department of Internal Medicine, Division of Dermatology, for Nova Southeastern University. He is also an expert reviewer for the Osteopathic Medical Board of California in dermatology.

Dr. Kirby lectures at national medical conventions, publishes articles in peer reviewed medical journals, authors and co-authors medical text book chapters and has the prestigious honor of serving on the editorial advisory board of popular dermatological publications including the Dermatologist and the Journal of Aesthetic and Clinical Dermatology. He has served as the national spokesman for Johnson & Johnson’s Neutrogena Dermatologics and Kimberly Clark’s Truist Skin Care lines.

A licensed osteopathic physician and surgeon in the state of California since 2002, Dr. Kirby is recognized as a Fellow by the American Osteopathic College of Dermatology. He is currently a member of the American Osteopathic College of Dermatology (AOCD) and the American Osteopathic Association (AOA). In the past, Dr. Kirby has held membership in the American Medical Association (AMA), the American Society for Laser Medicine and Surgery (ASLMS), the American Society of Dermatologic Surgery (ASDS) and the American Academy of Dermatology (AAD). Having appeared on more than 35 different television shows, Dr. Kirby was a featured physician on E! Entertainment Television’s “Dr. 90210” and has frequently been seen on “The Doctors,” where he showcases his dermatology practice. Other TV appearances have included “The Young & The Restless,” “LA Ink,” “The Real Housewives of Orange County,” “Regis & Kelly,” “The Talk” and “Chelsea Lately” and has appeared on QVC more than 100 times. Dermatologist, professor, spokesman, researcher and author, Dr. Kirby is well-recognized as one of the country’s leading dermatologists!

Creative Ways to Maximize Professional Efforts Most Effectively and Avoid Practice Pitfalls

Objectives:
1. Exploration of state rules and regulations
2. Discussion of compliance mechanisms
3. Review of medico-legal issues

Needs:
1. Legislative, regulatory, or organizational changes effecting patient care

References:
1. JAOC
2. JAOF
3. TalkRx, Five Steps to Honest Conversations that Create Connection Health and Happiness. N. Sangwan, MD.

Core Competencies: 4, 5

Disclosures: No relevant financial relationships to disclose.

Mark Lebwohl, MD

Dr. Mark Lebwohl graduated summa cum laude from Columbia College in 1974 and graduated from Harvard Medical School in 1978. He completed residencies in internal medicine and dermatology, both at Mount Sinai. Dr. Lebwohl has been practicing dermatology since 1983. He is professor and chairman of the Kimberly and Eric J. Waldman Department of Dermatology of the Icahn School of Medicine at Mount Sinai. Dr. Lebwohl is the President of the American Academy of Dermatology. Dr. Lebwohl is chairman emeritus of the medical board of the National Psoriasis Foundation. He is the founding editor of Psoriasis Forum, as well as a medical editor of the bulletin of the National Psoriasis Foundation, Psoriasis Advance. He is editor of the Dermatology Section of Scientific American Medicine. Dr. Lebwohl has chaired numerous symposia and has written, edited or co-edited several books including Psoriasis, Mild-to-Moderate Psoriasis and Moderate-to-Severe Psoriasis. He has authored or co-authored over 500 publications.
including peer-reviewed articles, invited articles and book chapters. Dr. Lebwohl is actively involved in clinical trials of many new dermatologic treatments.

**A Dermatologist’s Guide to Using Anti-IL-17 Agents to Effectively Manage Patients with Challenging Psoriatic Disease**

**A Multidisciplinary Approach to Cutaneous Squamous Cell Carcinoma**

**Objectives:**
1. Recognize advantages of anti-IL-17 therapy
2. Recognize clinical scenarios where anti-IL-17 therapy is ideal
3. Analyze the long term past improvements of these drugs compared to other psoriasis therapies

**Needs:**
1. New advances in dermatologic treatment
2. Availability of new medication(s) or indication(s)

**References:**

**Core Competencies:** 2, 3, 4, 5, 6, 7

**Disclosures:** Abbvie, Amgen, Arcutis, Bausch Health (Valeant), Boehringer Ingelheim, Celgene, Eli Lilly, Incyte, Johnson & Johnson (Centocor, Janssen), Leo Pharmaceuticals, Medimmune/Astra Zeneca, Novartis, Pfizer (anacor), Regeneron, Sciderm, UCB, Inc., and ViDac. Consultant – Allergan, Aqua, Arcutis, Inc., Boehringer-Ingelheim, Bristol-Myers Squibb, LEO Pharma, Menlo, Mitsubishi, Neuroderm, Promius, Theravance, and Verrica

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**Michael Nowak, MD**

Dr. Michael Nowak is board-certified in anatomic and clinical pathology and dermatopathology. Dr. Nowak earned a Bachelor of Science degree from Xavier University, followed by a medical degree from Wright State University. After completion of an internship in internal medicine, he served as chief resident and cancer fellow at Western Reserve Care System where he completed a residency in anatomic and clinical pathology. Dr. Nowak finished his training in Providence, RI at Brown University, where he completed fellowship training in dermatology and dermatopathology.

After his training, Dr. Nowak joined Palm Beach Dermatology in 1999. He organized the development and staffing of Palm Beach Dermatology’s CLIA-certified diagnostic dermatopathology laboratory, where he serves as Medical Director. The laboratory is equipped with the latest technology and staffed with five ASCP-certified and state-licensed technologists who specialize in dermatology samples.

Dr. Nowak has authored over 15 articles in major medical journals on a variety of subjects including malignant melanoma, extramammary Paget’s disease, generalized pruritis and infectious diseases of the skin. He is also a member of the medical staff at Columbia Hospital in West Palm Beach, FL, where he conducts weekly teaching sessions with dermatology residents and has been awarded “Dermatology Attending of the Year” by his peers at the hospital. He is also a reviewer for the journal titled the *Physician and Sports Medicine*, and is on the editorial board for the journal *Postgraduate Medicine*. Resident lectures and review of journal articles ensures that Dr. Nowak stays current with the latest advances in the science of dermatopathology. Dr. Nowak’s practice is dedicated to the evaluation and diagnosis of skin pathology samples.

**Biopsy Technique**

**Objectives:**
1. Gain a better understanding of skin biopsy techniques including helpful guidelines on choosing the best lesion(s) for biopsy and adapting the appropriate biopsy type
2. Gain a better understanding of practical considerations to avoid causing artifacts that could hinder optimal dermatopathology interpretation
3. Gain a better understanding of skin biopsy techniques in specific disease categories including inflammatory and neoplastic diseases
Needs:
1. New methods of diagnosis or treatment
2. Advances in medical knowledge

References:

Core Competencies: 1, 2, 3, 4, 5, 6, 7

What are These Stains?

Objectives:
1. Gain a better understanding of conventional special stains and immunohistochemical stains used in dermatopathology
2. Gain a better understanding of immunohistochemical stains used in specific situations or histologic patterns in dermatopathology
3. Gain a better understanding of notable associations of immunohistochemical stains and specific diagnoses

Needs:
1. New methods in dermatologic treatment
2. Advances in medical knowledge

References:

Core Competencies: 1, 2, 3, 4, 5, 6, 7

Disclosures: Modernizing Medicine

Robert Piccinini, DO

Robert G.G. Piccinini, DO, is an AOA board-certified psychiatrist in private practice.

Outside of his involvement with the AOA, Dr. Piccinini is the past president of the Michigan Osteopathic Association. He also is past president of the American College of Osteopathic Neurologists and Psychiatrists, the Macomb County Osteopathic Medical Association and the Michigan Osteopathic Service Corporation.

Dr. Piccinini did a forensic psychiatry fellowship at the Medical College of Wisconsin. He received the Distinguished Fellow award from the American College of Osteopathic Neurologists and Psychiatrists in 2007. Dr. Piccinini has given numerous presentations on domestic violence, violence in the workplace and physician wellness.

Dr. Piccinini earned his osteopathic medical degree from the Michigan State University College of Osteopathic Medicine in East Lansing. He completed an internship at Genesys Regional Medical Center followed by residency training at Henry Ford Health Systems.

Physician Wellness

Objectives:
1. Understand the difference in learning across the generations
2. Understanding balance between work (career) and home
3. Recognize avenues to gain wellness, throughout the lifespan
Needs:
1. Legislative, regulatory, or organizational changes effecting patient care

References:

Core Competencies: 1, 4, 5

Disclosures: No relevant financial relationships to disclose.

Harold Rabinovitz, MD
Harold S. Rabinovitz, M.D., is a Board Certified Dermatologist with his office located in Plantation, Florida. He performs Mohs Micrographic Surgery and Skin Surface Microscopy. Dr. Rabinovitz provides patients with the latest technology and expertise in the field of skin cancers.

Dr. Rabinovitz graduated Cum Laude from Princeton University and received his medical degree from University of Miami School of Medicine. He performed his internship at Mt. Sinai Medical Center and completed his dermatology residency at New York University Medical Center.

Dr. Rabinovitz served as Assistant Clinical Professor and as Associate Clinical Professor of Dermatology at University of Miami School of Medicine. Presently, he serves as Clinical Professor of Dermatology.

The Role of Dermoscopy in the Evaluation of Facial Lesions on Sun Damaged Skin

Objectives:
1. Formulate management plans for patients with pigmented facial lesions on sun damaged skin.
2. Describe how the dermatoscope can be incorporated into daily practice.
3. Identify the benefits of utilizing the dermatoscope to aid in diagnosis.

Needs:
1. New advances in dermatologic treatment
2. New methods of diagnosis or treatment
3. Development of new technology

References:
1. Rabinovitz H. Dermoscopy: A Practical Guide. 1999

Core Competencies: 2, 3, 4

Disclosures: 3 Gen, American Dermoscopy Meeting, Dermasensor

Babar Rao, MD
Dr. Rao is board certified in both dermatology and dermatopathology and is a leading authority on pigmented lesions, as well as a pioneer in dermoscopy and confocal microscopy.

He completed residency training and fellowships at the University of London, University of Texas Southwestern, New York University and Cornell University. Dr. Rao currently serves as the acting chair and a clinical professor of dermatology and dermatopathology in the department of dermatology at Rutgers Robert Wood Johnson Medical School.

Dr. Rao is also an associate clinical professor of dermatology at Weill Cornell Medical College at Cornell University.

Reflectance Confocal Microscopy
Objectives:
1. Learn the importance of reflectance confocal microscopy in your practice
2. Why physicians should consider the use of reflectance confocal microscopy in your practice

Needs:
1. New methods of diagnosis or treatment
2. Development of new technology
3. Advances in medical knowledge

References:

Core Competencies: 2, 3, 4, 5, 6

Disclosures: Celgene, L’Oreal, Novartis, Caliber ID, Aire, NIDIskin

Leslie Rojas, JD
Leslie A. Rojas, JD, is an attorney who focuses her practice on healthcare regulatory, transactional and corporate matters. Ms. Rojas devotes a substantial portion of her practice to HIPAA and state privacy and security law matters, healthcare fraud and abuse issues, healthcare joint ventures and business transactions, practitioner employment agreements, and Medicare enrollment, billing and reimbursement issues.

Ms. Rojas represents a variety of healthcare businesses and professionals, including physicians and other practitioners, group practices, laboratories, imaging centers, pharmacies, hospitals, home health agencies, assisted living facilities, and healthcare compliance consultants. Through collaboration with other attorneys and law firms, Ms. Rojas ensures that her healthcare clients receive full-service legal representation, including representation related to real estate law, tax law, intelecton property law, litigation services, etc.

Ms. Rojas currently serves on the Governing Council for the Health Care Law Section of the State Bar of Michigan, and serves on the Health Care Law Section’s Medical Legal Subcommittee and Technology Subcommittee. She is also a committee member for the American Health Lawyer Association’s Physician Organization Practice Group. Additionally, Ms. Rojas has authored many articles for the American Bar Association’s Health Law Section.

In her spare time, Ms. Rojas serves as the President of the Paraguayan-American Association of Physicians, a non-profit charity that raises money for the medical school in Asuncion, Paraguay, as well as for other causes in Latin America. Ms. Rojas also serves as Vice-President of the Mental Illness Research Association, which raises money for mental illness related research grants and mental illness educational presentations in secondary schools throughout Michigan and Ohio.

Minimizing HIPAA Liability

Objectives:
1. An understanding of basic HIPAA requirements and how to identify potential business risks associated with HIPAA
2. How to minimize HIPAA liability, including an overview of the severe government fines
3. Proper HIPAA breach response

Needs:
1. Legislative, regulatory, or organizational changes effecting patient care
Robert Signore, DO, FAOCD
Dr. Signore was born in Blue Island, IL and was raised on the southwest side of Chicago. After graduating from Bogan High School, he studied at Loyola University of Chicago, where he graduated in 1982 with a bachelor's degree in biology. Dr. Signore attended medical school in Des Moines, IA at the University of Osteopathic Medicine and Health Sciences (now known as Des Moines University) and graduated in 1986 with his D.O. degree.

He completed a rotating internship at Pontiac Osteopathic Hospital in Pontiac, MI. Dr. Signore then completed a family practice residency at St. Joseph Hospital in Flint, MI. He returned to Pontiac Osteopathic Hospital for his dermatology residency, which he completed in 1992. During residency, he received the P.O.H. Resident of the Year Award. Additionally, he trained with John von Weiss, MD at the von Weiss Skin Center in Salem, MA.

Dr. Signore is board-certified in dermatology by the American Osteopathic Board of Dermatology. Dr. Signore has also studied classical homeopathic medicine through the British Institute of Homeopathy (USA). He received his Diploma of Homeopathy (DiHom) from the British Institute of Homeopathy (USA) in 2009. He has implemented classical homeopathic medicine into his dermatology practice for the treatment of patients with skin conditions.

One Simple Homeopathic Medicine That Can Help Our Dermatology Patients

Objectives:
1. Homeopathic sodium chloride is a readily available, over-the-counter medicine which can be very helpful in the treatment of acne, warts, molluscum, atopic eczema, and hyperhidrosis
2. For best results, the patient’s signs and symptoms should match the clinical criteria for the “sodium chloride” patient type
3. The sodium chloride clinical criteria will be discussed and successfully treated patient cases will be presented

Needs:
1. New advances in dermatologic treatment
2. Advances in medical knowledge

References:

Stacy Spizuoco, DDS
Dr. Stacy Spizuoco is a graduate of New York University College of Dentistry. She works in private practice, serves as a clinical instructor at the Columbia University College of Dental Medicine and performs charitable work including annual dental missions to La Romana, DR, as well as local missions such as Give Kids a Smile Day. Dr. Spizuoco is a member of the ADA, the American Association for Women Dentists, New York Academy of Dentistry, Fellow of American College of Dentists and various other dental organizations.
What she loves about dentistry: “I fell in love with dentistry because it’s a unique combination of art and science that calls for creativity and technical dexterity. Equally as unique are the patients who require practical and personal solutions for their dental needs. Having the ability to bridge this divide by providing esthetic dental solutions is my passion and purpose in life.”

**Understanding Oral Lesions**

**Objectives:**
1. Understand lesions that occur in the oral cavity
2. Differentiate lesions in the oral cavity
3. Understand treatment of lesions in the oral cavity

**Needs:**
1. New advances in dermatologic treatment
2. New methods of diagnosis or treatment
3. Availability of new medication(s) or indication(s)
4. Advances in medical knowledge

**References:**
1. JADA
2. International Journal of Dentistry

**Core Competencies:** 2, 3, 4, 5, 6

**Cosmetics and Dentistry**

**Objectives:**
1. Understand teeth and supporting structures
2. Understand teeth and how smile design relates to facial architecture
3. Improve facial cosmetic dermatology with assistance from dentistry

**Needs:**
1. New advances in dermatologic treatment
2. New methods of diagnosis or treatment
3. Development of new technology
4. Advances in medical knowledge

**References:**

**Core Competencies:** 2, 3, 4, 5, 6, 7

**Disclosures:** No relevant financial relationships to disclose.

**Lisa Swanson, MD**

Dr. Swanson is a board-certified dermatologist and pediatric dermatologist. She was born in New Orleans, Louisiana, and raised in Scottsdale, Arizona. She attended college at the University of Colorado at Boulder, graduating with honors as an English major. After that she obtained her medical degree from Tulane University School of Medicine in New Orleans. She performed her internship at Mayo Clinic in Scottsdale, Arizona, and went on to complete her dermatology residency at Mayo Clinic in Rochester, Minnesota. After that, she completed a fellowship in Pediatric Dermatology at Phoenix Children’s Hospital in Arizona.

She is a past Treasurer, Vice President and President of the Colorado Dermatologic Society. She is an active lecturer at conferences discussing pediatric dermatology with audiences across the country. She has been selected as a “Top Doc” by 5280 Magazine in 2012 through 2019. She is on staff at Rocky Mountain Hospital for Children where she sees hospital consults and performs procedures.

**Updates in Pediatric Dermatology**
Objectives:
1. Increase understanding of the pathogenesis of atopic dermatitis and various treatment options for the disease
2. Gain increased comfort in the treatment options for pediatric psoriasis
3. Learn about treatment options for common issues in kids like molluscum and warts

Needs:
1. New advances in dermatologic treatment
2. New methods of diagnosis or treatment
3. Availability of new medication(s) or indication(s)
4. Advances in medical knowledge

References:

Core Competencies: 2, 3, 4, 5, 6, 7

Disclosures: Speaker for Valeant, Bayer; Advisory board: Allergan; Off-label: JAK inhibitors for various derm conditions, topical beta blockers for vascular lesions, topical Rapamycin for derm conditions, Gleevac for stiff skin syndrome.

Alison Tam, DO, FAOCD
Dr. Alison Tam is a board certified Dermatologist who specializes in the field of cosmetic dermatology and laser therapy. Dr. Tam currently is a fellow of both the American Osteopathic College of Dermatology and the American Academy of Dermatology. Dr. Tam attended medical school at Western University of Health Sciences in Pomona, California and graduated in 2001. A traditional internship and her first residency in Family Medicine was done at Midwestern University-Mesa General Hospital. During her training, she was chosen to serve as the Chief Resident of the Family Medicine Residency.

After finishing a Family Medicine Residency in 2004, Dr. Alison Tam finished a second residency in Dermatology at Midwestern University in 2007. Dr. Tam served as Chief Resident of the Dermatology Residency. Upon finishing residency, Dr. Tam passed board certifications for the American Osteopathic Board of Dermatology and the American Society of Mohs Surgery.

Dr. Alison Tam currently holds medical licenses with the Nevada Board of Osteopathic Medicine, Arizona Board of Osteopathic Examiners in Medicine and Surgery, and the Osteopathic Medical Board of California. Other active licenses include the Nevada State Board of Pharmacy and the United States Department of Justice Drug Enforcement Agency. Active affiliations include the American Academy of Dermatology, American Osteopathic College of Dermatology, American Osteopathic Association, and American Society of Mohs Surgery.

Dr. Tam was awarded the “Top Beauty Expert” Award in 2013, 2014, and 2015 by New Beauty. Dr. Tam has sat on multiple Advisory Board Committees, spoken at conferences about laser therapy, served as a test writer for the American Board of Osteopathic Dermatology, and recently co-authored and published an article.

Currently, Dr. Alison Tam practices in Las Vegas, Nevada and serves as Medical Director at Laser Away Las Vegas. Most of her free time is devoted to her husband, child, and extended family and friends.

The Use of Compounded High Percentage Hydroquinone and Oral Tranexamic Acid for the Treatment of Resistant Melasma

Objectives:
1. A new and novel way to treat resistant melasma with compounded hydroquinone
2. Learning to use oral tranexamic acid in the treatment of melasma
3. View case series of active patients that have been treated with high percentage hydroquinone and/or tranexamic acid

Needs:
1. New advances in dermatologic treatment
2. New methods of diagnosis or treatment
References:

Core Competencies: 2, 3, 4, 6

Disclosures: No relevant financial relationships to disclose

Matthew Zirwas, MD
Matthew Zirwas, MD is an American Board of Dermatology certified physician. He is a nationally known expert who specializes in allergy patch testing, psoriasis, and eczema.

Dr. Zirwas founded the Bexley Dermatology Research Clinic in 2017. The research clinic treats qualifying patients with the newest cutting-edge treatment options prior to the medications becoming available on the market for normal physicians to prescribe. He is also a physician contributor and dermatologic advisor for companies such as ALL Laundry Detergent, CeraVe, AsepticMD, *Women's Health Magazine*, and many more.

He received his Bachelor of Science in Biology from the University of Pittsburgh in 1996. Dr. Zirwas also received his Doctor of Medicine from the University of Pittsburgh in 2000. He then completed his dermatology residency at the University of Pittsburgh in 2003 where he became Chief Resident.

Dr. Zirwas is married to Dr. Jill Fichtel, who practices in Nashville, TN.

Dr. Zirwas professional memberships include: American Academy for Dermatology, American Medical Association, American Contact Dermatitis Society, Association of Professors of Dermatology, Central Ohio Dermatology Society, Columbus Medical Association, European Society of Contact Dermatitis, North American Contact Dermatitis Group, Ohio Dermatology Association, Ohio Dermatological Association, The Ohio State Medical Association

In Dr. Zirwas’s free time, he enjoys tending to his wild cats, as well as spending time with his family and traveling with his wife, Dr. Jill Fichtel.

*Cosmetic Procedures and Contact Dermatitis*

Objectives:
1. Cause of contact dermatitis
2. Best treatments for contact dermatitis

Needs:
1. Advances in medical knowledge
2. New advances in dermatologic treatment
3. New methods of diagnosis or treatment

References:

Core Competencies: 2, 3, 4, 5, 6, 7

Disclosures: Abbvie, Aclaris, Aructis, Asana, Avillion, DS Biopharma, Foamix, Incyte, Janssen, Leo, Lilly, Ortho Dermatologics, Pfizer, Regeneron Sanofi, UCB, Aerolase, Fit Bit, L'Oreal, Menlo, AsepticMD, Genentech/Novartis
Resident & Student Board Review Session Speaker Biographies

Courtney Bernett, DO
Dr. Courtney Bernett, DO, PGY-4 is currently the Chief Resident for the Dermatology Residency Training Program at Orange Park Medical Center in Orange Park, FL. She graduated from the Philadelphia College of Osteopathic Medicine in 2016 and completed a Traditional Rotating Internship at O’Bleness Memorial Hospital in Athens, OH. Dr. Bernett has a special interest in complex medical dermatology, vulvar dermatology, and pediatric dermatology.

**Vulvar Dermatology**

**Objectives:**
1. Management of vulvovaginal involvement in SJSTEN
2. Management of lichen sclerosus
3. Management of vulvovaginal lichen planus

**Needs:**
1. New advances in dermatologic treatment
2. Advances in medical knowledge

**References:**

**Core Competencies:** 2, 3, 6

**Disclosures:** No relevant financial relationships to disclose.

Katherine Braunlich, DO
Kate Braunlich, DO, PGY-4 is a dermatology resident at Largo Medical Center under the direction of Dr. Richard Miller. Her undergraduate education was completed at Indiana University. She then attended medical school at Kansas City University of Medicine and Biosciences. At the conclusion of this academic year, she hopes to pursue a fellowship in Complex Medical Dermatology. She would like to thank the AOCD for the opportunity to present at this year’s meeting. She looks forward to a wonderful meeting filled with education and networking.

**Bullae & Vesicles: A Review of Differential Diagnoses and Treatment Options**

**Objectives:**
1. Important differential diagnoses for patients who present with primary skin finding known as bullae
2. Important differential diagnoses for patients who present with primary skin finding known as vesicles
3. Treatment options for patients with bullous or vesicular skin disease

**Needs:**
1. New advances in dermatologic treatment
2. Availability of new medication(s) or indication(s)
3. Advances in medical knowledge
4. New methods of diagnosis or treatment

**References:**

**Core Competencies:** 2, 3, 4, 6, 7

**Disclosures:** No relevant financial relationships to disclose.
Falon Brown, DO
Dr. Brown is a third-year dermatology resident at Campbell University – Sampson Regional Medical Center in Wilmington, North Carolina. She completed her undergraduate studies at the University of Alabama and attended medical school at the Georgia Campus of Philadelphia College of Osteopathic Medicine in Suwanee, Georgia. Dr. Brown completed an Internal Medicine residency with a concentration in hospital medicine from Louisiana State University – New Orleans Health Sciences Center in 2016. She practiced as a hospitalist at Touro Infirmary in New Orleans, Louisiana prior to beginning her dermatology residency in 2017. Her dermatologic interests include medical, surgical and cosmetic dermatology.

**Update in Melanoma Therapies**

**Objectives:**
1. Review basic science with regard to melanoma tumor biology
2. Provide an overview of melanoma related therapies
3. Review of medical management of advanced or unresectable cutaneous melanoma

**Needs:**
1. New advances in dermatologic treatment
2. Availability of new medication(s) or indication(s)
3. Advances in medical knowledge
4. New methods of diagnosis or treatment

**References:**

**Core Competencies:** 2, 3

**Disclosures:** No relevant financial relationships to disclose.

David Cleaver, DO, FAOCD
Dr. David Cleaver graduated from Truman State University with a BS in Biology. He graduated from KCOM in 2006 as a Doctor of Osteopathy. He did his Internship at Richmond Heights/Case Western University Hospital in Cleveland, OH and he completed his Dermatology training at St. Joseph Mercy Health System of Michigan State University in Ann Arbor, Michigan in June 2010. Dr. Cleaver is board-certified in dermatology and a Fellow of American Osteopathic College of Dermatology.

**Preserving Osteopathic Dermatology**

**Objectives:**
1. Gain an understanding of the history of Osteopathic dermatology
2. Understand the significance of Osteopathic dermatology
3. To discuss the future of Osteopathic dermatology

**Needs:**
1. New advances in dermatologic treatment
2. New methods of diagnosis or treatment
3. Legislative, regulatory, or organizational changes effecting patient care

**References:**
Core Competencies: 1, 4, 5, 7

Disclosures: Director, Officer or Employee of: KCOM (Assistant Professor); Speaker: Abbvie; Sibling is speaker for: Abbvie

Jonathan Crane, DO, FAOCD
A dedicated board-certified dermatologist who specializes in diagnosing and treating rare skin conditions, Dr. Crane is a graduate of New York Institute of Technology and New York College of Osteopathic Medicine. He completed his residency in dermatology at Lower Cape Fear Dermatology Clinic in Wilmington, North Carolina and his internship at Peninsula General Hospital in Far Rockaway, New York.

A trustee and former president of the North Carolina Osteopathic Medical Association, Dr. Crane has been Associate Editor of the Journal of the American Osteopathic College of Dermatology since August 2011. Born in Huntington, New York Dr. Crane is married with two sons and enjoys boating, fishing, water skiing, camping, and white-water wilderness canoeing.

Biologics: A Review for the Boards

Objectives:
1. Be better prepared for the Dermatology Boards
2. Be better prepared for the In-Service Exams
3. Have a greater understanding of how biologics work

Needs:
1. New advances in dermatologic treatment
2. Availability of new medication(s) or indication(s)
3. Advances in medical knowledge
4. Development of new technology

References:
1. Journal of the American Academy of Dermatology
2. Journal of Drugs in Dermatology

Core Competencies: 1, 2, 3, 6, 7

Disclosures: Research, Consultant, or Speaker’s Bureau for: 3M, Allergan, Candella Laser Company, Fujisawa, Genetech Inc., Glaxo Smith Klein, Novartis

Joanna Emilio, DO
Dr. Joanna Emilio, DO is a second year dermatology resident at St. John’s Episcopal Hospital in Far Rockaway, NY. She completed her undergraduate degree in biology at Wagner College with a minor in chemistry and psychology. She attended medical school at New York Institute of Technology College of Osteopathic Medicine. Dr. Emilio completed her intern year at St. John’s Episcopal Hospital in 2018 and currently lives in Brooklyn, NY. Her dermatological interests include medical and cosmetic dermatology.

Morphea in Pediatrics

Objectives:
1. The common clinical presentations of pediatric morphea
2. How to diagnose morphea in pediatrics
3. Therapeutic approaches for morphea in pediatrics

Needs:
1. New advances in dermatologic treatment
2. New methods of diagnosis or treatment

References:
Core Competencies: 2, 3, 4, 5, 6, 7

Disclosures: No relevant financial relationships to disclose.

Michael Fong, DO
Michael Fong was raised in the Northwest and received his degree from Western University of Health Sciences in 2018. Since that time he has completed his internship training in Miami and is in his first year of residency in Missouri.

Nerve Sheath Myxoma

Objectives:
1. Currently accepted nomenclature of Nerve Sheath Myxoma
2. Histologic characteristics of Nerve Sheath Myxoma
3. Current recommendations in treatment

Needs:
1. Advances in medical knowledge

References:

Core Competencies: 2, 3, 5, 6, 7

Disclosures: No relevant financial relationships to disclose.

Chelsea Harper, DO
My name is Chelsea Harper. I am from Huntsville, TX. I attended medical school at Texas College of Osteopathic Medicine in Fort Worth, TX. I am a PGY-1 dermatology resident at Sampson Regional Medical Center, Atlantic Dermatology in Wilmington, NC.

Dermatologic Manifestations of Diabetes Mellitus

Objectives:
1. Recognize various dermatologic manifestations of diabetes mellitus
2. Clinical recognize entities of dermatologic associations of diabetic mellitus
3. Discussion pathogenesis of selected dermatologic manifestations of diabetes mellitus

Needs:
1. Advances in medical knowledge

References:

Core Competencies: 2, 3

Disclosures: No relevant financial relationships to disclose.
Jessica L. Jones, DO
Dr. Jessica Jones completed her undergraduate studies at Saginaw Valley State University of Saginaw, MI where she earned a Bachelor of Science, finishing with honors while maintaining a position on the woman's tennis team. She then graduated with a Doctorate of Osteopathic Medicine as part of the inaugural class of Campbell University’s School of Osteopathic Medicine located in Buies Creek, NC. As a native to Michigan, she then returned to complete her internship training year in the Detroit Metropolitan area at Beaumont Farmington Hills. Before continuing to residency, Dr. Jones took a year to complete a Dermatology Research Fellowship in Miami, Florida under the supervision of Mark Nestor, MD, PhD and Brian Berman, MD, PhD. Here she served as a sub-investigator on both FDA and investigator initiated trials, helping to solidify her appreciation for the important of research in the field. Dr. Jones is currently training with Northeast Regional Medical Center’s Dermatology Residency Program in Kirksville, MO and continues to be honored to be a part of this community.

**Keloids and SRT**

**Objectives:**
1. Review the pathophysiology of keloid formation
2. Explore SRT as a treatment modality
3. Discuss mechanism of action on how SRT is thought to work to prevent keloid recurrence

**Needs:**
1. New advances in dermatologic treatment
2. Availability of new medication(s) or indication(s)
3. New methods of diagnosis or treatment

**References:**

**Core Competencies:** 2, 3

**Disclosures:** No relevant financial relationships to disclose.

Logan Kolb, DO
Logan Kolb is a PGY-4 dermatology resident at Orange Park Medical Center in Jacksonville, FL. He is originally from Plainview, MN, received his B.S. from the University of Wisconsin - La Crosse, graduated medical school from Des Moines University College of Osteopathic medicine, and completed his internship at Larkin Community Hospital. He is the founder of the Learn Derm Podcast and is passionate about dermatology medical education.

**You Don’t Know What You’re Missing: The Utility of the Wood’s Lamp**

**Objectives:**
1. Understand the wavelengths of light emitted by the Wood's lamp
2. Learn tips for proper use of the Wood's lamp and to make it more accessible
3. Learn ten practical dermatologic uses for which a Wood’s lamp can be used

**Needs:**
1. Advances in medical knowledge
2. New methods of diagnosis or treatment

**References:**

**Core Competencies:** 1, 2, 3, 5

**Disclosures:** No relevant financial relationships to disclose.
Ann Lin, DO
Dr. Ann Lin is a second year dermatology resident at St. John’s Episcopal Hospital in the New York City Metropolitan area. She completed a double degree in Film Production and Psychology at the University at Buffalo. Following her brief career in film and entertainment, she returned to school for a post baccalaureate in premedical studies at Hunter College in New York City. She continued her science and medicine career at Rush University in Chicago where she earned her masters of science in biotechnology. She attended William Carey University School of Osteopathic Medicine and went on to complete her intern year at St. John’s Episcopal Hospital. Her dermatologic interests includes complex medical dermatology, global health, and dermatologic surgeries.

**Hidradenitis Suppurativa Update**

**Objectives:**
1. Disease state and definition
2. Current treatments
3. New treatments on the horizon

**Needs:**
1. New advances in dermatologic treatment
2. Availability of new medication(s) or indication(s)
3. Advances in medical knowledge
4. New methods of diagnosis or treatment
5. Development of new technology

**References:**
1. Uptodate
2. Dermatology. 4th Ed. Bolognia Jean L. MD, Schaffer Julie V. MD, Cerroni Lorenzo MD.

**Core Competencies:** 2, 3, 6, 7

**Disclosures:** No relevant financial relationships to disclose.

Erin Lowe, DO
Erin Lowe grew up outside of NYC. Medicine is actually not her first career; in her formative years she worked as a professional actress. Her most notable role was Maureen Stabler on Law and Order: SVU, a character she played for nearly a decade. But as she grew up she knew acting wouldn't be her forever career. She became inspired by the medical work of many family members, and thus the journey began. She attended Columbia University for pre-medical education, spent a few years working in HIV research, and went to medical school at LECOM in Pennsylvania. Dr. Lowe is currently in her final year of dermatology residency training under Dr. Richard Miller at Largo Medical Center in Largo, FL.

**An Update on Atypical Mycobacterial Infections**

**Objectives:**
1. Clinical presentations of cutaneous atypical mycobacterial infections
2. Initial treatment options for a working diagnosis of cutaneous atypical mycobacterial infections
3. Work up and treatment options for cutaneous atypical mycobacterial infections

**Needs:**
1. Advances in medical knowledge
2. New methods of diagnosis or treatment

**References:**

**Core Competencies:** 2, 3

**Disclosures:** No relevant financial relationships to disclose.
Luke Maxfield, DO
Degrees include a bachelor of science from the University of Colorado at Colorado Springs and doctorate of Osteopathic Medicine from Lake Erie College of Osteopathic Medicine.

Related Work Experience
• University of Colorado Anschutz Medical Campus 2010-2012 Department of Pathology Student Researcher
• Editor and Author for USMLE/Comlex Combank/Truelearn 1/2018-2019
• Editor and Author for Wolvers Kluwer USMLE Firecracker 1/2019-current
• Reviewer for the *Journal of Dermatological Treatment*
• Reviewer for the *Journal of Dermatology* and *Dermatologic Surgery*

Publications
• Eighteen peer reviewed and indexed publications including *Cutis, BMJ Cases, Journal of the American Osteopathic College of Dermatology*, and the *JAOA*.
• Over 25 publications and presentations with a particular emphasis on infectious dermatology and tropical medicine.

**Vaccine Preventable Infections and Dermatosis**

**Objectives:**
1. Identify barriers to vaccination in the United States
2. Identify vaccine preventable diseases with dermatosis
3. Summarize cutaneous findings of vaccine preventable diseases

**Needs:**
1. Availability of new medication(s) or indication(s)
2. Advances in medical knowledge
3. Legislative, regulatory, or organizational changes effecting patient care

**References:**

**Core Competencies:** 1, 2, 3, 4, 6, 7

**Disclosures:** No relevant financial relationships to disclose.

John Moesch, DO
Dr. John Moesch is currently a senior dermatology resident at Largo Medical Center. He is a native of Savannah, GA. He graduated in 2012 from Furman University in Greenville, SC and in 2016 from Philadelphia College of Osteopathic Medicine. After completing dermatology residency he will be completing a Dermatopathology Fellowship at University of Pittsburgh Medical Center. He enjoys all aspects of dermatology including complex medical dermatology and hair loss diagnosis and treatment. In Dermatopathology, his interests include cutaneous lymphomas, melanocytic tumors, and soft tissue tumors. Dr. Moesch is an avid music and sports fan and loves spending the weekends at live concerts/sporting events in the Tampa Bay Area with friends.

**Cutaneous B-Cell Lymphomas: A Practical High-Yield Review**

**Objectives:**
1. Discuss the clinical findings of the four most common primary cutaneous B-cell lymphomas
2. Discuss basic pathology and diagnostic workup of primary cutaneous B-cell lymphomas
3. Discuss treatment of primary cutaneous B-cell lymphomas

**Needs:**
1. Advances in medical knowledge
References:
2. McKee’s Pathology. Ch. 29. Lymphomaproliferative disorders.

Core Competencies: 2, 6

Disclosures: No relevant financial relationships to disclose.

Serge Petrosian, DO
Serge Petrosian is a native of New York City and currently a third year dermatology resident at St. John’s Episcopal Hospital. He completed his bachelors at SUNY Stony Brook University and then continued his medical education at New York College of Osteopathic Medicine.

Interesting Cases from the Big City

Objectives:
1. Common presentations of leishmaniasis
2. Subtle cutaneous findings in scleroderma
3. Treatment and management of leishmania and scleroderma patients

Needs:
1. Advances in medical knowledge

References:

Core Competencies: 1, 2, 3, 6

Disclosures: No relevant financial relationships to disclose.

Ashley Rice, DO
Dr. Ashley Rice is a second-year dermatology resident at the Campbell University/Sampson Regional Medical Center program in Clinton and Wilmington, NC. Dr. Rice also completed her Traditional Rotating Internship at Sampson Regional Medical Center. She is originally from Ohio, where she received a Bachelor of Science Degree in Microbiology at The Ohio State University and her Doctor of Osteopathy Degree at Ohio University Heritage College of Osteopathic Medicine.

Surgical Anatomy for the Boards

Objectives:
1. Relevant anatomy in dermatologic and cosmetic surgery
2. Important danger zones in dermatologic and cosmetic surgery
3. Management options for surgical complications

Needs:
1. Advances in medical knowledge

References:

Core Competencies: 2

Disclosures: No relevant financial relationships to disclose.
Dahlia Saleh, DO
I grew up in Troy, Michigan (a suburb about 15 miles from Detroit). I earned my bachelor’s degree at Michigan State University with a degree in Human Biology. I then moved to Florida to attend medical school at Lake Erie College of Osteopathic Medicine- Bradenton campus. I then completed my transitional year at Broward Health Medical Center (formerly Broward General Hospital) in Fort Lauderdale, FL.

After five years in Florida, I moved to Wilmington, NC for dermatology residency at Sampson Regional Medical Center/ Campbell University School of Osteopathic Medicine. It has truly been a dream come true and every day I am so thankful for the opportunity to connect with patients and make a positive impact on their lives, because they truly make a positive impact on mine. I feel honored to provide care for patients in both Clinton and Wilmington. When I am not working, I enjoy doing most things that involve the outdoors like going to the Wrightsville beach, or trips to the mountains. I have a passion for traveling, both domestically and internationally; I love exploring new places and meeting new people. I have always held the belief that life is too short and the world is too large to stay in one place for too long!

I have published dermatology articles on topics including recurrent aphthous stomatitis, tumid lupus erythematosus, anagen effluvium, guttate psoriasis, herpes simplex virus, Vohwinkel syndrome, and hypertrichosis. After residency, I plan on practicing a mix of general, surgical, and cosmetic dermatology.

Basic Sciences for the Boards

Objectives:
1. Structure and Function of the skin
2. Interactions of selected molecules within the basement membrane
3. Commonly tested blistering disorders as they pertain to components of the basement membrane

Needs:
1. Advances in medical knowledge

References:

Core Competencies: 2, 3

Disclosures: No relevant financial relationships to disclose.

Muneeb Shah, DO
Muneeb Shah grew up in New York and attended medical school at Nova Southeastern University in Fort Lauderdale, Florida. He then completed his internship in internal medicine at Albany Medical Center. He is currently a first year Dermatology resident at Campbell University in North Carolina.

Principles of Electrosurgery

Objectives:
1. Different electrosurgical techniques (electrodessication, electrofulguration, etc)
2. Indications and contraindications of electrosurgery
3. Complications of electrosurgery

Needs:
1. New methods of diagnosis or treatment

References:
Core Competencies: 2, 3, 6, 7

Disclosures: No relevant financial relationships to disclose.
Thursday, September 26, 2019

8:00 a.m. - 12:00 p.m.  Exhibitor Set Up
                          Broadway Ballroom F

11:30 a.m. - 12:30 p.m. Lunch on Your Own

12:30 p.m. - 1:00 p.m.  Break with Exhibitors
                          Broadway Ballroom F

1:00 p.m. - 2:00 p.m.  The Use of Compounded High Percentage Hydroquinone and Oral Tranexamic Acid for the Treatment of Resistant Melasma
                      Alison Tam, DO, FAOCD

2:00 p.m. - 3:00 p.m.  One Simple Homeopathic Medicine That Can Help Our Dermatology Patients
                      Robert Signore, DO, FAOCD

3:00 p.m. - 3:30 p.m.  Hey, Doctor, What about CBD Oil?
                      Derrick Adams, DO, FAOCD

3:30 p.m. - 4:00 p.m.  Break with Exhibitors
                          Broadway Ballroom F

4:00 p.m. - 5:00 p.m.  The Role of Dermoscopy in the Evaluation of Facial Lesions on Sun Damaged Skin
                      Harold Rabinovitz, MD

5:00 p.m. - 5:30 p.m.  Minimizing HIPAA Liability
                      Leslie Rojas, JD

6:00 p.m.             Reception
                          Broadway Ballroom F
The Use of Compounded High Percentage Hydroquinone and Oral Tranexamic Acid in the Treatment of Resistant Melasma

ALISON TAM, D.O., F.A.O.C.D., F.A.A.D.

No conflicts of interest.

Which of the following are cost effective treatments for melasma?

- A. Compounded high percentage hydroquinones.
- B. Oral tranexamic Acid.
- C. None of the above.
- D. A and B.

Which of the following can be used in the treatment of recalcitrant melasma?

- A. Tranexamic Acid 650 mg ½ tablet BID.
- B. Hydroquinone 12% Kojic Acid 6% BID.
- C. Hydroquinone 16% Kojic Acid 6% BID.
- D. All of the above.

SUBJECTIVE

- Brown spots
- Sun Damage
- Acne Scars
- My pigment is worsening or spreading.
- I had a laser treatment and the brown spots are worse.
- I had a chemical peel and the brown spots are worse.
- I have been on Hydroquinone 4% for over a year and it doesn’t work.

38 year old female
60 year old female
What history should you elicit now? *Your clinical dx should be done *

- Family history of "hyperpigmentation"?
- When you had treatments, were you put on hydroquinone to prep your skin before and after the treatment? What was the %?
- When did the start? (Pregnancy, Perimenopause, OCP, Bioidentical hormones, acne/scar removal, post peel/procedure).
- What else in your skin care regimen? Vit C/antioxidants, SPF #, reapplication of SPF, makeup with SPF (don't count), retinoids (too irritating), aesthetician appointments.
- Allergic? (not mushroom)
- What's your ethnicity/genetic makeup? Have you done genetic testing?
- What are some successful treatments that you have had in the past?

You already know the diagnosis (at least in your head):

- Melasma
- PIH due to procedure
- Acne Scars (PH)
- Combination of any of the above

Plan--Melasma

- Although we all categorize our patients into a Fitzpatrick Skin Type, should we refine our categorization based on ethnicity?
- There are so many mixed races now that it becomes important to know how someone scars, develops PIH or erythema, and responds to procedures.
- Have they done any "genetic testing" such as Ancestry or 23&Me? Why is this important in treatment considerations?
- Consider increasing their Fitzpatrick skin type to highest skin type based on ethnicity or genetic testing for treatment and procedures.
- Photodocumentation at the office

Melasma Treatment with Hydroquinone

- Fitz 1-3 HQ 4% Vit C 5 BD
- Fitz 4-6 HQ 10-12% KA 6% Vit C 5 BD
- Fitz 6-8 HQ 12-18% Vit C 5 BD 30 BD
- PEARL: Don’t use KA if allergic to PCN/mushrooms
- PEARL: Rosacea or Atopic patients may not be able to tolerate KA
- PEARL: Highly sensitive patients may not be able to tolerate KA
- PEARL: HQ 20% cannot be made with any other components since adding additional ingredients lowers the concentration of the HQ.
- Follow up in 4-6 weeks to check for compliance, photos, and irritation

What happens at the 6 week follow up?

- Photos
- Tolerance of dosage
- Is it possible to start maintenance?
- Consideration and discussion of future procedures
- Consideration for oral Tranexamic Acid
- What about a summer time flare?
- What if there is irritation?
- What other types of counseling should be done?
Photos

- If your system allows for evaluation of erythema, check for a vascular component of melasma. If present, consider a vascular laser or low level IPL starting in the fall/winter.
- If patient had a procedure that created a flare, consider erythema/melasma flare vs. true vascular melasma. Consider changing to HQ/HC 2.5% x 1 month and recheck in 4-6 weeks.
- The addition of HC 2.5% or TAC 0.1% requires follow up in an effort to take patient off of the steroid and go back to a product without a steroid.
- If the patient has vascular melasma, consider oral Tranexamic Acid.

Titration of dosage

- For patients, a 4% change in HQ is much more visible clinically than a 2% change.
- Use this chart and titrate upwards:
  - Fitz 1-3 HQ 8/ KA 6/ VIT C 5 BID
  - Fitz 3-4 HQ 10-12/ KA 6/ VIT C 5 BID
  - Fitz 4-5 HQ 12-18/ KA 6/ VIT C 5 OR HQ 20 BID
- If it is almost summer, consider a 4% increase in HQ for the summer.

Is it possible to start maintenance therapy?

- You can start maintenance whenever you and the patient are happy with the result and stable on a specific dosage.
- Options:
  - Decrease HQ % by 2-4% every 6 weeks
  - Decrease frequency of usage to qd
  - Decrease frequency of weekly usage to QI.
- Bridge with a proprietary topical like Lytera or other non-HQ cosmeceutical compounded topical Tranexamic acid 7%, Cysteamine.
- Control the pills (no pills without refill).
- Don’t get too stressed because patients will run out, use longer than the expiration date, or just stop their HQ.

Future procedures

- Always prep a melasma patient with HQ for 2-4 weeks before doing a procedure. They have to stay on HQ throughout the procedure process.
- Intense pulse light x 3
- Peels
- Vascular lasers
- Microneedling
- Laser toning (Genesis)

Tranexamic Acid

- So far, I love using it.
- Contraindications: Thromboembolic hx or risk, Family hx Thromboembolic dx, Hypersensitivity
- Dosing: 650 mg 1/2 tablet QD-BID
- How long do we let patients take it off?
- The risk of clotting is approximately the same as an OCP.
- OCP > Transdermal/patch hormones > vaginal hormonal therapy for risk of clotting.
- No one knows the risk of clotting for bioidentical hormones but FDA will be studying this soon.

Summer time flare

- It is expected, it will happen and it’s normal.
- Don’t take a patient off HQ during the summer.
- Decrease the brown as much as you can before summer starts.
- Increase SPF usage/reapplication.
- HQ is not photosensitizing so it’s ok to take it with you on a sunny vacation.
- Consider adding Tranexamic Acid for the summer.
- Sunglasses with metallic rims.
- Blue filters for buses and planes.
What if there’s irritation with your compound?

Change the base and go to a gel
Take out KA
Add HC 2.5%
Make sure the patient is using a pea size mixed in with moisturizer for the WHOLE face (more is not better)
Decrease the HQ%
Call your compounding pharmacist—they are the best resource. Have they changed the formula, base, bad batch?

Counseling

- SPF >45 with reapplication
- Makeup doesn’t count (you know this)
- Iron Oxide/Mineral sunscreen
- Blue filters on phone
- Metallic finish on glasses
- Hats
- Ongoing condition that requires ongoing maintenance. Ex: HTN, DM
- What about hormone therapy or withdrawal of it?
- What is the patient’s melasma phenotype? What about pseudo-ochronosis?

Pearls

- The first predictor that HQ is working is the ability to wear less makeup or that application of makeup takes less time
- Tinted HK2% by 2-4% each time
- Happy patients—start tapering usage and consider non-HQ alternatives
- Expectations: Advise patients it takes 1 year to figure out their correct dose (no tapering). It may take 3 months to expect measurable improvement (do not give up!)
- Higher Fitz = Higher HQ = Longer treatment time required
- Severe melasma takes longer to treat than mild so don’t get caught up in “new drug” you keep a patient on HQ

- Expiration of product in compounding: 6 weeks=100% efficacy; 8 weeks=90% efficacy; 12 weeks <75% efficacy and requires a refill.
- Do not use Kojic Acid in a PCN/mushroom allergic patient
- Add Vit C to the compound for stability and further lightening of skin
- Add a cosmeceutical antioxidant qAM during most of the year and BID during summer months
- Consider procedures (reserved for fall and winter in melasma patients)

Where do you find high percentage HQ?

- Doc Jul 29, 2019, 09:59.pdf
- 230.8kB
How else can you get HQ?

- Skinmedicinals
- Your local compounding pharmacy
- Patient cost is approximately $60-70 for a 12 week supply (30 gm)
- Ask for samples if starting with your local compounding pharmacy
- Try out the texture
- Look for “grittiness” (the pharmacy isn’t running the powder through the mill long enough)
- Look for texture (too thick, too thin, the base that it’s made in)
- Try different bases (light lotion, versabase, gel base, pracasil)
- Call your pharmacist—they are a wealth of information!
LEARNING OBJECTIVES:
1. EXPLORE ROLE OF HOMEOPATHIC MEDICINE IN TX OF SKIN DISEASES OCCURRING AFTER LOSS & GRIEF
2. DESCRIBE CLINICAL CRITERIA FOR “SODIUM CHLORIDE” PATIENT TYPE
3. REVIEW CASES: 8 PATIENTS WITH SKIN DISEASES AFTER LOSS & GRIEF SUCCESSFULLY TREATED WITH P.O. HOMEOPATHIC SODIUM CHLORIDE

WHICH SKIN DISEASES RESPOND MOST READILY TO HOMEOPATHIC TX?

1. ACNE VULGARIS
2. WARTS (COMMON & PLANTAR)
3. MOLLUSCUM CONTAGIOSUM
4. ATOPIC ECZEMA
5. HYPERHIDROSIS

HOW CAN HOMEOPATHIC MEDICINE HELP OUR DERMATOLOGY PATIENTS?
1) HELP PATIENTS AVOID THE USE STEROIDS AND ANTIBIOTICS
2) SEEMINGLY UNRELATED MEDICAL ISSUES MAY ALSO IMPROVE
   • SIMPLE, SAFE, AVAILABLE (OTC)
   • INEXPENSIVE
   • EASY TO ADMINISTER (DISSOLVE SUBLINGUALLY)
   • PLEASANT TASTE

CLASSICAL HOMEOPATHIC MEDICINE

1. WHAT IS CLASSICAL HOMEOPATHIC MEDICINE?

   • GREEK: Homoios – ‘similar’ or ‘like’
   Pathos – ‘suffering’
   • LOW-COST, NON-TOXIC SYSTEM OF MEDICINE
   • USED BY OVER 200 MILLION PEOPLE
   • USES MICRODOSES OF NATURAL SUBSTANCES
   • DERIVED FROM PLANTS, MINERALS, ANIMALS
   • STIMULATING THE NATURAL HEALING RESPONSE.
   • LENGTH OF TRAINING - 4 TO 5 YEARS
HOMEOPATHIC MEDICINES & FDA
• REGULATED BY THE FDA AS DRUGS SINCE 1938
• FOOD, DRUG, & COSMETIC ACT OF 1938
• MANUFACTURED IN ACCORDANCE WITH HOMEOPATHIC PHARMACOPOEIA OF THE UNITED STATES (HPUS)
• MANUFACTURED ACCORDING TO GOOD MANUFACTURING PRACTICES (GMP)

HOW DO HOMEOPATHIC MEDICINES WORK?
• SCIENCE: BEGINNING TO UNDERSTAND HOW HOMEOPATHIC MEDICINE WORKS
• MANUFACTURING MUST INCLUDE: VIGOROUS SHAKING AFTER DILUTION
• PRODUCES NANOPARTICLES
• NANOPARTICLES HAVE ENERGETIC PROPERTIES (electromagnetic, optical properties)

SODIUM CHLORIDE NANOPARTICLES ARE PRESENT IN HOMEOPATHIC NaCl SOLUTION (1x10^-400)!

How is Homeopathic Medicine DIFFERENT?

Conventional Tx
• Based on: DIAGNOSIS
• Looking for: DISEASES

Homeopathic Tx
• Based on: HOMEOPATHIC CLINICAL TYPE (“Constitution”)
• Looking for: PERSONS!
2. MOST COMMON HOMEOPATHIC PATIENT TYPE IN MY PRACTICE:

SODIUM CHLORIDE

There are over 9000 different homeopathic remedies!

PREVALENCE OF SODIUM CHLORIDE:
(MOST COMMON PATIENT TYPE)

NaCl 33%
All Other Patient Types 67%

“How to Find A NaCl Patient”


TO ACHIEVE CLINICAL RESULTS, WE STRIVE TO MATCH AS MANY CLINICAL CRITERIA OF THE HOMEOPATHIC REMEDY AS POSSIBLE WITH THE INDIVIDUAL PATIENT

HOM. SODIUM CHLORIDE – CLINICAL CRITERIA:

• RESERVED, HIDE THEIR EMOTIONS
• FASTIDIOUS, METICULOUS
• FEEL RESPONSIBLE FOR EVERYONE ELSE
• SADNESS (WANTS TO BE ALONE TO CRY)
• GUILT
• WORSE WITH CONSOLATION
• HIGHLY ETHICAL, HONEST, LOYAL, RESPONSIBLE
• DESIRE REVENGE (IF BETRAYED)
• OILY SKIN (esp. FACE)
• WARTS (PALMS, VOLAR, PERIUNGUAL)
• ECZEMA
• HERPES SIMPLEX
• INTOLERANCE TO SUNLIGHT / HEAT! (PHOTOPHOBIA, HEADACHES)
• CRAVE SALTY FOODS

Helpful Tips For Finding Sodium Chloride Patients:

• Loss manifests in a state of silent grief
• Pt. seeks solitude & desires to process loss alone
• Pt. carries a feeling of guilt (even if they didn’t actually have anything to do with loss)
• Pt. is generally worse from consolation (“No hugs, please leave me alone.”)
• Pt. often dwells on what happened and compound their grief and guilt around the situation.
• Silent rumination often progresses → feeling of anger over what happened
• Pt. may play sentimental music over and over (dwelling on their loss)
• Even when surrounded by loved ones during grief, Sodium chloride person feels alone & isolated.

Peter Swanz, ND: http://www.drswanz.com/2014/12/18/homeopathic-remedies-for-grief-and-a-broken-heart/
NEVER BEEN WELL SINCE?
• HELPFUL: RECALCITRANT DERM CASES
  • “HOW LONG HAVE YOU HAD THIS DZ.?”
  • “WHAT WAS GOING ON IN YOUR LIFE WHEN THIS
  SKIN DZ. OCCURRED?”
  → “WHAT DID YOU FEEL?”
  → “WHAT DID YOU DO?”
• BASED ON THE PT’S. ANSWERS, WE CHOOSE
  THE MOST FITTING HOMEOPATHIC MEDICINE,
  WHICH SOMETIMES TREATS BOTH THE SKIN
  DISEASE AND THE EMOTIONAL COMPONENT

For example, when NaCl Pts feel hurt (grief or rejection):
1. They feel better by themselves (being alone)
2. They feel worse when others try to comfort them / give sympathy
3. They don’t like to be pitied

Andre Saine, ND – Materia Medica (Natrum muriaticum)

How Do We Recognize Sodium Chloride Patient

PATIENT SMILES WHEN TELLING ABOUT
GRIEF (VALUABLE SIGN! → NaCl)

“You ask, ‘When your mother died, how was it?’
They smile and say, ‘Not so bad’. It means not good.”

“…they smile when sad or
smile when crying or
smile when talking of serious matters”

* Andre Saine, ND – Materia Medica (Natrum muriaticum)

Homeopathic NaCl Clinical Criteria:

Homeopathic NaCl Clinical Criteria:

TODAY WE REVIEWED
A CASE SERIES (n=8):
“SODIUM CHLORIDE”-TYPE PATIENTS
TREATED WITH HOMEOPATHIC NaCl

Two Patients: Exposure to Prenatal Loss & Grief
• All 8 Cases (100%): Skin Diseases Improved
• All 8 Cases (100%): Other Medical Issues
  Also Improved:
  (Anxiety, Guilt, Constipation, Joint Pain, Insomnia,
  Thirst, Hyperhidrosis, Low Energy, Dysmenorrhea,
  Allergic Rhinitis, Obsessive Compulsive Disorder)

Note:
• homeopathic sodium chloride is available
  over-the-counter at health food stores.
• also available online at www.boironusa.com:
  - click shop online
  - click our medicines
  - click boron single remedies
  - click natrum muriaticum (= the Latin name for
    sodium chloride)
  - click buy now
  - under “dilution” click: 30C (this is the “strength”)
  - usual starting dose: 2 pellets sublingually BID
  - for best clinical results, should take a classical
    homeopathic medicine distance learning class:
    e.g. http://www.bihint.com/course/foundations/

CLASSICAL HOMEOPATHIC MEDICINE
WHAT IS IT?
• NON-TOXIC, LOW COST NATURAL SYSTEM
  OF HEALING USED BY OVER 200 MILLION
  PEOPLE WORLDWIDE
CAN IT HELP PATIENTS WITH SKIN DISEASES
OCCURRING AFTER LOSS / GRIEF?
• YES (If patient type matches remedy type!)
WHAT ARE ITS ADVANTAGES?
• SAFE, AFFORDABLE, & EFFECTIVE
• HELPS REDUCE STEROIDS / ANTIBIOTICS
• CONCURRENT MEDICAL ISSUES MAY
  ALSO IMPROVE
THANK YOU!

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Minimizing HIPAA Liability: Tips from a healthcare law attorney

Leslie Rojas Whitworth
Rojas Law, PLLC
leslieanerojas@gmail.com

Healthcare = Risky Business

- HIPAA violations can result in significant monetary damages.
- Learn to manage the risk and minimize your HIPAA liability.

What are the risks?

HIPAA Breach Case Examples:

- A 12-physician dermatology practice paid $150,000 for alleged HIPAA violations arising out of a lost, unencrypted flash drive containing protected health information.
- An orthopedic clinic failed to execute a business associate agreement prior to turning over 17,300 patients' PHI to a potential business partner. The settlement included a monetary payment of $750,000 and a comprehensive corrective action plan.

HIPAA Liability Exposure:

- HIPAA breaches are inevitable.
- Liability can result from a breach report (whether reported by you pursuant to breach reporting requirements or reported by someone else), and a government investigation will likely follow.
- Routine Government HIPAA Compliance Audits.
- The government reports that the compliance issues discovered during investigations are:
  1. Insufficient HIPAA policies and procedures;
  2. Insufficient safeguards to protect PHI;
  3. Lack of an investigation after a possible breach;
  4. Lack of sanctions against responsible parties (e.g., employee discipline; terminated business associate).

No Conflicts

- I do not have any financial disclosures to make or conflicts of interest to report.
Minimizing Liability: Pre–Breach Checklist

1. A HIPAA Privacy Manual and HIPAA Security Policies that are tailored to your practice
2. Annual employee HIPAA training.
3. Updated Notice of Privacy Practices
   - Updated after 2013?
   - On practice’s website?
4. Updated Medical Record Release Authorizations
   - Updated after 2013?
   - Special rules for STDs, mental health, substance abuse

Minimizing Liability: Pre–Breach Checklist

6. Business Associate Agreements.
   - A “business associate” is a person or entity that performs certain functions or activities that involve the use or disclosure of protected health information on behalf of, or provides services to, a covered entity. (Excludes a member of the covered entity’s workforce.)
   - Examples:
     - Third-party billing company
     - IT company
     - Your healthcare attorney
     - EMR company
     - Medical record storage company (old paper charts)
   - Does not include those with incidental exposure to PHI. For example, janitorial staff or electrician.

Minimizing Liability: Pre–Breach Checklist

5) Annual Security Rule Risk Analysis (hire an IT forensic expert under the attorney client privilege)
   - Evaluate the risk to PHI when at rest. (ePHI on removable media, mobile devices, computer hard drives, as well as PHI on paper where are paper charts stored/secured? how are papers such as prescription refill requests disposed of?)
   - Store all e-PHI to a network
   - Encrypt data stored on portable devices & media
   - Remote device wipe to remove data when lost/stolen
   - Consider appropriate data backup procedures
   - Train workforce members on how to effectively safeguard data and timely report security incidents

Minimizing Liability: Pre–Breach Checklist

7. Documentation!!!
8. Follow-through.
   - Do not have any policies on paper that you are not going to follow in practice.
   - Not required by law… but DO IT!

Minimizing Liability: Pre–Breach Checklist

If you’ve implemented my Pre–Breach Checklist, then you have implemented basic HIPAA compliance in your practice.

But, HIPAA breaches are inevitable...

Minimizing Liability: Pre–Breach Checklist

What to do when there is a possible breach.

- Prompt investigations of possible breaches are critical.
- Work with health care legal counsel and IT experts.
- Document everything
- Rapid Response and Notification
  - “Without undue delay” rule
- Two Tracks (Important)
  1) Initial Breach Response
  2) Prepare for the investigation/get your house in order!
Minimizing Liability: Possible Breach Checklist

If you suspect a breach:

1) Immediately notify health care legal counsel and IT forensic experts to establish a plan of action for the investigation and analysis. (Attorney-client privilege)
2) Secure the premises.
3) Isolate affected system to prevent further intrusion, data release, or damage. Take infected machines offline, but leave the power on.
4) Use telephone to communicate. Attackers may be capable of monitoring email traffic.

5) Activate all auditing software (if not activated).
6) IT experts should preserve all pertinent system logs (firewall, router, intrusion detection system, etc.). Files on the affected systems should never be deleted, moved, or altered in any way.
7) If files are damaged/alterred, IT should create backup copies and store in a secure location.
8) Identify systems that connect to the affected system and where the affected system resides within the network topology.

9) Identify the programs and processes that operate on the affected system(s), pre-identify the associated IP address, MAC address, Switch Port location, ports and services required, physical location of system(s), the OS, OS version, patch history, safe shut down process, and system administrator or backup.
10) Locate backup, if any.
11) Take an inventory of missing items and their locations.
12) Review keycard and surveillance data for unusual activity.

13) Retain an external forensic IT expert to assist and to image the data.
14) Determine whether breach notification is required.

• Even if there has been an "incident," you have to determine, with the assistance of healthcare legal counsel, whether there has been:

A reportable breach under HIPAA.

Minimizing Liability: Breach Reporting and Investigation Checklist

15) If there is a reportable breach, after consultation with health care legal counsel, notify affected patients and the appropriate law enforcement agencies.

a) Document all conversations with law enforcement, if any, and the steps taken to restore the integrity of the system.

b) In the event the affected system is collected as evidence, make arrangements to provide for the continuity of services, i.e., prepare redundant system and obtain data back-ups.

16) Log the unauthorized use or disclosure in the patient’s disclosure tracking log.

Breach Notification Rule

General Rules:

› 500 or more = notify government within 60 days (but without unreasonable delay).
› More than 500 individuals in the same jurisdiction/state = notify media within 60 days (but without unreasonable delay).
› Less than 500 individuals = notify the government at least 60 days after the end of the calendar year.

• Usually March 1 or February 29 during a leap-year.

***Tip for breaches of less than 500: Don’t do it earlier than you need to.\n
Minimizing Liability:
Breach Reporting and Investigation Checklist

1. After the investigation and notification (if necessary) are completed, conduct a post-investigation review of the events and make necessary adjustments to the technology and/or response procedure to reflect the lessons learned.

Remember the 2 Tracks!
Track 1 was the initial breach response (steps 1-17 above). Now that the notifications have gone out, the government may come knocking, so...

Track 2 is... Get your house in order!!!
- Update Notice of Privacy Practices
- Update breach notification policies and implement safeguards
- Conduct and document employee training.
- Conduct and document risk assessment.
- Update BAAs and document satisfactory assurances from BAs.

OCR Investigation: Step-By-Step

1) OCR receives a complaint or a breach notice.
2) OCR conducts intake & review to determine if a possible Privacy or Security Rule violation occurred.
3) At any time, the OCR may refer the complaint to the DOJ if a possible criminal violation occurred.
4) If no Privacy or Security Rule violation, case closed.
5) If there is a possible violation, then OCR will notify the complainant and the CE.
   - The CE will receive a letter from the OCR asking for specific information to be submitted for review.
   - In some cases, the letter will not have any requests, but will instead list corrective actions for the CE to take.
6) Respond to OCR’s information requests/compliance demands.

Best Practices for Response Letter

- *Overreact:* Don’t underreact.
  - Show the OCR that the CE takes HIPAA compliance very seriously.
- Put your *best foot forward:*
  - Even if not specifically requested by the OCR, make sure to state the ways in which the CE is HIPAA compliant and the corrective actions taken to become/remain compliant.
  - Focus on reputable compliance history and what CE has done correctly.
- *Don’t highlight, but don’t hide from weaknesses:*
  - OCR wants to see that the CE is acting in good faith, learning, and taking corrective actions to ensure compliance. Highlight this when discussing your weaknesses.
- Highlight the CE’s *compliance barriers,* but don’t rely on this too heavily.
  - E.g., small practice with few employees/resources doing the best it can.
  - If breach was caused by a BA, seriously consider terminating relationship.

Government Investigation: Overview

HIllAA Privacy & Security Rule Complaint Process

OCR Investigation: Step-By-Step

7) After receiving CE’s response, OCR reviews evidence to determine if a violation occurred. If there was non-compliance, then OCR will suggest:
   - Voluntary compliance;
   - Corrective action; and/or
   - Resolution agreement.
8) Sometimes OCR will request additional information (e.g., follow-up requests about whether the CE actually took the corrective actions it said it would).
9) If CE does not take action to resolve the matter that is satisfactory to OCR, then OCR may impose fines and CE may request an evidentiary hearing.
10) If OCR is satisfied with the CE’s corrective actions, OCR will typically request a phone call to “offer HIPAA technical assistance” to the CE. This is usually an indication that OCR is closing the file.
11) Once investigation is closed, OCR will send a letter.

Summary: 3 takeaways

1. Good-faith efforts at HIPAA compliance before the government ever comes knocking will demonstrate to the government that you take HIPAA seriously.
   - This will lower your fines, and may result in no fines at all.
   - These include: HIPAA Privacy Manual, HIPAA Security Policies, Updated NPP (on website), Updated Release Forms, Updated BAAs.
2. Proper handling of the potential breach response (with IT forensic experts and healthcare legal counsel) will ensure that you put together an effective response to a government investigation. Follow the check list.
3. Timely notification of a reportable breach to patients, government, media and a well-thought out response to an investigation. This should include a response to the allegations, why you were lacking in certain compliance areas, in which areas you have robust HIPAA compliance (even if unrelated to the allegations), and the changes you have made since discovering the breach.
   - It looks best if these changes were implemented BEFORE the government investigation began, i.e., before the government had to request that you implement those changes.
Consider Cybersecurity Insurance

Cybercriminals are becoming more sophisticated. It’s no longer a matter of IF but WHEN you will be attacked. Security incidents are extremely expensive. Verify that you have cyber insurance (not always included in your basic policy) to help cover these costs.

Resources

- https://www.hhs.gov/hipaa/for-professionals/index.html
- https://www.hhs.gov/hipaa/for-professionals/privacy/laws-regulations/index.html
- https://www.hhs.gov/hipaa/for-professionals/privacy/index.html
- https://www.hhs.gov/hipaa/for-professionals/faq/index.html

Feel free to email me with questions at leslieannerojas@gmail.com.

FAQ’s

Does HIPAA require you to obtain a new acknowledgement of receipt of the Notice of Privacy Practices from patients if you change your privacy policy?

Answer:

Yes. A covered health care provider with a direct treatment relationship with individuals is required to make a good faith effort to obtain an individual’s acknowledgement of receipt of the notice only at the time the provider first gives the notice to the individual — that is, at first service delivery. See 45 CFR 164.520(c)(2).

FAQ’s

May physician’s offices use patient sign-in sheets or call out the names of their patients in their waiting rooms?

Answer

Yes. You may use patient sign-in sheets or call out patient names in waiting rooms, so long as the information disclosed is appropriately limited. HIPAA explicitly permits the incidental disclosures that may result from this practice, for example, when other patients in a waiting room hear the identity of the person whose name is called, or see other patient names on a sign-in sheet. However, these incidental disclosures are permitted only when the covered entity has implemented reasonable safeguards and the minimum necessary standard, where appropriate. For example, the sign-in sheet may not display medical information that is not necessary for the purpose of signing in (e.g., the medical problem for which the patient is seeing the physician). See 45 CFR 164.502(a)(1)(iii).

FAQ’s

Does the HIPAA Privacy Rule permit a doctor to discuss a patient’s health status, treatment, or payment arrangements with a person who is not married to the patient or is otherwise not recognized as a relative of the patient under applicable law (e.g., state law)?

Answer:

Yes. The HIPAA Privacy Rule at 45 CFR 164.510(b) permits covered entities to share information that is directly relevant to the involvement of that person in the patient’s care or payment for health care. In addition, HIPAA allows a covered entity to disclose information about a patient as necessary to notify, or assist in the notification of (including by helping to identify or locate), such a person of the patient’s location, general condition, or death. The Privacy Rule defers to a covered entity’s professional judgment in these cases and does not require the entity to verify that a person is a family member, friend, or otherwise involved in the patient’s care or payment for care.

FAQ’s

Does the HIPAA Privacy Rule permit a doctor to discuss a patient’s health status, treatment, or payment arrangements with the patient’s family and friends?

Answer:

Yes. The HIPAA Privacy Rule at 45 CFR 164.510(b) specifically permits covered entities to share information that is directly relevant to the involvement of a spouse, family members, friends, or other persons identified by a patient, in the patient’s care or payment for health care. If the patient is present, or in some way available prior to the disclosure, and has the capacity to make health care decisions, the covered entity may share information with the family and these other persons if the patient agrees or, when given the opportunity, does not object. The covered entity may also share relevant information with the family and these other persons if it can reasonably infer, based on professional judgment, that the patient does not object.
FAQ’s

Does the HIPAA Privacy Rule permit a doctor to discuss a patient’s health status, treatment, or payment arrangements with a person who is not married to the patient or is otherwise not recognized as a relative of the patient under applicable law (e.g., state law)?

Answer:

Yes. The HIPAA Privacy Rule at 45 CFR 164.510(b) permits covered entities to share with an individual’s family member, other relative, close personal friend, or any other person identified by the individual, the information directly relevant to the involvement of that person in the patient’s care or payment for health care. In addition, HIPAA allows a covered entity to disclose information about a patient as necessary to notify, or assist in the notification of (including by helping to identify or locate), such a person of the patient’s location, general condition, or death. The Privacy Rule defers to a covered entity’s professional judgment in these cases and does not require the entity to verify that a person is a family member, friend, or otherwise involved in the patient’s care or payment for care.
6:30 a.m. - 7:00 a.m.  Breakfast with Exhibitors
Broadway Ballroom F

7:00 a.m. - 7:30 a.m.  CLIA Proficiency Exam
Gregory Papadeas, DO, FAOCD

7:30 a.m. - 8:00 a.m.  Allergy for the Dermatologist, Part 2
Ujwala Kaza, MD

8:00 a.m. - 8:30 a.m.  Antioxidants: The Enemy of My Enemy?
Derrick Adams, DO, FAOCD

8:30 a.m. - 9:00 a.m.  Cosmetic Procedures
Jill Fichtel, MD

9:00 a.m. - 10:00 a.m.  Creative Ways to Maximize Professional Efforts Most Effectively and Avoid Practice Pitfalls
Will Kirby, DO, FAOCD

10:00 a.m. - 10:30 a.m.  Break with Exhibitors
Broadway Ballroom F

10:30 a.m. - 11:00 a.m.  Allergy for the Dermatologist, Part 1
Ujwala Kaza, MD

11:00 a.m. - 11:30 a.m.  Essential Oils: Is There a Scent of Evidence?
Derrick Adams, DO, FAOCD

11:30 a.m. - 12:30 p.m.  Cosmeceuticals: The Science of Designing a Skincare Regimen
Leslie Baumann, MD

12:30 p.m. - 1:30 p.m.  Lilly Product Theater
Dawn Sammons, DO, FAOCD
Broadway Ballroom G-H
(Note: **No CME Awarded for This Session**)
2:00 p.m. - 3:00 p.m.  Acne Devices
Michael H. Gold, MD

3:00 p.m. - 3:30 p.m.  Break with Exhibitors
Broadway Ballroom F

3:30 p.m. - 4:30 p.m.  Physician Wellness
Robert Piccinini, DO

4:30 p.m. - 5:00 p.m.  Cosmetics and Dentistry
Stacy Spizuoco, DDS

5:00 p.m. - 5:30 p.m.  What Are These Stains?
Michael Nowak, MD
ALLERGY FOR THE DERMATOLOGIST PART II

UJWALA KAZA, M.D., FACAAI, FAAAAI
CLINICAL ASSISTANT PROFESSOR, NYU LANGONE

DISCLOSURES
• Astra Zeneca
• Pfizer
• Sanofi Genzyme
• Regeneron

ALLERGIC DISEASE STATES DERMATOLOGISTS CAN ENCOUNTER
• Urticaria – presented in an earlier lecture
• Angioedema
• Drug Allergy
• Insect Allergy
• Atopic Dermatitis
• Contact Dermatitis – patch testing is the gold standard for testing and given dermatologist’s familiarity of this disease, will not address here

ANGIOEDEMA

• Manifests as bouts of asymmetric nondependent swelling involving cutaneous or mucosal surfaces
• Typically is not pruritic
• Usually mast cell-mediated, often seen with urticaria
• Many of the same treatments and principles apply as with urticaria, so will defer to lecture on urticaria
• Hereditary angioedema and bradykinin angioedema are other types of isolated angioedema independent of urticaria. Will not be covered here

DRUG ALLERGY (1/2)

Morbilliform drug rash from antibiotics

Adapted from www.dermnetnz.org/topics/cutaneous-adverse-reactions-to-antibiotics

Adapted from Janjua at DermnetNZ.png
DRUG ALLERGY

**WHO DEFINITION**

- World Allergy Organization recommends:
  - Use of the terms immediate and delayed to refer to the onset of symptoms
  - Immediate: within or later than 1 hour after dosing
  - Delayed: these are helpful in distinguishing whether the probable immunologic mechanism is antibody mediated (e.g., immunoglobulin E or T lymphocyte mediated)


**INCIDENCE**

- ADRs (adverse drug reactions) affect 10% to 20% of hospitalized patients and up to 25% of outpatients
- Most are type A reactions
- Type B reactions are much less common, with an estimated frequency of 10% to 15% of all ADRs
- Immune-mediated drug reactions may constitute 6% to 10% of ADRs

1. Gomes ER, and Demoly P. Curr Opin Allergy Clin Immunol 2005

**MOST COMMON CAUSES**

- The most common drugs causing hypersensitivity reactions: β-lactam antibiotics and nonsteroidal antiinflammatory drugs (NSAIDs)
- Others common causes: radiocentrost media, neuromuscular blocking agents, and antiepileptic drugs


**MANIFESTATIONS**

- Most common cutaneous eruption is a generalized maculopapular exanthem which accounts for up to 90% of all cutaneous eruptions caused by drugs
- Most severe reactions are Stevens–Johnson syndrome and toxic epidermal necrolysis
- In diagnosing drug allergy, the history is very important
- Diagnosis of drug allergy is largely based on clinical history because diagnostic tests are limited

3. Abernase DF, Khan DR. Diagnosing and managing Drug Allergy. 2018
STEVEN JOHNSON’S SYNDROME/TOXIC EPIDERMAL NECROLYSIS

www.dermnetnz.org/topics/stevens-johnson-syndrome-toxic-epidermal-necrolysis/

ANTIBIOTIC DRUGS THAT CROSS REACT (1/3)

• Penicillins, cephalosporins, and carbapenems share a bicyclic nucleus which conveys an appreciable but variable immunologic cross-reactivity in immune responses to these drugs.
• Cephalosporins are similar to penicillins immunochemically, but individual immune responses vary greatly.

1 Torres MJ, and Blanca M. Med Clin North Am 2010

ANTIBIOTIC DRUGS THAT CROSS REACT (2/3)

• Third-generation cephalosporins (e.g., ceftriaxone) appear less likely to have cross-reactivity responses than first-generation cephalosporins (e.g., cephalothin).
• Carbapenems have a similar degree of cross-reactivity by skin testing to 1st generation cephalosporins but studies have consistently shown that penems are well tolerated clinically.
1 Romano A, Gaeta F et al. J Allergy Clin Immunol 2010

ANTIBIOTIC DRUGS THAT CROSS REACT (3/3)

• The monobactam class (aztreonam) very weakly cross-react with other β-lactams.
• Aromatic sulfonamides, often with antimicrobial activity (i.e., sulfamethoxazole, sulfadiazine, sulfisoxazole, and sulfacetamide), differ from other sulfonamide-containing medications.
• Based on chemical structure, cross-reactivity between these two groups of sulfonamides is considered unlikely, and studies have indicated this is rare.
1 Hernstreet BA, and Page RL. Pharmacotherapy 2006

ONE MEDICATION CAN HAVE MULTIPLE TYPES OF REACTIONS – GELL AND COOMBS – PENICILLIN

Pen-Gell-Coombs Classification
I Anaphylactic (IgE-mediated)
II Complement-dependent cytolysis (IgG/IgM)
III Immune complex damage
- Serum sickness
- Drug fever
- Some cutaneous eruptions and vasculitis
IV Delayed or cellular hypersensitivity
- Contact dermatitis
- Morbilliform eruptions
- SJS/TEN
- Pneumonitis

Adapted from Celik GE; Pichler WJ et al.. Middle’s Principles of Allergy and Immunology

PENICILLIN ALLERGY

• + PCK skin test results do not occur more frequently for atopic individuals.
• ~10% of the US population reported allergies to penicillin.
• Clinically significant IgE-mediated or T lymphocyte-mediated penicillin hypersensitivity rates are <5%.
• IgE-mediated penicillin allergy wanes over time, with 80% of patients becoming tolerant after a decade.

1 Adkinson NF: J Allergy Clin Immunol 1984
2 Shenoy ES, Macy E. JAMA. 2019
PENICILLIN AND CEPHALOSPORINS

- An oral challenge with amoxicillin in patients with low-risk penicillin allergy histories: optimal method to confirm current tolerance
- Clinically significant immunologically mediated penicillin-cephalosporin cross-reactivity is rare
- May still avoid using a β-lactam with a shared side chain in an individual with proven IgE-mediated β-lactam−associated anaphylaxis (Ex: avoiding ceftriaxone in someone with cefepime anaphylaxis; or avoiding cefotaxime in someone with history of cefuroxime anaphylaxis 1)

ALLERGY TO CORTICOSTEROIDS

- Allergic reactions to corticosteroids: rare, testing complicated by the need to test for excipients too
- Li et al. reported on a study of 64 patients - evaluated through skin tests and drug provocation testing - only 9 (14%) were allergic, most cases due to an excipient 1,2
- In all cases confirmed sensitive to methylprednisolone, due to the excipient polyethylene glycol.
- In at least 2 cases positive to triamcinolone, due to carboxymethylcellulose

LOCAL ANESTHETICS (1/2)

- Local anesthetic agents are relatively good sensitizers when applied topically (i.e., contact allergy) 1
- Antibody-mediated allergic reactions to these agents are rare events 2,3,4
- Nonallergic responses to local anesthetics, particularly in dentistry, often lead to allergy consultations 1

LOCAL ANESTHETICS CONTINUED (2/2)

- Vasovagal syncope may mimic anaphylaxis - dental settings.
- Paresthesias and lightheadedness can be explained on the basis of the pharmacologic toxicity of the "caines," and the symptoms are more common in drug-intolerant patients1
- Episodes of anxiety or panic associated with a procedure contribute to some reactions
- IgE responses to local anesthetics are rare 1
- Intradermal skin testing followed by a series of provocative dose challenges is the recommended approach to diagnosis and management 2,3

CONTACT ALLERGY TO ANESTHETIC

Adapted from: www.dermnetnz.org/topic/allergy-to-bezocaine

CLASSIFICATION OF NONSTEROIDAL ANTI-INFLAMMATORY DRUG (NSAID) HYPERSensitivity Reactions

<table>
<thead>
<tr>
<th>Timing of reaction</th>
<th>Type of reaction</th>
<th>Clinical manifestations</th>
<th>Putative mechanism</th>
</tr>
</thead>
<tbody>
<tr>
<td>Acute (≤ 24 h)</td>
<td>Cross-reactive</td>
<td>Asthma, rhinosinusitis</td>
<td>Cox-1 inhibition</td>
</tr>
<tr>
<td>Delayed (48–96 h)</td>
<td>Single drug</td>
<td>Urticaria, angioedema,</td>
<td>IgE-mediated</td>
</tr>
</tbody>
</table>

**NSAIDS**

Classification of Nonsteroidal Anti-Inflammatory Drug (NSAID) Hypersensitivity Reactions:

- Clinical manifestations: Asthma, rhinosinusitis, urticaria, angioedema, 
- Putative mechanisms: Cox-1 inhibition, IgE-mediated

- Delayed: Type IV delayed-type Cytotoxic T-cell, NK cells, others
LIP ANGIOEDEMA FROM DICLOFENAC IN A WITH CHRONIC SPONTANEOUS URTICARIA AND ANGIOEDEMA

Adapted from Park HS, Kowalski ML, et al. Middleton: Principles and practice. 2013

NONSTEROIDAL ANTIINFLAMMATORY DRUGS (NSAIDS)

• Up to 35% of patients with chronic spontaneous urticaria (CSU) experience exacerbation of skin symptoms when exposed to ASA/NSAIDs. The term AECD (aspirin exacerbated cutaneous disease) has been recently proposed for this condition.
• Urticaria and angioedema appear ~1 to 4 hours of drug ingestion, although late reactions occurring up to 24 hours after can occur.

NONSTEROIDAL ANTIINFLAMMATORY DRUGS (NSAIDS)

• Delayed or non-immediate reactions involving the skin and other organs develop more than 6 hours after drug ingestion.
• Symptoms usually emerge several days to weeks after initiation of a new drug, but they can develop earlier when induced by reintroduction of the drug.
• The recovery period lasts from several days to weeks.

NSAIDS

• Immediate-type skin testing has been usefully applied to β-lactam antibiotics and a variety of other immunogenic drugs, but the rate of clinically false-negative test results is well established only for penicillins.1,2
• Although a positive cephalosporin skin test result implies the presence of drug-specific IgE antibodies, a negative result does not exclude immediate hypersensitivity.1,4

DRUG ALLERGY TESTING (1/3)

1Krause B, and Aberer W. Immunol Allergy Clin North Am 2009

DRUG ALLERGY TESTING (2/3)

• + results for intradermal skin tests have been reported for imipenem and other β-lactams, but validated skin testing protocols are not available.
• Sulfamidoyl-poly-L-tyrosine, a synthetic multivalent sulfonamide antigen, has been shown to elicit positive type I skin test results in a few patients with sulfonamide allergy by history.5,6

5Wang E, J Allergy Clin Immunol 1993
DRUG ALLERGY TESTING (3/3)

- Only with penicillin allergy have in vitro test results been systematically compared with those of skin tests.
- Potential IgE-mediated reaction - validated skin testing reagents exist only for penicillin and not for any of the other low-molecular-weight drugs.
- Immunoassays for documenting IgE antibodies to quinolone antibiotics, rocuronium, and other drugs have been reported, but their validity is unknown.


INSECT BITES

- Few credible reports of allergic reactions to biting insects.
- Sensitization to salivary proteins from insects can cause abnormal local swelling following insect bites.
- Anaphylaxis is rarely reported with insect bites.
- Registry at the American Academy of Allergy and Immunology: Only a small number of cases with a convincing history of systemic reactions and detectable allergen-specific IgE antibodies were found.


CULICOIDAE (MOSQUITO)

- Few cases of anaphylaxis have been reported.
- There has also been increased recognition of the clinical impact of large local reactions to mosquito bites in children (i.e., Skeeter syndrome).
- Mosquito extracts commercially available are of unreliable composition and activity, not useful.


MOSQUITO REACTIONS

- Current research is looking at the major allergens in mosquito extracts, they have also looked at recombinant allergens.
- Natural desensitization may occur with frequent and numerous bites.
- Treatment with second-generation antihistamines can be used to prevent and treat reactions to mosquito bites.

**TRIATOMA (KISSING BUG, CONE-NOSE BUG)**

- Most common confirmed cause of systemic reactions to insect bites is the kissing bug\(^1\)
- Found throughout the areas of the southwest states and California
- Feed exclusively by sucking the blood of vertebrate animals, may find shelter in homes at night
- Bite causes an erythematous plaque, but because it is painless, the person may be unaware of the cause of an allergic reaction
- Immunotherapy had success in a small group of patients\(^2\)


**OTHER INSECT BITES**

- Tabanidae (Horsefly, Deerfly)
  - Tabanid species are large flies that suck blood and inflict painful bites.
  - Widespread distribution in rural and suburban areas.
  - Affects humans and animals.
  - Allergic reactions to insect bites from horseflies and deerflies have been reported.
- Allergic reactions to other insects – usually large local reactions, rarely anaphylaxis, for example with black flies
- Flea bites usual in humans, more in pets. Reactions, usually popular urticaria, above the ankle and can last for weeks, sometimes months

**VENOM ALLERGY**


**VENOM STINGS**

- Types of venom – paper wasp (Polistes spp.), honeybee (Apis mellifera), yellow jacket (Vespula spp.), white faced hornet (Dolichovespula maculata), yellow hornet (Dolichovespula arenaria)
- Anaphylaxis to insect stings occurs in 3% of adults and 1% of children, and even the first reaction can be fatal\(^1\)
- Cutaneous-systemic reactions are most common in children
- Hypotensive shock is most common in adults
- Respiratory complaints occur equally in all age groups.


**VENOM – HONEY BEE**


**VENOM STINGS (1/3)**

- Chance of a systemic reaction to a sting is low in those with large local reaction and in children with mild (cutaneous) systemic reactions; in adults it varies from 23% to 70% depending on the severity of previous systemic sting reactions.
- Venom skin tests are most accurate for diagnosis, but the specific IgE test can be an important complementary test.
- Degree of sensitivity on skin or serum tests does not predict the severity of a sting reaction. History is important because venom sensitization can be detected in up to 25% of adults.
VENOM STINGS (2/3)

• Venom immunotherapy is 75% to 98% effective in preventing sting anaphylaxis.
• Large local reactions are not usually a precursor of systemic reactions. The risk of eventual anaphylaxis in those with large local reactions is only 5% to 10%.
• Prevalence rates are up to 3.3% in the United States.


VENOM STINGS (3/3)

• Indication for Venom immunotherapy (VIT):
  • History of previous systemic allergic reaction to a sting AND evidence of venom-specific IgE antibodies with a + venom skin test result or increased specific IgE level.
  • Some patients with + skin test results do not require VIT because they are judged to be at relatively low risk for anaphylaxis.
  • Those with recent and severe anaphylaxis are at highest risk (40% to 70%).
  • Low risk (<10%) has been found for children and adults with a history of large local reactions and for children with reactions limited to cutaneous signs and symptoms but with no respiratory or vascular manifestations.


VENOM STING IMAGES

Left: wasp sting. Right: bee sting

adapted from https://www.dermnetnz.org/

FIRE ANTS

• Anaphylaxis to fire ants has been reported.
• Currently there is no standardized IT for fire ants.
• Although has been reported to be effective, there have been no trials with placebo controls.


ATOPIC DERMATITIS AND ALLERGIC TRIGGERS

Adapted from www.dermnetnz.org/topics/atopic-dermatitis/

ATOPIC DERMATITIS AND FOODS (1/4)

• Patients with AD and + food allergen skin tests could have negative food challenges to the implicated allergens, this distinguishes between symptomatic and asymptomatic hypersensitivity.
• Triggers for clinical disease cannot be predicted by testing alone.
• Double-blind placebo-controlled food challenges have demonstrated that food allergens can cause exacerbations in a subset of patients with AD.

[1] Sampson HA, and McCall CC. J Allergy 1985
ATOPIC DERMATITIS AND FOODS (2/4)

• About 1/3 of infants and young children with AD will show clinically relevant reactivity to a food allergen

• Lesion induced by single challenges are usually transient

• Repeated challenges can result in eczematous lesions.

• Food-specific T cells have been cloned from lesional skin and peripheral blood of patients with AD

• Elimination of food allergens results in amelioration of skin disease and a decrease in spontaneous basophil histamine release


ATOPIC DERMATITIS AND FOODS (3/4)

• Food allergy testing usually done more for an immediate type reaction

• In AD that is resistant, if food is being considered a trigger, a limited panel of foods may be tested

• An avoidance diet:
  • Indicated in patients clearly identified as food allergic by an appropriate diagnostic food challenge
  • After adequately informing the family of the limited benefits, and possible harms of an elimination diet

1 Eigenmann PA, Beyer K et al.. Pediatr Allergy Immunol. 2019

ATOPIC DERMATITIS AND FOODS (4/4)

• Food-avoidance can improve AD but does not cure it
  • It can have detrimental effects:
    • Progression to an immediate type reaction
    • May reduce the quality-of-life of the patient and the family.


ATOPIC DERMATITIS AND AEROALLERGENS (1/3)

• Evidence supports a role for aeroallergens in AD. These findings include both allergen-specific IgE antibodies and allergen-specific T cells

• Exacerbation of AD can occur with house-dust mites, animal danders, and pollens.

• It has been estimated that as many as a third of AD patients with HDM hypersensitivity experience worsening of AD or respiratory symptoms with dust exposure


ATOPIC DERMATITIS AND AEROALLERGENS (2/3)

• In DHRPCT, a subgroup of patients with AD underwent bronchoprovocation with a standardized house-dust mite extract. They developed unequivocal cutaneous lesions after inhalation of dust mite

• All the patients with dust mite-induced atopic dermatitis had a history of asthma, so it’s possible the respiratory route may be important in the induction and exacerbation of AD.

ATOPIC DERMATITIS AND AEROALLERGENS (3/3)

- Direct contact with inhaled allergens can also result in eczema lesions.
- Using the atopy patch test, Langeveld-Wildschut and coworkers showed that positive reactions to house-dust mite were associated with IgE+ Langerhans cells in the epidermis of AD patients.
- The severity of AD has been correlated with the degree of sensitization to aeroallergens.
- Environmental control measures aimed at reducing dust mite allergens have shown clinical improvement in AD patients.


SUMMARY

- Drug allergy – we do not have very good testing. Best testing we have is for Penicillin. The most important diagnostic tool we have is the history.
- Insect allergy – usually localized.
- Venom allergy – usually when it is a systemic reaction, immunotherapy has a good outcome in reducing the recurrence of another systemic event. Localized reactions do not predict systemic reactions.
- Atopic dermatitis – although airborne allergens as well as food sensitization can be involved, need to be careful about what we remove from patient’s diet.
Creative Ways to Maximize Professional Efforts Most Effectively and Avoid Practice Pitfalls

Disclosures:
No relevant disclosures.
No irrelevant disclosures.

Dr. WILL KIRBY
• Chief Medical Officer, LaserAway
• Fellow, American Osteopathic College of Dermatology
• Clinical Assistant Professor and CosmetiC Director, Western University of Health Sciences Dermatology Residency Program
• Expert Reviewer, Osteopathic Medical Board of California, Division of Dermatology
• Expert Witness, Legal Cases involving Aesthetic Dermatology

Other thoughts...
• My goal is to analyze the business of dermatology efficiently so as to maximize effort
• At least some (if not all) of the action items I’ll discuss today are applicable to your own practice
• Mantra: Money ≠ Success. Freedom = Success!
• Sincere thanks to the AOCD for having a meeting in California!

Relevant References
1 http://www.ncbi.nlm.nih.gov/pmc/articles/PMC1337555/pdf/westjmed00264-0092.pdf
2 http://scholarship.law.edu/cgi/viewcontent.cgi?article=1529&context=jchlp
5 http://www.ombc.ca.gov/
6 http://www.mbc.ca.gov/Consumers/Complaints/Complaints_FAQ/Practices_and_Protocols_FAQ.aspx

This lecture is not...
• A risk management lecture
• All inclusive
• An economic/business discussion
• Applicable to all state rules/regs
• A substitute for legal advice
This lecture is...

- Encouragement to use non-traditional ways to improve your (not practice but) life...
- A means by which to open the audience’s minds to alternative methods of navigating the current medicolegal environment
- A teaching mechanism for insider tips from the speaker who has spent a decade working in the most litigious city in the most litigious state with the most challenging patient population in the universe

Most Importantly...

- This lecture is intended to give you ten real, actual, inexpensive, tangible action items to strongly consider incorporating into your own practice
- Not only might they prevent an unwanted interaction with a patient but they may likely increase patient satisfaction, employee retention and you might be pleasantly surprised to find that they are emotionally rewarding
- While you’ll experience many academic lectures over the next few days, it is my contention that this one in particular can offer a positive impact on your practice

And...

- You don’t need to take notes
- The material is dense and will be peppered with captivating personal stories and enchanting anecdotes
- I’ll emphasize anything important
- There will be a one page recap at the end highlighting all ten action items!
- I’ll leave time for Q and A as well
- Do NOT call or e-mail me next week!
- (I’m half joking - you can email me next week: DrWillKirby@yahoo.com)

Offense Vs. Defense

- Practitioners are often focused on the maximization of patient volume, management of patient schedule, reimbursement and collections.
- The leaves less time (or the outright neglect) of compliance with rules/regulations, and medicolegal considerations.
- The point?
- Offense makes money and defense protects the practice. And a good practice has both!

What to do?

- Many ways to improve “offense” (which means just increase revenue): Hire practice manager, marketing, advertising, expanding hours, hiring allied health care professions, see more patients, work faster, work longer hours, accept more insurance plans, etc.
- But how do you best manage the “defense”?
- Better put: “Winterize your beach house”

Winterize Your Beach House
But Why?

- Why does it matter if I am familiar with the state rules and regulations? My competitors aren’t.
- Lawsuits are scary... but the State Medical Boards are like Mike Tyson on Redbull and Roofies!
- “If you aren’t at the table then you are on the table”

State Rules and Regulations

- Quagmire
- Overregulated Industry
- Conflicting rules and regulations
- Lack of compliance by competitors
- Absence of uniformity in rules/regs enforcement
- Dearth of transparency in the review process

Hire Health Care Attorney to Review Your Practice

- Paid by the hour. Thus, one could argue that they are incentivized to work slowly and inefficiently
- Often bill to obtain information that you can easily obtain by yourself
- Emotionally taxing for you: Fear based practices lead unnecessary and excess work
- But some are excellent and can help ensure that your “defense” is strong

What to Do About Rules/Regs?

- Simply go to your state medical board website to begin:
  I. Subscribe to the monthly newsletter. They often make useful announcements
  II. Go to the FAQ section of the website.
  III. Review the posted (monthly or quarterly) newsletters to see why others ran into issues.
  IV. Apply to become an expert reviewer...

Expert Reviewer

- The Medical Board will contact you to review cases
- You’ll be financially compensated for your time and effort
- You are protected from lawsuits related to the case
- In some states you remain anonymous
- They often provide training as to how to be an expert reviewer
- You get to participate in the review process and enforcement of rules/regs = best way to truly understand how to keep your practice in compliance

Example:
Quarterly Medical Board Meetings
- Attend in-person
- Most have teleconferences where you can listen in remotely
- You can monitor and assembly bills or senate bills coming down the pike so you can be prepared
- You’ll build professional relationships with lobbyists and influence rules reg

LAW SUITS

Discussion of Lawsuits
- Expensive to defend
- Time consuming
- May coincide with complaint (and subsequent investigation) to the medical board
- Your insurance company may force you to settle
- Emotionally taxing
- Listed as a malpractitioner in the National Practitioner Data Bank (NPDB) pursuant to the Healthcare Quality Improvement Act of 1986 (HCQIA).

Medical Malpractice Insurance
- DUH
  - You obviously need med-mal insurance
  - Your insurance broker is not your friend
  - They will try to sell you more insurance than you need or could ever use
  - But annual med-mal review is a good idea; you might be performing procedures that your policy doesn’t even cover

Medical Malpractice Claims

<table>
<thead>
<tr>
<th>Cause of Action</th>
<th>No. (%) of 174 Cases</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lack of informed consent</td>
<td>20 (20%)</td>
</tr>
<tr>
<td>Fraud</td>
<td>15 (8.6)</td>
</tr>
<tr>
<td>Loss of consortium</td>
<td>13 (7.5)</td>
</tr>
<tr>
<td>Assault/battery</td>
<td>9 (5.2)</td>
</tr>
<tr>
<td>Strict products liability</td>
<td>9 (5.2)</td>
</tr>
<tr>
<td>Breach of contract</td>
<td>8 (4.6)</td>
</tr>
<tr>
<td>Infusion of emotional distress</td>
<td>8 (4.6)</td>
</tr>
<tr>
<td>Negligent misrepresentation</td>
<td>7 (4.0)</td>
</tr>
<tr>
<td>Gross negligence</td>
<td>5 (2.9)</td>
</tr>
<tr>
<td>Negligent per se</td>
<td>5 (2.9)</td>
</tr>
<tr>
<td>Deceptive trade practices</td>
<td>4 (2.3)</td>
</tr>
<tr>
<td>Other</td>
<td>15 (8.7)</td>
</tr>
</tbody>
</table>

Informed Consent
- Informal:
  - Often an informed consent comes informally in the course of discussion with a patient during a routine office visit or similar situation.
- Format:
  - Informed consent can also be given formally, by having a patient sign a document that states that the health care provider has fully discussed a treatment or procedure and that the patient fully acknowledges and agrees to the risks.
What Happens if You Perform a Treatment Without an Informed Consent?

• You could be charged with battery or fraud.
• Battery is defined as an unlawful act of applying force to the person of another without consent.
• You could be accused of negligence in a medical malpractice lawsuit.

Imperative Parts of a Solid Informed Consent

• Layman’s terms
• Offer alternatives to the treatment
• Alternatives included no treatment at all
• Side effects of treatment
• Chances of developing side effects
• Section for patient to document that they understand the informed consent
• Acknowledgement that they aren’t under the influence or were coerced into signing
• Acknowledgement that all questions were answered.
• Acknowledgement that patient requests treatment

Arbitration Agreement

• Check with a health attorney in your respective state
• If it is allowed in your state then consider incorporating an arbitration agreement
• An arbitration doesn’t take rights away from patients – it just solves disputes much more inexpensively and quickly
• Your fate isn’t in the hands of a jury
• Arbitration agreements many dissuade frivolous lawsuits

Obtain Proper Training

• Reps aren’t your friends nor are they qualified to teach you how to use a product or a device
• Your training must be documented and quantified
• Your training needs to meet or exceed the community standards
• Consider seeking out an accredited training facility that offers CME to quantify training

Purge Problem Patients

• Ten percent of your patients cause 90 percent of your problems. And that takes up a lot of time!
• It is very conceivable to think that those problems patients take a big emotional toll on your employees and you.
• Just like any toxic relationship, you can’t change them.
• Make a list of your problem patients and legally dismiss them.
• Dismissal must meet specific state requirements
PATIENT DISMISSAL LETTER
- Must provide letter to patient (USPS letter, certified letter, fax, email)
- Must provide emergency care for 14 days
- Must provide them information as to where they can also receive care
- Provide information on how to obtain medical records

Providing Medical Records
- Patients may have a copy (not original)
- You have 14 days to provide records
- You can (and should!) charge for the records
- You should always mail the records to the patient

Hire an NP or a PA
- Add some diversity to your practice
- Allows you to connect with patients you have little in common with
- It’s the future
- Can be a great income source
- Allows you to concentrate on more important things in life
- Dermatology is the holy grail of medicine because of its relationship with NPs or PAs!

Preventing Burnout
- Financial arguments with patients are stressful.
- Consider giving refunds anytime a patient complains.
- When you do give a refund, require that the patient sign a Refund Release form.
- The Refund Release form requires them to agree to your terms (no disparagement, no complaint to the medical board, no legal action, etc.)
- When the patient cashes the refund check...

Refund Release Form
- Patients love having young doctors present
- You keep your skills sharp by teaching
- Education is backbone of our profession
- Dermatology residents have a command of the rules/regs!!!

Join the Academic Faculty of a Dermatology Residency Program
- The time commitment is extremely flexible
- The time commitment is extremely flexible
- Patients love having young doctors present
- You keep your skills sharp by teaching
- Education is backbone of our profession
- Dermatology residents have a command of the rules/regs!!!
Improve Your On-Line Reputation
- Wide next vs. efficient net
- Having an excellent on-line reputation will allow you to capture the best patients
- Perception is Reality
- Social media is the future
- Putting your head in the sand won’t make it go away
- You can easily improve your Yelp, Facebook and Google reviews

Case Study: My Practice

Social Media
- Never use the phrase “the best”
- Don’t use brand names (unless necessary)
- Educate the patient
- Provide relevant but diverse content
- Make the material personal

Social Media Examples

Values, transparency, ethics...
The Next Slide...

...is the only slide you need to pay attention to!

TEN POINT RECAP:
1. Monitor updates from your state medical board!
2. Apply to become an expert reviewer!
3. Review your med-mal insurance policy annually!
4. Improve your Informed Consent (and add an Arbitration Agreement if your state allows it!)
5. Obtain Proper Training!
6. Purge Problem Patients (dismiss properly and possibly provide medical records)!
7. Add a Refund Release form to your practice!
8. Hire an NP or a PA!
9. Join the faculty of a dermatology residency program
10. Improve your on-line professional reputation and focus on social media!

Questions and Answers (Tip: All the answers are “no”)
• May I have a copies of your Informed Consents?
• May I have a copy of your Arbitration Agreement?
• May I have a copy of your Refund Release form?
• May I have a copy of your Patient Dismissal Letter?

Questions and Answers (Tip: All the answers are “maybe”)
• Can you refer me to a med-mal insurance broker?
• Can you refer me, or my NP/PA get proper training?
• Can you refer me to a site to improve my on-line professional reputation?

Real Questions and Answers
• Ask me anything you like...
• I’m an open book...
• I’d rather you ask me now than accost me while I eat a stale muffin in eight minutes...
• I might say “no”.
• But you can always email me at anytime in the future if you think of a question at a later date: DrWillKirby@hotmail.com

Thank you!
• Will Kirby, D.O., FAOCD
Urticaria

Overview

- Pruritic, erythematous papules or plaques, with superficial swelling of the dermis
- 1 in 5 patients will experience urticaria in their lifetime

Acute urticaria
- Lasts less than 6 weeks

Chronic urticaria
- Chronic urticaria (CU) - urticaria on most days of the week, for 6 weeks or longer
- 40% of patients with CU have accompanying episodes of angioedema or deeper swelling of dermal or mucosal tissues
- 10% have angioedema as their main manifestation


Urticaria - incidence and recurrence (1/2)

- Estimated incidence in general population: 4.9% over 10 years
- In 10 years of follow up, 7.8% of those with urticaria developed CU
- Of those that developed CU, 52.6% achieved remission at 1 year and 88.9% at 5 years

Urticaria - incidence and recurrence (2/2)

- In patients attending an allergy clinic at an academic center:
  - 13% of patients with CU developed recurrent CU (return of CU >6 months after cessation of controller therapy)
  - Patients who developed recurrent CU were more likely to require treatment other than anti-histamines

Kim JK, Har D et al. J Allergy and Clin Immun In Practice 2018

Acute urticaria

- Occurs in up to 20 percent of the population
- Can be caused by medications, foods, infections
- Generally it is self-limited

Chronic urticaria

- ~ 20 % have a reversible physical trigger: Physical urticarias:
  - Dermographism
  - Cholinergic
  - Vibratory
  - Heat or cold induced contact urticaria
  - Aquagenic urticaria
  - Solar urticaria
  - Exercise induced urticaria
  - Delayed pressure urticaria


Chronic urticaria/chronic spontaneous urticaria (CSU)

- 80 percent: no external or allergic cause can be identified
- Chronic idiopathic urticaria - some people classify 30-40 % in this group as having an autoimmune etiology
- The remaining 60% to 70% of patients with CU (chronic idiopathic urticaria) are classified as having chronic spontaneous urticaria(CSU)


Chronic urticaria: Natural course of the disease

- CU is usually self-limited. Average duration is 2-5 years
- If no trigger is identified, rates of spontaneous remission at 1 year - approximately 30% to 50% have been reported
- In 1/10 of patients symptoms have persisted beyond 5 years

CIU and autoimmunity

- 30% - 40% of patients with CIU have an autoimmune disease or CAU.
- This is driven by IgG autoantibodies to either IgE or the α subunit of the high-affinity IgE receptor.
- Leads to activation of mast cells and basophils.

Mast Cells and CIU

- Mast cell number in patients with CIU: not increased in either lesional or nonlesional skin compared to healthy controls.
- Culture-derived mast cells grown from peripheral blood CD34+ cells from those with CIU.
- Elevated spontaneous histamine release over that observed in mast cells from healthy donors.

Chronic urticaria and autoimmunity

- These antibodies cannot be used as diagnostic in identifying the source.
- These antibodies may also be present in patients without CIU.
- Although autoantibodies have been identified, the relationship of antibody titers to disease activity has not been clearly demonstrated.

Chronic Urticaria: Patient History

- Urticarial skin lesions typically last less than 24 hours without residual change to the skin area.
- Scratching and rubbing skin → development of ecchymosis.
- Multiple ecchymotic lesions or painful lesions should alert to possible urticarial vasculitis.
- Pruritus of CU → often noticeable at night.
- Severe symptoms during periods of stress can occur.

Chronic urticaria - ? Triggers

- Foods are extremely rare triggers.
- Food elimination may help in some patients.
- Exacerbating factors: NSAIDs, hormones etc.
- Patients may suspect food allergies, but it is rarely proven.
- Pseudoallergens, or chemicals in foods, have occasionally been linked to urticaria → restricted diets may assist patients these particular patients.
- Exacerbating factors: aspirin or other NSAIDs, hormones, or particular drugs, should be sought and eliminated.
Urticaria: Physical appearance

- Urticaria are pruritic, raised, and erythematous and they can have central pallor
- Shape can be round, oval, or serpiginous, and can be confluent
- H1 blocker antihistamines, can cause the appearance to be flat
- Size may range from < 1 cm to several centimeters

Chronic idiopathic urticaria


Urticaria and angioedema: Physical appearance

- It can be deeper tissue swelling which is what occurs more with angioedema
- Angioedema involving the face, lips, tongue, extremities, or genitalia may occur in conjunction with hives or as a separate entity

Angioedema


Lab evaluation and testing in chronic urticaria (1/3)

- Diagnosis is usually made clinically
- In 80-90% of cases a source is not identified
- Consensus statements recommend limited testing:
  - A complete blood count with differential to assess for eosinophilia
  - C-reactive protein or erythrocyte sedimentation rate to identify the risk of underlying rheumatic disease
  - TSH level
- Results of these laboratory studies are normal in most patients who lack signs and symptoms of systemic disease

1 Tarbox JA, Gutta RC et al. Ann Allergy Asthma Immunol 2011

Lab evaluation and testing in chronic urticaria (2/3)

- Value of testing for thyroid autoantibodies is thought to be linked to the idea of identifying an underlying association with possible chronic autoimmune urticaria (CAU)
- Allergen skin testing is of little value in CU except in rare cases. A high rate of false positive results may be expected, owing to the higher rate of dermographism
Lab evaluation and testing (3/3)

- A meta-analysis involving 29 clinical studies and more than 6000 cases found no association between # of tests ordered and diagnosis reached
- An underlying disease was found in 1.6% of cases tested (105 of 6462):
  - Cutaneous vasculitis (60 cases)
  - Thyroid disease (17 cases)
  - SLE (7 cases)
  - Connective tissue disease (16 cases)
  - Paraproteinemia (3 cases)

When to do a skin biopsy?

- Uncertain diagnosis
- No response to standard therapies
- Therapies that involve significant toxic effects are to be used

Differential diagnosis

- Systemic lupus erythematosus (SLE)
- Urticarial vasculitis: Hives are more painful and not so much pruritic. In addition:
  - Lesion last longer than 48 hours
  - Lesions leave residual pigmentation changes
  - Lesions recur whenever glucocorticoids are tapered

Typical findings with skin biopsies

- Histopathology of urticarial lesion:
  - Skin mast cells that have degranulated in the dermis
  - Perivascular leukocyte infiltrate:
    - Lymphocytes
    - Eosinophils
    - Neutrophils
    - Basophils
  - All migrate to skin lesion

Findings on skin biopsies

- Mast cells and basophils release histamine and other mediators (prostaglandins, leukotrienes, cytokines) on activation
- Result: local vasoconstriction, itch, and swelling in the skin.
- Histamine: central mediator, as suggested by the prominent clinical symptom of pruritus and response to antihistamines

Urticaria - H and E staining

- Skin mast cells that have degranulated in the dermis
- Perivascular infiltrate: lymphocytes, eosinophils, neutrophils, and basophils
Differential diagnosis and other causes (1/2)

- Drug- or food-based reaction
- Unrecognized infections such as hepatitis or mononucleosis
- Contact urticaria
- Insect bites leading to papular urticaria
- Urticaria pigmentosa
- Urticarial vasculitis
- Familial cold urticaria, Muckle-Wells syndrome, Schnitzler syndrome, Gleich syndrome (eosinophilic dermatitis).
- Cryoglobulinemia causing cold-induced urticarial or vasculitic lesions can be seen in hepatitis B or C infection.

Differential diagnosis and other causes (2/2)

- Acute urticaria/anaphylaxis to foods, drugs, and other agents frequently demonstrate skin symptoms within 2 hours.
- Bacterial/viral infections are common causes of acute urticaria in children.
- Of 88 children at an emergency department with delayed presentation of cutaneous eruption including urticaria, 66% were found to have evidence of a viral infection.

NSAIDs in urticaria

- NSAIDs: aspirin, naproxen and ibuprofen can trigger urticaria.
- Reaction is related to inhibition of cyclooxygenase by these agents.
- Reported frequency of NSAID-induced exacerbations of skin disease ranges from 25% to 50%.
- In some patients, period of aspirin sensitivity ends when the urticarial disease resolves.
- Up to 35% of patients with chronic spontaneous urticaria (CSU) experience exacerbation of skin symptoms when exposed to ASA/NSAID.

Treatment (1/4)

- Recent evidence-based guidelines support that the most effective, first-line therapy for CU is the use of the nonsedating second generation antihistamines:
  - fexofenadine, loratadine, desloratadine, cetirizine, and levocetirizine.
- In up to 50 percent of patients, this may only be partially effective and other therapies can be considered in addition.

Differential - urticarial vasculitis


Treatment (2/4)

- When nonsedating 2nd generation antihistamines are not enough consideration:
  - Increasing the dose of the nonsedating antihistamine.
  - Combine nonsedating antihistamine with:
    - A sedating older-generation antihistamine (diphenhydramine, hydroxyzine)
    - Or a tricyclic antidepressant such as doxepin, which blocks both H1 and H2 receptors.
    - Or cyproheptadine taken at bedtime.
  - Addition of an H2 blocker.
  - Consideration of a trial of a leukotriene pathway inhibitor.
Treatment (3/4)

- Although systemic corticosteroids work, the optimal dose and course has not been studied and repeated use can raise concerns regarding adverse reactions.
- Alternatives to corticosteroids in antihistamine-refractory cases of CU:
  - Omalizumab1,2,3


Treatment (4/4)

- Sulfasalazine1
- Dapsone, hydroxychloroquine2
- Calcineurin inhibitors such as cyclosporine and tacrolimus1
- Mycophenolate3,4,5

1McGirt LY, Vasagar K et al. Arch Dermatol. 2
3Trojan TD, Khan SIA. Curr Opin Allergy Clin Immunol 2012.

Chronic urticaria and Omaluzimab (1/3)

- In a study done by A. Kaplan et al., timing and duration of omaluzimab response was assessed.
  - 975 patients in the study; received either: placebo, omaluzimab 75 mg, 150 mg or 300 mg.
  - Urticaria Activity score (UAS) (range, 0-6):
    - Comprises a sum of daily ratings for itch severity and number of hives (0-3 points for each).
    - The weekly Urticaria Activity Score (UAS7) sums UAS scores during a 7-day period, and possible values for the UAS7 range from 0 to 42.

Kaplan et al. Journal of Allergy and Clinical Immunology, 2016.

Chronic urticaria and omaluzimab (2/3)

- The following definitions of response to treatment were based on the UAS7:
  - Either complete response (itch and hive free, UAS7=0)
  - Or well-controlled urticaria (UAS7 < 6)
  - 19 percent of patients achieved complete response (UAS7 = 0) in the 300 mg dose of omaluzimab by week 4, and 37 percent achieved well controlled (UAS7 < 6) urticaria in this group by week 4.

Chronic urticaria and omaluzimab (3/3)

- The timing of well-controlled and complete response suggests that there might be 2 categories of responders to omaluzimab:
  - Those who respond early (before week 4) and those who require more than 3 monthly doses to respond.
  - The median time to complete response was observed between 8 and 10 weeks for 300 mg of omaluzimab.

Response to omaluzimab in chronic urticaria

P < 0.05, **p<0.001. Reference 44
Kaplan et al. Journal of Allergy and Clinical Immunology, February 2016, Volume 137, Issue 2, pages 474-481.
Omaluzimab's effectiveness

- Meta-analysis of 67 studies and over 1000 patients treated with omaluzimab:
  - Reported a complete response rate of 72.2%
  - Partial response rate of 17.8%
  - This confirms that outside of clinical trials omaluzimab appears highly effective in treating CSU.

1Tharp MD, Bernstein JA et al. JAMA Dermatology 2018
2Miller RL, Shtessel M et al. J Allergy Clin Immunology. 2019

Omaluzimab resistance

- Omaluzimab resistance is poorly understood
- In a retrospective observation study done based on chart review: Resistance to omaluzimab in severe CSU appeared to be associated with the following factors:
  - Obesity
  - Arterial hypertension
  - High plasma C3 level
  - High CRP level

1Magen E, Chikovani T et al. Allergy Asthma Proc. 2019

Cyclosporine and urticaria (1/2)

- A meta-analysis of 18 studies investigated the efficacy and safety of cyclosporine in 332 treatment of CSU refractory to anti-histamines:
  - Though limited by quality and quantity of prior studies, the authors reported response rates for cyclosporine:
    - Up 73% with moderate dose therapy (4-5 mg/kg/day) for 12 weeks

1Kulthanan K, Chaweekulrat P et al. J Allergy Clin Immunology In Practice 2018
2Miller RL; Shtessel M; Robinson LB; Banerji A. J Allergy Clin Immunology. 2019

Urticaria - summary

- Typical urticaria are very pruritic, erythematous and raised
- Urticaria can be acute or chronic
- In acute urticaria, causes can be foods, medications, infections or other processes
- In chronic urticaria 80-90 percent of time causes are not identified
- Consensus regarding lab testing is limited to a few tests

Cyclosporine and urticaria (1/2)

- Adverse event rates occurred in 23% in the low dose group (2 to <4 mg/kg/day) and 57% in the moderate dose group.
- The number of adverse events increased with increasing dosage
  - However, rates of major adverse events (hypertension and elevated creatinine) did not significantly differ between the very low-dose group (6%) and the moderate dose group (10%).

1Kulthanan K, Chaweekulrat P et al. The J Allergy Clin Immunology In Practice 2018
2Miller RL; Shtessel M; Robinson LB; Banerji A. J Allergy Clin Immunology. 2019

Chronic urticaria summary

- Many patients will spontaneously remit, typically may last 2-5 years, with 1/10th of patients persisting beyond 5 years
- Treatment is focused initially on:
  - Second generation antihistamines, sometimes in multiple doses
  - Next moving on to 1st generation antihistamines
  - H2 blockers and LTRA.
- Other agents for treatment have been used more in limited cases
- Omaluzimab has been FDA approved since 2014
SKIN BIOPSY
TECHNIQUE

MICHAEL A NOWAK, MD

CONFLICTS

- No conflicts with the content of this lecture

SKIN BIOPSY

- Helps in cases of dilemma
- Opportunity to find something extraordinary
- Document diagnosis and justify treatment

CHOOSING THE LESION

- Classical, well-formed lesion
- Exception (early lesions in HSP, DH, BP)
- Avoid lesions that are modified

CHOOSING THE SKIN BIOPSY TECHNIQUE

- Size of Lesion
- Anatomic location
- Disease category or clinical diagnosis
- Proficiency or preference of clinician

SKIN BIOPSY TECHNIQUES

- Punch biopsy
- Shave biopsy
- Saucerization biopsy
- Wedge biopsy
- Incisional biopsy
- Excisional biopsy
SPECIAL CIRCUMSTANCES

- Urticaria pigmentosa
- Scalp biopsy trichoglyphics
- Immunofluorescence
- Lupus erythematosus

LOCAL ANESTHESIA

- Lidocaine: Infiltration, ring, or field block
- Topical: EMLA with 2 hours occlusion = 5 mm
- Lidocaine with epinephrine:
  - Use with caution in digital block
  - Use with caution in pregnancy

COMPLICATIONS

- Hypersensitivity to local anesthetic
- Discomfort associated with local anesthetic
- Bleeding
- Scarring
- Infection

PRACTICAL CONSIDERATIONS

- Optimal strength (10%) and volume of formalin solution (10X)
- Minimal handling of tissue to avoid crush artifact from forceps
- Place tissue in bottle (not in the cap)
- Infiltrate with optimal local anesthetic to avoid "artificial edema"
- Avoid electrocautery or cryotherapy artifact
- Avoid secondary or treated lesions

PRACTICAL CONSIDERATIONS

- Non-specific findings or misdiagnosis can result from improper:
  - Biopsy site selection
  - Lesion selection
  - Technique (superficial specimen)
  - Choice of transport media
  - Examples include:
    - False-negative DIF and sampling error in NMSC or large pigmented lesions
    - Multiple biopsies in polymorphic lesions and large pigmented lesions

SKIN BIOPSY IN SPECIFIC DISEASES

- Bullous diseases
- Lupus erythematosus
- Vasculitis
- Parapsoriasis
- Hair disorders
- Epithelial neoplasms
- Malignant melanoma
- Dermatofibrosarcoma Protuberans
- T and B cell lymphomas
SKIN BIOPSY IN SPECIFIC DISEASES
Panniculitis

- Deep incisional biopsy (not shave biopsy)
- Double punch technique or electric rotary power punch
- 6 mm punch minimum size divided for culture and H&E

SKIN BIOPSY IN SPECIFIC DISEASES
Hair Disorders

- More than 1 biopsy is helpful
- Established lesion
- 4 mm punch is ideal:
  - Remember trichoglyphics
  - Remember to obtain adequate depth (4-5 mm)
- Normal scalp biopsy is not essential
- Transverse sections, vertical sections, or both

SKIN BIOPSY IN SPECIFIC DISEASES
Hair Disorders

- Tyler Technique - 3 pieces, vertical and transverse
- HoVert Technique - 4 pieces, vertical and transverse
SKIN BIOPSY IN SPECIFIC DISEASES
Epithelial Neoplasms

- Shave biopsy: Epidermal lesions
- Punch biopsy: Dermal lesions
- Curettage: Potential interpretation limitations
- Special considerations: Microcystic adnexal carcinoma

SKIN BIOPSY IN SPECIFIC DISEASES
Malignant Melanoma

- Complete excision
- Saucerization: Macular lesions
- Orientation: Score, ink, or suture
- Partial biopsy specimens are associated lower yield
- Lentigo Maligna: Broad shave or shaves (not punch)
- Multicolor lesions: Multiple shaves

SKIN BIOPSY IN SPECIFIC DISEASES
Dermatofibrosarcoma Protuberans

- DFSP requires a deep biopsy to show the characteristic honeycomb pattern involving the subcutis
- Deep incisional biopsy is recommended
- Superficial biopsies result in a dilemma
  - Cellular dermatofibroma vs. DFSP
  - Immunostains can be helpful in superficial specimens
SKIN BIOPSY IN SPECIFIC DISEASES
Cutaneous T-cell Lymphoma

- T = T-cell and Top Heavy
- Broad shave biopsies include a wide area of the dermoepidermal junction to help demonstrate epidermotropic lymphoid cells
- Broad shave biopsies are also ideal for immunostains and gene rearrangement studies
- Multiple shave biopsies from different anatomic sites may be needed to establish the diagnosis

SKIN BIOPSY IN SPECIFIC DISEASES
Primary Cutaneous B-cell Lymphoma

- B = B-cell and Bottom Heavy
- B-cell lymphoma requires a deep biopsy showing the infiltrate’s architecture and zonal immunostaining patterns
  - B-cell lymphoid hyperplasia vs. B-cell lymphoma
- Superficial biopsies can result in misinterpretation due to insufficient sampling of the deep dermal infiltrate
- Deep incisional biopsy is recommended
Summary

- Choosing the biopsy site and lesion:
  - Lesional vs. perilesional
  - Early vs. established
  - H&E vs. DIF

- Choosing the skin biopsy technique:
  - Superficial vs. deep

- Practical considerations: Avoid artifacts
- Skin biopsy techniques for specific diseases
- Multiple biopsies especially in polymorphic lesions

Michael A. Nowak, MD
man2004@comcast.net
Physician Wellness an Osteopathic Approach
Robert G.G. Piccinini, DO, DFACN

Objectives
- Understand the scope of the problem that confronts physicians today in regards to burnout/moral injury/damaged resiliency.
- Recognize the factors which are impacting your own career development and progression.
- Learn practical solutions to aid in the road to recovery and rebuild resiliency.

"I will remember that there is art to medicine as well as science, and that warmth, sympathy, and understanding may outweigh the surgeon’s knife or the chemist’s drug” - Louis Lasagna
- The dynamic is to point out the symptoms, and give training to extinguish it.
- Teach them to maintain life balance, physical health and rest as they go through training.
- You do that …. They will maintain their empathy

Disclosure:
- I have no conflict of issues in the Presentation of this information.

Doctors are programed to be workaholics, Superhero perfectionists operating with a pair of cardinal rules:
- The patient comes First- Praise worthy but extremely unhealthy if you never learn the off switch
- Never show weakness
- As a doctor burns out (or loses resiliency), one of the symptoms is loss of empathy, compassion, and being cynical and sarcastic about your patients

Percentage of physicians burned out
54
Percentage of resident physicians suffering from depression
29
Estimated number of physicians committing suicide every year
400

Sources: Shanafelt et. al., 2012 & 2015; Mata et al., 2015; American Foundation of Suicide Prevention, 400; Andrew, 2015
• Mental Well-Being & Osteopathic Medical Students (n=10,187)
  - 46% of students screened for depression fell into the Clinical Concern range
  - 10% indicated they had suicidal ideations within 30 days prior to being surveyed
  - 147 were classified as high risk for suicide.

Burnout study in osteopathic residents (otolaryngology) (n=684)
- 10% experienced high burnout
- 76% moderate burnout
- 14% low burnout

Sources: Yeh et al. (2008)

Causes of Burnout
- Workload
- Perceived Lack of Control
- Reward or lack of
- Community
- Fairness
- Values mismatch

Sources: Yost et al. (2014)

In the 2015 Medscape Physician Lifestyle Report physician were asked to rank the causes of burnout in order of significance.
- 1. Too many bureaucratic tasks
- 2. Too many hours at work
- 3. Insufficient income
- 4. Increasing computerization of practice

Burnout by Region

White Males, 26 US states, 1984-92

Peterson & Burnett, 2007
Baby Boomers (Boomers or Me Generation)

Born: 1945-1964
Commonly described as:
• Optimistic
• Collaborative
• Team-oriented
• Tech-savvy
• Culturally diverse
• Globally oriented

For DOs: Represents 24% of osteopathic physicians
This generation is known to occupy the majority of positions of authority.
Equate work with self-worth, contribution, and personal fulfillment

Work Values/Ethics:
• Respect authority
• Loyalty to their organizations
• Hardworking
• Promotion earned over time
• Focused on leaving legacy

Teaching & Learning:
• Focus is on leaving a legacy
• Wants their knowledge to have been an asset to the organization

Generation X

Born: 1965-1980
Commonly described as:
• Independent
• Self-directed
• Pragmatic
• Flexible

Holds most mid-level positions
For DOs: Represents 35% of osteopathic physicians.

Millennials

Born: 1981-1999
Commonly described as:
• Optimistic
• Collaborative
• Team-oriented
• Tech-savvy
• Globally oriented
• Majority of medical students and residents today.

For DOs: Represents 36% of the osteopathic physicians (includes students).

Work Values/Ethics
• Believe can make an impact at any age
• Seek to contribute immediately to an organization
• Do not want to wait for years before being heard
• Value being connected with others

Teaching & Learning:
• Expect frequent feedback and need praise
• Prefer information presented individually or via technology

Traditionalists (Silent Generation)

Born: 1925-1945
Commonly described as:
• Dedicated
• Patriotic
• Conventional
• Respecting order
• Altruistic

For DOs: Comprise 6% of all osteopathic physicians.

Work Values/Ethics:
• Formality
• Hierarchy
• Loyalty to their organizations
• Hardworking
• Expect rewards for hard work

Teaching & Learning:
• Sense of duty to teach
• Comfortable with teaching and learning in a large group
• Lecture format
• bedside teaching is anecdotal
• Keepers of institutional memory
• Feedback given only to correct or improve mentees

William G. Anderson, DO
(ADA President, 1994)

Comfort with Technology

<table>
<thead>
<tr>
<th>Generation</th>
<th>Birth Years</th>
<th>Pop.</th>
<th>Major Societal Events</th>
<th>Childhood Characteristics</th>
<th>Personal Characteristics</th>
<th>Work Characteristics</th>
<th>Comfort with Technology</th>
</tr>
</thead>
<tbody>
<tr>
<td>Traditionalist</td>
<td>1925–1945</td>
<td>37 million</td>
<td>Great Depression, Pearl Harbor, WWII, bombing, Civil War</td>
<td>Nuclear family, married young, divorce uncommon</td>
<td>Dedicated, patriotic, respect order, altruistic</td>
<td>Formality, hierarchy, and loyalty; very hard-working</td>
<td>Low</td>
</tr>
<tr>
<td>Baby Boomers</td>
<td>1946–1964</td>
<td>77 million</td>
<td>Civil rights and women’s rights movements, Vietnam War, Woodstock, TV, economic prosperity</td>
<td>Nuclear family with stay-at-home mom and working father</td>
<td>Elite, competitive, driven, anti-authoritarian</td>
<td>Workaholics, competitive, consensus builders, mentor</td>
<td>Medium</td>
</tr>
<tr>
<td>Millennials</td>
<td>1981-1999</td>
<td>80 million</td>
<td>9/11, Digital Age, natural disasters, Columbine shootings</td>
<td>Protective “helicopter parents”</td>
<td>Confident, high-achieving, creative, practical</td>
<td>Feedback-oriented, team-oriented, appreciate cultural diversity, idealistic, want to make impact quickly</td>
<td>Very high</td>
</tr>
</tbody>
</table>
Signs of Burnout

4 areas characterized by certain signs and symptoms, some of which overlap

- **Exhaustion**
- **Depersonalization**
- **Lack of Accomplishment**
- **Ineffective Mentoring**

Cultural Differences (Diversity)

*Diversity* means all the ways we differ. It includes the readily visible differences and the underlying differences that may be below the surface.

Address Biases

- Use online tools to enhance personal communication, not replace face-to-face interaction
- Recognize biases and respect differences
- Change mentoring style based on mentee to enable mentoring across differences
- Incorporate information-sharing and shared problem solving; offer frequent and frank feedback; and refrain from comparing today to the glories of yesterday
- Create an atmosphere of reciprocal mentoring
- Model professionalism

Roadmap to Your Reset Point

**Where are you starting?**

**Where do you want to go?**

**What is the process in between?**

Wellness Culture

- Create Framework
- Develop a program
- Foster at an individual level
- Empower faculty and Trainees to confront burnout
- Create a sustainable culture of wellness and resiliency

Some events may be Physician-centric. But remember at all stages if we can include other members of the family the experience may be more fulfilling and rewarding. It's about building memories not just doing things.

- Movie Nights or dinners
- Holiday Potlucks
- Recreational Classes
- Charity Work
- Lunchtime exercise or yoga
- Sporting events
- Exploring the local or regional attractions

Concept: Edward T. Hall (1976)
Osteopathic Approach focuses on the 8 Dimensions of Wellness:

- **EMOTIONAL**
  - A positive self-concept, which includes dealing with feelings constructively and developing positive qualities such as optimism, trust, self-confidence, and determination.

- **ENVIRONMENTAL**
  - Health by occupying pleasant, stimulating environments that support well-being.

- **INTELLECTUAL**
  - Recognizing creative abilities and finding ways to expand knowledge and skills.

- **PHYSICAL**
  - Recognizing the need for physical activity, healthy foods, and sleep.

- **OCCUPATIONAL**
  - Personal satisfaction and enrichment from one's work.

- **SPIRITUAL**
  - Expanding a sense of purpose and meaning in life.

- **SOCIAL**
  - Developing a sense of connection, belonging, and a well-developed support system.

- **FINANCIAL**
  - Satisfaction with current and future financial situations.

Applying Wellness Adapted from SAMHSA's Eight Dimensions of Wellness

Source: https://www.samhsa.gov/wellness-initiative/eight-dimensions-wellness

Golden Opportunity

- **Members Now**
  - Issues facing physicians who have graduated and are full members
  - Provide services now

- **Members in the Future (students, interns & residents)**
  - Concerted effort to assist
  - Cannot abdicate our responsibility (i.e., wait for others to act, or just not participate)
  - Be present at these crucial times

- **Osteopathic Conundrum**
  - Very few practicing osteopaths have the time and resources to devote to another program

- **Perfect Opportunity**
  - For the COMS, AOA, State Affiliates and Specialty Societies to step up.
  - Provides relevance for all organizations and a reason for members to join and exist
  - Can be complementary and non competitive

Key Elements of Effective Transgenerational Leadership

- Be flexible.
- Be a good listener.
- Develop your emotional intelligence.
- Be less defensive and more open, and assume people have good intentions.
- Learn to delegate, build teams, and use consensus.
- Create and communicate the big picture and “why” you are doing what you’re doing.
- Provide timely feedback to your team, and thank them for all their hard work.
- Honor work-life balance.
- Help people see the “what” and “why,” but let them determine the “how.”
- Maintain a sense of humor.
- Invest in your people, and help them continue to grow.
- Figure out how technology can help people without enslaving them.

Other Ideas

- Set expectations early
  - Ethics and standards
  - When/how to communicate
  - Rules for cellphone/computer use
  - What you expect in a formal email
  - Consequences for unacceptable behaviors
  - Feedback assessing the quality of trainees’ performance and suggesting alternate behaviors, if necessary
  - Tell them what they need to learn and why they need to learn it
  - Establish a sense of personal responsibility
  - Use peers effectively

Source: (Moreno-Walton)
What’s Utopia?

- Enhancing the medical team... what does the utopia look like?
  - Working with other health professionals
  - Treating every person on the floor and in the organization like you want to be treated
  - The golden rule of life translates to medicine – EVERYONE HAS VALUE
  - Putting self in the other person’s shoes and understanding where everyone has challenges and how to assist.
  - Leverage everyone’s abilities and time
  - Create an atmosphere of mutual respect and appreciating everyone’s efforts as well as recognizing where we are all accountable.

Physicians are not the center of the universe. It takes a team to ultimately provide the best patient care.

Common Drivers and Selected Organizational-Level Solutions for Physician Burnout

| Excessive workload | Fair productivity targets
|                    | Duty hour limits
|                    | Appropriate distribution of job roles
| Work inefficiency and lack of work support | Optimized electronic medical records
|                                                                 | Non-physician staff support to offload clerical burdens
|                                                                 | Appropriate interpretation of regulatory requirements
| Lack of work-home integration | Respect for home responsibilities at setting work and meeting schedules
|                                                                 | Include all required work tasks within expected work hours
|                                                                 | Support flexible work schedules, including part-time employment
| Loss of control and autonomy | Physician engagement in setting work requirements and structure
|                                                                 | Physician leadership and shared decision-making
| Loss of meaning from work | Promote shared core values
|                                                                 | Protect physician time with patients
|                                                                 | Promote physician communities
|                                                                 | Other professional development opportunities
|                                                                 | Leadership training and awareness around physician burnout
LIFE

Born 00/00/000  Died 00/00/0000
Live the Dash!

Shanafelt et al., 2012 & 2015; Mata et al., 2015; American Foundation of Suicide Prevention, ND; Andrew, 2015
- AAMC Mental Health Awareness Taskforce (2015)
Petersen & Burnett, 2007

Cosmetics and Dentistry
Stacy Spizuoco, DDS FACD

I have no disclosures

Twin

- Current Photo Amy & Stacy
- Baby photos

Identical Twins

Amy Spizuoco DO, FAOCD
Stacy A. Spizuoco DDS, FACD
Basketball
Photo of us playing

Teamwork

Dentist
- Video of Harmionie
Teeth

- 32
- 28
- Molars
- Premolars
- Incisors

Orthodontics

- The branch of dentistry concerned with the correction and prevention of irregularities and malocclusion of the teeth
- General, Perio, Endo, Pedo, Oral Surgery, Pathology

Class Types

Arch Types

X-ray with 32 and 28
Orthodontics

- Clear aligners
- Invisalign
- Braces

Dental Esthetics

- Structure
- Smile arc
- Cant Buccal corridors
- Lip competency

Facial Esthetics

- Lip Contour: fullness, symmetry, competency
- Maxillary Protrusion
- Mandibular Position
- Facial Height
- Appearance of chin
- Nasal Prominence
WHAT ARE THESE STAINS?

Michael A Nowak, MD

CONFLICTS

- No conflicts with the content of this lecture

TYPES OF STAINS

- Routine H&E
- Conventional special stains
- Immunohistochemical stains
- Immunofluorescent stains

ROUTINE STAINS

- H&E = Hematoxylin and Eosin
  - Hematoxylin stains cell nuclei blue
  - Eosin stains extracellular matrix and cytoplasm pink

ROUTINE STAINS

“Bug” Stains

- PAS: Periodic acid Schiff
- GMS: Grocott’s methanamine silver
- AFB: Ziehl-Neelsen stain, Wade-Fite
- Gram: Brown and Brenn
- Warthin-Starry
- Giemsa

ROUTINE STAINS

- PAS
- Giemsa
- Fontana-Masson
- Congo red
- Alcian Blue
IMMUNOHISTOCHEMICAL STAINS

- Antigen-Antibody reaction
- Different techniques
- Different chromogens
- Adjunct to H&E to confirm differentiation (not malignancy)
- Diagnostic
- Prognostic

IMMUNOHISTOCHEMICAL STAINS

- Panels or combinations of stains to determine differentiation
- Which lesions are positive with what stains?
- What stains are positive in which lesions?
- Specific situations: Histologic patterns
- Specific lesions: Melanoma and lymphoma
- Notable stains: CD vs CK

IMMUNOHISTOCHEMICAL STAINS
Specific Patterns

- Pagetoid pattern
- Clonal pattern
- Non atypical dermal spindle cell lesion
- Atypical dermal spindle cell lesion
- Small round blue cell tumor
- Large anaplastic epithelioid cell tumor

IMMUNOHISTOCHEMICAL STAINS
Pagetoid Pattern

- Atypical intraepidermal epithelioid cells involving upper and lower layers in small clusters of solitary cells.
- Prototype: Paget’s disease
- Paget’s disease, extramammary Paget’s disease, melanoma in situ, Pagetoid SCCs, sebaceous carcinoma, Pagetoid reticulosis, Merkel cell carcinoma, some amelanotic carcinomas metastatic to the skin.

IMMUNOHISTOCHEMICAL STAINS
Pagetoid Pattern

- Paget’s and extramammary Paget’s disease: CEA and CK7
- Melanoma in situ: S100, Mart-1 (Melan A), MITF, SOX10
- Pagetoid SCCs: Pan CK (not CK7)
- Sebaceous carcinoma: CEA, EMA (not CK7)
- Pagetoid reticulosis: CD3, CD4 or CD8 (not CD7)
- Merkel cell carcinoma: CK20 dot-like
**IMMUNOHISTOCHEMICAL STAINS**

**Clonal Pattern**

- Intrasitial epithelioid cells involving upper and/or lower layers in variably sized clusters of similar cells (clones).
- Borst-Jadassohn phenomenon
- Prototype: Basaloid carcinoma
- Poroma, melanoma in-situ (nested), clonal SCCIS, clonal SK, superficial basal carcinoma, porocarcinoma

**IMMUNOHISTOCHEMICAL STAINS**

**Clonal Pattern**

- Basal cell carcinoma
- Nevoid basal cell carcinoma syndrome (BCC syndrome)
- Poroma, melanoma in-situ (nested), clonal SCCIS, clonal SK, superficial basal carcinoma, porocarcinoma

**IMMUNOHISTOCHEMICAL STAINS**

**Non Atypical Dermal Spindle Cell Lesion**

- Uniform spindle-shaped cells involving the superficial and deep dermis (usually not the subcutis)
- Prototype: Fibrous histiocytoma (Dermatofibroma)
- Dermatofibroma, scar, leiomyoma, smooth muscle hamartoma, dermatomyofibroma, cellular blue nevus, and desmoplastic melanoma
IMMUNOHISTOCHEMICAL STAINS
Non Atypical Dermal Spindle Cell Lesion

- Dermatofibroma: Factor XIIa +, CD34 -, S100 -, Actin +/-
- Scar: Triple negative (Factor XIIa, CD34, S100) Actin +/-
- Leiomyoma/smooth muscle hamartoma: Actin +, Desmin +
- Dermatomyofibroma: Actin +, Factor XIIa -, CD34 -, S100 -
- Neurofibroma and cellular blue nevus: S100 +, SOX 10 +
- Desmoplastic melanoma: S100 +, SOX 10 +

IMMUNOHISTOCHEMICAL STAINS
Atypical Dermal Spindle Cell Lesion

- Non uniform (atypical) spindle-shaped cells involving the superficial and deep dermis (and frequently the subcutis)
- Prototype: Atypical fibroxanthoma
- AFX, poorly differentiated SCC, angiosarcoma, Kaposi’s sarcoma, leiomyosarcoma, DFSP, spindle cell melanoma

IMMUNOHISTOCHEMICAL STAINS
Atypical Dermal Spindle Cell Lesion

- AFX: CD10, CD68
- Poorly differentiated SCC: P63
- Angiosarcoma: CD31
- Kaposi Sarcoma: HHV-8
- Leiomyosarcoma: Desmin
- DFSP: CD34
- Spindle cell melanoma: S100, SOX10, Multiplex stains

IMMUNOHISTOCHEMICAL STAINS
Small Round Blue Cell Tumor

- Uniform "small" basophilic cells involving the superficial and deep dermis (usually no epidermal continuity)
- Prototype: Merkel cell carcinoma
- Merkel cell carcinoma, small cell carcinoma (metastatic), melanoma, lymphoma, basal cell carcinoma
IMMUNOHISTOCHEMICAL STAINS
Small Round Blue Cell Tumor

- Merkel Cell Carcinoma: CK20 dot-like
- Small Cell Carcinoma (metastatic): TTF-1
- Melanoma: SOX10
- Lymphoma: CD45
- Basal Cell Carcinoma: Pan CK, Ber-EP4

IMMUNOHISTOCHEMICAL STAINS
Large Anaplastic Epithelioid Cell Tumor

- Large non uniform epithelioid cells involving the superficial and deep dermis (usually no epidermal continuity)
- Prototype: Metastatic carcinoma
- Metastatic carcinoma, adnexal carcinoma, PO squamous cell carcinoma, anaplastic large cell lymphoma, melanoma

IMMUNOHISTOCHEMICAL STAINS
Large Anaplastic Epithelioid Cell Tumor

- Metastatic Carcinoma: P63 Negative
- Adnexal Carcinoma: P63 Positive
- Squamous Cell Carcinoma: CK5/6
- Anaplastic Large Cell Lymphoma: LCA (CD45) and Pan CK
- Melanoma - Nodular or Metastatic: S100, SOX10, etc

Use of p63 expression in distinguishing primary and metastatic cutaneous adnexal neoplasms from metastatic adenocarcinoma to skin.

Ivan D. Rashid, MD; Richard V. Edwards, MD; Laura D. Lilen, MD; Alan I. Loo, MD; Ali E. Loo, MD; Department of Pathology, University of Texas - M.D. Anderson Cancer Center
IMMUNOHISTOCHEMICAL STAINS
Specific Lesions

- Malignant melanoma
- Cutaneous T-cell lymphoma
- Cutaneous B-cell lymphoma

IMMUNOHISTOCHEMICAL STAINS
Malignant Melanoma

- SSMM: Pagetoid or nested pigmented epithelioid cells
- LM: Confluent pigmented epithelioid or spindle cells
- ALM: Pigmented cells with thicker dendrites
- DMM: Inconspicuous dermal spindle cells +/- LM
- Metastatic MM: Large epithelioid cells in dermis only

IMMUNOHISTOCHEMICAL STAINS
Malignant Melanoma

- SSMM: Atypia: Upward spread: MART-1, S100, etc
- LM: Minimal atypia high density: MITF, SOX10 > MART-1
- ALM: Thickened dendrites: MART-1 (aka Melan-A)
- DMM: Frequently negative for stains except: S100, SOX10
- Metastatic MM: Lacks epidermal component: S100, SOX10
- Adjunctive/Prognostic: HMB-45, BAP-1, P16, Ki-67, BRAF

The American Journal of Dermatopathology 2013 Jun;35(6)
Diagnostic Utility and Comparative Immunohistochemical
Analysis of MITF-1 and SOX10 to Distinguish Melanoma In Situ
and Actinic Keratosis.

Illustration by JD, Photo by RP, Jamie Galasso CA, Sailer S
Department of Pathology, University of Texas - MD Anderson Cancer Center
**IMMUNOHISTOCHEMICAL STAINS**

**CTCL**

- Top heavy (superficial)
- Intraepidermal lymphoid cells in basal layer or pagetoid
- Minimal epidermal spongiosis or interface alterations
- Dermal lymphoid cells and fibrosis

**IMMUNOHISTOCHEMICAL STAINS**

**CTCL**

- CD2, CD3, CD5, and CD7: Pan T-cell markers
- CD4: T-helper
- CD8: T-suppressor
- CD7: Expression is frequently lost in MF

**IMMUNOHISTOCHEMICAL STAINS**

**B-cell Lymphoma**

- Bottom heavy (deep)
- Monotonous dermal lymphoid cells with a Grenz zone
- Nodular, diffuse, or perivascular
- Lacks germinal centers

**IMMUNOHISTOCHEMICAL STAINS**

**B-cell Lymphoma**

- CD19, CD20, CD21, CD22, CD79: Pan B-cell markers
- BCL-6 and CD10: Follicular lymphoma
- CD5 and CD23: CL/USLL and mantle cell lymphoma (bad)
- BCL2: Marginal zone lymphoma (good)
- CD30: Large cell lymphomas
- SOX11: Mantle cell lymphoma
IMMUNOHISTOCHEMICAL STAINS
Notable Stains

- P63
- SOX10
- CD7
- CK7
- CD20
- CK20

P63
- Distinguishes primary skin cancers from metastatic cancers
- Distinguishes epithelial tumors from melanocytic lesions
- Distinguishes poorly differentiated SCC from AFX

SOX10
- Excellent melanocyte marker
- Nuclear stain similar to MITF
- Melan A frequently overemphasizes melanocyte density
- SOX10 and MITF very helpful in LM and unstable lentigo

CD7
- Pan T-cell marker
- Helpful in superficial dermal lymphoid infiltrates
- Distinguishes T cells from B cells
- Except expression is lost in MF

CK7
- Cytokeratin associated with breast cancer and Paget's
- Extramammary Paget's Disease (CK7 positive)
- Bowen's disease and MIS (CK7 negative)
CD20

- Pan B-cell marker
- Helpful in deep dermal lymphoid infiltrates
- Distinguishes B cells from T cells

CK20

- Cytokeratin associated with GI malignancies
- Merkel cell carcinoma is dot-like CK20 positive
- Subset of extramammary Paget’s disease is CK20 positive

Summary

- Why do we do immunohistochemical stains?
  - Identify or confirm differentiation via antigen expression
  - Specific patterns
  - Specific lesions
  - Notable stains
    - P63
    - SOX10
    - CK vs CD

Michael A. Nowak, MD
man2004@comcast.net
Saturday, September 28, 2019

6:30 a.m. - 7:00 a.m.  Breakfast with Exhibitors  
Broadway Ballroom F

7:00 a.m. - 8:00 a.m.  
**Dermatologic Surgery: Lessons Learned Along the Way**  
Stephen Kessler, DO, FAOCD

8:00 a.m. - 9:00 a.m.  
**JAAD Update & Cutaneous Infections**  
Dirk Elston, MD

9:00 a.m. - 10:00 a.m.  
**Do You Know JAK? New Applications for Janus Kinases Inhibitors in Dermatology**  
**Patients vs. Doctors: One-Star Reviews, Yelp and Social Media**  
Neal Bhatia, MD

10:00 a.m. - 10:30 a.m.  Break with Exhibitors  
Broadway Ballroom F

10:30 a.m. - 11:00 a.m.  
**Understanding Oral Lesions**  
Stacy Spizuoco, DDS

11:00 a.m. - 12:00 p.m.  
**Contact Dermatitis**  
Matthew Zirwas, MD

12:00 p.m. - 1:00 p.m.  Lunch in the Expo  
(Note: Limited quantities available)

1:00 p.m. - 1:30 p.m.  
**Osteopathic Continuous Certification Update**  
Lloyd Cleaver, DO, FAOCD

1:30 p.m. - 2:30 p.m.  
**Diagnostic and Therapeutic Pearls**  
Mark Lebwohl, MD

2:30 p.m. - 3:30 p.m.  
**Updates in Pediatric Dermatology**  
Lisa Swanson, MD

3:30 p.m. - 4:00 p.m.  
**A Dermatologist’s Guide to Using Anti-IL17 Agents to Effectively Manage Patients with Challenging Psoriatic Disease**  
Mark Lebwohl, MD

4:00 p.m. - 4:30 p.m.  
**A Multidisciplinary Approach to Cutaneous Squamous Cell Carcinoma**  
Mark Lebwohl, MD & Todd Schlesinger, MD

4:30 p.m. - 5:30 p.m.  
**What’s New in the Treatment of Hypertrophic Scars & Keloids - 2019**  
Michael H. Gold, MD
DISCLOSURES

I have no actual or potential conflict of interest in relation to this presentation.
CLOSURE OF SKIN LACERATIONS UNDER TENSION

S. Liddle, M. Davis, and B. Dheansa
Ann. R. Coll. of Surgeons of England 2012 Jan; 94

BASOSQUAMOUS CARCINOMA: CONTROVERSY, ADVANCES, AND FUTURE DIRECTIONS
Caroline Z. Tan, RA*, Kerr E. Rieger, MD, PhD, * and Kavita Y. Sarin, MD, PhD
© 2016 by the American Society for Dermatologic Surgery, Inc.
ISSN: 1076-0512 Dermatol Surg 2017;43:23-31
DOI: 10.1097

MARGIN STATUS IN SHAVE BIOPSIES OF NON-MELANOMA SKIN CANCERS
IS IT WORTH REPORTING?
- OBJECTIVE: TO ADDRESS THE ACCURACY OF MARGIN EVALUATION IN SHAVE BIOPSIES OF NON-MELANOMA SKIN CANCERS
- RESULTS: 47 CONSECUTIVE CASES WERE COLLECTED, INCLUDING 20 SQUAMOUS CELL (43%) AND 27 BASAL CELL (57%) CARCINOMAS. 11 OF 47 CASES (23%) WITH NEGATIVE MARGINS AT INITIAL DIAGNOSIS DEMONSTRATED POSITIVE MARGINS UPON DEEPER-LEVEL EXAMINATION. 8 OF 27 BASAL CELL CARCINOMAS (30%) AND 3 OF 20 SQUAMOUS CELL CARCINOMAS (15%) WERE ERRONEOUSLY CLASSIFIED AS “NEGATIVE”

Alicia Schneebelen, MD; Jerad M. Gardner, MD; Sara C. Shalin, MD, PhD
Department of Pathology, University of Arkansas for Medical Sciences
PREDICTIVE VALUE OF MARGINS IN DIAGNOSTIC BIOPSIES OF NON-MELANOMA SKIN CANCERS

Twelve of 148 squamous cell carcinomas (8.1%) had negative biopsy margins and all of the subsequent excisions were free of residual tumor. Nine of 87 basal carcinomas (10.3%) had negative biopsy margins, seven of those nine (77.8%) had residual tumor present in subsequent excisions.

JACKSON JE, KELLY B, PETITT M, UCHIDA T, WAGNER RF JR.

DIVISION OF DERMATOLOGY, DEPARTMENT OF MEDICINE, UNIVERSITY OF CALIFORNIA LOS ANGELES SCHOOL OF MEDICINE, LOS ANGELES, CALIFORNIA, USA.

EFFECTIVENESS AND SAFETY OF SURGICAL EXCISION IN THE TREATMENT OF DIGITAL MUCOID CYSTS

Multiple Therapies For Treating Digital Mucoid Cysts:

- Cryotherapy
- Injection with intralesional triamcinolone
- Drainage
- Excision
- CO2 Laser

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ISSN: 1076-0512 DERMATOL SURG 2017;43:928-933
DOI: 10.1097/DSS.1096
A CRYPTIC SET OF PHRASES DEVELOPED IN OUR OFFICE COMMUNICATING EITHER A GOOD ACTION OR A LESS THAN DESIRABLE ONE HAD JUST OCCURRED

BOXED VS PACKAGED GLOVES

COMPARISON OF STERILE VS NON STERILE GLOVES IN CUTANEOUS SURGERY AND COMMON OUTPATIENT DENTAL PROCEDURES: A SYSTEMATIC REVIEW AND META-ANALYSIS

DROPPING AN INSTRUMENT ON THE FLOOR
COMMUNICATE WITH PATIENTS THE EVENING AFTER A PROCEDURE

EXTRA THIN, HYDROCOLLOID DRESSING

Use of an extra thin, hydrocolloid dressing is designed to reduce the risk of further skin breakdown due to friction.

ASSISTING PATIENTS TO VEHICLES
USE OF A HYDROPHILIC POLYMER AND POTASSIUM FERRATE POWDER FOR HEMOSTASIS

NAIL GLUE FOR FISSURES

ADVANTAGE OVER NAIL GLUE IS IT MAY BE USED MULTIPLE TIMES

“STICKER” SIGN: SQUAMOUS CELL CARCINOMA

AFTER RESEARCHING OVER 30 REPUTABLE SOURCES, ONLY ONE SOURCE, POLLEY CLINIC, DERMATOLOGY & DERMATOLOGIC SURGERY LOCATED IN WILSON, NC WAS FOUND TO HAVE NOTED THAT SQUAMOUS CELL CARCINOMAS CAN FEEL LIKE A “STICKER” OR A “THORN” IS PRESENT INSIDE THE LESION. BELOW ARE JUST A FEW OF THE SOURCES THAT HAD NO INDICATION OF THE “STICKER” SIGN:

STANFORD, MAYO CLINIC, AMERICAN ACADEMY OF DERMATOLOGY, AOCD, UCSF, SKINCANCER.ORG, HEALTHLINE, WEBMD, DERMNETZ, HARVARD, AND MEDSCAPE

BLAST

A METHOD FOR DEALING WITH DISPLEASED PATIENTS

AUTHOR: ALBERT BARNETO, FOUNDER OF CAFÉ ROCKET
BLAST
WHEN DEALING WITH UPSET PATIENTS, WE OFTEN BECOME ANXIOUS, DEFENSIVE, OR ANGRY. BLAST IS QUITE EFFECTIVE FOR DEFUSING, REDIRECTING, AND CORRECTING THESE SITUATIONS. SIMPLY SAY TO YOURSELF “THIS IS GOING TO BE A BLAST,” AS YOU ENTER THE ROOM.

BELIEVE
EXPRESS BELIEF, EVEN IF THE PATIENT IS EXAGGERATING, LYING, OR INCORRECT.

LISTEN
STOP THE MIND AND MOUTH AND LISTEN TO WHAT THE PATIENT IS SAYING. GIVE THE PATIENT A “MAGIC MINUTE” TO EXPRESS ALL OF THEIR GRIEVANCES.

APOLOGIZE
APOLOGIZE FOR WHAT THE PATIENT IS EXPERIENCING AND THEIR UNMET EXPECTATIONS. A SINCERE APOLOGY CAN HELP TO DIFFUSE THE PATIENT’S FEAR, FRUSTRATION, AND ANGER.

SATISFY
ATTEMPT SOLVING THE PATIENT’S CONCERNS. EXPLAIN REASONABLE OPTIONS, THE TIME FRAME FOR IMPROVEMENT, AND WHETHER THE PROBLEM CAN BE COMPLETELY RESOLVED.

THANK
WRAP UP THE ENCOUNTER THANKING THE PATIENT FOR THE SECOND CHANCE; FOR ALLOWING YOU THE INFORMATION THAT SOMETHING OCCURRED THAT UPSET THEM; FOR STAYING IN YOUR PRACTICE.
Cost-effective Medicine

- Ogbechie-Godex OA, et al. 5 laboratory tests to reconsider. JAAD 2018; 78: 1332-5
- Potassium testing in young healthy women on acne doses of spironolactone
- For hirsutism doses or those with renal impairment watch kidney function, K, and possible sulfa interaction
- Monthly labs for isotretinoin
  - If no bump in 6–8 weeks, unlikely to bump
- Monthly labs for terbinafine
- Those with elevations were symptomatic

Cost-effective medicine

- Random TSH in vitiligo
  - Testing should be driven by signs or symptoms
  - Counsel patients about signs and symptoms
  - Screen ANA for biologics
  - CBC, metabolic panel for biologics
  - Even for TB, signs and symptoms are key

Hidradenitis suppurativa

- Retrospective study of 67 female patients
- Average 75 mg of spironolactone daily over 7.1-month
- Significant improvement in
  - pain [P = .01]
  - inflammatory lesions [P = .02]
  - HS-PGA score [P < .001]

Hidradenitis suppurativa

- No difference between <75 mg and >100 mg
- Lower doses appear to be effective and may be an appropriate option for patients with tolerability concerns
### Hidradenitis suppurativa

- 20 patients with moderate HS, 3:1 ratio blinded treatment with apremilast 30 mg twice daily or placebo for 16 weeks
- Clinical response 8 of 15 patients in the apremilast group (53.3%) and none of 5 patients in the placebo group (0%) (P = .055) at week 16.


### Apremilast-treated patients

- Significantly lower abscess and nodule count (mean difference, e2.6; 95% confidence interval, e6.0 to e0.9; P = .011)
- NRS for pain (mean difference, e2.7; 95% e4.5 to e0.9; P = .009)
- Itch (mean difference, e2.8; 95% confidence interval, e5.0 to e0.6; P = .015)

### Dupilumab

- Recalcitrant facial dermatitis during the dupilumab treatment
- **ACD:** Most frequent clinically relevant allergens
  - cocamidopropyl betaine (CAPB) (40%)
  - nickel (33%)
  - oleamidopropyl dimethylamine (27%)
  - Myroxylon pereirae (20%)
  - fragrance mix 1 (20%)

### Dupilumab

- Infamed atopic skin is predisposed to the development of TH2-mediated contact sensitization to weaker potency allergens, such as fragrances, emulsifiers, and surfactants
- Prevalent nickel sensitization was expected given the hapten’s ubiquity and atopic hand dermatitis association

**Brian C. Machler, Calvin T. Sung, Evan Darwin, Sharon E. Jacob.** J Am Acad Dermatol 2019;80:280-281

### Dupilumab

- Atopic patients may also have allergic contact dermatitis
- Untapped potential of IL-4 inhibitors in the treatment of recalcitrant and systematized ACD to certain allergens

**Brian C. Machler, Calvin T. Sung, Evan Darwin, Sharon E. Jacob.** J Am Acad Dermatol 2019;80:280-281
Methotrexate in alopecia areata

- A systematic review and meta-analysis performed according to recommended PRISMA (Preferred Reporting Items for Systematic Reviews and Meta-Analyses) guidelines.
- Reasonable efficacy
- Adults appear to be more responsive
- Combination treatment better

Best evidence for adults
Combination (methotrexate plus corticosteroids)


Paraneoplastic granuloma annulare

- Solid organ malignancies -- lung cancer (4/7)
- More often generalized disease, refractory to treatment, and perivascular inflammatory infiltrate
- HbA1c and age-appropriate screening


Vibration to reduce injection pain

- Use of a vibrating kinetic anaesthesia device reduces the pain of lidocaine injections
- Buffering and use of warm lidocaine.

William C. Xu, Jaimie C., Chiesa-Fusenich, Thurai Shin, Jeremy Edelson, Nicole Howe, Christopher J. Miller, Joseph P. Sobanko

https://doi.org/10.1016/j.jaad.2018.08.011

Psoriasis

- Psoriatic arthritis
  - TNF
  - IL-17
  - IL-12/23 (Ustekinumab) - less reliable


Psoriatic arthritis
  - Tofacitinib
  - Methotrexate (radiologic progression)
  - Cyclosporine (radiologic progression, modest peripheral, not axial)
  - Acitretin (modest at best)
  - Apremilast
  - Abatacept

Psoriasis

- Inflammatory bowel disease
  - Infliximab, adalimumab, certolizumab, and ustekinumab approved for the treatment of patients with CD.
  - Golimumab is approved for UC but not for CD.

- Methotrexate
- Cyclosporine
- IL-23 inhibitor use in CD has promising results.
- Etanercept is not as effective as other TNF-a inhibitors for CD.
- A direct causal relationship between IL-17 inhibitors or retinoids and CD has not been established.


Psoriasis

- Malignancy
  - Acitretin has preventative effects on NMSCs and is preferred.
  - Bev is avoid anti-TNF agents.
  - Ustekinumab with caution - carcinogenic potential in animal models.
  - Data are limited for apremilast and IL-23.
  - Avoid methotrexate and cyclosporine.


Psoriasis

- Obesity
  - Infliximab and ustekinumab dosed based on weight.
  - IL-17 inhibitors better clearance rates in nonobese patients.
  - Apremilast can cause weight loss.
  - Methotrexate carries a higher risk of fatty liver and hepatic fibrosis in obese patients.


Psoriasis

- Demyelinating disease
  - TNF-a inhibitors contraindicated.
  - Ustekinumab can be used – neutral.
  - IL-17 inhibitors - some benefit in MS symptoms.
  - Data are limited for apremilast and IL-23 inhibitors.


Psoriasis

- Lupus
  - Ustekinumab – safe.
  - Methotrexate and acitretin - good options.
  - Anti-TNF agents - watch for lupus induction and flare.
  - Data are limited for apremilast, IL-17, and IL-23 inhibitors.

Psoriasis

- Pregnancy
- Certolizumab - minimal transplacental transfer
- Enbrelcept - placental transfer less than infliximab and adalimumab
- Ustekinumab and secukinumab both category B
- minimal data available on ixekizumab, brodalumab, apremilast, and IL-23 inhibitors
- Methotrexate and acitretin absolutely contraindicated


Dermoscopy


Melanoma Guidelines

FROM THE ACADEMY
Guidelines of care for the management of primary cutaneous melanoma

https://doi.org/10.1016/j.jaad.2018.08.05

Immunization on tofacitinib

- Winthrop KL. T-cell-mediated immune response to pneumococcal conjugate vaccine and tetanus toxoid during tofacitinib treatment. JAAD 2018; 78: 1149-
- Most patients mount a response

Nonbullous pemphigoid: A systematic review

- Erythematous, urticarial plaques (32.3%) and papules/nodules (20.5%)
- The mean age at presentation was 74.9 years.

CARD14-associated papulosquamous eruption: A spectrum including features of psoriasis and pityriasis rubra pilaris

- Early age of onset:
- Prominent involvement of the cheeks, chin, and ears;
- Family history of psoriasis or PRP;
- Minimal response to conventional topical and systemic psoriasis therapies;
- Improvement with ustekinumab.
CARD14-associated papulosquamous eruption: A spectrum including features of psoriasis and pityriasis rubra pilaris

Pathologist characteristics associated with accuracy and reproducibility of melanocytic skin lesion interpretation

- Rates of diagnostic reproducibility and accuracy were highest among pathologists with
  - Board certification in dermatopathology
  - 5 or more years of experience.

Changing antimalarial agents after inefficacy or intolerance in patients with cutaneous lupus erythematosus

- Of the patients changed because of inefficacy, 56% were responders at month 3; however, the response decreased over time
- For patients switched because of adverse events, the second AM agent was well tolerated in 69% of cases.

Subclinical sensitization with diphenylcyclopropenone is sufficient for the treatment of alopecia areata

- 46 (28.9%) showed a complete response and 59 (37.1%) showed a partial response.

Dose escalation of doxepin for intractable pruritus

- JAAD ONLINE: THERAPEUTIC PEARL
- Optimal range in the plasma concentration 150-250 g/L, with risk for toxicity at 500 g/L
- Intractable pruritus where doxepin was considered a treatment failure,
- Dose can be titrated up to 300 mg/day, by using trough plasma levels to give doxepin a fair clinical trial

Clinical and histologic features of Mycoplasma pneumoniae related erythema multiforme

- M pneumoniae EM has a distinctive presentation with diffuse atypical target and severe and extensive mucositis.
- Histology is similar to toxic epidermal necrolysis.
Anti-MDA5 dermatomyositis
- Mucocutaneous ulceration, palmar papules, nonscarring alopecia, panniculitis, arthritis, and interstitial lung disease

Sweet syndrome in patients with and without malignancy
- Leukopenia, anemia, thrombocytopenia
- Absence of arthralgia
- Histiocytoid or subcutaneous histopathology were associated with malignancy

Pityrosporum folliculitis
- Pruritic, fine monomorphic papules and pustules on the face and back in patients previously treated with antibiotics suggest a diagnosis of Pityrosporum folliculitis.

Topical nitrates in the treatment of primary and secondary Raynaud’s phenomenon
- Meta-analysis shows that topical nitrates are effective in treating both primary and secondary RP

SPF 1001 sunscreen is more protective against sunburn than SPF 501 in actual use
- Natural sunlight, randomized, double-blind evaluation, SPF 1001 sunscreen was significantly more protective against sunburn than SPF 501 sunscreen.

Efficacy and tolerance profile of thalidomide in cutaneous lupus erythematosus
- Pooled rate of response was 90%, with similar response rates between severe cutaneous lupus erythematosus subtypes.
- Pooled rate of thalidomide withdrawal related to adverse events was 24%
Relative efficacy of systemic treatments for atopic dermatitis

- Strongest evidence currently exists for dupilumab and cyclosporine
- Clinical trials for lebrikizumab and tralokinumab

Seger, et al. JAAD 2019; Volume 80, Issue 2, Pages 411–416.e4

A systematic review of evidence-based treatments for prurigo nodularis

- Topical agents
  - corticosteroids, calcineurin inhibitors, calcipotriol, and capsaicin
- Photo- and photochemotherapy

Qureshi et al. JAAD 2019; Volume 80, Issue 3, Pages 756–764

A systematic review of evidence-based treatments for prurigo nodularis

- Thalidomide
  - 6 reports, only 2 of which were rated level 2b or greater
- Cyclosporine and methotrexate
  - 4 combined studies, albeit with level 4 evidence.
- Pregabalin, amitriptyline, paroxetine, fluvoxamine, and neurokinin-1 receptor antagonists
  - 5 level 2b studies

Qureshi et al. JAAD 2019; Volume 80, Issue 3, Pages 756–764

Tick bites and red meat allergy

- Hideo et al. Repeated Amblyomma testudinarium tick bites are associated with increased galactose-alpha-1,3-galactose carbohydrate IgE levels. JAAD 2018; 78:1135-41
  - Basophils serve as antigen presenting cells
  - Allergy to beef, pork, and lamb

Rethinking Biotin supplements

- Lipner, Shari R. Rethinking biotic therapy for hair, nail and skin disorders. JAAD 2018; 78:1236-8
  - Biotin: 30 microg/d from egg yolk, milk, nuts, grains, intestinal bacteria
  - Biotin deficiency: neuromuscular dysfunction, alopecia, dermatitis
  - Supplementation needed in holocarboxylase synthetase and biotinidase deficiency

Do we need extra?

- Average Western diet: 35 – 70 microg/d
- 5 mg/d may improve atopic dermatitis
- 2.5 - 5 mg/d may improve brittle nails, trachonychia uncombable hair
Downside

- Falsely elevated or depressed lab tests
- Troponin
Cutaneous Infections and Infestations

Dirk Elston MD
Professor and Chairman
Department of Dermatology and Dermatologic Surgery
Medical University of South Carolina

No relevant financial relationships to disclose

60 year old female
• Found an 18 year old bottle of eye drops in her medicine cabinet

60 year old female
• Found an 18 year old bottle of eye drops in her medicine cabinet
• Decided to use them

Necrotic vessel
Blue haze
No inflammation
Pseudomonas Sepsis

- Pseudomonas endophthalmitis
- Unresponsive to systemic antibiotics
- Globe removed
- Survived sepsis after surgery and aminoglycoside therapy

34 year old female

- Unresponsive tinea pedis
Secondary Syphilis with Prozone Phenomenon

RPR negative
1:64 dilution positive

Syphilis

Vacuolar interface dermatitis

Slender necrobiosis

Vessels lack apparent lumen

• 50 y.o. ice cream man
• Motor vehicle accident
• New fungal lesion
Zygomycete Infection

- Hyperglycemia predisposes to invasion
- Rapid vasculotropic invasion
- Surgical debridement
- Posaconazole

New Antifungals

- Caspofungin (echinocandin)
  - Candida and Aspergillus
- Voriconazole (triazole)
  - Candida, Aspergillus, Scedosporium and Fusarium
- Posaconazole
  - Zygomycetes
Stains poorly with fungal stains
Often stains well with tissue Gram stain

Child with pruritus
Courtesy of Quenby Erickson, MD

Antilycma americam

Ophthalmic eye
long mouthparts

D. Boon
Lone star tick
(*Amblyomma americanum*)

- Range expanding
- Larvae can attach by the thousands
- RMSF
- Tularemia
- Human monocytic ehrlichiosis (HME)
- *Ehrlichia ewingii* ehrlichiosis
- Southern tick-associated rash illness (STARI)

60 year old grandfather with pruritus

Membranous staining
Scabies

- CD30+ cells may be numerous
- Langerhans cells may be numerous (may mimic Langerhans cell histiocytosis)
- Bulla with eosinophils
  - IgG and C3 may be present at BMZ (may mimic pemphigoid)
2 year old Being Treated for Langerhans Cell Histiocytosis
Scabies Mimicking LCH


- 22 week gestation infant with bedsores
- Ventilator dependent
- Sores noticed on back / buttocks
Morphologic Diagnosis

- Thick walled, irregular, septate hyphae with prominent bubbly cytoplasm
  - Phaeohyphomycosis:
    - Surgery/antifungal

Black Molds

Aspergillosis
A Word of Caution

- Many fungi contain melanin
- Fontana positivity must be interpreted in the context of the fungal morphology

7 year of female

- 7 cm mass on scalp with draining sinus tracts
Microsporum canis

- In foster care
- Adopted a stray kitten

100 year old man

- Lesion on the arm
- Case donated by Antoinette Hood, MD

Reticular degeneration

Inclusion bodies
• Comatose, febrile patient
• Recent cardiac catheterization
• New murmur

• Echocardiogram and repeated blood cultures negative
Janeway lesion

• Osler’s nodes:
  – Tender pink papules
  – SBE
• Janeway:
  – Non-tender, angular infarct
  – ABE

• Transesophageal echo:
  – Vegetation
Dermatobia hominis (Warble Fly)
5 year old female

- Multiple subcutaneous nodules
**Histoplasma capsulatum**

Macroconidia are echinulate (tuberculate)

---

**African Histoplasmosis**

- Key features
  - Numerous large intracellular organisms in histiocytes and multinucleated giant cells
  - Evenly spaced distribution of organisms in giant cells
Hansen's disease:

Histiocytic infiltrate

Globi

Sausage-shaped granulomas following neurovascular bundle

Lymphoid infiltrate

Granuloma

Neutrophilic infiltrate

Onion skin fibrosis surrounding nerve
Do you know JAK?: New Applications for Janus Kinase Inhibitors in Dermatology

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2019 AOCD Fall Current Concepts in Dermatology

Dr. Bhatia’s Disclosures:
- Affiliations with Aclaris, Almirall, Biofrontera, BiopharmX, Dermira, Encore, EPI Health, Ferris, ISDIN, LaRoche, Leo, Mayne, Menlo, Novartis, Ortho-Derm, Pierre-Fabre, Pfizer, Regeneron/Sanofi, and Sun Pharma
- Some slides from industry were borrowed for explanation of data and scientific background, not for promotion; Off-label discussion is likely
- Copies of pdf or questions: bhatiaharbor@gmail.com

Important References:
Acknowledgments: Jim Del Rosso, D.O.
Mark Lebwohl, M.D.
Brett King M.D. PhD

So exactly is this JAK guy? Can they possibly be inhibited?

Janus Kinase...Signal Transducer and Activator of Transcription (JAK-STAT)

- Pathway utilized to transmit extracellular signals to the nucleus.
- Dysregulation of this pathway is responsible for immune deficiency syndromes and myeloproliferative neoplasms.
- Phosphorylation after ligand binding leads to translocation and gene expression regulation.
- JAK/STAT pathway suppresses:
  - dendritic cell activation, T-cell mediated inflammation

Starting Lineup:
- JAK 1
- JAK 2
- JAK 3
- TYK 2
- STAT 1
- STAT 2
- STAT 3
- STAT 4
- STAT 5a
- STAT 5b
- STAT 6

4 JAK (1,2,3, TYK 2) and 6 STAT (1-6) interact to send cytokine signals to nucleus

<table>
<thead>
<tr>
<th>Cytokine/Interactor</th>
<th>JAK</th>
<th>STAT</th>
</tr>
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<tbody>
<tr>
<td>IFNα</td>
<td>JAK3/2</td>
<td>STAT1</td>
</tr>
<tr>
<td>IFNγ</td>
<td>JAK3/2</td>
<td>STAT1</td>
</tr>
<tr>
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<td>JAK3/2</td>
<td>STAT1/5</td>
</tr>
<tr>
<td>IL-23</td>
<td>JAK2/3</td>
<td>STAT1/5</td>
</tr>
</tbody>
</table>

IL-12, TNF, and IL-17 do not signal through the JAK/STAT pathway.
How do these things work?
Janus Kinase Inhibitors

1. Cytokine binding to its cell surface-receptor leads to receptor polymerization.

2. JAKs are activated by the polymerized receptor.

3. JAKs (Janus Kinase) phosphorylate (add a phosphate group to) signal transducers and activators of transcription (STATs).

4. The phosphorylatedSTATs are then recruited to the surface of the cell and interact with the DNA to activate gene transcription.

Damsky W, and King, B. “The Promise of a New Drug Class,” J Amer Acad Dermatol, 2017; 76; 736-44
Are these safe?
- Perform all immunizations before starting
- Rare elevations of lipids (total, HDL, LDL)
- No reported strokes or worse
- Pancypopenia worse
- Ruxolitinib > tofacitinib
- JAK2 signaling mediated by erythropoietin, thrombopoietin, and GCSF
- Low CA risk across specialties
- Monitoring—TB test to start and annually
- Lipids and LFTs—4-8 wks after start
- CBC/diff at start and 4-8 wks then q 3 mo

Lipid Levels in Patients with PsA reported elevations with Tofacitinib
- n=783 pts, pooled data from two phase 3 studies and an ongoing long-term extension study
- Assessed fasting lipid levels, blood pressure, and MACE
- Percentage increases of LDL and HDL ranged from 9% to 14% for 5 mg and 10 mg doses of tofacitinib at 3 and 6 mo
- No meaningful changes in LDL/HDL or total cholesterol/HDL ratios. Blood pressure remained stable through 24 months
- Conclusions: Serum lipid level increases at month 3 following tofacitinib treatment in PsA were consistent with observations in rheumatoid arthritis and psoriasis

Risks more concerning in Transplant Patients?
- Concern with JAK inhibitors is a theoretical increased risk for malignancy—dampen antitumor surveillance?
  - Initial studies of tofacitinib in renal transplantation:
    - 1% of patients treated with tofacitinib developed post-transplant lymphoproliferative disease
    - Myelofibrosis and polycythemia vera treated with ruxolitinib
    - No increased risk for developing a second malignancy has been shown
  - Topical JAK inhibitors
    - Monotherapy in less severe disease (Vitiligo, AD, patchy AA)
    - Potential maintenance therapy after oral treatment
    - Safer options for children (Vitiligo, AD, AA) would prefer to a topical if viable and avoid systemic side effects.

JAK Inhibitors for Psoriasis
- JAK 1, 2, 3 and TYK 1
  - Ruxolitinib
    - JAK 1, 2
    - Phosphorylation due to IL-6, IL-12, or IL-23, resulting in suppression of Th17 differentiation
    - Reduced lymphocytic infiltration
    - Inhibited angiogenesis
    - Reduced IFN expression
  - Tofacitinib
    - JAK 1,3
    - Suppression of IL-23 receptors, IL-15, IL-17A, 17F in mouse models
    - IL-22 suppressed when T cells were stimulated with IL-6 and IL-23
    - Growth and activation signals sent to nucleus
Ruxolitinib for Psoriasis
Topical JAK1/2 inhibitor against IL-12/23, IFN
- Two 28-day studies: BSA 2-7%, 8-13%, and 14-20%
- Double-blind, vehicle controlled
  - 1% cream QD: 53% plaque reduction vs. 32% vehicle
  - 54% for 1.5% cream BID vs 27% for vehicle
  - 46% for 1.5% cream BID vs 40% for calcipotriene
- Open-label both for safety/tolerability/efficacy
- Mean total area 59% for treated lesions, 3% untreated
- Efficacy was seen as early as 1 week
- Plasma concentrations did not correlate to the BSA treated

BMS-986165
Oral selective tyrosine kinase 2 (TYK2) inhibitor
- Highly selective for IL-23, IL-12, and interferon alpha.
- 12-week, double-blind, dose-ranging placebo-controlled
  - PASI 75 rates: placebo 7%, 3 mg qod 9%, 3 mg daily 39%, 3 mg BID 69%, 6 mg BID 67%, 12 mg/day 75%
- PASI 75 response and other clinical benefits were retained one month after last dose
- Biopsies obtained on study days 1, 15, and 85: 3 mg bid or higher:
  - reduced expression of IL-19 and IL-36A, markedly decreased expression of genes in the Th17 pathway and essentially normalized expression of the proinflammatory genes beta defensin and S100A9.

JAK Inhibitors for Atopic Dermatitis
- Baricitinib: JAK 1-2 inhibitor: pro-inflammatory cytokine signaling, ph 3
- ASN002: JAK/SYK inhibitor: Reduce Th2/Th22 cytokine signaling, ph 3
- Upadacitinib: JAK-1 inhibitor: ph 3
- Abrocitinib: JAK-1 inhibitor: ph 3, ages > 12
- JTE-025 JAK-1 inhibitor: phase 2 studies in Japan
- Sienna SNA-125: JAK 3 inhibitor proof of concept 2018
- Aclaris 502-AD-201 topical: completing ph 3

Baricitinib Phase 2 Trial
- Oral JAK1-selective inhibitor
- 59x more selective JAK1> JAK2; 133X for JAK1> JAK3; 194 X for JAK1> TYK2
- No herpes zoster, malignancies, deaths or cases of thromboembolic dz
- Phase 2b dose-ranging study: upadacitinib 7.5 mg, 15 mg or 30 mg improvements overall
- Phase 2b study 32-week efficacy/safety patient-reported outcomes data evaluating upadacitinib once-daily
- Improved patient-reported itch and impact on sleep receiving upadacitinib 30 mg/daily vs placebo at week 16
- Phase 3 efficacy and safety n=2430 patients with moderate-to-severe AD (> 12 years old and ≤ 75 years old) + topical TCS
**ASN002 oral JAK and SYK inhibitor**

- n=36 moderate to severe AD
- random 20 mg, 40 mg, 80 mg or placebo qd x 4 weeks.
- EASI score ≥16, BSA ≥10%, and IGA of ≥3 at baseline.
- Dose-related declines in EASI75 (10%-55%) at 4 weeks
  - average decrease in EASI of 28%-68%
  - 19%-51% reduction in Itch Numeric Rating Scale score
  - Most common AEs being mild headache and nausea, most of which were transient and occurred on day 1 of dosing.

**Siphylotocous Dysbiosis Correlates of Success of Treatment of Atopic Dermatitis with the JAK/SYK Inhibitor ASN002**

- Skin microbiome analyzed for effect on *S. aureus* dysbiosis and correlates of success of treatment
- Double-blind randomized phase-1b study
- ASN002 for 28 days in with doses: 20, 40 and 80mg daily and placebo (N=9 per arm). Skin microbiome from lesional (LS) and non-lesional (NL) skin swabs at days 1, 29 and 43 were sequenced (16S V1-3).

**REFERENCES**

Avidan U. Neumann, PhD. AAD 2018 Late Breaking Abstract presentation

A Phase Ib, Randomized, Double-Blind, Placebo-Controlled, Sequential, Multiple-Dose Escalation, Study to Evaluate the Safety, Tolerability, Pharmacokinetics and Preliminary Efficacy of ASN002 in Subjects with Moderate-to-Severe Atopic Dermatitis.

**Higher S. aureus frequency associated with higher EASI score in lesional skin**

- Lower *S. aureus* frequency at baseline predicts (p=0.03) higher probability for a sustained EASI response 14 days after end-of-treatment, and in consequence significantly (p=0.001) predicts lower EASI at day 43.
- A significant (p<0.005) dose-dependent decline in *S. aureus* frequency in LS was exhibited at day 29 in 86% of patients, in comparison to placebo (33%), and correlated with EASI decline (R=0.7, p=0.003).

**Conclusions:**
- JAK/SYK inhibition with ASN002 not only improves clinical scores and Th2/Th22 inflammation markers but also reduces *S. aureus* frequency in lesion.
- Conversely, lower baseline *S. aureus* frequency in lesion correlated with ASN002 sustained efficacy post-treatment.

**Abrocitinib**

- Abrocitinib received Breakthrough Therapy designation from the FDA for moderate to severe AD in February 2018
- Phase 1 study: 79 healthy subjects single dose of placebo or 3, 10, 30, 100, 200, 400 or 800 mg (single ascending dose phase)
- BID for 10 consecutive days (multiple ascending dose phase)
- No deaths or serious AEs: headache, diarrhea, and nausea (n = 11)
- mean t½ 2.8-5.2 h after 10 days of QD or BID administration in the multiple ascending dose phase
- Less than 4.4% of the dose was recovered unchanged in urine.

**JTE-052 Pan JAK Inhibitor**

- Phase 2 study, n=327
- Well-tolerated with improvements in one day
  - 0.026% less effective

**Vitiligo**

- CD8+ T cells drive melanocyte destruction via IFN-γ signaling utilizes the JAK-STAT pathway
- Activated CD8+ T cells produce IFN-γ and other inflammatory mediators to target melanocytes
- JAK inhibition allows melanocyte regeneration by blocking IFN-γ-mediated inflammation

**REFERENCES**


Vitiligo

- Ruxolitinib: 1.5% cream applied bid, n=12
  - 4/11 (72.7%) patients showed >50% repigmentation
  - 7/12 patients showed facial repigmentation
  - With NB-UVB, further repigmentation occurred

- Oral Ruxolitinib 20 mg bid for over 20 weeks
  - Both vitiligo and AA responded
  - Significantly improved facial repigmentation

- ATI-502 (topical): open label phase 2 study
  - Anticipated release of data early 2019.

Janus Kinase Inhibitors for Vitiligo

- Tofacitinib:
  - 53-year-old pt with vitiligo covering her face, hands and body
  - 5 mg every other day, increased to 5 mg daily after 3 weeks.
  - 2 months: partial repigmentation
  - 5 months: "white patches nearly all gone"
  - Real world costs: $12,000/year

JAK2 inhibition is not required for efficacy in Alopecia Universalis

- IL-6 activates Janus kinase (JAK) which can reverse the IL-6-induced, STAT3-dependent proinflammatory effects on TGF-β1 and collagen I expression
- Autoreactive CD4+ T cells: JAK-STAT dependent cytokines, including IFN-γ
- JAK inhibitors induce restoration by:
  - Anti-inflammatory effects
  - Anagen initiation
  - Promotion of the activation of hair follicle stem cells
- Phase 2 trials with both an oral JAK1/3 inhibitor (ATI-502) and a topical JAK inhibitor (Ruxolitinib) were performed

Tofacitinib and Alopecia Universalis

- IL-6 induces STAT3-dependent proinflammatory effects
- JAK-STAT1/3 inhibition: STAT3 inhibition
- Phase 2 trials with both an oral JAK1/3 inhibitor (ATI-502) and a topical JAK inhibitor (Ruxolitinib)

JAK Inhibitors and Alopecia

- Open label phase 2 study n=66
  - Tofacitinib 5 mg bid
  - 32% of patients with 50% improvement
  - Relapse occurred after a median of 8.5 weeks after cessation
- N=90 AA patients treated with tofacitinib 5 mg bid reported 50% improvement in 42% of patients.
  - 9 of 13 adolescents treated showed significant hair regrowth.
  - AA>>universalis (81.9% vs 59.0%)

JAK Inhibitors for Granuloma Annulare

- Why?
  - Control against Th-1 inflammation cascade
  - TNF-α expression and release
  - Potential use for disseminated variants
- Why not?
  - Toxicity potential if above 5 mg
  - Long-term issues of compliance when unsure of endpoint
  - Costs
Where could JAK Inhibitors work in Derm?

- Tofacitinib: FDA approved for RA and PsA
- Ruxolitinib: Myelofibrosis and Polycythemia Vera
  - Oclacitinib: approved for atopic dermatitis...in dogs
- In trials:
  - Psoriasis
  - Atopic Dermatitis
  - Vitiligo
  - Alopecia Areata
- Anecdotal:
  - CTCL
  - Mastocytosis
  - Granulomatous disorders
- Autoimmune disorders
  - Lupus/Dermatomyositis
- Where we haven’t had any luck:
  - Lichen Planus
  - GVHD
  - Erythema Multiforme
- Instead of Steroids? Biologics?

Don’t Be Afraid...

Fight!

Thank You
Patients vs. Doctors: One-Star Reviews, Yelp, and Social Media
Neal Bhatia, M.D.
Director of Clinical Dermatology
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Customers or Patients?
- Customers choose to do business with a certain establishment
  - As a result, the establishment’s revenue increases.
  - Customer service is a business idea developed to attract and retain profits.
- Physician-Patient relationship is profit driven
- Patients needing health care may positively, neutrally or even negatively affect revenue.
  - Patients may carry good, poor or no insurance.
  - Institutions are legally obligated to care.

Physicians’ obligations for their patients do not translate to the language of customer service.

Where does patient care end and customer service begin?
- Customers or Patients?
  - There is a fine line between customer service and people taking advantage of you.
  - "Remember that you run your business, you make the rules, you follow your contract obligations, and you should never have to lose money on any claim because a patient tells you to…"

88% of consumers trust online reviews as much as a personal recommendation
- Countless other studies have proved the same thing over and over again – “consumers” trust online reviews and those reviews heavily influence their “purchasing” decisions.
- 72% of consumers, will make purchasing decisions ONLY after they’ve read a positive review. If you can have both a high volume and a good score, you become the defacto choice for many consumers

Freedom of Speech now means you can say anything you want
- Can any physician advertise any skill?
- Can the term “dermatology” or “skin care specialist” be applied when the services are not from a dermatologist?
- Who is a “cosmetic specialist?” “Cosmetic Surgeon?”
- And how can any organization stop the proliferation of the “fake derms?”

DISCLOSURES
Neal Bhatia MD
None; Copies of pdf or questions: bhatiaharbor@gmail.com
Customer-generated negative online reviews on hospitality employees and businesses

- Multiple research questions demonstrate how negative online reviews can have adverse and diverse effects on restaurant industry employees and businesses.
- Practical implications - Four types of countermeasures are presented: preventative, protective, positive and palliative.
- Social implications - Negative online reviews can exact a hefty toll:
  + Reduced customer patronage → company profitability
  + Human consequences → adverse stress reactions, loss of face, damaged personal and professional relationships


https://www.physicianspractice.com/patient-relations/5-smart-ways-handle-negative-online-reviews
What prevention strategies should we consider?

- Be your patient’s advocate
- Share frustrations about current state of healthcare
- Walk out for a break if discussions get adversarial
- Accept that we can’t be everything to every patient
- Don’t go to the next patient after a bad encounter, find a minute to take a breath no matter how behind you are

Reputation protection services
- Attempting to be your online bodyguard
- Expensive and ongoing

Search Engine Optimization
- This is what every practice needs and not all think about

Call patients routinely about their progress
- Medical, surgical, aesthetic

How to Navigate the Review Storm

- Set systems in place that automatically monitor relevant review sites and alert you when new reviews are out
- Thank positive reviewers and reinforce some key points when appropriate.
- Negative reviews should also be acknowledged. You should try to thank the reviewer for the feedback when constructive, and try to reach out to him/her to find a way to mitigate this negative experience whenever possible.
- Some reviewers will delete their negative reviews once they realize you have been sincere and tried to find a positive resolution.

Social Media Extortion

- Do we now have to hang ourselves out and submit to predators that want free or marked down service?
- Why is medicine now a commodity like coffee and plumbing?
- What protection does a small practice have?
- What protection does a small practice have?
- Why do we let these petty antics ruin our day?
- Or do they slowly chip away at our drive and focus?
Why do we let patient reviews and bad behavior ruin our day?

1. Annoying remarks
   - Negative reviews can affect your personal frame of mind.
   - They can be retaliatory.

2. One Star Reviews: Weapon of retaliation?
   - 91% of consumers read online reviews before a purchase.
   - According to a survey performed by Softwareadvice.com in 2013 and 2014, the percentage of patients using online reviews to find their physician increased from 25% to 42%.
   - 1 star review from misunderstanding of healthcare system?

Prior Auths, processing referrals, additional path stains all take time, regardless of efforts to ensure good patient experience


<table>
<thead>
<tr>
<th>Characteristics of 152 Five-Star Reviews</th>
<th>Characteristics for 112 One-Star Reviews</th>
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<tbody>
<tr>
<td>Characteristic</td>
<td>No. (%)</td>
</tr>
<tr>
<td>Bedside manner</td>
<td>40 (26.3)</td>
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<tr>
<td>Knowledge</td>
<td>33 (21.7)</td>
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<tr>
<td>Results</td>
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<tr>
<td>Honest/pressure</td>
<td>17 (11.2)</td>
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<td>Office staff</td>
<td>17 (11.2)</td>
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<tr>
<td>Wait times/scheduling</td>
<td>12 (7.8)</td>
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<tr>
<td>Cost</td>
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<td>Consultation fee</td>
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<td>Characteristic</td>
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<td>25 (22.3)</td>
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<tr>
<td>Consultation fee</td>
<td>3 (2.7)</td>
</tr>
</tbody>
</table>


How to Not Handle Bad Reviews

- Don’t get the mob and riot
- Don’t take them on alone
- Don’t burn the house down

If you discover a dreaded 1-star review, don’t light your torches and form a mob. Instead, take a breath, calm down, and determine the authenticity of the review.

Put out the fire... but does this solve burnout?

- Contact the reviewer directly
  - If you can get people on the phone, they’ll see you as a human and not a faceless business.
- Take the proper tone
  - The right tone can turn a complaint into a positive review.
  - Don’t make excuses. Don’t get defensive.
- Never threaten a lawsuit
  - Technically, you can sue someone for online defamation
  - Lawsuits draw negative attention, and the media backlash from suing a consumer will scare off other clientele.
The "real" fake review problem

- As of 2017: estimated 267 million Facebook users were fake or duplicates
- unclear how many were involved in fake review schemes.
- doesn't include fake reviews from real accounts
- Fake reviews on Google is a "massive problem"

Spot the Fakes:

- Tricks to spot a fake Google review

  - Compare suspicious complaints with your sales records to find any matching transactions
  - The buyer isn’t in your point of sale software system
  - The transaction date doesn’t match their complaint
  - Lack of detail (e.g., they haven’t named any specific employees)
  - There is a surge of bad reviews in a short period of time
  - There’s a connection between the reviewer and a competitor
  - The reviewer recommended a competitor in their complaint

Imposters Everywhere

Real Story of Sabotage: Cameron Woodsum

- Woodsum is an e-commerce entrepreneur who owns a bill reduction company called Bill Slasher. They renegotiate customers’ phone and cable bills to save them money.
- In early 2018, Woodsum launched a Google Adwords campaign targeting the leading competitor in the space, Billshark. When people searched for “Billshark,” they found Bill Slasher’s advert at the top of search results.
- Billshark wasn’t happy. Within 12 hours of launching that advert, Woodsum says the Bill Slasher Facebook page was flooded with 1-star reviews.
- Upon closer inspection, he noticed that they came from Billshark’s executive team, employees, and family members. Even the CEO’s wife was involved.
- As Woodsum watched his company’s review average plummet from a 4.9 to a 2.5, he realized he had a full-fledged crisis on his hands.
Improve chances of removing fake reviews

- Woodsum eventually followed this advice. He turned Bill Slasher’s Facebook reviews back on and responded to the competitors’ reviews openly, calling them out for what they did. He also encouraged his existing base of happy customers to leave positive reviews.
- The loyal support of Bill Slasher’s happy customers did get the company’s review average back above a 4.0.
- Make sure you have access to all of your social media and review site profiles and listings; Claim duplicate pages
- “If a fake reviews crisis happens before these are done, there will be more work to do before getting the situation resolved.”

What is burning us? Or burning us out?

- Small practices don’t have IT departments, lawyers on retainer, or systems in place for constant trolling
- There’s a difference between working the system (i.e. asking patients to review you) and gaming the system (posting fake reviews yourself, etc)
- Failing to work the system likely means that you’re going to get your clock cleaned by inferior practices that play the game better.
- It’s bad enough that the best and worst doctor in town get paid the same for the same work.
Commonly Encountered Lesions of the Oral Mucosa

**Goals:**
1. Review a sample of commonly encountered oral lesions & discuss the clinical characteristics, etiology, risk factors & recommended management
2. Discuss indications for biopsy and biopsy technique
3. Explain biopsy workflow—what happens to your specimen—and dental codes/billing

Oral Candidiasis

- **Etiology:** C. albicans
- Opportunistic infection
• Sloughing from superficial chemical burn – Colgate total is a common culprit
• Pt complains of pulling “strings” out from mouth in am

Oral Candidiasis

• Diagnostic Key
  ▫ Plaques are removable/wipe-able
  ● Exception being: erythematous & hyperplastic variants
  □ take smear or culture

Oral Candidiasis

• Treatment:
  ▫ Rx antifungal treatment
    ● Nystatin
    ● Mycelex troches
    ● Diflucan
    ● Mycolog II Cream

Also treat the denture

Herpes Labialis aka “Cold Sore”

• Etiology: Reactivation of HSV
• Stimuli that can reactivate the virus

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<thead>
<tr>
<th>Stimuli</th>
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<tr>
<td>Fever</td>
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<td>High-stress event</td>
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<tr>
<td>Hormonal changes</td>
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<tr>
<td>Fatigue</td>
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<tr>
<td>Upper respiratory infection</td>
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<tr>
<td>Suppressed immune system</td>
</tr>
<tr>
<td>Extreme temperature</td>
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<tr>
<td>Recent dental work or surgery</td>
</tr>
</tbody>
</table>
Herpes Labialis

- The lifetime prevalence in U.S. is estimated at 20-45% of the adult population
  - 1/3 experience recurrent outbreaks

- Prodromal symptoms

- Although the fluid-filled vesicles are most infectious, all stages can be contagious
  
  Must decide whether it is wise to treat a pt with an active infection, the dentist must avoid spreading the virus to other areas of the pt’s mouth and/or face

Herpes Labialis

- Treatment
  - OTC antiviral
    - Abreva cream 10% (docosanol)
  - Rx antiviral cream
    - Zovirax Ointment 5% (acyclovir)
  - Rx systemic antiviral
    - Valtrex (valacyclovir)
  - Laser
Impetigo

- Infection from staph or strep
- Can last for many weeks
- Contagious
- Rx: topical or systemic antibiotics
  - 7-10 day course of an oral antibiotic (e.g., erythromycin, cephalexin)
  - strong topical antibiotic, such as mupirocin (Bactroban)
- Wash (do not scrub) the skin several times a day with antibacterial soap to remove crusts

Aphthous Ulcers/“Canker Sore”

- Occurs most frequently in children & young adults
- Etiology = unknown
  - Reported Triggers/Predisposing Factors
    - Allergies/hypersensitivity
    - Stress
    - Trauma
    - Nutritional deficiency
    - Hormones
    - Immunological factors
    - Hematological abnormalities
    - Genetic predisposition

Major

Herpetiform

Aphthous Stomatitis
**Aphthous Ulcers**

- **Diagnosis:** Clinical

- **Treatment:**
  - No treatment
  - OTC anesthetic
  - Topical corticosteroid
  - Topical Apthasol
  - Cauterization
  - Systemic steroid (for multi-focal outbreak)
  - Laser CO2 or YSGG Er

*Rx: Fluocinonide Gel 0.05%
Disp: 15g
Sig: Apply a thin layer to lesion 4 times per day until resolved*

*Rx/o systemic disorder

**Herpangina**

- Most often caused by coxsackie virus A (aka “herpangina virus”)
  - Can also be caused by coxsackievirus B or echoviruses

- Most cases of herpangina occur in the

- Mostly affects children, occasionally occurs in adolescents and adults

- Self limiting, palliative care

**Deep Fungal Infection**

- Usually in immunocompromised

- Clinical mimics
  - SCC
  - Wegner’s Granulomatosis
  - Oral TB
  - Syphilitic gumma
  - Sarcoidosis

- Biopsy and/or fungal culture required for diagnosis
TUGSE

- Traumatic ulcerative granuloma with stromal eosinophilia
- Unknown etiology
- Most common oral location: tongue
- May resemble a traumatic ulcer clinically

Key histology finding—numerous eosinophils within the CT and extending into muscle

Necrotizing Sialometaplasia

- Often hard palate following trauma
  - Ex) from injection of local anesthesia
  - Local ischemia and infarction of minor salivary glands
  - Early lesions= palatal swelling
  - Late lesions= ulcer or defect in palate
  - “A piece of my palate fell out”
  - Usually occurs over a course of weeks
  - Clinical mimic= SCC
PRIMARY HERPES
Acute Herpetic Gingivostomatitis

- Diagnostic Keys
- Ulcers occur on BOTH keratinized & non keratinized tissue
- Gingiva is puffy, painful, & erythematous
- Pt currently has or recently had fever, sore, throat, malaise
- Pt has never had a “cold sore”

Acute Herpetic Gingivostomatitis

- Diagnosis: Clinical
- Treatment: Self limiting, palliative

Lichen Planus

- Chronic, muco-cutaneous condition thought to be the result of a cell-mediated immune response of unknown origin
- Affects ~2% of the adult population
- Presents intraorally as white striae on an erythematous background
  - &/or white patches, erosions & desquamative gingivitis
- An association w/ Hep C has been reported
  - More recalcitrant to Tx ???
- A slight increase risk for OSCCa has been reported
  - Keep LP patients on follow-up indefinitely
  - *** Document that Pt was informed of risk & need for long-term follow-up

Lichen Planus
Lichen Planus

- Features to Aid in Clinical Diagnosis
  - Lesions should be **multifocal & bilateral**
  - Lesions _wax & wane_
  - _Whickham's striae_
  - Any skin lesions
  - Family Hx
  - Medical Hx

- Other mimickers if erosive LP
  - Erythema multiforme
  - Pemphigoid
  - Pemphigus

- Management
  - Biopsy to establish a diagnosis
  - Treat only if symptomatic
  - Long-term clinical follow-up

- Treatment
  - Topical or systemic steroids
    - 1. Dexamethasone Elixir
    - 2. Fluocinonide Gel 0.05%
    - 3. Clobetasol Ointment 0.05%
    - 4. Prednisone 30mg (1wk), 15g (1wk), 5g (1wk)
  - Secondary fungal infection

Erythema Multiforme

- Spectrum of lesions:
  - EM Minor—EM Major—Stevens Johnson Syndrome—Toxic Epidermal Necrolysis
  - Most often a reaction to an infectious agent (TB, HSV) or an offending drug
  - Clinical buzzwords: “black crusted lips,” “bullseye” or “targetoid” skin lesions

- Management
  - Treatment
  - Secondary fungal infection
Erythema Multiforme

- Autoimmune condition causing vesiculobullous lesions of skin and oral mucosa
- Autoantibodies against BP180, laminin-5 in the basement membrane zone
- Causes subepithelial clefting
- DIF: Linear band of IgG and complement along BMZ

Mucosal Membrane Pemphigoid

- Patients may have ocular lesions—symblepharon
  - Plaques of bulbar conjunctiva
  - If untreated, progression to blindness

MMP

- Autoantibodies against Desmoglein 1 and Desmoglein 3
  - Desmoglein 3= seen in oral cavity
  - Desmoglein 1= seen in upper layers of epidermis (skin)
- Histology: acantholysis with “tombstone” appearing cells
- DIF: “Chickenwire” pattern of IgG and Complement

Pemphigus Vulgaris

- Intraepithelial clefting
- Autoantibodies against Desmoglein 1 and Desmoglein 3
  - Desmoglein 3= seen in oral cavity
  - Desmoglein 1= seen in upper layers of epidermis (skin)
- Histology: acantholysis with “tombstone” appearing cells
- DIF: “Chickenwire” pattern of IgG and Complement
Pemphigus Vulgaris

Desquamative Gingivitis

“Butterfly Rash”

Oral findings: often non-specific
Lupus Chelitis

Skin: scaling, atrophy, pigmentary disturbances

Systemic Manifestations:
- Renal: ESRD
- Cardiac: Libman-Sacks endocarditis

Diagnosis
- DIF: Shaggy or granular band of IgG, IgM, c3 at BMZ
- Positive lupus band test
- Positive ANA
  - Anti-dsDNA
  - Anti-Sm (small nuclear RNA)

Geographic Tongue
**Geographic Tongue**

- Diagnosis: Clinical
- Treatment: No treatment
Hairy/Coated Tongue

- **Diagnosis:** Clinical

- **Treatment:**
  - Gentle tongue brushing or scraping
  - Increase water consumption
  - Avoid certain food/beverages (ie coffee), bismuth containing medications, chlorhexidine, etc.

Leukoplakia

- “A white patch or plaque that cannot be characterized clinically or pathologically as any other disease” WHO
- Strictly a clinical term, does not imply a specific histological diagnosis

Leukoplakia:

- Keratosis
- Benign epithelial hyperplasia +/- hyperkeratosis
- Dysplasia
- SCC
Leukoplakia

- Considered a "pre-malignant" lesion
  - Even though histologic features of dysplasia not always present
    - Dysplasia in 4 -15%
    - Tongue & FOM = high risk

- Management:
  Any leukoplakia that is present for +2 wks and for which a cause can not be identified & removed should be biopsied

Not all leukoplakias are the same!

Leukoedema

Chronic cheek bite

Smokeless tobacco keratosis

Lichen planus

Contact stomatitis - cinnamon

Frictional keratosis

Lichen planus

FOM: Frictional Oral Mucosa

Photos: Dr. C.D. Johnson, University of Texas Dental Branch at Houston

Leukoplakia

Leukoedema

Chronic cheek bite

Smokeless tobacco keratosis

Lichen planus

Contact stomatitis - cinnamon

Frictional keratosis

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Smokeless tobacco keratosis

Photos: Dr. C.D. Johnson, University of Texas Dental Branch at Houston

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Osteonecrosis of the Jaw (ONJ)

- Formerly bisphosphonate-related (BRONJ), now referred to as medication-related (MRONJ)
- Required features for diagnosis:
  - Current or previous treatment with antiresorptive or antiangiogenic agent
  - Exposed bone for longer than 8 weeks
  - No history of radiation therapy or obvious metastatic disease to the jaws

- Most strongly associated with nitrogen containing bisphosphonates (aminobisphosphonates) and denosumab
  - Usually for treatment of multiple myeloma (rare in patients treated for osteoporosis)
- IV administration > oral administration
- Mandible > maxilla
- Often occurs following local trauma (dental extraction)

Treatment

- #1 treatment is prevention!
  - Promote good oral hygiene
  - Conservative treatments when applicable/practical (RCT vs. exo)
- Managing exposed bone
  - Managing patient symptoms
    - Smooth roughened edges
    - Gentle curettage
    - Chlorhexidine rinse
- Timing is key
  - If larger dental procedures indicated, try to schedule for when drug half life is at its lowest
- Role of hyperbaric oxygen therapy
INDICATIONS FOR BIOPSY

- Any lesion that **cannot** be diagnosed clinically, including the following:
  - Lesions with no identifiable etiology that persist for >14 days, despite local therapy
- Any lesion that is felt to have **malignant or premalignant characteristics**, including the following:
  - Any lesion that has grown or is growing rapidly for no obvious reason
  - Any lesion that feels firmly attached or fixed to adjacent structures
  - Any unknown lesion in a **high-risk area**
- Confirmation of clinical diagnostic suspicions

What to biopsy

- **Soft tissue**
  - Any clinically suspicious leukoplakic or erythroplakic lesion
  - Pigmented lesions
  - Non-healing ulcer (present after two weeks)
  - Soft tissue masses
- **Intraosseous**
  - Tissue curetted from extraction sockets/periapical debridement

Pigmented lesions

- Concern is melanoma
- Consider biopsying all pigmented lesions to definitively rule out melanoma
  - Even innocuous appearing pigmented lesions may be melanoma in situ!
  - Exception—racially pigmented gingiva/mucosa need not be biopsied

Soft tissue masses

- Numerous soft tissue/salivary lesions, most common of which clinically encountered include:
  - Fibroma
  - Lipoma
  - Papilloma
  - Mucocoele
  - Pyogenic granuloma/peripheral ossifying fibroma/peripheral giant cell granuloma
  - Denture polyp
**Periapical Pathology**

**Clinical Diagnosis:** Periapical pathology

**Microscopic Diagnosis:**

- 99% PAP (797 out of 805 cases; periapical granuloma, radicular cyst)
- 1% non-PAP (8 out of 805 cases)
  - 2 CGCG
  - 1 nasopalatine duct cyst
  - 1 lateral periodontal cyst
  - 1 benign fibro-osseous lesion
  - 1 Pindborg tumor
  - 1 odontogenic myxoma
  - 1 Multiple myeloma

**Dental Extractions**

- Impacted teeth
  - Should consider submitting curetted tissue for pathologic analysis, especially if there is a large radiographic lucency associated with the tooth
  - Most likely a dentigerous cyst, but need to rule out odontogenic keratocyst, ameloblastoma

**Periapical Pathology—A note on residual cysts**

- Residual cyst = radiolucency that remains in an area after extraction of the infected tooth
- Majority of SCC occurring within cystic jaw lesions are in residual cysts!

**Q1. Does the lesion require a biopsy?**

**Q2. What is your differential diagnosis?**

**Q3. Does the patient have any contraindications for biopsy?**
Q4. Will it be Excisional vs. Incisional

- Differential Dx *
- Size of lesion < or > 5mm
- Awareness of regional anatomy/significant anatomic structures (ie labial artery)
- Awareness of potential esthetic compromise as a result of scarring

Small, pedunculated, exophytic masses (ie papilloma, fibroma) in accessible areas are excellent candidates for excisional biopsy

Incisional Biopsy

- * Site selection — Want to acquire the most representative sample
- The minimal requirements for an adequate specimen vary
  - Want to have at some connective tissue in all specimens, should have bleeding
  - Ulcerated vs. non-ulcerated lesions
  - A small amount of local anesthetic infiltrated at 4 points around the lesion
  - Anesthetic should not be injected directly into lesion

Biopsy Armamentarium for Mucosal Biopsies

- Blade handle with a No. 15 blade, +/- punch
- Tissue forceps with teeth
- Local anesthetic (preferably w/ epi) and syringe
- Silk suture for traction & silk or chromic gut suture for closure, if needed
- Needle holder
- Fine-tipped scissors
- Silver-nitrate sticks
- Gauze sponges
- Ruler or perio-probe for measuring
- Specimen bottle containing formalin and biopsy requisition form
Ulcerated lesions

• When biopsing ulcerated lesions INCLUDE ADJACENT NON-ULCERATED/NORMAL TISSUE

Biopsy paperwork and workflow

Please remember to label formalin bottle with patient's name!

Quick notes on paperwork:
1) Fill out the clinical info to the best of your ability
2) Please provide a radiograph/clinical photograph if available/applicable
3) The actual biopsy procedure is a dental code/billed through dental insurance, but the lab tissue processing is billed through medical insurance—put this one on the form you send to us, but the regular dental codes/fees apply to your office charts/EMR!
Biopsy workflow—what happens to your specimen

- 1) Mailed requisition form and formalin bottle are received by our lab (fedex shipping labels and mailing envelopes are included in the biopsy kits)
- 2) Tissue is processed, embedded, and fixed on a slide (by lab techs)
- 3) Oral pathologist (us) receive the slide and render a diagnosis
- 4) Report with diagnosis is faxed and mailed to your office. Biopsy reports are also available through an online portal

Biopsy workflow

- Normal turnaround time is one day after we receive the specimen (needs to fix overnight)
  - Possible exception—if diagnosis requires additional stains/workup
    - In this case we will contact you and let you know
  - If you have any questions about the diagnosis or how to relay results to the patient, just give us a call!

Biopsy Coding

<table>
<thead>
<tr>
<th>Dental Procedure</th>
<th>Dental Code</th>
</tr>
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<tbody>
<tr>
<td>Excision of oral hard tissue</td>
<td>D7285</td>
</tr>
<tr>
<td>Excision of oral soft tissue</td>
<td>D7286</td>
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<tr>
<td>Excision of oral hard tissue</td>
<td>D7285</td>
</tr>
<tr>
<td>Excision of oral soft tissue</td>
<td>D7286</td>
</tr>
</tbody>
</table>

The Biopsy Report & what to do next

- Evaluating, documenting and following up oral pathological conditions: A suggested protocol
  JADA, Vol. 132, March 2001

THANK YOU ANY QUESTIONS?
Peds Derm Updates

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Disclosures

Speaker
- Sanofi Regeneron
- Amgen
- Almirall
- Pfizer
Advisory Board
- Janssen

Powerpoints are the peacocks of the business world; all show, no meat.
— Dwight Schrute, The Office

What’s New In Atopic Dermatitis?

Impact of Atopic Dermatitis
- Eczema causes stress, sleeplessness, discomfort and worry for the entire family
- Treating one patient with eczema is an example of “trickle down” healthcare
- Patients with eczema have increased risk of:
  - ADHD
  - Anxiety and Depression
  - Suicidal Ideation
  - Parental depression
  - Osteoporosis and osteopenia (due to steroids, decreased exercise, and chronic inflammation)

Impact of Atopic Dermatitis
- Sleep disturbances are a really big deal
- Parents of kids with atopic dermatitis lose an average of 1-1.5 hours of sleep a night
- Even when they sleep, kids with atopic dermatitis don’t get good sleep
  - Don’t enter REM as much or as long
  - Growth hormone is secreted in REM (JAAD Feb 2018)
Atopic Dermatitis and Food Allergies
- Growing evidence that food allergies might actually be caused by atopic dermatitis
  - Impaired barrier allows food proteins to abnormally enter the body and stimulate allergy
- Avoiding foods can be harmful
  - Proper nutrition is important
  - Avoidance now linked to increased risk for allergy and anaphylaxis
- Refer severe eczema patients to Allergist before 4-6 mos of age to talk about food introduction

Atopic Dermatitis: Standard Treatment
- Sensitive skin care
  - ALL free and clear detergent, no dryer sheets/fab soft
  - Dove sensitive skin or cetaphil soap
  - Vanicream/Vaseline/Aquaphor as moisturizers
  - Robathol bath oil
  - Bleach baths—1/4 cup bleach in full tub water

Atopic Dermatitis: Steroid Burst
- Topical steroid burst for severe eczema/significant flares
  - Clobetasol bid for 5 days
  - Fluocinonide bid for 10 days
  - Triamcinolone bid until clear or followup appt

Pathogenesis of Atopic Dermatitis
- Skin barrier is “broken”
- Overactive immune system process
  - Reaction to microbiome on skin?
    - Studies show microbiome in lesional/nonlesional atopic dermatitis on the same patient is different from people without atopic dermatitis (JAMA Derm March 2018)
    - Result of a “bored” immune system?

Atopic Dermatitis- Standard Treatment
- Topical steroids—always do OINTMENTS in little kids
  - HC 2.5
  - Triam 0.1
  - Fluocinonide 0.05
  - Clobetasol 0.05
Aron Regimen

- Originated with dermatologist in UK
- Peter Lio doing some studies on it
- Compounded medicine:
  - Betamethasone Valerate 0.1% cream- 30 gms
  - Mupirocin cream- 24 gms
  - Vanicream 400 gms
  - Mix to total 454 gms
- Use it 4-5 times daily to start and with improvement you slowly decrease the frequency of application

Calcineurin Inhibitors

- Elidel (pimecrolimus) 1% ointment
- Protopic (tacrolimus) 0.1% ointment
- Great for areas like face and folds
- Can be used as part of a maintenance routine

Calcineurin Inhibitors- Safety

- Black Box Warning
- Pimecrolimus study from Pediatrics
  - 2418 patients age 3-12 mos old
  - Pts followed for 5-10 years
  - Found no evidence of lymphoma, malignancy or immune system impairment
  - Concluded it was safe even in the younger age group
- Pediatric Allergy and Immunology June 2015
  - Review of 21 studies of almost 6000 pediatric patients with atopic dermatitis on TCIs
  - Conclusion: safe and effective

Eucrisa (Crisaborole)

- Boron based topical ointment
- Inhibits phosphodiesterase-4 activity (PDE4) and decreases production of proinflammatory cytokines
- Efficacy
  - Stinging and burning
  - Nice for maintenance
  - 78% of patients went a whole year without needing topical steroids (Eichenfield, JAAD)
- Might have a niche for hands
- Contains propylene glycol (Contact Allergen of Year in 2018)

Eucrisa (Crisaborole)

- JAAD May 2019
- Retrospective review of pain and Eucrisa
  - 41 patients
  - 13/41- 31.7%- had pain
  - 5/10 who used it on the face had pain

Dupixent (Dupilumab)

- Blocks IL-4 and IL-13 (decreases the TH2 inflammatory response)
- About 70% of patients achieve EASI 75
- Very tolerable
- Good side effect profile
  - Injection site reactions
  - Conjunctivitis- increased risk in severe AD and if patient has h/o eye symptoms such as allergic conjunctivitis
- 300 mg subcutaneously every other week
- Real life results better than study data
- Decreases risk of skin infections (JAAD Jan 2018)
**Dupixent (Dupilumab)**

- Approved for 12 and up for atopic dermatitis on March 11, 2019!
- Phase 3 data - really tough atopic derm patients
- Severe > Moderate
- Could not use topical steroids
- Same side effects as in adults
- Dupixent improved the signs, symptoms and quality of life in adolescents that didn’t achieve IGA 0/1 (SPD July 2019 Poster)

**Dupixent in Kids under 12**

- Studies in 6-11 yr olds have been completed
- My personal experience using it in kids down to 6 yrs old has been quite good
- I use 300 mg every 2 wks without loading dose
- Once clear, I often decrease to every 4 wks for maintenance
- I don’t often use the 200 mg dose
- It is a fight, but a fight that is worthwhile

"Just a little update...we started dupixent last Thursday when it arrived. We are AMAZED!!! He has felt great, just 1 wet wrap needed all week, just using vanicream. All blisters are gone and new skin is emerging. I just wanted to say thank you! Already an enormous difference.

He has been active this week, even wanting to go out to dinner, go out to play, and even on a shopping outing (which is rare for him). We have seen a huge increase in his happiness and hope. Thank you so much for helping us get here!"

**Getting Dupixent Approved**

"Most of the rules impacting access to medications are about cost, masquerading as safety."

- ELAINE SIEGFRIED, MD
- SPD MEETING JULY 2019
Getting Dupixent Approved

JAAD Sept 2018
- Burden of ER visits for patients with atopic dermatitis
- Association of atopic dermatitis with depression, anxiety, and suicidal ideation in kids and adults

JAAD Feb 2019
- Atopic Dermatitis and Suicide- metaanalysis of 15 studies
  - Patients with atopic derm were 44% more likely to have suicidal ideation and 36% more likely to attempt it

JAAD Feb 2019
- Both children and adults with atopic dermatitis have increased risk of other various autoimmune diseases
- Systematic review and meta analysis showing the relationship between atopic dermatitis and depression/suicidal ideation
  - Atopic derm is association with increased depression, antidepressant use, suicidal ideation and parental depression
- LACK OF FDA APPROVAL SHOULD NOT LIMIT ACCESS TO APPROPRIATE TREATMENT by Elaine Siegfried, et al

SPD Journal Jan/Feb 2019
- Impact of atopic dermatitis on families
  - Decreased quality of life of families in terms of sleep, finances and relationships
- ***Case Series by Peter Lio looking at 6 patients age 7-15 with atopic dermatitis on Dupixent***
  - All had improvement in IGA and no side effects reported
- ***Use of Dupixent in Pediatric AD: Access, dosing and implications by Elaine Siegfried***

JAMA Derm May 2019
- Sleep disturbances and exhaustion in mothers of children with atopic dermatitis- profound effect for the first 11 years of the child’s life

JAMA Derm Feb 2019
- Include pictures!
- Don’t take no for an answer
- Request a peer to peer
- Request an external review
- Have the parents call their insurance company 1-2 times a week to tell them how their child and family are suffering

Don’t take no for an answer
Request a peer to peer
Request an external review
Have the parents call their insurance company 1-2 times a week to tell them how their child and family are suffering
New Regional Dermatoses with Dupixent

- JAMA Derm July 2019
- Looked at New Regional Dermatoses (NRDs) in patients on dupixent for atopic dermatitis
- 17/124 patients developed new regional dermatoses
- 14/17 were on the face
- 12/17 were eczematous, 4/17 were erythema
- Is this allergic contact? Rosacea? Demodex?

Treatments on the Horizon

- Tapinarof 1% cream- activates the aryl hydrocarbon receptor
  - JAAD Jan 2019- EASI 75 in 50% of patients
- Lebrikizumab- IL 13- Q 4 wks
- Tralokinumab- IL 13- Q 2 wks
- Nemilizumab- IL 31
- Pozakinumab- IL 22- IV infusion
- JAK Inhibitors
  - Baricitinib JAK 1/2
  - Upadacitinib JAK 1- breakthrough status, Abbvie
  - Hopefully topical JAKs

Atopic Dermatitis: Natural Therapy

- Coconut oil
  - Has good antibacterial properties, but might not help the eczema itself
- Sunflower seed oil
  - Does appear to help with eczema- difficult to find a good preparation
  - Aroma Workshop in Chicago
  - hello@aromaworkshop.com
  - Patients can call 773-871-1985
  - 8 oz spray bottle for $22 plus $5.50 shipping

- CBD Oil
  - Probably does have some efficacy for itch and maybe for inflammation
  - Good formulation available thru Aroma Workshop in Chicago, formulated with help of Peter Lio, MD
- Mahonia Aquifolium
  - JCAD Dec 2018; looked at 8 small studies (7 for psoriasis, 1 for atopic dermatitis)
  - Statistically significant improvement with minimal side effects
  - On amazon, but super fragrant

Atopic Dermatitis: Prevention

- Probiotics
  - Taken by a child with eczema appear to have no impact
  - But if a pregnant woman takes probiotics 2 weeks prior to having a baby and for 3 mos after having the baby, it reduces the risk of eczema in that baby by 20-30%
- Transepidermal Water Loss (TEWL)
  - TEWL in first weeks of life associated with increased risk of eczema
  - Families with h/o eczema should be managing their new baby with the same sensitive skin care strategies to try to prevent the eczema
  - 50% reduction in eczema by simply using sensitive skin care in first weeks of life
What’s New in Pediatric Allergic Contact Dermatitis?

Contact Dermatitis in Kids
- Either on the rise or being recognized more commonly
- 1 exposure to the triggering agent causes a rash for 3 weeks (patients cannot intermittently use their allergen)
- Patch testing can be considered, but most of the time, we try to identify the culprit based on the pattern of the rash

Wet Wipe Contact Dermatitis
- Due to preservative MCI/MI (Kathon CG)
- Also think about it in cases of persistent facial dermatitis
- There are now 3 brands of wipes that don’t contain the allergen
  - Honest Brand
  - Earth’s Best Hypoallergenic
  - Water wipes

A Quick Comment about Parabens
- Parabens were the "Non Allergen" of the Year in 2018
- Parabenphobia was unnecessary in the first place
- Based on estrogen effects seen in animals given 25000 times the dose of parabens used as a preservative in topicals
- Most manufacturers replaced it with MCI/MI

Nickel Contact Dermatitis
Nickel Contact Dermatitis

- Most common allergen
- Present in almost anything metal
  - Jewelry
  - Snaps on jeans
  - Belt buckles
- Strict avoidance is the only option
  - www.nickelsolution.com makes a clear lacquer that is better than nail polish and can be effective
  - www.nonickel.com
- Dimethylglyoxime test
- Can trigger an id reaction

Id Reaction

- An id reaction is a sympathy rash to the primary problem
- Most commonly triggered by allergic contact dermatitis, but can be triggered by molluscum or tinea

Gianotti Crosti

- Also causes monomorphic skin colored to pink papules all over arms, legs and cheeks
- Check the ears
- Typically caused by EBV but several viruses or vaccinations can do it
- Can take up to 8 wks to resolve
- Topical steroids help if itchy

Gianotti Crosti- ear involvement
Toilet Seat Dermatitis

- Either a reaction to a cleanser being used on the seat or to the components of the seat itself
- Characteristic distribution on the lateral buttocks and post thighs
- “Soft and Comfy” toilet seat covers - Amazon $5.99
- Treat with hydrocortisone or desonide

Slimer’s Dermatitis

- Making slime has become quite the trend
- The ingredients in slime are all irritating - borax, glue, soap
- Produces a rash on the palms that looks like dyshidrotic eczema with red scaly patches and sometimes teeny vesicles
- Typically extends into webspaces from squeezing the slime
- “A Slime of the Times” Peds Derm Jan/Feb 2019

What’s New in Pediatric Psoriasis?

- It’s out there
- Plaque psoriasis
- Guttate psoriasis - often triggered by strep
- Inverse psoriasis - nearly always mistaken for yeast/tinea cruris in kids/teens
- Check the nails, check the tongue
Psoriasis/Eczema Overlap
(aka Psoriasiform Dermatitis)

- Very common in kids
- Might look like eczema in the antecub fossa but looks like psoriasis on the scalp
- Check the fingernails, check the tongue, check the belly button
- Look for Koebner phenomenon
- Sometimes family history of psoriasis helps
- Lack of response to mild topical steroids is suggestive of psoriasis
- Treat and observe the patient over time

Pediatric Psoriasis- Topical Treatment

- Clobetasol cream/ointment- body
- Clobetasol foam (Olx/Olux E Foam)- scalp
- Taclonex suspension or Enstilar foam
- Elidel or Protopic- face and folds
- I personally don’t think calcipotriene alone or tazorac is that helpful
- Light therapy

Psoriasis

- Systemic treatment of pediatric psoriasis is probably 10 years behind adult treatment, but we’re trying to catch up!
- Systemic effects of psoriasis are making it more advantageous to consider systemic therapy, even in children
- Old school treatments like methotrexate and soriatane carry significant risks
  - JAMA Derm Sept 2017- compared MTX or TNF (mostly Enbrel) for peds psoriasis
  - Far fewer adverse events in the TNF inhibitor group

Biologics in Kids

- Enbrel (etanercept)- NOW APPROVED FOR KIDS >4 YRS OLD!!
  - Approved in Europe for psoriasis in kids >6 yrs old
  - Approved in US for JIA in kids >2 yrs old
  - 1 study in US in children- 2008- 211 patients age 4-17
    - 0.8 mg/kg/wk
    - 57% achieved PASI 75
    - This study has been continued to date and has great long term safety data (JAAD Feb 2016)

- Stelara (Ustekinumab)
  - Approved in kids 12 and up!!
  - Going for approval down to 6- study has completed and FDA approval is pending
  - Several case reports of effectiveness and safety
  - 1 clinical trial- patients age 12-18, 110 patients
    - 80% reached PASI 75 at 12 wks (JAAD Oct 2015)
  - I have several pediatric patients on it- it’s my treatment of choice for pediatric psoriasis

Biologics in Kids
Psoriasis is a Systemic Disease
Guidelines in Kids (JAMA Derm July 2017)

- #1 association in children is obesity
- Talk to them about weight
- Screen for diabetes every 3 yrs at age 10
- Screen for NASH every 3 yrs at age 10
- Screen for HTN annually starting at age 3
- Screen lipids at age 10 and again at 18
- Ask about arthritis
- Ask about depression and anxiety
- Ask about smoking, stress, substance abuse in older kids

Psoriasis is a Systemic Disease

- For every year that you have psoriasis, your risk of cardiovascular disease goes up by 1%
- That’s a big deal if you get psoriasis when you are a kid
- We hope systemic treatment reduces the risk
  - Aortic root inflammation is key marker of cardiovascular disease (measured on PET CT)
  - Ustekinumab decreased aortic root inflammation by 19% in 12 weeks

Impact of Pediatric Psoriasis on Parents

- JAAD Feb 2017
- 65% said their own emotional wellbeing was affected
- 50% were sad or frustrated
- 20% were depressed or anxious
- They felt burdened by all the topical management
- Had to make activity accommodations 2/3 of the time because of child’s skin disease

What’s New with Pediatric Rashes?

Diaper Rashes
Diaper Rashes

- Most common causes are irritant contact derm and yeast
- Symmetrical, moist appearing pinkness with satellite pustules suggests yeast
- Dermatitic like symmetrical rash that involves contact with soiled areas, frictional creases suggests irritant contact
- Regardless, I suggest zinc oxide barrier cream (Desitin) with each diaper change
- Pick one (go with your gut) and treat
  - Hydrocortisone 2.5% ointment bid
  - Econazole 1% cream bid

Diaper Rashes- Irritant Contact!

Diaper Rashes- Yeast!

Diaper Rashes- Yeast again!

Hand Foot and Mouth Disease

- Causes somewhat annular red-purple-gray patches on hands, feet, and around the mouth sometimes with intraoral lesions
- Previously coxsackie A16 and enterovirus 71 were the most common causes
- Coxsackie A6 has emerged over the past 4-5 yrs as the primary causative agent
- Produces more severe rash with prominent diaper area involvement
- Adults have been getting it
- Commonly produces onychomadesis 1-2 mos later (SPD July/Aug 2016)
HFMD and Onychomadesis

What’s New with Acne?

Mid Childhood Acne
- Acne in kids age 3-7 is NOT normal
- Ask about inhaled steroid use- can be the cause
- Good idea to order labs and/or refer to peds endocrinology
  - Total/free testosterone
  - DHEA-S
  - LH/FSH
  - Bone age- plain film of left hand and left wrist

Acne
- Happening younger and younger
- Used to be abnormal before age 9, now abnormal before age 7
- Most acne medicines are technically approved for age 12 and up
- Helpful to work through the mail order pharmacies in these situations
  - DermRx is my favorite for acne meds

Seysara (Sarecycline)
- New oral antibiotic to treat acne
- First new oral antibiotic for acne in over 40 years
- Approved for kids 9 and up
- Once daily weight based dosing
- Seems to avoid side effects of doxycycline and minocycline
  - No esophageal discomfort, no sun sensitivity
  - No vertigo, dizziness, lupus like syndrome, blue skin
- Has been studied and found to have minimal effect on gut flora
### Oral Contraceptive Pills
- Given desire to decrease oral antibiotic use, the use of OCPs has become more appealing.
- FDA Approved for acne: Ortho Tri Cyclen, EstroStep, Yaz.
- My counseling routine:
  - How to start the pill
  - Weight gain, nausea, mood issues
  - Blood clots, heart attack, stroke
  - Health benefits
  - Timeliness is important

### OCPs
- Retrospective review of 2147 patients on OCPs for acne (JDD June 2016):
  - All OCPs help with acne
  - Triphasics probably help a little more than monophasics
  - Non estrogen component matters for efficacy:
    - Best: Drospirenone (Yaz, Yasmin)
    - 2nd Best: Norgestimate/desogestrel (ortho tri cyclen, ortho cyclen/ mircette, desogen)
    - 3rd Best: Norethindrone/levonorgestrel (loestrin, ortho novum/seasonale)

### OCPs
- Typically want to try to avoid OCPs in girls less than 14 yrs old or girls that have had their period for less than 2 yrs.
- Rifampin and Griseofulvin are the only antifungals that decrease the efficacy of OCPs when preventing pregnancy.
- Risk of clots is greatest when a patient is first starting the pill
- Practical Derm May 2018:
  - Baseline DVT risk: 3/10000 women/yr
  - On OCPs: 6/10000
  - On Yaz: 9/10000
  - Pregnant: 12/10000

### Contraindications to OCPs (W.H.O.)
- Pregnancy
- Current breast cancer
- Breastfeeding < 6 wks postpartum
- Age > 35 yrs and a heavy smoker
- HTN
- Diabetes with end organ damage
- Diabetes > 20 yrs duration
- History of or current DVT/PE
- Major surgery with prolonged immobilization
- Ischemic heart disease or Valvular heart disease with complications
- History of CVA
- Headaches (migraine with focal neuro symptoms at any age or without aura if > 35 yrs old)
- Active viral hepatitis
- Severe decompensated cirrhosis
- Liver tumor (benign or malignant)

### Other Hormone Tidbits
- Progesterone only methods of birth control tend to increase acne:
  - Implanon
  - Mirena IUD
  - Progesterone mini pills
- Spironolactone can be helpful in the teenage population, especially if the patient:
  - Is on a progesterone only method of birth control
  - Has features of or a diagnosis of PCOS
  - Mayo study: 80 teenage girls- Spiro worked well, but most had to use 100 mg daily

### Changes in Isotretinoin Monitoring
- A number of studies in 2017 showed that we have been “over monitoring” with labs for isotretinoin.
- New recommendations are to check lipids and LFTs at baseline and then at 2 mos into therapy. If normal, that is all that is necessary.
- No need to check CBC
Changes in Treatment of Scarring

- A number of studies showed that we don’t have to delay treatment for the scarring
- Practical Derm July 2017, JAAD July 2017, JAMA Derm August 2017
- Don’t have to wait for things like microderm, superficial chemical peels, skin surgery, LHR, fraxel
- I still wait until acne is under control and or/accutane therapy has been completed

Isotretinoin and Depression

- JAAD June 2017- Isotretinoin and depression- a systematic review and metaanalysis done in Taiwan
- Reviewed 31 studies
- DID NOT show an association
- Most kids had an improvement in their mood

Accutane and Depression

- From 2015-2019, I have had 4 male patients and 2 female patients become severely depressed on accutane. None of them had h/o mood issues prior.
- Appears to happen acutely
- All 6 admitted that they felt the symptoms early on, but had lied to me about it because they saw the improvement the accutane was having with their skin
- 2 of them were cutting themselves unbeknownst to their friends and family
- All 6 of them expressed suicidal ideation
- 1 of them was admitted to the hospital on a psych hold
- 1 of them attempted to commit suicide by jumping off a ladder head first
- All 6 of them stopped the accutane and their mood returned to normal

Food and Acne

- Diet with a high glycemic index (high carb, high sugar) appears to worsen acne for some people

What’s New with Moles?

Eclipse Nevi
**Eclipse Nevi**
- Very common on the scalp of children
- Frequently biopsied because of somewhat atypical coloring, large size, history of changing
- Often read out as atypical on pathology, but these are known to be completely benign
- Probably a “special site” that isn’t currently recognized as a special site

**Pediatric Melanoma**
- Fortunately rare
  - JAAD Feb 2018 and SPD May/June 2018 wrote about it
- In kids older than 10ish:
  - They tend to look like adult melanomas with the ABCDs, etc
  - Can be deadly
- In kids younger than 10ish:
  - They tend to be spitzoid
  - Clinically amelanotic. Biopsied thinking they are a spitz or a pyogenic granuloma or a wart or an angiokeratoma
  - They tend to look worse on path - deeper, more mits, neural invasion - but their prognosis is excellent
  - Supports hypothesis that MM in young kids is biologically distinct from MM in adults

**Infantile Hemangiomas**

**What’s New with Vascular Things?**

**Infantile Hemangiomas**

**Infantile Hemangiomas**

**Propranolol is Still Great!**
- Suspension is 20 mg/5 ml
- 2 mg/kg/day divided TID
  - If you are doing the math correctly, the dose ends up being around 1 ml TID for most babies
- Always give with food
  - To prevent hypoglycemia
- Don’t be afraid - if the hemangioma needs it, use it!
- Typically used during growth period (1st 8-12 mos of life), but can work even beyond the proliferative phase (SPD May/June 2015)
### Which Hemangiomas Need Propranolol?
- Large hemangiomas
- Ulcerating hemangiomas
- Hemangiomas in functional locations that will interfere with crawling, walking, etc
  - Knees, hands, elbows
- Special site hemangiomas
  - Eyelids, nose, lips, parotid glands, genital area
- Dome shaped hemangiomas
  - Even when they involute, there is usually residual fibrofatty tissue

### Infantile Hemangiomas
- Long term studies show no risk of developmental adverse effects or growth impairment at age 4 in pts treated with at least 6 mos of propranolol (JAAD July 2016)
- More recent study shows the same at age 7, specifically no cognitive impairment (SPD Sept/Oct 2017)
- Topical timolol 0.5% gel forming solution can work for superficial hemangiomas- applied BID

### Pyogenic Granulomas
- “Little ball of capillaries”
- Common in kids and pregnant women
- Some people remember trauma to the area prior to its growth
- 2 Treatment Options
  - Shave removal
  - Topical timolol bid

### Pyogenic Granuloma
- Initial study in March/April 2014 SPD journal using timolol 0.5% gel forming solution BID
- Great results with clearance after 2-3 mos
- Bleeding stopped relatively instantly
- Likely working by vasoconstriction
- Important to followup these patients to ensure improvement (spitz nevi, even melanoma in ddx)
What’s New with Warts and Molluscum?

Warts
- Countless treatment options
  - Liquid nitrogen
  - Cantharidin
  - OTCs
  - Candida
  - Laser
  - Bleomycin
- Best Thing Ever- WartPeel!
  - Nucara Pharmacy- Iowa
  - Sal acid + 5FU
  - Magic in a bottle
  - Applied at bedtime under “sticky tape”
  - $89 and worth every penny!

Ring Phenomenon
- Typically associated with cantharidin
- Can happen with liquid nitrogen
- The treated wart may or may not go away and then a ring of warts develops around the initial wart
- If you continue that treatment, the ring gets bigger
- I feel it is happening more and more commonly with cantharidin these days
Mounting number of case reports showing that when pre-teens and teens are given HPV vaccine, their warts go away. It will be interesting to see if we notice a decrease in incidence of warts over time as more and more people get immunized.

Warts vs Corn: A Handy Trick

- Press on top of it
  - If it hurts, it is a callus/corn
- Press on the sides of it (squeeze it)
  - If it hurts, it is a wart

Molluscum Contagiosum

- Caused by a poxvirus
- Very common in kids - pretty much all kids get them
- Spread by direct contact and spread like crazy in water (including swimming pools)
- Treatment is not mandatory as they will go away with time
  - Can take up to 2 yrs to resolve on their own
  - 2015 study of 170 kids - half treated, half not treated
    - Molluscum resolved in the same amount of time

Molluscum Treatment Options

- Imiquimod?
- Zymaderm
  - All natural OTC product, botanical based
  - Applied BID
- Candida antigen injections
  - Injected into 1-2 of the molluscum every 3 wks
  - Tolerable; typically 3-5 treatments
  - Side effect profile favorable
- Cantharidin
  - Never use it in the axilla
  - Blisters can be bad
  - 50% resolution with each treatment is success
  - Hard to get these days
- WartPeel?
  - Terydab MWF at bedtime
  - Just treat 3-4
  - No sticky tape
- Curettage
- Liquid Nitrogen
- Topical retinoids
- KOH 10% daily?

On the Horizon

- Verrica - 0.7% cantharidin
  - Due for approval shortly
- SB206 - Nitric oxide product applied daily
  - Complete clearance at week 12 for most patients in phase 2
  - Phase 3 trials in 2019
- Some kids will get an eczema like rash around the molluscum
- Important to treat it as it itches so kids scratch and then spread the molluscum

Molluscum Dermatitis

Pseudofurunculoid Molluscum
- Look like pimples/boils
- Due to body's immune system response
- Not infected, just inflamed
- BOTE sign - Beginning Of The End

Pseudofurunculoid Molluscum

PF Molluscum and Id Reaction
- Treat the Id Reaction with topical steroids
- Treat the PF molluscum with oral antibiotics or bleach baths
- F/u 2-3 wks
- Usually everything is "all better"

PF Molluscum and Id Reaction

What's New in Spots?
"Midline Anterior Neck Inclusion Cyst"

Giant milia in the suprasternal notch at birth

Benign

Can be removed or can resolve on own

Shave can be used to remove

Miscellaneous Tidbits

Princess Package at Disneyland
SPD May/June 2018

Topical Rapamycin for lymphangiomas

JAAD Feb 2019:
Lack of FDA Approval should not limit access to appropriate treatment

- Peds Derm July/Aug 2018 - topical sirolimus 0.1% useful for lymphangiomas. Response in less than 3 mos with bid application
- JAAD Feb 2019 - Topical rapamycin effective for lymphangiomas
- Prescribe 15 1 mg tablets to usual pharmacy (typically covered). Bring them to compound pharmacy and have them crush and compound it into emollient cream base to make 15 gms of rapamycin 0.1% cream. 1-2 month supply. Cost usually $45.- SPD Sept/Oct 2015.
Topical Rapamycin for Lymphangiomas

- Chemistry Rx compounds it
  - Can get it with glitter!
- Systemic sirolimus levels are NOT detectable when used topically for vascular malformations (SPD July 2019 Poster)

Xepi (Ozenoxacin) Cream
Approved for impetigo in patients >2 mos old-
BID x 5 days

MAM Air Pacifier

- For kids that have persistent dermatitis around the mouth, drool and irritation from pacifiers are a common cause
- Recommend the MAM Air Pacifier which is more open than most

The End!

- Feel free to contact me with any questions
  - lisawansonmd@gmail.com
Tips on the use of anti-IL-17 Drugs in Psoriasis

Mark Lebwohl, MD
Waldman Professor
And Chairman
Kimberly and Eric J. Waldman
Department of Dermatology
Icahn School of Medicine at Mount Sinai

Mark Lebwohl is an employee of Mount Sinai which receives research funds from: AbbVie, Amgen, Astellas, Boehringer Ingelheim, Celgene, Eli Lilly, Incyte, Janssen / Johnson & Johnson, Kadmon, Leo Pharma/Anacor, Medimmune, Novartis, Pfizer, Sideris, UCB, Ortho-Dermatologics, and Vizzav.

Dr. Lebwohl is also a consultant for Allergan, Almirall, Astellas, BMS, Boehringer Ingelheim, Bonten Meyer Aquilini, Carle, Castle Biosciences, Dermici, Enca, Immune, IQ Pharma, Med1, Menlo, Mitsubishi Pharma, Neuroderm LTd./Pfizer, Promius/Dr. Reddy, Theravance Biopharma, and Vifor.

Updated 04/23/19

Time to Achieve 50% Improvement in PASI

Time for 25% of Patients to achieve PASI 75

Marked URGENT!

Dear Dr. Lebwohl,
I prescribed Secukinumab for a 175 pound patient with severe psoriasis. We showed her how to administer the shots on Monday. She called me on Friday to say that she had administered two shots each day from Monday through Friday and was now out of Secukinumab. What should I do?

Sincerely,
Dr. XXXXXX

Inadvertent Overdose of Secukinumab, Consequences, and Cautions
Mark Taliercio, Dana Alessa, David B. Kessler
JOURNAL OF PSORIASIS AND PSORIATIC ARTHRITIS.

Dr. Lebwohl,
Thank you for taking the time to speak to me at the Mt Sinai Winter meeting. I have reported to Novartis, my patient a 70 year old white female with PMH of diabetes and hypertension with severe psoriasis with prior treatment of Enbrel, Humira, StellaRa and methotrexate who was recently started on Cosentyx. Her medications included; Insulin, coreg, amloidpine, aspirin, citalopram, fenofibrate, and hydrochlorothiazide; She is 5’5”, 190 lbs, bp 150/90.
She gave herself SQ shots of Cosentyx 150 mg x 2 daily on 8/11,8/12,8/13,8/14, 8/15/2015. She called after one week complaining of her Skin “dry and peeling” and when was her next shot due.She had no adverse effects and her labs were normal except her usual elevated glucose. Her PGA went from 4->2 after 2 weeks. I restarted her next Cosentyx one month after her last dose and continues to do well.
Once again thank you for your time. I wish you and your family a Happy and healthy New Year.
Sincerely,
"In post-marketing database, we have 100+ cases of "overdoses" which are divided as unintentional, intentional and prescribed. ...there are no reports of unusual AEs — most times we don't see any AE." From Novartis

Pearl #2 Anti-IL-17 antibodies are safe
Maximum dose not known

Secukinumab package insert

10 OVERDOSAGE
Doses up to 30 mg/kg intravenously (i.e., approximately 2000 to 3000 mg) have been in clinical trials without dose-limiting toxicity. In the event of overdosage, it is recommended that the patient be monitored for any signs or symptoms of adverse reactions and appropriate symptomatic treatment be instituted immediately.

Response to question about ixekizumab overdose

- Doses up to 180 mg have been administered subcutaneously in clinical trials without dose-limiting toxicity. Overdoses up to 240 mg, subcutaneously, have been reported without any serious adverse events.1
- In the event of overdosage, monitor the patient for any signs or symptoms of adverse reactions and institute appropriate symptomatic treatment immediately.2

Enclosed Prescribing Information
TALTZ® (ixekizumab) injection, for subcutaneous administration, Lilly

References
1. Data on file, Eli Lilly and Company and/or one of its subsidiaries.

Treatment of Moderate to Severe Psoriasis With High-Dose (450-mg) Secukinumab: Case Reports of Off-Label Use.

Immunity to infection in IL-17-deficient mice and humans.

Chronic mucocutaneous candidiasis in humans with inborn errors of interleukin-17 immunity.

Oral fluconazole 150 mg single dose versus intravaginal clotrimazole treatment of acute vulvovaginal candidiasis.

• "independent risk factors for post-transplant included cyclosporine-based immunosuppressant agents during the first year after kidney transplantation (odds ratio [OR]: 1.98, P"
• "high proportion of extrapulmonary spread

Pearl #4 IL-17 not assoc. w/ TB reactivation

• 70/147,000
• 48 < 3 infusions
• Test for TB!

• Tumor necrosis factor-alpha is required in the protective immune response against Mycobacterium tuberculosis in mice. Flynn JL, et al

TNF is necessary for normal granuloma formation and function.

• ↑ TB risk up to 4x
Risk of tuberculosis is higher with anti-tumor necrosis factor monoclonal antibody therapy than with soluble tumor necrosis factor receptor therapy: the three-year prospective French research axed on tolerance of biotherapies registry. Tubach F et al. *Arthritis & Rheumatism* 2009;60:1884-94.

- IFX: SIR 18.6
- ADA: SIR 29.3
- ETN: SIR 1.8

**WARNING**

*RISK OF INFECTIONS TUBERCULOSIS (FREQUENTLY DISSEMINATED OR EXTRAPULMONARY AT CLINICAL PRESENTATION), INVASIVE FUNGAL INFECTIONS, AND OTHER OPPORTUNISTIC INFECTIONS, HAVE BEEN OBSERVED IN PATIENTS RECEIVING REMICADE. SOME OF THESE INFECTIONS HAVE BEEN FATAL (SEE WARNINGS). PATIENTS SHOULD BE EVALUATED FOR LATENT TUBERCULOSIS INFECTION WITH A TUBERCULIN SKIN TEST. TREATMENT OF LATENT TUBERCULOSIS INFECTION SHOULD BE INITIATED PRIOR TO THERAPY WITH REMICADE.*

**TB Rates in Adalimumab Clinical Studies**

<table>
<thead>
<tr>
<th></th>
<th>Pre-screening</th>
<th>Post-screening</th>
</tr>
</thead>
<tbody>
<tr>
<td># cases</td>
<td>EU: 13</td>
<td>EU: 11</td>
</tr>
<tr>
<td></td>
<td>North America:</td>
<td></td>
</tr>
<tr>
<td>Exposure</td>
<td>534 pt-yrs</td>
<td>3078 pt-yrs</td>
</tr>
</tbody>
</table>

**BOXED WARNING**

*Patients need to be evaluated for tuberculosis risk factors and for latent or active tuberculosis infection with a tuberculin skin test both before and during treatment.*

*Cases of tuberculosis have occurred in patients who received etanercept; therefore, treatment of latent infection should be started before etanercept initiation.*

*Consider antituberculosis therapy before etanercept initiation in patients with a past history of latent or active tuberculosis in whom an adequate course of treatment cannot be confirmed.*

*Some patients who tested negative for latent tuberculosis before etanercept receipt have developed active tuberculosis.*

**Low penetrance, broad resistance, and favorable outcome of interleukin 12 receptor beta 1 deficiency: medical and immunological implications.**


- 41 patients - IL12 receptor β1 deficiency
- Salmonellosis
- Tuberculosis
“Individuals genetically deficient in interleukin (IL)-12/IL-23 are particularly vulnerable to disseminated infections from mycobacteria (eg, nontuberculous, environmental mycobacteria), salmonella (eg, nontyphi strains), and Bacillus Calmette-Guerin (BCG) vaccinations; consider appropriate diagnostic testing. Evaluate for tuberculosis (TB) infection prior to, during, and after treatment; do not administer to patients with active TB. Consider anti-TB therapy prior to initiation in patients with history of latent or active TB when an adequate course of treatment cannot be confirmed.”

**Ustekinumab PI**

**Tuberculosis**

Evaluate all potential recipients of guselkumab for tuberculosis infection before initiating treatment. Do not administer guselkumab to patients with active tuberculosis infective patients with latent tuberculosis, antituberculosis therapy should be administered before initiating guselkumab. C antituberculosis treatment for patients with a past history of latent or active tuberculosis in whom an adequate course of treatment cannot be confirmed. Monitor patients closely for signs and symptoms of active tuberculosis infection during and after treatment.

**Guselkumab PI**

Essential role of IL-17A in the formation of a mycobacterial infection-induced granuloma in the lung.


- IL-17A deficiency may reduce formation of granulomas

IL-23 compensates for the absence of IL-12p70 and is essential for the IL-17 response during tuberculosis but is dispensable for protection and antigen-specific IFN-γ responses if IL-12p70 is available.


- depletion of IL-17A-producing CD4+ T cells → no effect on disease progression during primary *M. tuberculosis* infection

**Secukinumab in patients with LTBI**

- At BL, 25 subjects who received SKB had a past history of either pulmonary TB, LTBI or a positive TB test
- Tested negative for LTBI by QFN Gold at screening
- None were on anti-TB medication during the psoriasis study
- None experienced reactivation of TB; median SKB treatment duration was 363 days

**Subjects diagnosed with LTBI during screening Phase 3 SKB trials**

<table>
<thead>
<tr>
<th>Treatment</th>
<th>Subjects diagnosed with LTBI in screening (%)</th>
<th>Median duration of treatment with SKB (days)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Any SKB 150 mg</td>
<td>52</td>
<td>364</td>
</tr>
<tr>
<td>Any SKB 300 mg</td>
<td>55</td>
<td>364</td>
</tr>
<tr>
<td>Any SKB 450 mg</td>
<td>100</td>
<td>364</td>
</tr>
</tbody>
</table>

Tsai T-F, et al. AAD 2015, P607 Sponsored by Novartis Pharma AG
Secukinumab shows no evidence for reactivation of previous or latent TB infection in psoriasis patients: Pooled Phase 3 safety

- 1 TB-negative subject (at BL; in ERASURE) was diagnosed with LTBI following retest according to local guidelines (Argentina) on Day 141 while on SKB 150 mg; treated with isoniazid 300 mg daily and completed the study without SKB dose interruption

Secukinumab PI

No reactivation of tuberculosis in psoriasis patients with latent tuberculosis infection while on ixekizumab treatment: a report from 11 clinical studies

Elisabeth Riedl1,2, Stefan Winkler3, Wen Xu2, Noah Agada2, Mark G Lebwohl4

Inborn errors of human IL-17 immunity underlie chronic mucocutaneous candidiasis.

Puel A, et al.

“Pre-treatment Evaluation for Tuberculosis
Evaluate patients for tuberculosis (TB) infection prior to initiating treatment with SECUKINUMAB. Do not administer SECUKINUMAB to patients with active TB infection. Initiate treatment of latent TB prior to administering SECUKINUMAB. Consider anti-TB therapy prior to initiation of SECUKINUMAB in patients with a past history of latent or active TB in whom an adequate course of treatment cannot be confirmed. Patients receiving SECUKINUMAB should be monitored closely for signs and symptoms of active TB during and after treatment.”

Secukinumab PI

Autosomal recessive deficiency in the receptor IL-17RA (due to mutations in the IL17RA gene) or autosomal dominant mutations in IL17F→Chronic mucocutaneous candidiasis

Safety in Psoriasis Patients with Latent Tuberculosis (TB) Tr with Guselkumab and Anti-TB Treatments in the Phase 3 VO Trials

Luis Puig, Tsen-fangTsai, Tina Bhutani, Jonathan Uy, Paraneedh Ramachandran, Michael Song, Yin You, Melinda Gooderham, Mr Lebwohl

130 patients randomized to PBO, GUS or ADA at baseline tested positive for concomitant anti-TB treatments.

No cases of TB reactivation

Presented at ECON
IL-23 compensates for the absence of IL-12p70 and is essential for the IL-17 response during tuberculosis but is dispensable for protection and antigen-specific IFN-γ responses if IL-12p70 is available.


- depletion of IL-17A–producing CD4+ T cells — no effect on disease progression during primary *M. tuberculosis* infection

**Pearl #4** IL-17 blockade not associated with Tb reactivation

---

**Secukinumab**

**FUTURE 1:** Radiographic progression in PsA patients stratified by MTX use

<table>
<thead>
<tr>
<th>MTX: Yes</th>
<th>MTX: No</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean change in vdB-mTSS</td>
<td>Overall population</td>
</tr>
<tr>
<td>MTX: Yes</td>
<td>MTX: No</td>
</tr>
<tr>
<td>Overall</td>
<td>MTX: Yes</td>
</tr>
</tbody>
</table>

-0.03 | 0.4 | 0.2 |

-*P*<0.05 vs PBO

Change in mTSS >0.5 considered progression of radiographic disease

**Gottlieb AB, et al. EADV 2015, P0348 Sponsored by Novartis Pharma AG**

---

**Secukinumab**

**FUTURE 2**  
**ACR20 Response Through Week 52**

- TNFi-Naive
- TNFi-Exposed

<table>
<thead>
<tr>
<th>Treatment</th>
<th>TNFi-Naive</th>
<th>TNFi-Exposed</th>
</tr>
</thead>
<tbody>
<tr>
<td>Secukinumab 75 mg SC</td>
<td>31.3%</td>
<td>26.9%</td>
</tr>
<tr>
<td>Secukinumab 150 mg SC</td>
<td>27.4%</td>
<td>21.6%</td>
</tr>
<tr>
<td>Placebo</td>
<td>14.3%</td>
<td>9.2%</td>
</tr>
</tbody>
</table>

**Kavanaugh A, et al. Presented at American College of Rheumatology Annual Meeting; Nov 6-11, 2015; San Francisco, CA; Oral 2146.**

Brodalumab Phase 2 PsA study: Clinical response and improvement in psoriasis in subjects with PsA

Mease P, et al. AAD 2014, P7605

ACR20 response rate at Week 12

<table>
<thead>
<tr>
<th></th>
<th>Placebo (n=55)</th>
<th>BRO 140 mg q2w (n=57)</th>
<th>BRO 280 mg q2w (n=56)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Patients (%)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>0</td>
<td>0</td>
<td>18.2</td>
<td>36.8</td>
</tr>
<tr>
<td>5</td>
<td>1</td>
<td>50.0</td>
<td>54.4</td>
</tr>
<tr>
<td>10</td>
<td>9</td>
<td>27.3</td>
<td>21.3</td>
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<tr>
<td>15</td>
<td>2</td>
<td>18.2</td>
<td>18.3</td>
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<tr>
<td>20</td>
<td>1</td>
<td>15.5</td>
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</tr>
<tr>
<td>25</td>
<td>0</td>
<td>12.5</td>
<td>12.5</td>
</tr>
<tr>
<td>30</td>
<td>0</td>
<td>0.0</td>
<td>0.0</td>
</tr>
<tr>
<td>35</td>
<td>0</td>
<td>0.0</td>
<td>0.0</td>
</tr>
<tr>
<td>40</td>
<td>0</td>
<td>0.0</td>
<td>0.0</td>
</tr>
<tr>
<td>45</td>
<td>0</td>
<td>0.0</td>
<td>0.0</td>
</tr>
</tbody>
</table>

*P<0.05 vs placebo

ACR20 response rate at Week 24

<table>
<thead>
<tr>
<th></th>
<th>PBO (n=55)</th>
<th>BRO 140 mg q2w (n=57)</th>
<th>BRO 280 mg q2w (n=56)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Patients (%)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>0</td>
<td>19.6</td>
<td>32.7</td>
<td>33.3</td>
</tr>
<tr>
<td>5</td>
<td>20.0</td>
<td>27.7</td>
<td>27.3</td>
</tr>
<tr>
<td>10</td>
<td>18.2</td>
<td>29.3</td>
<td>23.3</td>
</tr>
<tr>
<td>15</td>
<td>9.5</td>
<td>18.2</td>
<td>13.3</td>
</tr>
<tr>
<td>20</td>
<td>5.0</td>
<td>12.5</td>
<td>10.0</td>
</tr>
<tr>
<td>25</td>
<td>0.0</td>
<td>12.5</td>
<td>0.0</td>
</tr>
<tr>
<td>30</td>
<td>0.0</td>
<td>0.0</td>
<td>0.0</td>
</tr>
<tr>
<td>35</td>
<td>0.0</td>
<td>0.0</td>
<td>0.0</td>
</tr>
<tr>
<td>40</td>
<td>0.0</td>
<td>0.0</td>
<td>0.0</td>
</tr>
<tr>
<td>45</td>
<td>0.0</td>
<td>0.0</td>
<td>0.0</td>
</tr>
</tbody>
</table>

*Enrolled set. After Week 12, patients receiving placebo or bimekizumab 16 mg were re-randomized (1:1) to receive bimekizumab 160 mg or 320 mg; all other patients continued on their previous dose;

**Patients with <10% improvement from baseline in TJC and SJC are eligible for rescue therapy. CASPAR, Classification Criteria for Psoriatic Arthritis; LD, loading dose; Q4W, every four weeks; SJC, swollen joint count; TJC, tender joint count; TNF, tumor necrosis factor

Bimekizumab BE ACTIVE study

Study design

Key inclusion criteria
- Active PsA
- TJC ≤33 or ≥34
- CRP <10
- CASPAR ≥3
- Patients with <10% improvement from baseline in TJC and SJC are eligible for rescue therapy.

Efficacy and safety at Week 76

Therapeutic response of PsA to TNFi and brodalumab

- No head-to-head trials
- BRO trial is Phase 2, not placebo controlled
- Others are Phase 3 and placebo controlled

*ADA ETN IFX GLM BRO

Screening

Primary endpoint

ACR50 response

Study week

Placebo Q4W

Bimekizumab 160 mg Q4W

Bimekizumab 320 mg Q4W

Bimekizumab 16 mg Q4W

Bimekizumab 160 mg Q4W

Bimekizumab 320 mg Q4W

N/A = not available
ACR50 response rates at Week 12 (NRI)

<table>
<thead>
<tr>
<th>Treatment</th>
<th>ACR50 response (%) (SE)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Placebo</td>
<td>21.8 (4.1)</td>
</tr>
<tr>
<td>BKZ 16 mg</td>
<td>41.3 (4.0)</td>
</tr>
<tr>
<td>BKZ 160 mg (100 mg)</td>
<td>46.3 (4.0)</td>
</tr>
<tr>
<td>BKZ 200 mg (160 mg)</td>
<td>24.4 (4.1)</td>
</tr>
</tbody>
</table>

ACR50 response rates increased up to Week 24 and were maintained through Week 48 (NRI)

<table>
<thead>
<tr>
<th>Treatment</th>
<th>ACR50 response (%) (SE)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Placebo</td>
<td>20.7 (3.8)</td>
</tr>
<tr>
<td>BKZ 16 mg</td>
<td>53.8 (4.0)</td>
</tr>
<tr>
<td>BKZ 160 mg</td>
<td>46.4 (4.0)</td>
</tr>
</tbody>
</table>

ACR20 and ACR70 response rates at Weeks 12 and 48 (NRI)

<table>
<thead>
<tr>
<th>Treatment</th>
<th>ACR20 response (%) SE</th>
<th>ACR70 response (%) SE</th>
</tr>
</thead>
<tbody>
<tr>
<td>Placebo</td>
<td>11.8 (1.8)</td>
<td>7.1 (0.9)</td>
</tr>
<tr>
<td>BKZ 16 mg</td>
<td>11.2 (1.8)</td>
<td>7.6 (0.9)</td>
</tr>
<tr>
<td>BKZ 160 mg (100 mg)</td>
<td>11.4 (1.8)</td>
<td>7.8 (0.9)</td>
</tr>
<tr>
<td>BKZ 200 mg (160 mg)</td>
<td>10.2 (1.8)</td>
<td>7.5 (0.9)</td>
</tr>
</tbody>
</table>

ACR50 response rates at Week 12 (NRI)

- There was a significant dose-response at Week 12 for ACR50 response rates (primary outcome; p=0.031).

ACR50 response rates increased up to Week 24 and were maintained through Week 48 (NRI)

ACR20 and ACR70 response rates at Weeks 12 and 48 (NRI)

<table>
<thead>
<tr>
<th>Treatment</th>
<th>ACR20 response (%) SE</th>
<th>ACR70 response (%) SE</th>
</tr>
</thead>
<tbody>
<tr>
<td>Placebo</td>
<td>11.8 (1.8)</td>
<td>7.1 (0.9)</td>
</tr>
<tr>
<td>BKZ 16 mg</td>
<td>11.2 (1.8)</td>
<td>7.6 (0.9)</td>
</tr>
<tr>
<td>BKZ 160 mg (100 mg)</td>
<td>11.4 (1.8)</td>
<td>7.8 (0.9)</td>
</tr>
<tr>
<td>BKZ 200 mg (160 mg)</td>
<td>10.2 (1.8)</td>
<td>7.5 (0.9)</td>
</tr>
</tbody>
</table>

PASI90 response rates increased up to Week 24 and were maintained through the study (NRI)

<table>
<thead>
<tr>
<th>Treatment</th>
<th>PASI90 response (%) SE</th>
</tr>
</thead>
<tbody>
<tr>
<td>Placebo</td>
<td>100 (0)</td>
</tr>
<tr>
<td>BKZ 16 mg</td>
<td>20.7 (3.8)</td>
</tr>
<tr>
<td>BKZ 160 mg</td>
<td>53.8 (4.0)</td>
</tr>
<tr>
<td>BKZ 200 mg (160 mg)</td>
<td>46.4 (4.0)</td>
</tr>
</tbody>
</table>

PASI90 response rates increased up to Week 24 and were maintained through the study (NRI)

Percentage of patients with resolution of enthesitis at Weeks 12 and 48 (NRI)

<table>
<thead>
<tr>
<th>Treatment</th>
<th>Resolution of enthesitis (%) SE</th>
</tr>
</thead>
<tbody>
<tr>
<td>Placebo</td>
<td>39.6 (2.3)</td>
</tr>
<tr>
<td>BKZ 160 mg (100 mg)</td>
<td>58.1 (2.6)</td>
</tr>
<tr>
<td>BKZ 200 mg (160 mg)</td>
<td>53.1 (2.6)</td>
</tr>
<tr>
<td>BKZ 200 mg (160 mg)</td>
<td>34.6 (2.3)</td>
</tr>
</tbody>
</table>

Adverse events for special monitoring up to Week 48

- There were no cases of inflammatory bowel disease, major cardiovascular events or hypersensitivity and anaphylactic reactions during the study.
- All candida infections were of mild or moderate intensity and did not lead to treatment discontinuation.
Pearl #6 IL-17 blockers are effective in obese patients

<table>
<thead>
<tr>
<th>Baseline Body Weight Group</th>
<th>PASI 90 Response Rate (%)</th>
<th>PASI 100 Response Rate (%)</th>
<th>Week 12</th>
<th>Week 52</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt;80 kg</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>PBO (n=50)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>ETN (n=111)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>IXE Q2W (n=187)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>IXE Q4W (n=187)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>ETN (n=111)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>IXE Q2W (n=187)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>IXE Q4W (n=187)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Across all baseline body weight categories, IXE-treated patients achieved significantly greater PASI 90 response rates vs. PBO at Week 12.

Across all baseline body weight categories, IXE-treated patients achieved significantly greater PASI 100 response rates vs. PBO at Week 12.

Skin Clearance Response Rates improve over time on treatment with Brodalumab 210 mg Q2W in Non-obese and Obese Patients

<table>
<thead>
<tr>
<th>AMAGINE-1</th>
<th>Nonobese and obese patients who received continuous brodalumab 210 mg Q2W</th>
</tr>
</thead>
<tbody>
<tr>
<td>52 weeks</td>
<td>PASI, TEAEs +</td>
</tr>
</tbody>
</table>

- Rates of achieving sPGA 0/1, PASI 75, PASI 90, and PASI 100 were higher among nonobese patients than obese patients at weeks 12 and 52.
- The percentage of patients achieving PASI 100 increased from week 12 to week 52 in both nonobese and obese patients.
- The safety associated with brodalumab 210 mg Q2W was comparable between nonobese and obese patients (data not shown).

Activity of secukinumab, an anti-IL-17A antibody, on brain lesions in RRMS: results from a randomized, proof-of-concept study.

Havrdová E, et al

IL-17 Mediated Inflammation Promotes Tumor Growth and Progression in the Skin

D. He, et al

IL-23→IL-17→tumor growth

Could blocking IL-17 be protective against cancer?

Secukinumab
PUBMED search
1/10/18
NO ↑ MALIGNANCIES
Exposure-Adjusted Malignancy Event Rates Through 52 Weeks Were Lower in the All-Brodalumab Group Than Those in the Ustekinumab Group

**Figure.** Malignancy events in psoriasis studies (52-week results).

<table>
<thead>
<tr>
<th>Exposure-Adjusted Malignancy Event Rates</th>
<th>All-Brodalumab</th>
<th>Ustekinumab (N=613; 495 PY)</th>
<th>Constant brodalumab 210 mg Q2W (N=1335; 1042 PY)</th>
<th>All brodalumab (N=4019; 3446 PY)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Basal cell carcinoma</td>
<td>0.2</td>
<td>2.6</td>
<td>1.8</td>
<td>1.6</td>
</tr>
<tr>
<td>Squamous cell carcinoma</td>
<td>0.3</td>
<td>0.4</td>
<td>0.5</td>
<td>0.4</td>
</tr>
<tr>
<td>Bowen’s disease</td>
<td>0.3</td>
<td>0.3</td>
<td>0.3</td>
<td>0.4</td>
</tr>
<tr>
<td>Adjudicated malignancies</td>
<td>2.6</td>
<td>2.6</td>
<td>2.6</td>
<td>2.6</td>
</tr>
</tbody>
</table>

The above bar charts illustrate that the all-brodalumab group had significantly lower malignancy event rates compared to the ustekinumab group. This indicates that exposure-adjusted malignancies were lower in the all-brodalumab group than those in the ustekinumab group through 52 weeks.

Safety of Secukinumab in Hepatitis B Virus

SL Bevans, TT Mayo, BE Elewski, in press

- Reports of HBV infection (5 patients), HCV infection (3 patients), and HBV and HCV co-infection (1 patient), all without viral reactivation or significant elevation in liver enzymes.

Pearl #9 IL-17 blockers have been used in hepatitis/HIV; anecdotal

Impact of Secukinumab on Endothelial Dysfunction Other Cardiovascular Disease Parameters in Psoriasis Patients over 52 Weeks.

- At w. 52 “secukinumab might have a beneficial effect on CV risk by improving the endothelial function of plaque psoriasis” as measured by flow-mediated dilation.

Pearl #10 Do IL-17 blockers protect against cardiovascular disease?
Suicidal ideation and behavior, including 4 completed suicides, occurred in subjects treated with SILIQ in the psoriasis clinical trials. There were no completed suicides in the 12-week placebo-controlled portion of the trials. SILIQ users with a history of suicidality or depression had an increased incidence of suicidal ideation and behavior as compared to users without such a history [see Adverse Reactions (6.1)]. A causal association between treatment with SILIQ and increased risk of suicidal ideation and behavior has not been established.

Pearl #11: REMS program is easy and worthwhile; only for brodalumab

Suicidal Ideation and Behavior Rates in Psoriasis Studies

RESULTS (cont)

Table 4. Summary of Completed Suicides (Known and Unknown Cause)

<table>
<thead>
<tr>
<th>Age, y/ Sex</th>
<th>Brodalumab dose</th>
<th>Clinical response (PASI score)</th>
<th>Clinical Information</th>
</tr>
</thead>
<tbody>
<tr>
<td>59/Male</td>
<td>210 mg</td>
<td>100</td>
<td>329 days after first dose of brodalumab</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>History of financial stress (not able to work) due to brodalumab response and inability to obtain unemployment benefits</td>
</tr>
<tr>
<td>39/Male</td>
<td>210 mg</td>
<td>73</td>
<td>140 days after first dose of brodalumab</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Informed investigator he had legal difficulties and was likely to be incarcerated</td>
</tr>
<tr>
<td>56/Male</td>
<td>210 mg</td>
<td>100</td>
<td>Ongoing treatment for depression and anxiety</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Described recent stress and isolation due to relocation</td>
</tr>
<tr>
<td>Indeterminate case</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>56/Male</td>
<td>210 mg</td>
<td>100</td>
<td>History of depression and anxiety</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>History of stress and depression due to brodalumab response and inability to work</td>
</tr>
<tr>
<td>56/Male</td>
<td>210 mg</td>
<td>100</td>
<td>28 days after first dose of brodalumab</td>
</tr>
</tbody>
</table>

C-CASA, Columbia classification algorithm of suicide assessment; HADS, hospital anxiety and depression scale; PASI, psoriasis area and severity index.
Changes in HADS Severity at Week 12

RESULTS

Table 1. Changes in Depression and Anxiety as Measured by HADS During Short-term Psoriasis Treatment in the AMAGINE-1 Trial

<table>
<thead>
<tr>
<th>Placebo</th>
<th>Brodalumab 140 mg Q2W</th>
<th>Brodalumab 210 mg Q2W</th>
<th>Placebo</th>
<th>Brodalumab 140 mg Q2W</th>
<th>Brodalumab 210 mg Q2W</th>
</tr>
</thead>
<tbody>
<tr>
<td>Depression</td>
<td>Anxiety</td>
<td>Depression</td>
<td>Anxiety</td>
<td>Depression</td>
<td>Anxiety</td>
</tr>
<tr>
<td>Placebo</td>
<td>Brodalumab 140 mg Q2W</td>
<td>Brodalumab 210 mg Q2W</td>
<td>Placebo</td>
<td>Brodalumab 140 mg Q2W</td>
<td>Brodalumab 210 mg Q2W</td>
</tr>
<tr>
<td>Placebo</td>
<td>Brodalumab 140 mg Q2W</td>
<td>Brodalumab 210 mg Q2W</td>
<td>Placebo</td>
<td>Brodalumab 140 mg Q2W</td>
<td>Brodalumab 210 mg Q2W</td>
</tr>
<tr>
<td>Placebo</td>
<td>Brodalumab 140 mg Q2W</td>
<td>Brodalumab 210 mg Q2W</td>
<td>Placebo</td>
<td>Brodalumab 140 mg Q2W</td>
<td>Brodalumab 210 mg Q2W</td>
</tr>
<tr>
<td>Placebo</td>
<td>Brodalumab 140 mg Q2W</td>
<td>Brodalumab 210 mg Q2W</td>
<td>Placebo</td>
<td>Brodalumab 140 mg Q2W</td>
<td>Brodalumab 210 mg Q2W</td>
</tr>
<tr>
<td>Placebo</td>
<td>Brodalumab 140 mg Q2W</td>
<td>Brodalumab 210 mg Q2W</td>
<td>Placebo</td>
<td>Brodalumab 140 mg Q2W</td>
<td>Brodalumab 210 mg Q2W</td>
</tr>
</tbody>
</table>

* In the AMAGINE-1 study, the proportion of patients who experienced improvement in depression or anxiety, as determined by HADS, was appreciably greater with brodalumab than with placebo.

HADS: Hospital Anxiety and Depression Scale; Q2W: every 2 weeks.

Data are through week 12 of the AMAGINE-1 study. Baseline to endpoint. Baseline to highest score.

75th Annual Meeting of the American Academy of Dermatology; March 3-7, 2017; Orlando, FL.

---

Pearl #13 Getting approvals for biologics isn’t that hard

70 yo ♀, Psoriasis 15% BSA & PSA

- Prescribe etanercept

DENIED

National Psoriasis Foundation
Leah McCormick Howard
Health Policy Manager
Lhoward@psoriasis.org
(503) 546-5553

AAD Practice Management Center
Office of Access to Care and Treatment
Rachna Chaudhari
www.aad.org/priorauth
Are we finally at the point where we can successfully treat these lesions?

This presentation will review some of these new therapies

Some new modalities showing much better overall responses in 2018

Recurrence rates of 40-55% reported

Some new modalities showing much better overall responses in 2018

This presentation will review some of these new therapies

Are we finally at the point where we can successfully treat these lesions?

- Consultant to many pharmaceutical, cosmetic, laser and energy-based device companies
- Consultant, performs research and speaks on behalf of numerous pharmaceutical and medical device companies
- For the benefit of this presentation: consultant, investigator, Dr. Gold is a consultant for and speaks for Lumenis, Teana and Strathmore
Silicone Gel Sheetings
Plast Reconstr Surg 2002; 110:560

Updated International Clinical Recommendations on Scar Management:
Part 2 - Evaluating the Evidence
Dermatology Surgery, Vol. 40, No. 8, August 2014, p. 825

Special Topic
International Clinical Recommendations on Scar Management

Topical Silicone: Mechanism of Action

Therapies
Silicone Gel
Despite initial skepticism, there is now good evidence of its efficacy and it is now one of the best treatments for scar therapy.
Combination therapy is most preferable with silicone gel:
- Eight randomized, control studies and a meta-study of 27 trials demonstrate its safety and efficacy
- Totally occlusive dressings and semi-occlusive dressings have not shown evidence of efficacy, and evidence from other non-silicone based dressings is mixed

Topical Silicone Gel Sheetings in the Treatment of Hypertrophic Scars and Keloids –
A Dermatologic Experience

After two to three months of silicone gel treatment, collagen deposition has normalized, and there is no scar hypertrophy.
Radiotherapy - OLD
Radiotherapy continues to be reserved for secondary management in adults with hypertrophic or keloid scarring after other treatment options have proven ineffective
- Monotherapy remains controversial
- Combination therapy with surgery often reported
- Most trials retrospective
- Most trials do not define recurrences

Radiotherapy - NEW
Superficial Radiation Therapy WORKS with excisional surgery
- Literature very supportive
- Recurrence rates low – reports from 1-10%
  - Changes the game for many
  - And with combination with topical therapies??????

Low Rate of Keloid Recurrence Following Treatment of Keloidectomy Sites with Biologically Effective Dose 10 of Superficial Radiation
Efficacy of SRT for Recurring Keloids

Keloid scars are considerably challenging as they are often refractory to treatment and the recurrence rate using surgical excision alone is 45 to 100%.

Recurrence rates after excision with adjuvant radiation therapy range from 0% to 8.9%.

Studies suggest that x-ray radiation may prevent keloid recurrence by controlling fibroblast proliferation, arresting the cell cycle, and inducing premature cellular senescence.

<table>
<thead>
<tr>
<th>Author</th>
<th>N lesions</th>
<th>Treatment dose</th>
<th>Follow up period</th>
<th>Non-recurrence</th>
</tr>
</thead>
<tbody>
<tr>
<td>Reaven (1986)</td>
<td>21</td>
<td>80 Gy</td>
<td>1 yr</td>
<td>95%</td>
</tr>
<tr>
<td>Horgan et al. (2000)</td>
<td>61</td>
<td>30 Gy in 20% of lesions</td>
<td>1 yr</td>
<td>98%</td>
</tr>
<tr>
<td>Benamou (2015)</td>
<td>21</td>
<td>9 Gy x 40 Gy, total 40 Gy</td>
<td>1 yr</td>
<td>98%</td>
</tr>
<tr>
<td>Jones et al. (2017)</td>
<td>50</td>
<td>13-18 Gy + PRP, 2yr</td>
<td>94%</td>
<td></td>
</tr>
<tr>
<td>Ragoowansi (2003)</td>
<td>80</td>
<td>10 Gy in 24h of excision</td>
<td>1yr</td>
<td>91%</td>
</tr>
</tbody>
</table>

Optimal Treatment for the Prevention of Keloids after Surgery: BED 30

A literature search was performed to identify studies dealing with the efficacy of various treatment regimens for the prevention of keloids after surgery. Biologically effective doses (BEDs) of the various radiation regimens were calculated. To effectively treat keloids postoperatively, relatively high doses must be applied in a short overall treatment time.

The optimal treatment is a radiation scheme resulting in a BED value of at least 30 Gy obtained with:
- A single acute dose of 15 Gy
- Two fractions of 8 Gy
- Three fractions of 6 Gy
- A single dose of 27 Gy at low dose rate

Radiation treatment should be administered within 2 days after surgery.

One Year Retrospective Chart Review Study

Sixty-one patients, 96 excised keloids with at least 1 year follow up at 4 US sites. BED 30 of SRT 70 or 100kV, usually 3 Frs of 60 Gy on POD 1, 2 and 3.

10.4% (10/96) treated keloids were noted to recur within 12 months. Of the 11 recurring treated keloids:
- 8 (73%) recurred within the first 6 months;
- 2 (18%) recurred within 6 to 12 months post-treatment; and
- 1 (9%) recurred within 12 to 18 months post-treatment.

Kaplan-Meier Survival Probability cure rate of 85.6% from 24 months post-SRT treatment onwards.

Greater rate of recurrence if keloid had previously recurred or was on the chest.

Long term Safety

Although ionising radiation may have long term effects, the development of calibration techniques and better treatment modalities show an incidence of radiation therapy-related cancers of less than 0.3%.

A recent review of evidence suggests that the risks of cancer following Radiation Therapy for benign disease are small, especially in older patients. However, the balance of risk vs benefit needs to be considered in younger adults and especially if RT is being considered in adolescents or children.

For the specific treatment of recurrent keloid scars, a systematic review concluded that the risk developing a neoplasm from keloid radiotherapy was low.
Laser Therapy – becoming more popular as efficacy has increased

- Carbon dioxide Lasers: +/-
- Argon Lasers: -
- ND: YAG Lasers: +
- Pulsed-Dye Lasers and IPL: ++
- Fractional Lasers: +++
  - Non-Ablative Fractional Lasers
  - Ablative Fractional Lasers
- Picosecond Lasers

Ablative fractional (CO₂)
(for severe, large, contracted scars)

Three for Hope Foundation

CO₂ Ablative Resurfacing for Scars

Angel Faces

CO₂ Ablative Resurfacing for Scars

Waibel J, Beer K.
Ablative Fractional Laser Resurfacing for the Treatment of a Third-Degree Burn

The Katie Piper Foundation
Fractional CO2 lasers with or without pulsed dye lasers in the treatment of hypertrophic scars

Pulsed Dye Laser (PDL) has been shown to be effective for the treatment of raised hypertrophic scars. Both 585 nm and 595 nm lasers have been shown to be effective in the treatment of hypertrophic scars.

- **Mechanism:** PDL both destroys and inhibits the formation of small vessels within the scar.
- **Penetration depth:** 0.4 to 1.2 mm
- May not penetrate deep enough to effectively destroy or prevent dilated capillaries in the deeper regions of the scar.
- PDL is not effective in stopping the proliferation of fibroblasts and the deposition of collagen.

Fractional CO2 has become very popular for treating hypertrophic scars. It penetrates deep into the skin, promotes fibroblast apoptosis, and collagen degradation.

Combination of PDL and Fractional CO2 may be better than PDL alone. Current study, performed at The Department of Plastic and Laser Cosmetic, Hunan Provincial People's Hospital, Changsha, Hunan, China.

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Fractional CO2 lasers with or without pulsed dye lasers in the treatment of hypertrophic scars

56 patients with immature hypertrophic scars were selected for inclusion into the clinical trial.

- **35** males, 21 females
- **Age range:** 3-51 years

All scars were less than 3 months old to be enrolled into the study.

20 scars were from burns, 17 from scalding from hot liquid, 12 from scratches or contusions, and 7 from surgical procedures.

All patients (or guardians) signed an Informed Consent.

**Control Group – PDL only (V Beam Perfecta)**

Laser settings: 7-15 J/cm² fluence, 1.5-3 ms pulse width, 7 mm spot size, and 30-millisecond spray, 20-millisecond delay of dynamic cooling device (DCD). The endpoint for the PDL treatment was purpura occurring in the treatment area. The patients received 2 laser treatments at 1-month intervals.

Fractional CO2 lasers with or without pulsed dye lasers in the treatment of hypertrophic scars

**Treatment Group:** Hypertrophic scars were treated with the 595 nm adjustable pulse width PDL plus the UltraPulse fractional CO2 laser. The PDL treatment parameters and the endpoint for this therapy were the same as the treatment group. The UltraPulse fractional CO2 laser was used in the scanner mode so that all of the scars were treated with the following laser settings: Mode: Deep FX, Energy: 30-50 mJ, Frequency: 300 Hz, Density 90, Scan Shape, and Spot Size were determined by the shape and the area of the scar, following treatment with the 595 nm adjustable pulse width PDL.

The time between laser treatments in the Treatment Group was 3 months.

Photographs were obtained after the second laser treatment in both the Treatment Group and the Control Group (Treatment group: Photographs were taken 2 months after the first treatment, and Control group: Photographs were taken 6 months after the first treatment).

Clinical efficacy was evaluated by the physicians who were not directly involved in the actual laser treatments and follow-up visits using the Vancouver Scar Scale (VSS).

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Vancouver Scar Scale (VSS)

<table>
<thead>
<tr>
<th>Feature</th>
<th>Treatment</th>
<th>Control</th>
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</thead>
<tbody>
<tr>
<td>Epidermal</td>
<td>Full thickness</td>
<td>Full thickness</td>
</tr>
<tr>
<td>Inflammation</td>
<td>Partial</td>
<td>Partial</td>
</tr>
<tr>
<td>Hydration</td>
<td>Mild</td>
<td>Mild</td>
</tr>
<tr>
<td>Texture</td>
<td>Rough</td>
<td>Rough</td>
</tr>
<tr>
<td>Mobility</td>
<td>Good</td>
<td>Good</td>
</tr>
</tbody>
</table>

Fractional CO² lasers with or without pulsed dye lasers in the treatment of hypertrrophic scars

The total score of VSS and score of melanin, height, vascularity, pliability of the Control Group, and the Treatment Group both demonstrated an obvious decrease in the VSS scores after the treatments when comparing the before and after score numbers on the VSS.

Both groups showed statistically significant differences between the before and after treatment levels by the statistical analysis performed (P < .05).

The total score of the VSS, as well as the scores of melanin, height, vascularity, pliability in the Treatment Group, decreased more than that of Control Group when the groups were compared, and was also statistically significant (P < .05).

VSS Score of Treatment Group and Control Group

Discussion

We choose to use first the PDL to coagulate or close the scar’s blood vessels which gives these scars the red color and then use the fractional CO² laser to prevent the scar’s continued growth as this laser deals primarily with inhibiting proliferating fibroblasts and the deposition of abnormal collagen.

The advantage of this combination approach is the definite clinical improvements that we see with the two technologies together. The disadvantage of this is that it takes several months to achieve these acceptable results.

Fractional CO² lasers with or without pulsed dye lasers in the treatment of hypertrrophic scars

A consensus within our plastic surgical team and in our clinical practice is that 3 months is the optimal scar duration for successful scar therapy.

As the thickness of a fresh immature scar is limited, the Deep FX can be used to penetrate to deep layers of the scar tissue, produce the appropriate heat effect, and establish a drug delivery channel.

Conclusions

In summary, the 595 nm adjustable pulse width PDL combined with the UltraPulse CO² Fractional Laser appears to have a beneficial clinical effect on fresh red hypertrrophic scars.

The PDL combined with the CO² fractional laser treating fresh red hypertrrophic scars is worthy treatment method, which should be used regularly in treating these fresh red hypertrrophic scars.
Management choices should depend on the patient’s individual requirements and evidence-based findings.

There remains a significant need for further randomized, controlled trials of all available scar therapies and systematic, quantitative reviews of the literature to ensure optimal management of scarring.

Recommendations given are based on best available evidence to date.

2019 – we are able to treat hypertrophic scars and keloids better today than what has been available in the past.
Resident & Student Board Review Session Schedule

Friday, September 27, 2019
9:00 a.m. - 4:00 p.m.  Preparing for Boards and Beyond (Including roundtable discussion on board preparation, job search, contract negotiations, and starting your own practice)

Saturday, September 28, 2019
8:00 a.m. - 8:15 a.m.  Biologics: A Review for the Boards
Jonathan Crane, DO, FAOCD

8:15 a.m. - 8:30 a.m.  Vulvar Dermatology
Courtney Bernett, DO

8:30 a.m. - 8:45 a.m.  Bullae & Vesicles: A Review of Differential Diagnoses and Treatment Options
Katherine Braunlich, DO

8:45 a.m. - 9:00 a.m.  Update in Melanoma Therapies
Falon Brown, DO

9:00 a.m. - 9:15 a.m.  Morphea in Pediatrics
Joanna Emilio, DO

9:15 a.m. - 9:30 a.m.  Nerve Sheath Myxoma
Michael Fong, DO

9:30 a.m. - 9:45 a.m.  Dermatologic Manifestations of Diabetes Mellitus
Chelsea Harper, DO

9:45 a.m. - 10:00 a.m.  Keloids and SRT
Jessica L. Jones, DO

10:00 a.m. - 10:30 a.m.  Break with Exhibitors
Broadway Ballroom F

10:30 a.m. - 10:45 a.m.  You Don't Know What You're Missing: the Utility of the Wood's Lamp
Logan Kolb, DO

10:45 a.m. - 11:00 a.m.  Vaccine Preventable Infections and Dermatosis
Luke Maxfield, DO
11:00 a.m. - 11:15 a.m.  
**Hidradenitis Suppurativa Update**  
Ann Lin, DO

11:15 a.m. - 11:30 a.m.  
**An Update on Atypical Mycobacterial Infections**  
Erin Lowe, DO

11:30 a.m. - 11:45 a.m.  
**Cutaneous B-Cell Lymphomas: A Practical High-Yield Review**  
John Moesch, DO

11:45 a.m. - 12:00 p.m.  
**Interesting Cases from the Big City**  
Serge Petrosian, DO

12:00 p.m. - 1:00 p.m.  
Lunch on Your Own

1:00 p.m. - 1:15 p.m.  
**Surgical Anatomy for the Boards**  
Ashley Rice, DO

1:15 p.m. - 1:30 p.m.  
**Basic Sciences for the Boards**  
Dahlia Saleh, DO

1:30 p.m. - 1:45 p.m.  
**Principles of Electrosurgery**  
Muneeb Shah, DO

1:45 p.m. - 2:00 p.m.  
**Preserving Osteopathic Dermatology**  
David Cleaver, DO, FAOCD
Biologics for the Boards
Jonathan Crane, DO FAAD FAOCD
Campbell University at Sampson Regional Medical Center
Dermatology Residency Program Director
Wilmington, NC

Immunopathogenesis of Psoriasis

- T-cell disorder, primarily CD8+ in epidermis and mix of CD4+/CD8+ in dermis
- Immune Profile
  - Increased Th1 cytokines (IFN-γ and IL-2), IL-1, IL-6, and TNF-alpha
  - Decreased IL-10
  - IL-23 from dendritic cells
    - Th 17 stimulation
    - IL-17 and IL-22 release
    - dermal inflammation and keratinocyte replication
  - Increased CXCL8
    - neutrophil chemotaxis
    (spongiform pustules of Kogoj and microabscesses of Munro)
  - VEGF
    - angiogenesis of superficial dermal vessels
  - STAT-3 expression
    - keratinocyte proliferation

Growth of Biologics

TNF-alpha Inhibitors: Indications

- Infliximab
  - Chimeric monoclonal IgG antibody targeting TNF-alpha receptor only
- Etanercept
  - Fully human dimeric fusion protein (TNF receptor linked to Fc portion of IgG) that blocks both TNF-alpha and TNF-beta
- Adalimumab
  - Fully human monoclonal IgG antibody against transmembrane TNF receptor

TNF-alpha inhibitors for psoriasis

- None
Which of the following medications should be avoided in patients on TNF-alpha inhibitors?

a) Cyclosporine  
b) Azathioprine  
c) Rituximab  
d) Methotrexate

Question: Which of the following medications should be avoided in patients on TNF-alpha inhibitors?

a) Cyclosporine  
b) Azathioprine  
c) Rituximab  
d) Methotrexate

Fatal hepatosplenic T-cell lymphoma may be seen in patients on TNF-alpha inhibitor + azathioprine

TNF-alpha inhibitors: Side Effects and Monitoring

- Caution if family history of demyelinating disease (MS, Guillain-Barre, optic neuritis)
- New onset psoriasis, palmoplantar pustulosis, and cutaneous vasculitis have been reported
- May increase the risk of MALT lymphoma
- Increased risk of lymphoma
- Hepatosplenic T-cell lymphoma (FATAL!) can be seen in patients on TNF inhibitor + azathioprine
- CHF exacerbation (particularly with infliximab)
- TNFs contraindicated in Class III or IV CHF
- Echocardiogram should be done before treatment initiation in class 1 or 2 CHF and should be avoided in patients with EF<50%
- Increased risk of TB (primary and reactivation), invasive fungal infections, and opportunistic infections such as legionella and listeria
- Increased risk of NMSC (namely SCCs); best to avoid anti-TNF alpha agents in patients with concurrent malignancy or history of malignancy, especially multiple cutaneous SCCs

IL-23 inhibitors

- Tildrakizumab (Ilumya®)
- Guselkumab (Tremfya®)
- Risankizumab-rzaa (Skyrizi™)

All target the p19 subunit of IL-23

IL-23 is produced by antigen-presenting dendritic cells, and the normal function of IL-23 is to stimulate differentiation, activation, proliferation, and survival of Th17 cells
IL-17 inhibitors (Ixekizumab, Secukinumab, Brodalumab)

- Ixekizumab (Taltz)
- Secukinumab (Cosentyx)
- Brodalumab (Siliq)

Neutralize IL-17A 
Blocks IL-17 receptor

Which of the following side effects can be expected in a patient on an IL-17 inhibitor?

a) Lymphoma
b) Congestive heart failure
c) Neuromuscular disorders
d) Oral thrush

Candidiasis and herpes infections have been reported with IL-17 inhibitors.

The most commonly reported side effect of IL-17 inhibitors is nasopharyngitis. Other common side effects include URI, injection site reactions, and headache.

There is NO increased risk of lymphoma, heart failure, or neuromuscular disorders.

Apremilast (Otezla)

- FDA approved for both psoriasis and psoriatic arthritis
- A PDE-4 inhibitor involved in modulating production of inflammatory mediators
- Phosphodiesterase 4 (PDE4) degrades cyclic AMP (cAMP) in many immune cells
- Includes eosinophils, neutrophils, macrophages, T cells, and monocytes

Apremilast (Otezla): Side Effects and Monitoring

- Most common side effects are diarrhea (25%) and nausea - typically resolve on their own within 4 weeks.
- Depression and weight loss (5-10% decrease in weight in 10% of patients) have also been reported.
- No laboratory monitoring required.
- Dose may need to be adjusted in patients with severe renal impairment.

Janus Kinases (JAKs)

- There are four JAKs in humans: JAK1, JAK2, JAK3, and tyrosine kinase 2 (TYK2)
- Play a key role in the transduction of cytokine-mediated signals leading to activated transcription of inflammatory proteins implicated in the pathogenesis of inflammatory and autoimmune diseases, including RA, PsO, and PsA, atopic dermatitis, alopecia areata, and vitiligo
- Examples of JAK-dependent cytokines include:
  - JAK1/2: IFN-γ, IL-6
  - JAK1/3: IL-2, IL-4
  - JAK1/TYK2: IFN-α/β, IL-4/13
  - JAK2/TYK2: IL-12/23

- Drugs include Ruxolitinib, Baricitinib, Tofacitinib, Ponatinib, and Olaparib
JAK Mechanism of Action

- Cytokines bind various combinations of JAK receptors, causing the JAKs to phosphorylate STATs.
- Upon phosphorylation, STATs dimerize, translocate to the nucleus and activate target genes.

Inhibitors
- Tofacitinib (Xeljanz) targets JAK 1,3
- Baricitinib (Olumiant) and Ruxolitinib (Jakafi) target JAK 1,2

JAK Inhibitors

- Tofacitinib (Xeljanz)
  - 5 mg BID or 11 mg ER daily
  - Psoriatic arthritis
  - Rheumatoid arthritis
- Baricitinib (Olumiant)
  - RA, unresponsive to TNF-alpha
- Ruxolitinib (Jakafi)
  - Myelofibrosis
  - Dermatologic

FDA Approved Indications
- Tofacitinib (Xeljanz)
  - RA 5 mg BID or 11 mg ER daily
  - Psoriatic Arthritis
  - Ulcerative colitis
- Baricitinib (Olumiant)
  - RA, unresponsive to TNF-alpha
- Ruxolitinib (Jakafi)
  - Myelofibrosis
  - Dermatologic

Dermatologic Uses
- Tofacitinib (Xeljanz)
  - Psoriasis-Phase III studies
  - AA–5 mg BID x 3 mo
  - TOPICAL 2% cream
- Baricitinib (Olumiant)
  - Psoriasis–phase II
- Ruxolitinib (Jakafi)
  - AA–20 mg BID x 3-6 mo
  - TOPICAL 2% cream
  - Vitiligo

Other dermatologic conditions for which successful oral JAK inhibitor therapy has been reported include GVHD, dermatomyositis, STAT1-related chronic mucocutaneous candidiasis, and interferonopathies.

JAK Inhibitors Monitoring

- Prior to treatment:
  - PPD/IFN-y release assay
  - CBC and CMP
  - Hepatitis B and C profiles
  - HIV
  - Lipid profile
- During treatment:
  - Annual PPD/IFN-y release assay
  - CBC, CMP and lipid profile within 1-2 months and then every 3 months.

References

- https://www.psoriasis.org/
- National Psoriasis Foundation
- Journal of the American Academy of Dermatology
- Journal of Drugs in Dermatology
- Bolognia 4th edition
- Up To Date
VESICLES & BULLAE:  
A REVIEW OF DIFFERENTIAL DIAGNOSES AND TREATMENT OPTIONS  
Kate Braunlich, DO, PGY4  
Program Director: Dr. Richard Miller

I have no relevant disclosures

All photos are taken from Andrews’ Diseases of the Skin Clinical Atlas unless otherwise specified. The rights/copyright to these photos remains with the authors of this text.

<table>
<thead>
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<th>History</th>
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<tr>
<td>• How long have the bullae or vesicles been present?</td>
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<tr>
<td>• Has the patient had bullae/vesicles before?</td>
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<tr>
<td>• If chronic, does the eruption occur at the same site each time?</td>
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<tr>
<td>• Are the bullae/vesicles symptomatic?</td>
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<tr>
<td>• Is the patient taking medications? If so, which medications?</td>
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<tr>
<th>Physical Exam</th>
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<tbody>
<tr>
<td>• Patient age</td>
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<tr>
<td>• If female, childbearing status, i.e. pregnant, recently post-partum etc.</td>
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<tr>
<td>• Bullae/vesicle distribution</td>
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<tr>
<td>• Is there mucosal involvement?</td>
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<tr>
<td>• Are the bullae/vesicles isolated or is there concomitant desquamation, erosions, fissures, scale or erythema?</td>
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<tr>
<td>• Is there evidence of scarring</td>
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<table>
<thead>
<tr>
<th>Fragile or tense bullae?</th>
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</thead>
<tbody>
<tr>
<td>• Fragile Bullae</td>
</tr>
<tr>
<td>• Bullous impetigo</td>
</tr>
<tr>
<td>• Pemphigus (all variants)</td>
</tr>
<tr>
<td>• Hailey-Hailey disease</td>
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<tr>
<td>• Tense Bullae</td>
</tr>
<tr>
<td>• Contact dermatitis (allergic or irritant)</td>
</tr>
<tr>
<td>• Bullous drug eruption</td>
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<tr>
<td>• Bullous pemphigoid</td>
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<tr>
<td>• Porphyria cutanea tarda (PCT)</td>
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<tr>
<td>• Erythema multiforme (EM)</td>
</tr>
<tr>
<td>• Smallpox/Vaccinia</td>
</tr>
<tr>
<td>• TEN</td>
</tr>
<tr>
<td>• Second degree sunburn</td>
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</tbody>
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Please Note
**Etiologies**

- **Infectious:** bacterial & viral
- External
- Autoimmune
- Genetic
  - Porphyria cutanea tarda (PCT)
  - Epidermolysis bullosa (EB)
  - Epidermolysis bullosa acquisita (EBA)
- Medication
- Overlap/Multiple etiologies ➔ Hospital consults
- Bullous Erythema multiforme
- Stevens-Johnson syndrome/Eosin Epidermal Necrolysis

**Infections**

- Honey-colored, weeping plaque on the chin with vesiculation on the lower lip.

**Bullous Impetigo**

**Pathogenesis:**

- S. aureus phage propagating strain group II (Pneu SE and 71) ➔ produce exfoliatins A and B (ETA and ETB) ➔ cleaves desmoglein 1 ➔ subcorneal acantholysis

**Clinical features:**

- Children > adults; flaccid bullae + erosions w/ collarette of scale, minimal surrounding erythema + “honey-colored” crust

**Diagnosis:**

- Clinical
- Bacterial culture
- Histology: subcorneal/intragranular acantholysis, neutrophils in blister cavity. Gram(+) cocci

**Subcorneal Pustule DDX on H&E**

- CAT SIPS
- Contagia
- Acropustulosis of infancy
- Transient neonatal pustular melanosis
- Steadman-Wilkinson
- Impetigo
- Pustular psoriasis
- Epiderfoliaceous acnecystic skin syndrome

**Bullous Impetigo: Tx**

**Treatment:**

- **Localized:** topical Mupirocin 2% ointment 1-2 x 5 days [or] retapamulin
- **Widespread:**
  - 1st generation: Cephalexin 500mg – 10 days
  - 2nd/3rd generation: Cefpodoxime 200mg – 10 days
  - 1st generation: Erythromycin 250mg – 10 days
- **Complicated:** IV Ceftriaxone
- Penicillin allergic: Ciprofloxacin or Doxycycline

*NB: erythromycin-resistant staph are prevalent (19-50% of strains) in many regions of the USA*

*If culture returns positive: Ciprofloxacin, Bactrim, Doxycycline or topical Mupirocin (nasal) cream*
**Bullous Impetigo: Clinical Course**

**Clinical Course:**
- Self-resolves in two weeks
- If the patient continues to develop new lesions 3 days beyond starting antibiotics, the veracity of the dx should be reassessed
- Correlate with culture and susceptibility. CHANGE abx as necessary.

**Staph Scalded Skin Syndrome (SSSS)**

**Pathogenesis:**
- Infection by S. aureus phage group II (types 55 and 71) at distant site
- Produce exfoliatoxins A and B (ETA and ETB)
- Exfoliatoxins disseminate via bloodstream
- Widespread cleavage of desmoglein 1 → subcorneal acantholysis

**Clinical features:**
- Children esp those with renal clearance ↓ (low mortality <5%) > adults with ESRD (high mortality >50%)
- Prodrome: fever, skin tenderness, denuded areas on the face lead to characteristic periorificial radial fissuring → generalized flaccid bullae + Nikolsky sign → desquamation continues for up to 1 week, heals without scarring.

**SSSS: Dx**

**Diagnosis:**
- Clinical presentation is usually diagnostic
- Gram staining and cx of blister = Negative, because the process is caused by a systemic toxin (contrast to bullous impetigo)
- Nikolsky sign
- Histology: subcorneal acantholysis, inflammatory cell poor blister cavity

**SSSS: Tx**

**Treatment:**
- Skin care: bland emollients (white petrolatum) to promote re-epithelization
- Abx
  - Mild disease: B-lactamase resistant PCN or 1st generation CSN → Dicloxacillin or Cephalexin
  - Severe disease (or neonates): admission for IV abx

**Clinical Course:**
- Desquamation for 3-5 days
- Lesions heal without sequelae in 1-2 weeks
What medications known to impair renal function should be avoided in pts with SSSS?

- NSAIDs

HY Facts about SSSS

- SAME exfoliatotoxins as bullous impetigo (ETA/ETB) but hematogenously disseminated
- MC site of primary infection
- Kids = nasopharynx or conjunctivae
- Adults = pneumonia or bacteremia

Blistering Distal Dactylitis

- Pathogenesis
  - True or False: Staph aureus is the mc cause of BDD?
    - False: Strep pyogenes > Staph aureus
  - Often, impetigo or other cutaneous infection is present elsewhere.
- Clinical features
  - Children > adults
  - Darkening of the volar fat pad (distal finger > toes) → progresses to vesicle/bullae within one week
- Diagnosis
  - I&D & obtain gram staining or culture

Treatment

- Aspiration + PO Abx
- Also with b/p & drainage massage
- Orals: dicloxacillin, erythromycin, clindamycin
- Exudates erythematous or ulcerated
- Topical abx, avoid occlusive therapy, not recommended
- NB: there is no evidence that aspiration/I&D speeds recovery but it does improve symptomatology

Clinical Course

- After treatment, patient should demonstrate improvement within 72 hours

Important diagnostic clue to differentiate blistering distal dactylitis vs herpetic whitlow?

Unilocular blister = BDD
Multilocular blister = HW
Grouped vesicles/pustules on an erythematous base

Grouped vesicles on an erythematous base in a T3 distribution

Human Herpes Viruses: HSV vs VZV
- HSV = 8 distinct human herpesviruses (HHV-1 to HHV-8) all belong to Herpesviridae family, all are characterized by double-stranded DNA and replicate in host nucleus.
- Herpes simplex Virus = HSV-1 & HSV-2
- Varicella zoster virus = HHV-3

Human Herpes Viruses: HSV
- Recurrent vesicular eruptions occurring in orolabial (classically HSV-1) and genital (classically HSV-2) regions.
- Primary infection: Latency = virus is dormant in sensory (dorsal root) ganglia.
- Recrudescence/recurrence may = symptoms.
- 1° infection: Prodromal period = painful symptom before lesions appear.
- Recurrent infections: generally milder than 1° infections, have 24-hour prodrome of tingling/itch/burning.

Pathogenesis
- Infection can occur without clinical lesions & virus may still be shed.
- HSV can evade host immune system (e.g., ↓expression of CD1a by APCs, ↓TLR signaling).

Diagnosis
- Viral culture: high specificity, low sensitivity, direct fluorescent antibody assays, serology (Western blot = gold standard), PCR (most sensitive/specific), and Tzanck smear.

Treatment
- Orolabial: Oral valacyclovir, topical penciclovir, or topical acyclovir/hydrocortisone combination.
- Use meds w/in first 48 hours = ↓pain/healing time/viral shedding.
- Suppressive daily doses may be given in patients with >5 outbreaks of orolabial/genital HSV per year (also ↓viral shedding).
- May need IV acyclovir in eczema herpeticum, neonatal HSV, or severe HSV in immunosuppressed.
- Foscarnet or cidofovir for acyclovir-resistant HSV (more common in immunosuppressed patients).

Boards Factoid
What is the most common cause of EM minor? - HSV-1

Human Herpes Viruses: VZV
- Causes varicella (chickenpox) and herpes zoster (shingles).
- Primary varicella (chickenpox) = illness associated with chickenpox eruptions; prodrome = rash before skin lesions appear.
- Reactivation (herpes zoster) = painful grouped vesicles on red base in a dermatomal pattern, typically in the thoracic dermatome (most common), can involve >20 lesions outside the dermatome (disseminated disease).

Diagnosis
- Cerebrospinal fluid analysis: increased white blood cells, protein, and reduced glucose.
- Imaging: MRI of brain to identify lesions.

Treatment
- Oral acyclovir for primary varicella.
- Intravenous acyclovir for severe varicella.
- Foscarnet or cidofovir for acyclovir-resistant VZV.

Vaccines
Human Herpes Viruses: VZV

Diagnosis
- Tzanck smear
- PCR (sensitive, fast)
- Viral culture (specific, not sensitive), serology (four-fold increase in IgG titer can retrospectively confirm prior infection), and skin biopsy

Treatment
Primary varicella
- Systemic acyclovir or valacyclovir within 3 days of lesion onset → ↓severity/duration disease
- Primary prevention = varicella vaccination
  - Contraindicated in pregnancy and in immunocompromised patients

Herpes zoster
- Antiviral treatment with acyclovir, famciclovir, or valacyclovir is best given within 72 hours; prednisone helps with acute pain but has no effect on course or development of PHN
- ↓duration of lesions/pain
- ↓rate of postherpetic neuralgia (PHN) in patients >50 years old
- PHN: tricyclic antidepressants (e.g., nortriptyline), gabapentin, 8% capsaicin patch, pregabalin, opioid analgesics, and lidocaine patch

Hand Foot and Mouth Disease (HFMD)

Pathogenesis
- Coxsackie A16 virus
  - Enterovirus A71
  - Echovirus 16

Clinical features
- MC in Children >> Adults
- Outbreaks usually occur from June to October
- An acute, self-limited viral illness characterized by an oral enanthem with accompanying vesicular eruption
- Erythematous macules & vesicles on hands/feet/buttocks and can eventually become more widespread

Transmission
- Fecal-oral & respiratory secretions → highly contagious and commonly transmitted in day care centers, schools, summer camps, and hospitals
- The incubation period for the virus is approximately 3-6 days, symptoms last 7-10 days
- Individuals can shed the virus via GI passage for 4-6 weeks or via the upper respiratory tract for 3 weeks

Diagnosis
- Usually a clinical diagnosis
- Polymerase chain reaction (PCR) can be obtained from vesicular or nasopharyngeal swab
- Serum antibody testing may also be performed
- CBC may reveal leukocytosis

Treatment
- Supportive care & Prevention
  - Hand hygiene
  - Special consideration should be taken by caregivers changing diapers since CV16 and EV71 can be shed in feces for weeks following infection
  - Disinfection of surfaces with secretions or feces is necessary to prevent secondary transmission

What treatment decreases the risk of postherpetic neuralgia?
- Antiviral treatment or prednisone?
  - Antiviral tx

Hand Foot and Mouth Disease (HFMD)

- Diagnosis
  - Usually a clinical diagnosis
  - Polymerase chain reaction (PCR) can be obtained from vesicular or nasopharyngeal swab
  - Serologic antibody testing may also be performed
  - CBC may reveal leukocytosis

- Treatment
  - Supportive care & Prevention
    - Hand hygiene
    - Special consideration should be taken by caregivers changing diapers since CV16 and EV71 can be shed in feces for weeks following infection
    - Disinfection of surfaces with secretions or feces is necessary to prevent secondary transmission

Hand Foot and Mouth Disease (HFMD)

- Pathogenesis
  - Coxsackie A4 virus
  - Coxsackie A6
  - Coxsackie EV71

- Clinical features
  - MC in Children >> Adults
  - Outbreaks usually occur from June to October
  - An acute, self-limited viral illness characterized by an oral enanthem with accompanying vesicular eruption
  - Erythematous macules & vesicles on hands/feet/buttocks and can eventually become more widespread

- Transmission
  - Fecal-oral & respiratory secretions → highly contagious and commonly transmitted in day care centers, schools, summer camps, and hospitals
  - The incubation period for the virus is approximately 3-6 days, symptoms last 7-10 days
  - Individuals can shed the virus via GI passage for 4-6 weeks or via the upper respiratory tract for 3 weeks

- Prevention
  - Hand hygiene
  - Special consideration should be taken by caregivers changing diapers since CV16 and EV71 can be shed in feces for weeks following infection
  - Disinfection of surfaces with secretions or feces is necessary to prevent secondary transmission

- What treatment decreases the risk of postherpetic neuralgia?
  - Antiviral treatment or prednisone?
  - Antiviral tx
Bullous Insect/Arthropod reactions

- Pathogenesis
  - Hypersensitivity reaction to allergens such as venom (bedbugs, fleas), feces (scabies)
  - Common cause of bullous arthropod reactions = bedbugs
- Clinical features
  - Exposed areas of the body typically affected = urticarial plaques & bullae
  - Lesions may be solitary or grouped
- Treatment
  - Antihistamines, supportive care
  - Oral antibiotics if secondary bacterial infection suspected

Bullous Allergic Contact Dermatitis

- Pathogenesis
  - Contact allergens penetrate the skin, cause a delayed cutaneous response
- Clinical features
  - Linear vesicles coalescing into an erythematous plaque in a unilateral distribution
- Clinical Course
  - Self-limiting

The patient rinsed her hair with lime juice in Mexico and subsequently went in the sun.
- The patient developed linear vesicles followed by hyperpigmentation.
Phytophotodermatitis

- Caused by furocoumarins in plants + UVA light (320–400nm) → erythema +/- blistering (24–72hrs post-contact) followed by hyperpigmentation (1 to 2 weeks later)
- 4 MC plant families in the USA
  - Apiaceae/Umbelliferae
  - Rutaceae
  - Moraceae
  - Fabaceae (legumes)

Phytophotodermatitis

- Apioles or Umbelliferae phytophotodermatitis = Ape holding an Umbrella-looking plant to stay protected from sun

Phytophotodermatitis

- Flowers easily identified as they are clustered on a stalk and arise from a single point
- Hogweed (Heracleum), cow parsley, and wild chervil: "strimmer dermatitis" after weed whacking
- Parsley, parsnip, celery, and carrots: "harvester’s dermatitis" in gardeners
- Mnemonic: "Apioles or Umbelliferae phytophotodermatitis = Ape holding an Umbrella-looking plant to stay protected from sun"

Phytophotodermatitis

- Rutaceae
  - Citrus (lemon, lime, grapefruit), rue
  - Common cause in bartenders and spring breakers
  - Citrus bergamia (bergamot orange): causes berloque dermatitis
  - Pelea anisate (Hawaiian leis)
  - Lime beer dermatitis: phytophotodermatitis variant that may be widespread rather than linear, due to aerosolization of lime-beer mixture

Phytophotodermatitis

- Moraceae
  - Fig and fig leaves
  - Mulberry
  - Fabaceae (legumes)
    - Bavachee/scurf pea (used as vitiligo treatment)
    - Balsam of Peru (Myroxylon balsamum, Myroxylon pereirae)
Impetigo-like crusted erosions on an erythematous base in a seborrhoeic distribution \( \rightarrow \) “cornflakes”

Pemphigus Foliaceus

Pathogenesis

Autoantibody: IgG4 to Dsg1

Clinical features

- 2nd mc form of pemphigus
- Exceptions = Brazil, Tunisia and Finland
- Adults >>> children
- Predilection for Head and neck & lacks mucosal involvement
- Recurrent, superficial, often ruptured blisters with a background of erythema and scaling
- \( \rightarrow \) “Impetigo-like crusted erosions on an erythematous base in a seborrhoeic distribution” \( \rightarrow \) “cornflakes”
- Two important variants
  - Fogo selvagem: endemic variant. Highest incidence in areas near rivers rich in black flies (\( \text{Simulium pruinosum} \)); more common in children
  - Senear-Usher syndrome: lupus + PF \( \rightarrow \) malar and seborrhoeic areas

Diagnosis

- Culture, r/o secondary infection (may cause flare)
- Shave/punch of AA
- Perilesional DIF placed in immunofluorescence transport media or normal saline (for periods of <24-48 hrs)
- IIF using guinea pig esophagus as substrate
- R/o causes of drug induced PF

- Thiols: ACE inhibitors (captopril >> enalapril, lisinopril), Penicillamine = common
- Non-thiols: B-lactams, CCBs, BBs, gold, sulfasalazine = rare

Treatment

- PO prednisone (0.5 – 1.0 mg/kg per day) with a SLOW taper.
- Don’t forget Ca and Vit D supplementation, consider DEXA scan and recommend f/u with PCP for osteoporosis monitoring
- While tapering steroid begin steroid spearing agent: mycophenolate mofetil, MTX, azathioprine, dapsone, cyclophosphamide or rituximab

Clinical Course

- Chronic
- In contrast to PV, more benign course but can greatly affect QOL

Intracellular IgG & C3: FT or PV

Corticosteroid Taper: recommended regimen

The goal of tapering is to use a rate of change that will prevent both recurrence activity of the underlying disease and symptoms of cortisol deficiency due to prolonged HPA-suppression. The generally recommended stable decrement of 10 to 20 percent, while accommodating convenience and individual patient response. The dose is tapered by:

- The 10 mg/day every one to two weeks from an initial dose of 60 mg of prednisone or equivalent per day
- Taper by 10 mg every one to two weeks and maintain dose between 10 and 20 mg/day
- 5 mg/day every one to two weeks and maintain dose between 10 and 20 mg/day
- 2.5 mg/day every one to two weeks and maintain dose between 10 and 20 mg/day
- Taper every two to four weeks and maintain dose between 5 and 10 mg/day
- This can be achieved by alternating daily doses, eg. 5 mg on day one and 1 mg on day two.
Calcium & Vit D supplementation if patient taking glucocorticoids for >3 months

- Calcium and vitamin D - We agree with the American College of Rheumatology (ACR) Task Force recommendations, which suggest that all patients taking glucocorticoids 9.8 mg/day or an equivalent duration of 3.0 mg/day should receive a total calcium intake of 1000 to 1200 mg/day and vitamin D use of 800 to 1200 international units daily through either diet and/or supplements [1]. (See “Calcium and vitamin D supplementation in osteoporosis”)

Which two entities, previously discussed, have the same target within the epidermis as Pemphigus foliaceus?

- Dsg1 = PF
- Bullous impetigo
- Staphylococcal scalded skin syndrome

Characteristic sunflower-like arrangement of vesiculopustules

IgA Pemphigus

Pathogenesis
- 2 Subtypes:
  - Intraepidermal Neutrophilic Type:
    - IgA to Desmoglein-1 & 3
    - Characteristic sunflower-like arrangement of vesiculopustules
    - + DIF intercellular IgA staining throughout entire epidermis
    - Suprabasilar pustules → neutrophilic infiltrate confined to the lower epidermis
  - Subcorneal Pustular Dermatosis Type:
    - IgA to Desmocollin-1
    - Mimics Sneddon-Wilkinson clinically and histologically (need DIF/IIF)
    - + DIF intercellular IgA staining in upper epidermis
    - Subcorneal pustules → neutrophilic infiltrate confined to the upper epidermis

Clinical features
- Pruritic vesicles or pustules in an annular/circinate pattern w/ central crusting; MC on axillae, groin; no mucosal involvement
- Ax: IgA gammopathy & possible multiple myeloma

Diagnosis
- Shave/punch of AA + perilesional DIF
- DIF+ in 100%
- IIF+ in 50%

Treatment
- Tx: Dapsone (TOC) (resolution w/in 48hrs).
- Other options: PO corticosteroids & sulfapyridine.

Sneddon-Wilkinson:
classic subcorneal pustular dermatosis

- Variant of IgA pemphigus, analogous to the SPD type. Only difference is DIF/IIF.
- IgA anti-Desmocollin-1
- SPD + DIF
- Sneddon-Wilkinson = - DIF
- Ts: Dapsone
Pemphigus Vulgaris

Pathogenesis:
- MC form of pemphigus in most of world (PV: PF ~3:1)
- M:F: 30:40yo; Jewish 10x incidence
- Associated diseases: myasthenia gravis, thymoma, & AI thyroiditis
- 2 subtypes:
  - Mucosal dominant: IgG to Dsg-3 = mucosal dominant pemphigus
  - Mucocutaneous: IgG to Dsg-1 & Dsg-3 (160kDa, 130kDa) = mucocutaneous pemphigus

Clinical features:
- Oral erosions
- Skin involvement (50%): flaccid vesicles/bullae: widespread denudation may result in death from fluid imbalance or secondary infection
- Nikolsky & Asboe-Hansen signs: heals without scarring

Pemphigus Vulgaris

Diagnosis:
- Histology:
  - Eosinophilic spongiosis (early) → intraepidermal acantholysis (tombstoning of basal layer).
  - Acantholysis down hair follicles and adnexa (Hailey-Hailey ≠ adnexal involvement)
- DIF: most reliable test (~100%) perilesional biopsy
  - Intercellular "chicken wire" IgG (100%) +/- C3, lower epidermis most strongly stained
- IIF:
  - Patient's serum on Monkey Esophagus tests for patient's anti-IgG DSG2 levels correlate with disease activity (useful for monitoring)
  - ELISA: assesses patient's serum for IgG anti-Dsg1 & 3; levels correlate w/ disease activity (useful for monitoring)

Treatment:
- First line: PO steroids + Azathioprine
- TCNs + nicotinamide if mild
- Tx resistant: IVIG, rituximab
- Monitor tx response with IIF or ELISA

Pemphigus Vegetans

- Vegetative variant of PV affecting intertriginous areas (h<del>p</del> & facial): reactive phenomenon to friction; pustules w/multifocal plaques
- 2 subtypes:
  - Neumann: severe, generalized
  - Hallopeau: local, less severe
- Histology: pemphigus vegetans
- DIF/IF/ELISA/Tx same as pemphigus vulgaris
Drug Induced Pemphigus

- IgG anti-DSG 1 & 3
- PF-like presentation (6:1 PF:PV); most commonly induced by Thiol (sulfhydryl-containing drugs)
- MC = Thiol (sulfhydryl) drugs >> Non-thiols

Thiols:
- Penicillamine
- ACE inhibitors (Captopril > enalapril, lisinopril), ARBs

Non-thiols:
- B-lactams, gold, CCB, BBlockers, piroxicam, rifampin

Subepidermal split

Bullous Pemphigoid

Pathogenesis
- MC autoimmune blistering disorder
- IgG autoantibodies against BP180 (Type XVII collagen) a 180kD transmembrane protein, the main pathogenic target is the NC16A domain & BP230 (BPAG1) a 230kD cytoplasmic protein belonging to the plakin family

Clinical features
- MC >60 y/o, M>F
- Non-bullous phase (early): urticarial pruritic plaques
- Bullous phase: tense bullae on the trunk
- Oral involvement 10-30%
- Peripheral eosinophilia (50%)

Clinical Course
- Chronic, may be w/s significant mortality but usually low mortality
- Documented IgG levels or +DIF (linear C3 & IgG) at time of therapy cessation → higher risk of relapse

Diagnosis
- Biopsy for H&E → subepidermal split with eos. DIF = most sensitive for BP with 100% sensitivity
- Linear C3 (n-serrated pattern) & IgG
- Biopsy from perilesional, uninvolved skin
- IIF: 80% linear IgG at BMZ, epidermal or roof on SSS → no correlation with disease activity
- ELISA (80-90% sensitivity): serum test for detecting circulating IgG to BP180 & BP230
- Levels correlate strongly w BP dz activity → useful for monitoring response
- High ELISA levels &/or positive DIF at time of tx cessation → high chance of relapse

Treatment
- First line = 0.5-1mg/kg/d x 1-2 weeks with a 6-9 month taper + steroid sparing agent immunosuppressive (MTX, mycophenolate mofetil, azathioprine or cyclophosphamide)
- Other options
  - TCN + Nicotinamide 500mg TID (mild disease)
  - Dapsone (mucosal predominate BP)
  - Rituximab (recalcitrant cases)
  - IVIG

BP Variants

- Pemphigoid vegetans → plaques in intertriginous areas
- Childhood pemphigoid → acral bullae w/ increase facial/genital involvement
- Pemphigoid nodularis
- Lichen planus pemphigoides → LP-BP overlap
- Pemphigoid gestationis → linear C3 on DIF
- Anti-p200 pemphigoid → often w/s PPO
- Anti-p105 pemphigoid → resembles SJS/TEN
- Drug induced pemphigoid → fat abdomens covered by pemphigoid
Drug induced BP

- **Mnemonic**: “Fat Abdomens Covered By Pemphigoid”
  - Finasteride
  - ACE inhibitors
  - Cephalosporins
  - β-lactams
  - Penicillamine/PD1 inhibitors
  - NSAIDs
  - Gold
  - Sitagliptin/Sulfa (DPP-4 inhibitors)

Serration Patterns in Subepidermal Blistering Diseases

- Ni-serrated Linear DIF = BP
  - Others: p200, anti-LN-332

- Gi-serrated Linear DIF = EBA
  - Others: SSLE

Cicatricial Pemphigoid aka mucous membrane pemphigoid (MMP)

**Pathogenesis**
- Autoreactive IgG abs directed against anchoring filament zone (vs hemidesmosomal plaque in BP)
- 4 subgroups:
  - Ocular MMP = β4 subunit of α6β4 integrin (transmembrane component of hemidesmosome)
  - Anti-BP MMP: Mucosal + skin dz = BPAg2 (distal C-terminal)
  - Anti-epiligrin MMP: strongly a/w underlying solid organ malignancy (1=adenocarcinoma)
  - Brunsting-Perry variant: limited to head/neck, scaring alopecia. NO mucosal involvement.

**Clinical Presentation**
- 60-80yo
  - #1 MC site oral (85%); desquamative gingivitis
  - #2 MC Conjunctiva; symblepharon, trichiasis → blindness
  - Skin (25%): MC scalp/face/neck, upper trunk; erythematous plaques, recurrent blisters/erosions heal w atrophic scars (not seen in BP)

**Associated Dxs: Adenocarcinoma** (Laminin 332/5/anti-epiligrin MMP)

Cicatricial Pemphigoid aka mucous membrane pemphigoid (MMP)

**Diagnosis**
- Biopsy for H&E → subepidermal split with EOS
  - DIF = most reliable test = Linear IgG, IgA, &/or C3 along BMZ (perilesional; more frequently + in oral lesions)
  - IIF: only 20-30% will have detectable circulating Abs

**Treatment**
- Dapsone (1st line for ORAL + CUTANEOUS sx) and Steroids
- Severe/progressive ocular dz: Cyclophosphamide (TOC) + systemic steroids or steroid sparing immunosuppressive (MMF, Azathioprine)
  - “cyclops-phosphamide”
  - IVIG & biologic agents for severe dz
  - Surgical correction of ocular scarring - only AFTER dz controlled medically!
Linear IgA Bullous Dermatosis (LABD)

Pathogenesis
- IgA antibodies against 2 related antigens, both derived from BPAG2
- MAb (Measles virus-attachment protein 1) known to associate with LABD

Clinical features
- Acute or chronic urticaria, urticarial plaques, blisters arranged in annular, polycyclic, or herpetiform pattern
- MC in flexures of lower trunk/thigh/groin/buttocks, & face (kids)
- Childhood Variant (4yo) = Chronic Bullous Disease of Childhood
- Adult onset LABD is usually drug-induced (ave >60yo): MC Vancomycin > PCN/CSN, captopril (>other ACEIs), NSAIDs >phenytoin, sulfonamides >many others (furosemide, lithium)

Diagnosis
- Histology: Early urticarial lesions → neutrophils diffusely lined up along BMZ w/ basal vacuolar change (represents early separation) +/- neutrophilic papillitis; Fully developed bullae → Subepidermal Blister w/ Neutros +/- neutrophilic papillitis.
- CANNOT distinguish from DH on H&E need DIF.
- DIF: Linear IgA +/ -C3 along BMZ
- IIF: + in 65% of cases, Linear IgA, stains epidermal side/roof on SSS

Treatment
- Dapsone (TOC) or Sulfapyridine → rapid response (<72hrs)
- Add PO corticosteroids & immunosuppressants in refractory cases (uncommon)

Clinical course
- Usually spontaneous remission in a few years

DIF Review

<table>
<thead>
<tr>
<th>Disease Antigen Size (kDa)</th>
<th>DIF Salt-Split Skin</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bullous pemphigoid</td>
<td>BPAG1 (plakin) 230</td>
</tr>
<tr>
<td></td>
<td>Linear C3 and IgG along BMZ in &quot;n-serrated&quot; pattern Epidermal</td>
</tr>
<tr>
<td>Pemphigoid gestation</td>
<td>BPAG2 (collagen XVII) 180 Linear C3 &gt; IgG along BMZ Epidermal</td>
</tr>
<tr>
<td>LABD</td>
<td>LAD-1 (120kD cleaved portion of BPAG2) Linear IgA +/ -C3 along BMZ Epidermal (IgA)</td>
</tr>
<tr>
<td></td>
<td>LABD97 (97kD cleaved portion of LAD-1)</td>
</tr>
<tr>
<td>Mucous membrane pemphigoid</td>
<td>BPAG2 (C-terminus) 180 Linear IgG and C3 along BMZ Epidermal (or both sides with stronger staining on epidermal side)</td>
</tr>
<tr>
<td>Ocular -predominant MMP</td>
<td>β4 integrin NA Linear IgG and C3 along BMZ Epidermal</td>
</tr>
<tr>
<td>Anti-epiligrin MMP</td>
<td>Laminin 332 400-440 Linear IgG and C3 along BMZ Dermal</td>
</tr>
<tr>
<td>p200 pemphigoid</td>
<td>Laminin γ1 200 Linear IgG and C3 along BMZ Dermal</td>
</tr>
<tr>
<td>p105 pemphigoid</td>
<td>NA 105 Linear IgG and C3 along BMZ Dermal</td>
</tr>
<tr>
<td>Bullous SLE</td>
<td>Type VII collagen(anchoring fibrils) 290 Granular to linear staining w/multiple reactants (IgG, IgA, IgM, C3) Dermal</td>
</tr>
</tbody>
</table>

Etiologies
- Infectious: bacterial & viral
- External
- Autoimmune
- Genetic
- Porphyria cutanea tarda (PCT)
- Psoriasis vulgaris/bullous (PV/BV)
- Epidermolysis bullosa (EB)
- Epidermolysis bullosa acqua (EBA)
- Medication
- Overlap/Multiple etiologies → hospital consultations
- Bullous Syphilis/Multiforme
- Stevens-Johnson syndrome/Toxic Epidermal Necrolysis
- Porphyria cutanea tarda (PCT)
- Epidermolysis bullosa (EB)
- Epidermolysis bullosa acqua (EBA)

Resources
THANK YOU

Kate Braunlich, DO, PGY4
Morphea in the Pediatric Patient

JOANNA EMILIO DO, PGY-3
SUZANNE SIROTA ROZENBERG, DO, PROGRAM DIRECTOR
ST. JOHN’S EPISCOPAL HOSPITAL

Disclosures
I have no disclosures for this presentation

For today’s lecture:
• Definition, epidemiology, pathogenesis, classifications
• Clinical presentations of plaque and linear variants, diagnosis, complications, & treatment
• Clinical Case

What is morphea?
Also known as localized scleroderma
Fibrosing disorder of skin and subcutaneous tissues
Inflammatory disorder characterized by skin hardening
- Caused by increased collagen density from a combination of immune, genetic, and environmental factors
Systemic sclerosis-severe internal organ involvement, sclerodactyly, Raynaud phenomenon, and nail-fold capillary changes

Epidemiology
Incidence of morphea is 0.4 to 2.7% per 100,000 population
More common in Caucasians and females
Linear morphea has equal sex distribution
No lab markers are indicative of active disease but may correlate with disease extent
90% of children present between the ages of 2 and 14
Linear morphea is the most common form in children, but can have other types
  - Often have a history of other autoimmune diseases

Pathogenesis
Both autoimmune and environmental factors (infection/trauma) lead to inflammation
  - Increase in collagen production and deposition into the skin
Mononuclear cells infiltrate the skin and surrounding blood vessels in early morphea
  - Functional and structural changes to the microvascular system
Long stretches of disease remission are common, but most patients with morphea develop new lesions over time
Clinical Presentation

Initial inflammatory stage: erythematous or violaceous patches or plaques
- Central region becomes white and sclerotic from local edema and increased collagen deposition, while the active borders remain red
- Color changes caused by increase in local temperatures
Later stages: active stage subsides and leaving behind sclerotic plaques that are white or hyperpigmented, skin becomes indurated and bound down
- Overabundant collagen deposition destroys adnexal structures and hair follicles

Five subgroups of morphea-Mayo Clinic Classification

- Circumscribed (plaque) morphea
- Generalized morphea
- Bullous morphea
- Linear morphea
  - En coup de sabre
  - Progressive hemifacial atrophy
  - Linear Limb
- Deep morphea
  - Subcutaneous morphea, morphea profunda, disabling pansclerotic morphea, and eosinophilic fasciitis

Plaque Morphea

- < 3 discrete plaques, usually presents on the trunk in a well-circumscribed, oval shaped plaque
- Superficial or Deep
  - Superficial: involves the epidermis and dermis
  - Deep: dermis and SC tissue, +/- fascia and muscle
- Often affects pressure areas (hips, waist, bra line, and proximal extremities)
- No systemic symptoms

Linear Morphea Variant

- En coup de sabre - 1st described in 1854
  - Usually atrophy of forehead, frontoparietal dura (muscle, bone, and CNS)
  - Systemic symptoms: headaches, OIS or vision changes
  - Scalp: atrophic atrophic plaque of parietal scalp, usually shiny, smooth and often pigmented that may extend to cheeks, nose, and lip

Linear Morphea Variant

- Progressive hemifacial atrophy or Parry-Romberg syndrome
  - Minimal cutaneous change, significant atrophy of fat, muscle, bone, and neural elements
  - Usually following Blaschko lines
  - Mean age: 11
  - Male predominance in females
  - OIS involvement in 20%
  - Ophthalmic involvement in 15%

Linear Morphea Variant

- Linear Limb
  - Unilateral arms or legs +/- muscle atrophy
  - Usually following Blaschko lines
  - Systemic symptoms: limb-length discrepancy, joint contracture
  - Dermis and Subcutaneous tissue

- Most likely to have extracutaneous manifestations with linear morphea
Diagnosis

History and physical examination
Establish extent of skin involvement and signs of disease activity
Look for extracutaneous involvement

Skin biopsy
- Histology shows perivascular infiltrate of lymphocytes, admixed with rare eosinophils and plasma cells in the reticular dermis
- Thinned collagen bundles
- Later stages will have absent inflammatory cells, thickened collagen with atrophic eccrine glands, diminished number of blood vessels, trapped fat in dermis

Complications

In linear morphea: neurologic manifestations such as seizures, headaches, peripheral neuropathy, vascular malformations, brain calcifications, and CNS vasculitis
- Ocular complications: en coup de sabre (uveitis or episcleritis)
- Require screening even in the absence of symptoms

Depression and anxiety are more common
Associated autoimmune diseases reported in 2-5% of children with morphea

Treatment

Most lesions tend to regress spontaneously over 3-5 years leaving residual pigmented or atrophic changes
- Aim to treat active areas of involvement

Physical therapy to prevent contractures

Ocular examination

Few true evidence-based studies regarding treatment; few small retrospective or prospective studies involving MTX, systemic steroids, cyclosporine, and mycophenolate mofetil

Consensus shows to treat for at least 2-3 years allowing for minimum 1 year of disease inactivity before discontinuing. 15-28% will still have recurrence especially with linear lesions and older age of onset

Case 1

HPI: 10 year old Caucasian female presented, with her mother, with a 2 month history of two round red itchy rashes on the right and left abdomen. No color changes since it 1st appeared

PE: 9cm x 6cm round plaque with erythematous borders and slightly hypopigmented center on the right abdomen with a similar but smaller lesion on the left side

In the office: 2mm punch biopsy was done on the right abdomen

DDx: Morphea, Lichen sclerosis, MF, vitiligo, trauma

Pathology: Superficial and deep perivascular and focal predominate lymphocytic dermatitis. No sclerosis. Compatible with inflammatory stage of morphea

- DDx: erythema chronium migrans and less likely deep gyrate erythema or tumid lupus erythematosus. If clinically suspicious, suggest LYME and LE serology labs

Case 1

**Treatment:** Patient was placed on clobetasol ointment BID with calcipotriene ointment 0.005% BID and noted improvement.

**Patient Course:** After 2 months, clobetasol was weaned off but patient had a flare.

**Final recommendation:** calcipotriene BID with clobetasol 1-2 times per week and patient has since done well with no new flares.

References


Thank you!
PERIPHERAL NERVE SHEATH TUMORS

INTRODUCTION

- Uncommon neoplasia of peripheral nerves
- Signs and symptoms due to mass effect on surrounding tissue or direct nerve invasion
- Categorized according to cell type
- Most present as dermal or subcutaneous nodules
- Symptomatic if compresses nerve

Neuroma: Neurofibroma, Schwannoma

NERVE SHEATH ANATOMY

PRETEST

QUESTION

- What immunohistochemical marker is seen in nerve sheath myxoma?
  - S100
  - HMB-45
  - EMA
  - Melan-A

PRETEST

ANSWER

- What immunohistochemical marker is seen in nerve sheath myxoma?
  - S100
  - HMB-45
  - EMA
  - Melan-A

DISCLOSURES

- Nothing to Disclose
CELLULAR MARKERS

- S100, SOX 10 - Neural crest derivatives (Schwann cells, melanocytes)
- Synaptophysin - Presynaptic vesicles (neuronal and neuroendocrine cells)
- Epithelial membrane antigen (EMA) - Epithelial cells (including perineurial cells)
- NF - Neuron cytoskeleton
- CD34 - Hematopoietic cells (fibroblasts)
- Glial fibrillary acidic protein (GFAP) - Non myelinating schwann cells (function as astrocytes in PNS)

SCHWANNOMA

- Proliferation of Schwann cells
- Neurilemoma = Nerve sheath tumor
- Majority are solitary, some associated with NF2
- Most common in large nerve on flexor surfaces > head and neck
- Deep dermis or subcutis
- More frequent in adults, female predilection
- Likely deleterious mutation in NF2 (merlin protein)
- Tumor suppressor, multiple acoustic Schwannomas in neurofibromatosis type 2

SCHWANNOMA

HISTOLOGY

- Encapsulated by perineurium
- Hypercellular tissue (Antoni A-type)
- Proliferation of spindle cells, haphazardly arranged
- Indistinct cytoplasmic contours
- Verocay Body - elongated palisaded nuclei in two parallel lines, alternating with hyaline areas void of nuclei (cytoplasmic processes)
- Hypocellular tissue (Antoni B-type)
- Variable degree of degeneration (cystic, edematous, mucinous, fibrotic, vascular)
- Thin wispy cells
- Possible ancient cell changes - atypia
- + S100, + SOX10, + EMA (capsule), - synaptophysin, + collagen IV

Joshi 2012

SCHWANNOMA

HISTOLOGY

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**Schwannoma**

**Histology**
- Encapsulated by perineurium
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**Neurofibroma**

- Proliferation of Schwann cells triggers proliferation of other components of peripheral nerve (neuromesenchymal tissue)
- Perineurial cells, endoneurial fibroblasts, mast cells, undifferentiated cells
- Residual axons intermixed in tumor, helps differentiate from schwannoma
- Solitary cutaneous neurofibroma - common
  - No gender predilection, more frequent in adults
  - Easily invaginated - 'Buttonhole' sign

**Neurofibromatosis**
- Consider if multiple neurofibromas on exam
- Plexiform neurofibroma highly suggestive (not pathognomonic)
- Involves multiple nerve fascicles
- Only neurofibroma with risk of malignant transformation (~2-13%)
- Plexiform neurofibroma almost always indicative of Neurofibromatosis type 1

**Histology**
- Superficial - non-encapsulated
  - May be in a subcutaneous location
  - Haphazardly arranged Schwann cells with wavy nuclei
  - Fibroblasts form shredded carrot collagen in myxoid stroma
  - Rare cells present
  - Deep - enwrapped by perineurium or epineurium
  - More likely location of plexiform neurofibroma
  - Rarely surrounded by diffuse schwannomatous
  - Rarely scattered axons
+ S100, + SOX10, + EMA, + CD34, - NF
**NEUROFIBROMA**

**HISTOLOGY**
- Superficial - non encapsulated
- Usually in upper dermis
- Haphazardly spindled Schwann cells with wavy nuclei
- Fibroblasts form shredded carrot collagen in myxoid stroma
- Mast cells common
- Deep - encapsulated by perineurium or epineurium
- More likely location of plexiform neurofibroma
- Fascicles surrounded by hyalinized stroma
- Rare scattered axons
- + S100, + SOX10, + EMA, + CD34, + NF

**PERINEUROMA**

**HISTOLOGY**
- Elongated spindle cell with wavy nucleus
- Well circumscribed, cells may be epithelioid
- Soft tissue perineuroma
- Form fascicles, surrounding stroma may be hyalinized or myxoid
- Sclerosing perineuroma
- Form cords, surrounded by thin collagen bundles
- + EMA, S100

**DERMAL NERVE SHEATH MYXOMA**

- Schwann cell differentiation
- Originally though on spectrum with cellular neurothekeoma
- Wide range of ages (5 months - 84 years old)
- Average = 36 years old
- No gender predilection
- Most common dermal sites: fingers, knee, pretibial
- May be found in oral cavity, paranasal sinuses, intraspinal, paravertebral
DERMAL NERVE SHEATH MYXOMA

HISTOLOGY
- Multiple myxoid hypocellular nodules
- Cord / cluster of epitheloid Schwann cells aggregates
- Separated by fibrous connective tissue bands
- Stains with acidic mucopolysaccharides
- + S100, + SOX-10, + GFAP

MALIGNANT PERIPHERAL NERVE SHEATH TUMORS

HISTOLOGY
- Most commonly arise from plexiform neurofibroma (2-13%)
- Half of MPNST arise from plexiform neurofibroma
- Rapid enlargement of neurofibroma
- Most commonly found on extremities and trunk > head and neck
- Sporadic - no gender predilection, most frequent in 40 - 50 years old
- NF1 Associated - More common in males 20 - 40 years old
- Loss of NF1 + tumor suppressors (p53, p16)
- Fibroblasts often form majority of tumor (Perineurial or Endoneurial fibroblasts)

MALIGNANT PERIPHERAL NERVE SHEATH TUMORS

HISTOLOGY
- Proliferation of spindle cells
- Hypercellular
- Intersecting fascicles (‘herring bone’)
- Cells often uniform
- Hyperchromatic nuclei
- Mitoses
- Areas of necrosis
- Partial + S100
- Possible inactivation of NF1 or CDKN2A

TREATMENT
- Observation
- Biopsy may be therapeutic
- Symptomatic - Excision
- Malignant peripheral nerve sheath tumor - Treated as soft tissue sarcoma
  - Wide local excision + adjuvant radiotherapy
- Margins not well defined

POSTTEST

QUESTION
- What histologic characteristic distinguishes NSM from neurothekeoma?
  - Well Circumscribed
  - Multinodular
  - Hypocellularity
  - Fibrous Connective Tissue
What histologic characteristic distinguishes NSM from neurothekeoma?

- Well Circumscribed
- Multinodular
- Hypocellularity
- Fibrous Connective Tissue
Superficial Radiation Therapy Reduces Keloid Recurrences Post-Keloidectomy

Lecture Objectives:
- Review pathogenesis of wound healing
- Differentiate between hypertrophic scar vs keloid
- Discuss current modalities used for keloid treatment
- Discuss how radiation therapy may work on keloids
- Indications, contraindications, and risks of SRT

Hypertrophic vs Keloid:

<table>
<thead>
<tr>
<th>Hypertrophic Scar</th>
<th>Keloid</th>
</tr>
</thead>
<tbody>
<tr>
<td>Border confined to site of injury</td>
<td>Borders extend beyond site of injury</td>
</tr>
<tr>
<td>Develop within 4-8 weeks</td>
<td>Can develop years later</td>
</tr>
<tr>
<td>Asymptomatic</td>
<td>Symptomatic</td>
</tr>
<tr>
<td>May regress over time</td>
<td>Do not regress</td>
</tr>
<tr>
<td>Respond well to treatment with low recurrence rates</td>
<td>High recurrence rates</td>
</tr>
<tr>
<td>Microscopically: hairy pattern, papillary to the epidermis</td>
<td>Microscopically: Hypertrophied collagen in random pattern</td>
</tr>
</tbody>
</table>

The wound healing response:
3 phases
1. Inflammatory
2. Proliferative
3. Remodeling

Hypertrophic vs Keloid:
- Scar: Key features:
  - Fibroblasts with east-west orientation
  - Blood vessels with north-south orientation
  - Loss of elastic tissue

Authors:
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David W. Cleaver, DO
Jonathan L. Cleaver, DO
Lloyd J. Cleaver, DO

- No relevant financial relationships to disclose for each author listed above
Hypertrophic:

Key Features:
- Whorled proliferation of fibroblasts and blood vessels

Keloid:

Key Features:
- Whorled proliferation of fibroblasts and blood vessels
- Central bundles of amorphous “bubble gum” collagen

Keloid pathogenesis:

Impact on quality of life:

• Psycho-social impact
  – Self esteem
  – Poor body image
  – Social isolation

• Physical symptoms
  – Pruritus, pain
  – Limited range of movement

Conventional Therapies:

• Occlusive Dressings
  – Gel sheeting, Serica scar gel

• Compression Therapy
  – Elastic compression, pressure earrings, ACE bandage, elastic bandages

• Steroids
  – Topical
  – Intralosional: concentration of 10-40 mg/mL over 4-6 weeks

• Cryosurgery: 20-60 seconds freeze cycle
  – Contact
  – Intralosional

• Excision
Overview of recurrence rates of Keloids after Excision:

<table>
<thead>
<tr>
<th>Name</th>
<th>Year</th>
<th>Recurrence Rate</th>
</tr>
</thead>
<tbody>
<tr>
<td>Nisen</td>
<td>1942</td>
<td>31%</td>
</tr>
<tr>
<td>Arnold</td>
<td>1959</td>
<td>51%</td>
</tr>
<tr>
<td>Conroy</td>
<td>1960</td>
<td>45%</td>
</tr>
<tr>
<td>Coakley</td>
<td>1961</td>
<td>20%</td>
</tr>
<tr>
<td>Coakley</td>
<td>1972</td>
<td>7%</td>
</tr>
<tr>
<td>Coakley</td>
<td>1974</td>
<td>29%</td>
</tr>
<tr>
<td>Crampton</td>
<td>1974</td>
<td>41%</td>
</tr>
<tr>
<td>Esten</td>
<td>1983</td>
<td>23%</td>
</tr>
<tr>
<td>Mann</td>
<td>1999</td>
<td>43%</td>
</tr>
<tr>
<td>Kim</td>
<td>2003</td>
<td>44%</td>
</tr>
</tbody>
</table>

Emerging Therapies:
- Radiation Therapy*
- Interferon
- 5-Fluorouracil
- Imiquimod
- Tacrolimus
- Sirolimus
- Bleomycin
- Doxorubicin
- Transforming Growth Factor-Beta
- Epidermal Growth Factors
- Verapamil
- Retinoic Acid
- Tamoxifen
- Botulinum Toxin A

Introduction to Superficial radiation therapy (SRT):
- Has more than 100 years of research and development by dermatologists
- Compared to hospital based radiation therapy, SRT is the least expensive form of radiation
- What has it been used for?
  - Has been used to treat non-melanoma skin cancers for more than 100 years
  - BCC/ SCC
- Non-melanoma skin cancer therapy: accumulation of 45-50 Gy over 6-18 fractionations

SRT for Keloids:
- The SRT-100 device received FDA approval in 2013 for the specific treatment of keloid scar
- Indication: recurrent keloids, patients with high risk of recurrence, wider spread, unfavorable location
- Therapy should start within 24 hours of excision with more favorable results when radiation started on the same day after surgical removal
- Penetration of 5 mm depth – entirely targets the skin
- Low-dose (12–16 Gy) adjuvant radiotherapy delivered in three to four fractions, beginning within 24–48 hours following
- Cost of SRT treatments varies for each individual and is dependent on factors such as size and extent of treatment

SRT post keloidectomy outcomes:
- 297 Keloids treated with excision followed by 70-100kV with a 6Gy fractions on post-excision day 1,2,3
  - Recurrence rate at 3 years was 3.0%
- One study used 60kV for chest or 100kV for earlobes and dose of 10 Gy in a single fraction after excision
  - resulted in a probability of relapse of 7% at one year and 10% at 5 years
- 195 keloids treated with excision followed by radiation 50kV or 100kV depending on the site and a total of 10 Gy and 40 Gy dose
  - showed a 91% reduction in itching and a 96% reduction in pain

SRT + keloid protocol:
- 1 Gy = 100 cGy
Radiotherapy and how it works:

- Exact mechanism is unknown
- May act on fibroblasts— including prevention of fibroblasts repopulation after excisions or modulation of humoral or cellular factors that would otherwise recruit and stimulate fibroblasts, or by inhibition of angiogenesis

Contraindications:

- Pregnant patients
- Patients <12 years of age
- Previously radiated sites
- Radio sensitive syndromes
  - Ataxia-telangiectasia
  - Fanconi anemia
- For treatment of keloids in radiosensitive locations
  - Thyroid
  - Parathyroid

Risks:

- Radiation dermatitis— edema, erythema
  - Acute skin side effects are seen in almost all patients first
  - Edema and erythema resolved
- Hypo/hypopigmentation
- Rarely: desquamation, ulceration, necrosis
- Very few studies on the long-term consequences of SRT
- Radiation induced carcinogenesis?
  - In large studies of treatment for keloids, no radiation-induced cancers were reported.
  - A review of the literature by Ogawa et al. in 2009 showed a total of 5 cases of carcinogenic associated with radiation therapy for keloids—fibrosarcoma, breast carcinoma, thyroid carcinomas, and bone carcinomas

Pre and post pictures:


Pre and post pictures:

- https://www.lexingtonplasticsurgeons.com/procedures/scar-keloid-treatment
References:

You Don't Know What You're Missing: 10 Practical Uses for the Wood’s Lamp

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Orange Park Medical Center
Orange Park, FL

Learning Objectives

• Understand wavelengths of light emitted by Wood’s lamp
• Learn tips for proper use of the Wood’s lamp and to make it more accessible
• Learn ten practical dermatologic uses for which a Wood’s lamp can be used

The Wood’s Lamp

• Invented in 1903 by Baltimore physicist, Robert W. Wood.
• Emits long-wave UV radiation, aka “black light”, that is passed through “Wood’s filter” – it emits lights rays between 320 to 400nm with peak at 365nm
• Photons hitting skin are either reflected, scattered, transmitted, or absorbed by target chromophore
• Tissue fluoresces when photons of the Wood’s light wavelengths (340-450nm) are absorbed and longer wavelengths of visible light (e.g. yellow or red) are emitted.

Tips for Wood’s Lamp Use

• Make it accessible
• Room should be perfectly dark, preferably windowless; allow your eyes to become adapted to dark
• Topical creams, lint, soap residues can be gently wiped from area with dry gauze
• Light should be held 4-5” from lesion
• Normal skin is slightly blue
• Oily skin is slightly yellow
• Clothing lint is bright white

10 Practical Uses for the Wood’s Lamp

1. Identifying bacterial colonies: Wood’s lamp is used to visualize bacterial colonies on agar plates under long-wave ultraviolet light.
2. Detecting fungal infections: Wood’s lamp can help identify fungal infections on the skin by observing the fluorescence of fungal elements.
3. Identifying drug reactions: Wood’s lamp can assist in identifying drug reactions by observing the fluorescence of the lesion.
4. Assessing wound healing: Wood’s lamp can be used to assess wound healing by observing the fluorescence of the wound.
5. Detecting tattoo ink: Wood’s lamp can help detect the presence of tattoo ink by observing the fluorescence of the tattoo.
6. Identifying acantholytic dermatoses: Wood’s lamp can help identify acantholytic dermatoses by observing the fluorescence of the lesion.
7. Detecting phototoxic reactions: Wood’s lamp can be used to detect phototoxic reactions by observing the fluorescence of the lesion.
8. Identifying skin tags: Wood’s lamp can help identify skin tags by observing the fluorescence of the lesion.
9. Identifying epidermal nevi: Wood’s lamp can help identify epidermal nevi by observing the fluorescence of the lesion.
10. Identifying subungual debris: Wood’s lamp can be used to identify subungual debris by observing the fluorescence of the nail bed.
#6 Acne (vs Rosacea)
- Cutibacterium acnes produces coproporphyrin leading to orange-red fluorescence of comedones
- Example: 33 year old female comes to clinic with longstanding history of inflammatory papules on the mid-face.
  - You don’t appreciate comedones on exam
  - You grab your Wood’s lamp ...

#8 Hyperpigmented Rashes/Lesions
- Wood’s light is absorbed by melanin, white-leaved pigmented adjacent skin scatters the light, resulting in contrast between tissue with more melanin.
- NEVUS ANEMICUS
  - Hypopigmentation can be epidermal, dermal, or mixed
  - Delineate amount of epidermal pigment (which is usually more responsive to treatment)
  - Epidermal pigmented lesion (PHAKIC) scattered under Wood’s lamp while normal epidermally hypopigmented
  - Wood’s lamp is less helpful for Fitz V and VI patients 2/2 less contrast with surrounding skin
- Melasma
  - Identify clinical markers of lentigo maligna
  - Locate the site of a completely regressed primary cutaneous nevus

#9 Trichomycosis axillaris
- Trichomycosis axillaris is superficial bacterial infection of hair shaft caused by Corynebacterium tenuis
- 46 year old male with axillary malodour x 2 years
- Exam revealed waxy, adherent yellow deposits on hair shafts
- Wood’s lamp revealed green fluorescence
- Patient cured via shaving axillary hair and using 2% sodium fusidate ointment BID

#10 Milia
- Milia are tiny cysts filled with keratin
- At times, they can be difficult to distinguish from syringomas and verruca plana
- Keratin undergoes max excitation at 405 nm and emits fluorescence at 460-500 nm
- Due to bright yellow fluorescence, milia can be distinguished from other papules on the face
- Milia are tiny cysts filled with keratin
- Due to bright yellow fluorescence, milia can be distinguished from other papules on the face

#7 Hypopigmented Lesions
- Can be difficult to assess in lighter skin types
- Vitiligo
  - Depigmented patches are bright blue-white, which contrasts with surrounding normal skin
  - Helps distinguish from psoriasis, alopecia, nevus anemicus, & post-inflammatory hypopigmentation, all of which DO NOT ACCENTUATE with Wood’s lamp
  - Ash leaf macules of tuberous sclerosis
  - Can be difficult to see on light skin types; Wood’s lamp can help detect lesions
- Nevus depigmentosus vs nevus anemicus
  - Nevus depigmentosus, actually hypopigmented, mildly accentuates whereas nevus anemicus does not
#4 – Diagnosis of Porphyria

- Porphyrias result from enzyme deficiencies in biosynthetic heme pathways leading to accumulation of metabolites.
- Porphyria Cutanea Tarda (PCT)
  - Main symptom: photosensitivity; urine turns red/orange upon exposure to sun; darkening with age, 10% HCl
  - Congenital erythropoietic porphyria
  - Slurring of speech, fluorescent urine
  - Porphyrins used as photosensitizer in photodynamic therapy (PDT)
  - Shows fluorescence to ensure even distribution of levulinic acid and soybean lecithin.

#3 – Tinea Versicolor

- Superficial fungal infection caused by Malassezia furfur, which emits a yellow-white or copper-orange fluorescence upon illumination with Wood’s lamp.
- Not only helpful to make diagnosis, but for detecting extent of infection and response to treatment.
- May also help to distinguish Pityrosporum folliculitis from other causes of folliculitis.

#2 – Tinea Capitis

- Dermatophyte infection of scalp.
- 3rd order question: you suspect tinea capitis. You shine your wood’s lamp and you see fluorescence. How does this alter your differential diagnosis?
  - Not only helpful to make diagnosis, but for detecting extent of infection and response to treatment.
  - Superficial fungal infection caused by Trichophyton species, which emits a yellow-white or copper-orange fluorescence.
  - Wood’s lamp to ensure even distribution of levulinic acid and soybean lecithin.

#1 – Erythrasma

- Common, chronic, superficial bacterial infection with Corynebacterium minutissimum.
  - Corynebacterium is a common flora on everyone’s skin, however overgrowth leads to erythrasma.
  - Fluoresces coral red due to production of coproporphyrin III.
  - Why is the distinction of erythrasma from tinea important?
  - Erythrasma responds well to clindamycin or erythromycin; while tinea/candida responds better to topical imidazoles.

10 Practical Uses for the Wood’s Lamp

10. Milia
9. Trichomycosis axillaris
8. Hyperpigmented rashes and lesions
7. Hypopigmented rashes and lesions
6. Acne (vs rosacea)
5. Pseudomonal infections
4. Porphyrinas
3. Tinea versicolor
2. Tinea capitis
1. Erythrasma
Top 10 Non-dermatologic Wood's Lamp Uses

10. Illuminating your blacklight posters in your bedroom in 1993
9. Detecting counterfeit money used at ACOG Casino night, fall meeting 2018
8. Attracting mosquitoes into a bugzapper like samples attracting you all into the exhibit hall.

References

Hidradenitis Suppurativa Update 2019
Ann Lin, D.O. PGY3
Suzanne Rosenburg, D.O. Program Director
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Department of Dermatology

Disclosure
• No financial disclosures

Overview
• What is HS?
• Epidemiology
• Pathogenesis
• Genetic Factors
• Clinical Staging
• Associated Diseases
• Histopathology
• Diagnostic Criteria
• Differential Diagnoses
• Complications
• Treatments
• Updates

Introduction
• Definition of disease
• Acne Inversa
• A chronic inflammatory skin condition caused by follicular occlusion in the folliculopilosebaceous units of the skin.
• MC in the intertriginous areas such as the axillas, groin, perianal, perineal and inframammary regions.

WARNING
The content of this lecture is extremely dry and may cause xerosis.
The use of Vaseline is highly recommended.
Epidemiology
- Less than 1-4%
- Higher prevalence in US population database study
- 1.4 per 100,000
- Onset
  - Puberty – 40YO
  - MC: 20-30 YO
  - F:M: French and US studies
  - Higher in African Americans (US)

Pathogenesis
- ?
- New Evidence show follicle-centered pathology instead of defect in apocrine glands
- Follicular occlusion -> follicular rupture -> associated immune response -> HS clinical presentation

Genetic Factors
- 40% of HS patients have an affected first degree family member
- PSEN1, PSENEN, and NCSTN
- Gamma secretase mutations
- TNF gene polymorphisms
- Further studies needed to elucidate associated pathways

Other Factors
- Mechanical stress
- Obesity
- Hormonal role (androgen excess)
- Smoking
- Bacteria
- Controversial
- More as a complication

Clinical Staging
- Hurley clinical staging – MC used
- Stage I: Single or multiple abscess formation without sinus tracts and cicatrization/scarring
- Stage II: Single or multiple recurrent abscesses with sinus tracts and scarring
- Stage III: Diffuse distribution of lesions or multiple interconnected sinus tracts and abscesses across the entire area

What stage is this?
- Stage III: Diffuse distribution of lesions or multiple interconnected sinus tracts and abscesses across the entire area
Associated Diseases

- Metabolic syndrome
  - Increased co-occurrence
  - DM
  - Obesity
  - Dyslipidemia
  - Hyperglycemia
  - Hypertension
  - Insulin resistance
- These risks factors contribute to higher cardiovascular-associated death → Danish study, HS pts 2 fold greater CV death

- Inflammatory Bowel Disease
  - Crohn disease and ulcerative colitis
  - Multiple studies support this association
  - Gene association in both diseases: SUIT1B1 and SUIT1E1
  - Immune dysregulation and altered microbiota

- Acne Vulgaris
  - More severe and difficult to treat
  - Follicular occlusion tetrad
- PAPASH syndrome: PSTPIP1 gene

Histopathology

- Early features
  - Follicular hyperkeratosis, follicular plugging, follicular dilation and lymphocytic perifolliculitis

- Established lesions
  - Additional features include psoriasiform hyperplasia of the interfollicular epithelium
  - Sinus tracts lined inflammatory infiltrate involving the lower half of the dermis and subcutis

- Chronic lesions
  - With sinus tracts lined by stratified squamous epithelium
  - Granulation tissue with or without foreign body giant cells
  - Destruction of folliculopilosebaceous units
  - Pyogenic arthritis
  - Pyoderma gangrenosum
  - Acne
  - Suppurative hidradenitis

Dermatopathology

- Heavy mixed inflammatory cells in the reticular dermis
  - Extending into the subcutis.

- Abscesses are commonly present and may connect to
  - The skin surface via sinus tracts
  - Granulation tissue and occasional giant cells are sometimes present.
  - Acne scars, fibrosis and destruction of pilosebaceous follicles and sweat glands are evident.
Differential diagnosis
• Follicular pyoderma
• Acne vulgaris
• Intergluteal pilonidal diseases
• Crohn disease
• Granuloma inguinale

Complications
• Strictures and contractures
• Lymphatic obstruction, lymphedema of limbs and genitalia
• Malaise, depression, and suicide
• Anemia, hypoproteinaemia and amyloidosis
• Secondary to long term disease
• Infectious complications
  • Lumbosacral epidural abscesses
  • Sacral bacterial osteomyelitis
• Arthritis
• Squamous cell carcinoma

Treatments
• Oral antibiotics
  • Clindamycin and rifampin combo therapy
    • For pts refractory to tetracycline: Clindamycin 300mg BID, Rifampin 600mg QD for 10 weeks
    • Tetracycline
      • 100mg QD or BID for several months
      • Doxycycline 25-100mg per day
  • Intralesional corticosteroids
  • Punch debridement
    • I&D not performed due to recurrence nature of the disease
    • Topical resorcinol
      • Chemical peel: keratolytic and antiinflammatory
  • Oral retinoids
    • Acitretin 0.56mg/kg per day
    • Isotretinoin 44 mg/day
    • Alitretinoin (Canada)
  • Hormonal therapy
    • Oral contraceptive pills
    • Spironolactone
    • Finasteride
  • Surgery
    • Treatment of nodules and sinus tracts
  • Others:
    • TNF-alpha inhibitors- adalimumab, infliximab

What is new??
• North American Clinical Management Guidelines for HS (July 2019)
  • Data up to December, 1st 2018
  • Recommendation for evaluation, comorbidity screening and procedural treatment options based on strength
  • Overview of evidence-based recommendations for management and treatment based on strength
  • Topical and intralesional therapies
  • Systemic antibiotics
  • Retinoids
  • Systemic immunosuppressants
  • Biologics
  • Pediatric and pregnant patients

Treatments
• Patient's management
  • Avoid skin trauma or friction
  • Loose clothing
  • Smoking cessation
  • Weight management
  • Antiseptic washes
    • Chlorhexidine 4%
North American Clinical Management Guidelines for HS (July 2019)

• Topical and Intralesional Therapies
  • Most are used empirically
  • Clindamycin has been shown to reduce pustules but increase Staph a. resistance
  • ILK 10mg/dl shown to reduce inflamed lesions significantly

• Systemic Antibiotics
  • Some evidence for combo antibiotics
    • Rifampin and clindamycin
    • Rifampin, moxifloxacin, and metronidazole

• Hormonal Therapies
  • Androgen influence in HS has been established
  • Effective therapies in small studies
    • Ethinyl estradiol/noregestrol
    • Ethinyl estradiol
    • Cyproterone
    • Spironolactone 100-150mg QD
    • Metformin 500mg 2-3 times daily
    • Finasteride

• Systemic immunosuppressants
  • Data does not support efficacy
    • Methotrexate
    • Azathioprine
    • Cyclosporine
    • Colchicine
    • Systemic steroids: rapid response but large side effect profile

• Biologics: TNF Inhibitors
  • Adalimumab
    • Phase 3 trials demonstrated efficacy
    • PIONEER 1 and 2 studies showed significant clinical response compared to placebo at 12 weeks
      - 160mg at week 0
      - 80mg at week 2
      - 40mg at week 4, then weekly
    - Infliximab
      • Some evidence
      • Dosing suggestion 5mg/kg and 10mg/kg every 4 to 8 weeks
    • Etanercept
    • Data conflicting
    • Golimumab
      • Limited studies
North American Clinical Management Guidelines for HS (July 2019)

- Biologics
  - IL-1 Inhibitors
    - Anakinra – mixed results in studies
      - Can be considered only after failing TNF inhibitor
  - IL-12/IL-23 Inhibitors
    - Ustekinumab
      - Dosing needed to support efficacy

- Pediatrics and Pregnant Patients
  - Pediatrics
    - Endocrinologic evaluation
      - May be more severe than adult form
  - Pregnancy
    - First line
      - Topical treatments, procedural treatments and lifestyle modification
    - Systemic agents: second line
    - Retinoids and hormones: contraindicated

What's New?

- Spironolactone
  - 25-200mg QD
  - Evidence shows that androgen may play a role in HS
  - Limited efficacy data for antiandrogenic therapies in females HS
  - Spironolactone

- Apremilast - phosphodiesterase 4 inhibitor (JAAD 1/19)
  - 30mg BID
  - First randomized trial in 20 patients for 16 weeks
  - End point: ≥ 50% reduction in total abscess and inflammatory nodule count
  - Generally well tolerated. Common side effects:
    - Common cold
    - Headache
    - Diarrhea
    - Nausea
What’s New

- IL-1 Inhibitors
  - Anakinra
  - Bermekimab
  - Clinical trial for immunotherapy for colorectal cancer and atopic dermatitis
- IL-12/23 Inhibitors
  - Ustekinumab
  - Bermekimab
  - 1064nm Nd:YAG
  - Ongoing randomized controlled trials
  - Secukinumab
  - Bimekizumab - IL17a antibody
  - Guselkumab
  - Adalimumab + surgical interventions
  - IFX-1 - monoclonal anti-human complement factor C5a antibody

Key points

- New treatments include
  - North American Clinical Management Guidelines
  - First systematic review and meta-analysis supporting association between inflammatory bowel disease and HS (July 2019)

Closing Remarks

- HS is a debilitating chronic dermatologic disease that is challenging for physicians and devastating for patients
- More research is needed to find better treatment options for patients
- Important to refer patient to psychiatric counseling and support as it has profound psychosocial consequences

References:

d suppurativa&source=search_result&selectedTitle=2~64&usage_type=default&display_rank=2.

Thank you

- Annlin@ehs.org
An Update on Atypical Mycobacteria

Erin Lowe, DO, PGY-4

DISCLOSURES?
NONE

JOKES?
PLENTY

1999
• Don’t get into strangers’ cars
• Don’t meet people from the internet

2019
• Literally summon strangers from the internet
to get into their car

DIAGNOSIS (skin biopsy, left index finger):

MYCOBACTERIAL INFECTION (ACID FAST ROD-SHAPED ORGANISM) with
associated suppurative and granulomatous inflammation, granulation tissue, foreign body reaction and fibrosis

ACID FAST STAIN confirms the presence of ACID FAST ROD-SHAPED ORGANISM
keeping with a diagnosis of a MYCOBACTERIAL INFECTION

ACID FAST STAINS: Ziehl-Neelsen, Fite, Kinyoun’s
1. Mycobacterium tuberculosis
2. Mycobacterium leprae
3. Atypical mycobacterioses
+/- Nocardia
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### Mycobacterium marinum
- Primary contact
- Aquatic environments
- Single lesion or sporotrichoid lymphocutaneous nodules

### Mycobacterium avium complex
- AIDS patients
- Pulmonary infection m/c
- Disseminates to skin

### M. abscessus, M. chelonae, M. fortuitum = RGM
- Infection after trauma, surgery, or procedure
  - Liposuction
  - Implants
  - Tattoo, Acupuncture
  - Pedicures
  - Botox, Filler
  - CO2 Laser

### Work-up for a suspected Atypical Mycobacteria infection

#### TISSUE STUDIES
- Fresh tissue culture
- AFB-PCR identification

#### LAB STUDIES
- CBC
- CMP
- HIV
- PPD
- QuantiFERON-TB Gold

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**MycoBacteria, culture, with fluorochrome smear**
- **MICRO NUMBER:** 5104696
- **TEST STATUS:** FINAL
- **SPECIMEN SOURCE:** FT. LOWER LEG
- **SPECIMEN QUALITY:** ADEQUATE
- **SMEAR:** No acid fast bacilli seen.
- **RESULT:** No Mycobacterium species isolated after 6 weeks incubation.
**TREATMENT WHILE YOU WAIT?**

- Clarithromycin 500mg PO QD
- +/- a tetracycline

PEARLS

- Atypical mycobacteria are ubiquitous
- They are usually acquired through environmental exposure
- The microorganism is resistant to the standard levels of chlorination, therefore it can be found in tap water
- There have been no reports of spread via person-to-person contact
- You’re in for the long haul
- Get ID and your local TB clinic involved
- Sometimes it’s best to cut these out
PRIMARY CUTANEOUS B-CELL LYMPHOMAS: A PRACTICAL HIGH YIELD REVIEW

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FINANCIAL DISCLOSURES
• There are no financial disclosures for this lecture

T-CELL LYMPHOMAS
- Mycosis fungoides
  - MF variants
  - Foliculotropic MF (not in lecture)
  - Pagetoid reticulosis
  - Granulomatous slack skin
  - Sézary syndrome
  - Adult T-cell leukemia/lymphoma
  - Primary cutaneous CD30+ lymphoproliferative disorders
- Subcutaneous panniculitis like T-cell lymphoma
- Primary cutaneous gamma-delta T-cell lymphoma
- Extranodal NK/T-cell lymphoma, nasal type
- Primary cutaneous T-cell lymphoma, unspecified
- Primary cutaneous angiomatoid eosinophilic CD8+ T-cell lymphoma
- Hepatosplenic T-cell lymphoma
- Primary cutaneous acral CD8+ T-cell lymphoma
- Angioimmunoblastic T-cell lymphoma

T-CELL LYMPHOMAS
- Salivary gland-like T-cell lymphoma
- Primary cutaneous gamma-delta T-cell lymphoma
- Extranodal NK/T-cell lymphoma, nasal type
- Primary cutaneous T-cell lymphoma, unspecified
- Primary cutaneous angiomatoid eosinophilic CD8+ T-cell lymphoma
- Hepatosplenic T-cell lymphoma
- Primary cutaneous acral CD8+ T-cell lymphoma
- Angioimmunoblastic T-cell lymphoma

KEY POINT
• Primary cutaneous lymphomas are malignant lymphomas confined to the skin at presentation only after complete staging procedures
• Important because 6-10% of patients with systemic B cell NHL will develop cutaneous disease at some point in their illness

PRIMARY CUTANEOUS B-CELL LYMPHOMAS:
1. Primary Cutaneous Follicle Center B-Cell Lymphoma
2. Extranodal Marginal Zone Lymphoma of Mucosa-Associated Lymphoid Tissue-MALT lymphoma (aka Primary Cutaneous Marginal Zone B-Cell Lymphoma)
3. Primary Cutaneous Diffuse Large B-Cell Lymphoma, Leg type
4. Intravascular Diffuse Large B-cell Lymphoma
5. Precursor B lymphoblastic Lymphoma: Children/Young adults
6. Primary Cutaneous Large B-Cell Lymphoma, other
DIAGNOSTIC WORK-UP

- CBC with differential and platelet count
- LDH
- Flow cytometry of peripheral blood mononuclear cells
- HIV
- CT thorax/abdomen/pelvis or combined PET/CT. Include neck in palpable lymphadenopathy
- Unilateral bone marrow aspiration biopsy if patient has cytopenia and for all patient prior to initiation of systemic therapy

A QUICK WORD ABOUT CLONALITY

- PCR commonly used to check for clonality
- This can be performed on paraffin-embedded tissue
- Monoclonality does not equal malignancy. It has to take it in the neoplastic context. Also the absence of clonality does not exclude the presence of malignancy.
- Monoclonal population can identified when one or two dominant peaks substantially above that of the next highest background peak.

CASE #1

A 75-year-old male presents with pink-orange nodule with telangiectasias located on forehead

PRIMARY CUTANEOUS FOLLICULAR CENTER LYMPHOMA

- Solitary or grouped plaques and tumors preferably located on scalp/forehead or on the trunk (uncommon on legs)
- Tumor composed of neoplastic follicle center cells
- Predominance of large centrocytes (large cleaved cells) admixed with variable numbers of centroblasts (large, noncleaved cells with prominent nucleoli)
- Histopathology patterns: follicular, follicular and diffuse, or diffuse growth pattern
- B symptoms rare
- Lesions progress slowly. Indolent clinical course. Dissemination to extracutaneous sites uncommon (five-year survival rate at 95%)

Tx:
- Solitary lesions or lesions within one radiation field: radiation vs. surgical excision
- Extensive disease: rituximab
CASE #2

58-year-old male presenting with two well-circumscribed, erythematous nodules on the shoulder.

PRIMARY CUTANEOUS MARGINAL ZONE-MALT LYMPHOMA (PCMZL)

- Primary cutaneous immunocytoma/primary cutaneous plasmacytoma are now grouped under PCMZL
- Look for recurrent pink-violet to red-brown papules, plaques, or nodules
- Lesions tend to occur superannually over lower extremity
- B symptoms not present
- 5-year survival of 98-100%

Tx:
- Solitary lesion/localized lesions: radiation therapy vs. surgical excision
- Asymptomatic multifocal disease: initial observation period
- Symptomatic multifocal disease: symptomatic lesions with IL triamcinolone, low dose radiation, or surgical excision rather than chemotherapy due to indolent nature of these tumors
- There is an association with Borrelia species infection in European population: consider PCR testing and antibiotic therapy?

CASE #3

A 78-year-old female with ulcerated red nodule on lower ankle presents to clinic.

PRIMARY CUTANEOUS DIFFUSE LARGE B-CELL LYMPHOMA, LEG TYPE

- Think about this when you see solitary or clustered, erythematous to red-brown or blue nodules/tumors in elderly women
- In 30% of patients tumors with similar morphologic and phenotypic features can arise in sites other than lower extremities, but still called Diffuse Large B-Cell Lymphoma, Leg type
- Unlike other cutaneous B-cell lymphomas, these tumors commonly disseminate to extracutaneous sites
- 5-year survival of 40-50%

Tx:
- R-CHOP followed by involved field radiation therapy
CASE #4

80 y/o male presents with indurated violaceous plaques on the thigh mimicking panniculitis

INTRAVASCULAR DIFFUSE LARGE B-CELL LYMPHOMA

- Rare malignant proliferation of large B lymphocytes within blood vessels
- Most often systemic involvement (including CNS) from the onset and B symptoms common
- Look for indurated, erythematous or violaceous patches and plaques on trunk and legs with prominent telangiectasias can look like panniculitis or vascular tumor
- Poor prognosis, aggressive course
- Tx: R-CHOP systemic chemotherapy
Interesting Cases from the Big Apple

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Program Director: Dr. Suzanne-Sirota-Rozenberg

I have no disclosures or conflicts of interest

Case #1

HPI: 22 y/o Caucasian Female presented to our clinic complaining of an enlarging non-healing ulcer on the left wrist for about 5 months. She stated that the lesion initially started as a bump. She denied any previous trauma to the area.

ROS: Negative

PMHx: ADHD

Meds: Concerta

Allergies: NKDA

SocialHx: denied illicit drug use, tobacco use and sexual activity.

3.7cm ulcer with a rolled telangiectatic border and central crust/eschar

The patient had been seen by a PA at another dermatology clinic for a month. She was treated with intralesional kenalog, cryotherapy and topical sylvadene cream. The patient reported no improvement in the lesion.
An astute co-resident, who had coincidentally heard a lecture by an infectious disease specialist 1 week prior suggested asking a thorough travel history.

Travel history was investigated... AND the patient reported traveling to Israel 7 months prior.

A shave biopsy was done to rule out Cutaneous Leishmaniasis or other possible infectious etiologies.

Biopsy results:
"Pseudoepitheliomatous hyperplasia and granulomatous dermatitis with neutrophils, plasma cells and parasitized histiocytes consistent with Leishmaniasis."

According to the CDC:
Leishmaniasis should have a specimen sent to Atlanta, Georgia for additional testing and culture in special media (if possible) that can be sent out to the clinician prior to biopsy if requested.

Additional Information can be found on the CDC website.

Luckily for us, the infectious disease specialist who gave the lecture was a resident of NYC who practiced close by.

The patient was sent to the ID specialist, a second biopsy was sent to the CDC in Atlanta, Georgia; confirming the diagnosis as well as the species as Leishmania infantum.
Due to the localized nature and lack of systemic symptoms, the decision was made to treat the patient with topical preparation of paromomycin 15% and gentamicin 0.5% cream BID.

Over the span of 3 months, the lesion slowly resolved.
Cutaneous Leishmaniasis

Begins as a small papule at the site of inoculation that slowly enlarges over the span of a few weeks to months into a nodule or plaque. The lesions can ulcerate and form a rolled border or become verrucous.

Exposed sites such as arms, legs, neck and face are the most commonly involved.

Lesions are usually solitary, but maybe multiple with satellite or sporotrichoid spread.

Differential Diagnosis includes:
- BCC/SCC
- Arterial, Venous, Diabetic, Pressure ulcers
- Pyoderma Gangrenosum
- Vasculitis
- NLD
- Other infectious causes

Treatment of cutaneous Leishmaniasis depends on severity. Some lesions may spontaneously resolve with scarring.
- Oral medications include: Sodium stibogluconate, Pentamidine, Fluconazole, Liposomal amphotericin B.
- Topical therapies include: Paromomycin and MBC, Gentamicin, Gentamicin cream.
- Other: Cryotherapy, Heat therapy, PDT.

This case teaches us...
- To keep a wide differential diagnosis for a non-healing ulcer.
- Not every ulcer with a rolled border is a BCC.
- Yet again proves the most cliché lesson we have all heard since medical school: An extensive history is VERY IMPORTANT!

Case #2

HPI: A 32 y/o Caucasian male presented to clinic complaining of a 3 month history of new onset “red itchy spots.” He stated that they were increasing in number and occasionally had a burning sensation to them.

ROS: Negative for systemic symptoms. Positive pruritus.

PMHs: Negative

Meds: Negative

Allergies: NKDA

Social Hx: Denied illicit drug, tobacco use
Multiple scattered blanchable red to pink telangiectatic macules of varying sizes on the trunk. The remainder of the skin exam was normal.

Two 4mm punch biopsies were taken from the right scapula and lower back.

The patient was recommended zyrtec, allegra and a topical steroid to alleviate symptoms until follow up.

Dilated capillaries within the superficial dermis and sparse perivascular lymphocytic infiltrate with scattered mast cells.

Leder Stain utilizing chloroacetate esterase. Mast cells stain red.
After the biopsy results, the patient was sent for additional testing. CBC, CMP, TFTs, and Total tryptase levels. All results returned within normal limits.

Genetic testing was discussed, but the patient refused due to lack of systemic symptoms.

The constellation of symptoms, signs and testing suggested a diagnosis of Telangiectasia Macularis Eruptiva Persistans (TMEP).

The patient was counseled on avoiding triggers and continued on antihistamines with topical steroids on an as needed basis.

The lesions persisted but the pruritus subsided.

The patient was lost to follow up.

Mastocytosis

Mast cells are derived from pluripotent CD34+ precursors in the bone marrow. They also express the tyrosine kinase receptor KIT (CD 117). Alteration in KIT structure and activity are central to the pathogenesis of Mastocytosis.

Somatic mutations in KIT involving codon 816 represent the most common abnormality, seen in about 40% of cases. The result is a substitution of Aspartic acid with Valine, leading to constitutive activation.

Mastocytosis

The group disorders encompassing Mastocytosis can be as systemic or cutaneous.

Cutaneous variants include – Urticaria Pigmentosa, Nodular Mastocytoma, Diffuse cutaneous mastocytosis, Mastocytoma, and TMEP.

Onset can range from birth to adulthood. Childhood onset typically has cutaneous manifestations that resolve spontaneously, whereas adult onset disease is systemic and has a chronic course.
Urticaria Pigmentosa in a child can present as scattered, clustered, or confluent macules. There are also papular and papulonodular variants.

Diffuse Mastocytosis can present with blistering and erosions, especially in infants.
Telangiectasis Macularis Eruptiva Persitans

TMEP is a very rare cutaneous variant of Mastocytosis.
Most commonly presents in adulthood as symmetric red to brown telangectatic macules between 2-6mm in diameter on the trunk and proximal extremities.
Palms and soles are spared. Lesions are pruritic. Darier’s sign is commonly absent in this form.
Tryptase levels are usually normal, however if elevated can be a sign of possible systemic disease.
Systemic symptoms are rare, although a few cases have been reported.

DDx of TMEP

- Nevus telangectaticus
- CREST Syndrome
- Telangiectasia secondary to another cause (liver disease or hyperestrogen states)
- Freckles
- Nevi
- Rosacea
- Generalized essential telangiectasias
- Blotous impetigo

Evaluation and Management

- Ask and check for constitutional and systemic symptoms
- Check for lymphadenopathy and hepatosplenomagaly
- Additional testing (age dependent) - CBC, CMP, Tryptase, KIT gene analysis.
- If any of the above are positive, consider additional testing.
Evaluation and Management

There is no cure for mastocytosis, management is based on alleviating cutaneous and systemic symptoms. If there are no symptoms, no treatment is needed.

Avoidance of potential mast cell stimuli is key:
- Physical triggers such as friction, exercise, heat or cold
- Dietary triggers such as hot beverages, spicy foods, alcohol
- Medications: Aspirin, NSAIDs, Narcotics/Pain Killers, Anticholinergics, Polymixin B etc.

Evaluation and Management

Localized Therapy:
- Topical steroids, Topical Calcineurin inhibitors, Intralesional Steroids

Systemic Therapy
- Antihistamines, Cromolyn, Omalizumab, PUVA, NBUVB, Steroids,
- Patients with systemic symptoms should be given EPI-PEN in case of anaphylaxis

THANK YOU FOR YOUR TIME!
PRINCIPLES OF ELECTROSURGERY
Muneeb Shah, DO PGY-2
Campbell University
Sampson Regional Medical Center

OUTLINE
Definitions
Mechanism
Clinical uses
Cautions
Complications

DISCLOSURES
I have no financial disclosures that would be a potential conflict of interest with this presentation.

Electrocautery
• Not electrosurgery
• A metal wire is heated by resistance to flow of direct current
• The tip of the wire is hot
• Advantages:
  • Does not interfere with pacemakers / AICDs
  • Can achieve hemostasis in a wet field
• Disadvantages:
  • May lead to third-degree burns

Electrosurgery (synonym: radiofrequency surgery)
• High-frequency alternating currents passed via a cold tipped electrode
• Neuromuscular stimulation becomes negligible as frequency increases
• Tissue resistance to the passage of current converts electrical energy into heat
• Examples include electrocoagulation, electrodessication, electrosection, and electrofulguration

Electrosurgical unit (ESU)
• Electrosurgical unit (ESU) is the source of electrosurgical current
• The ESU takes a frequency of 60 Hz (standard outlet output) and converts it to over 300 kHz

CIRCUIT

Circuit is composed of:
- Generator
- Active electrode
- Patient
- Return electrode

Tissue provides resistance or impedance, generating heat.

-POLAR AND -TERMINAL

Monopolar: Electrode with 1 tip
- Bipolar: Electrode with 2 tips, active electrode and return electrode
- Monoterminal: 1 electrode, no grounding electrode
- Most frequently used in dermatology
- Biterminal: 2 electrodes are used, treating electrode and grounding electrode

OUTLINE

Definitions
Mechanism
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CURRENT WAVEFORMS

Current can be continuous or discontinuous
- Continuous current
  - Cutting mode produces heat very rapidly leading to vaporization
- Discontinuous current
  - "On" time is reduced
  - Instead of vaporization, a coagulum is produced

TISSUE EFFECT

Tissue effect (e.g., coagulation, cutting) is determined by the rate at which heat is produced
- High heat produced rapidly → Vaporization
- Low heat produced slowly → Coagulation
- Any waveform can accomplish both tasks

Electrocoagulation → Slow heating below the boiling point → Thermal denaturation of blood products
Electrosection → Slow heating above the boiling point → Tissue drying
Electrosurgery (cutting) → Rapid heating above the boiling point → Explosive vaporization of water content in tissue and tissue fragmentation
**ELECTROFULGURATION**

- High voltage, Low amperage
- Monoterminal circuit; Monopolar electrode
- Discontinuous, damped waveform is applied
- An electrical discharge arc (spark) bridges the gap of air between the electrode and the tissue
- Each spark acts as a very fine electrode
- Allows for rapid coagulation over a larger area, when compared to contact electrocoagulation
- Tissue destruction and coagulation is limited to superficial layer of tissue due to surface carbonization

**ELECTRODESSICATION**

- High voltage, Low amperage
- Monoterminal circuit; Monopolar electrode
- Discontinuous, damped waveform is applied
- Electrode is in direct contact with the tissue causing superficial ablation
- Less heat is generated and no cutting effect occurs
- Tissue is heated until the stage of tissue drying
- A popping sound will occur with desiccation

**ELECTROCOAGULATION**

- Low voltage, High amperage
- Biterminal circuit; Monopolar or bipolar electrode
- Damped waveform is applied
- Electrode is brought into direct contact with the tissue being treated
- Slow cellular heating leads to fluid evaporation, protein denaturation, and coagulation
- Higher amperage allows current to penetrate deeper than electrodesiccation

**ELECTROSECTION**

- Low voltage, High amperage
- Biterminal circuit; Monopolar electrode
- Continuous, undamped waveform produces a pure cutting effect
- Slightly damped, blended current can achieve cutting and hemostasis at the same time
- Sudden increase in temperature above the boiling point of water leads to explosive vaporization of water content in tissue and tissue fragmentation
- Useful for achieving relatively bloodless excisions

**OUTLINE**

- Definitions
- Mechanism
- Clinical uses
- Cautions
- Complications

**SUMMARY**

<table>
<thead>
<tr>
<th>Method</th>
<th>Voltage</th>
<th>Amperage</th>
<th>Waveform</th>
<th>Circuit</th>
</tr>
</thead>
<tbody>
<tr>
<td>Electrofulguration</td>
<td>High</td>
<td>Low</td>
<td>Discontinuous</td>
<td>Monopolar</td>
</tr>
<tr>
<td>Electrodesection</td>
<td>High</td>
<td>Low</td>
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<td>Monopolar</td>
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<tr>
<td>Electrocoagulation</td>
<td>Low</td>
<td>High</td>
<td>Variable</td>
<td>Variable</td>
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<tr>
<td>Electrosection</td>
<td>Low</td>
<td>High</td>
<td>Damped</td>
<td>Biterminal</td>
</tr>
</tbody>
</table>

ELECTRODESSICATION AND CURETTAGE

Electrosurgery and Curettage is useful for treating superficial malignancies:
- Preferred for treating small, uncomplicated primary basal cell carcinoma and squamous cell carcinoma
- Provides 90-95% cure rates

Area is scraped with a curette in all directions and then charred with the electrosurgery device
- Can be repeated 2 or more times to remove any residual tumor

Wounds are allowed to heal by secondary intention
Sequela should be anticipated and discussed with the patient prior to the procedure

OUTLINE

- Definitions
- Mechanism
- Clinical uses
- Cautions
- Complications

CAUTIONS

AICDs/Pacemakers
- Can theoretically lead to skipped beats, reprogramming of a pacemaker, or firing of an ICD
- To minimize risk:
  - Electrocautery: No risk
  - Bipolar forceps: Minimizes risk
  - Use short bursts of energy (< 5 seconds)
  - Avoid cutting currents (highest risk with electrosection)
  - Avoid use on skin around device
  - Hold magnet over device to avoid electrical interference


COMPLICATIONS

Fire or explosion
- Alcohol, oxygen, and bowel gases are flammable
- Note: Aluminum chloride solutions contain over 90% alcohol

Thermoelectric burns
- Burns can occur where the current exits the patient’s body
- Keep metal objects away from the patient’s body (E.g., ECG electrodes)

Microorganism transmission
- Can be transmitted by the electrode or smoke plume inhalation
- Use a new sterile tip for each patient
- Use an N95 mask and smoke evacuation system


RISKS OF SURGICAL SMOKE

Surgical smoke is comprised of 93% water and 5% particulate matter
- Particulate filters are effective for all sizes of particles except for smoke creation
- Particles less than 2 micrometers can deposit in the alveoli

Transmission of infection
- HPV, HIV, Staphylococcus, Corynebacterium, Haemophilus

Risk of mutagenesis
- Chronic inflammation
- Transmission of HPV infection
- Visible cancer cells in smoke


MINIMIZING RISK OF SURGICAL SMOKE

- High filtration masks (e.g., N95)
- Smoke evacuation system


May 2017

Awareness of Surgical Smoke Risks and Assessment of Safety Practices During Electrosurgery Among US Dermatology Residents

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Author Information

Upcoming Meetings:

2020 AOCD Spring Meeting
Hilton West Palm Beach
West Palm Beach, FL
February 17 - February 22

2020 AOCD Spring Cosmetic Workshop
Westin Detroit Metropolitan Airpor Hotel
Detroit, MI
April 25-26

2020 AOCD Fall Meeting
Hyatt Centric Chicago Magnificent Mile
Chicago, IL
October 8 - October 11

2021 AOCD Spring Meeting
Hilton West Palm Beach
West Palm Beach, FL
February 22 - February 27

2021 AOCD Fall Meeting
Westin Denver Downtown
Denver, CO
October 7 - October 10