Cutaneous Venous Hypertension

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Disclosure Information
AOCD
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• I have the following financial relationships to disclose:
  • Vein Experts
  • Dermaka, LLC
Cutaneous Venous Hypertension

• The manifestations of cutaneous venous hypertension range from:
  – Spider veins
  – Varicose veins
  – Stasis dermatitis
  – Atrophic blanche
  – Venous ulceration

• Most patients seek consultation for spider veins
Cutaneous Venous Hypertension

- Most spider veins are related to an area of venous hypertension
- Reticular veins are the final pathway for transmission of venous hypertension in the majority of patients but not all
- Volume overload in pregnancy is the main etiology of reticular vein dysfunction
Cutaneous Venous Hypertension

- However, reticular veins are usually connected to a deeper source of pathology.
- The branches of the reticular complex in the majority of clinical situations, are the final pathway leading to cutaneous telangiectasia.
Cutaneous Venous Hypertension

• The GSV, SSV, AAGSV, and Thigh Extension Branch if incompetent may transmit abnormal pressure to the skin via an incompetent branch that may or may not connect to a reticular vein

• Refluxing perforators may also transmit abnormal pressure to the skin resulting in cutaneous venous dilatation (Spider Veins)
Cutaneous Venous Hypertension

• Conversely, a refluxing branch may arise from a normal truncal vessel
• This is not an uncommon occurrence
• In this case, the truncal vessel was not treated but, only the refluxing vessel that will be discussed later in the presentations
Cutaneous Venous Hypertension

• It is imperative to treat the etiology of the cutaneous manifestation, or one of the following events may happen:
  – Non-Clearance
  – Recurrence
  – Angiogenesis
Cutaneous Venous Hypertension

• The US is indispensable in diagnosing deeper pathology. Use the US at 2 cm depth to trace reticulars to origin of hypertension

• **All pathologic pathways will reflux**

• Skin illumination only allows visualization to a depth of 1 mm but, is helpful as a mapping aide for reticular veins

• Only an US can determine pathology of the reticular vein
Cutaneous Venous Hypertension

• The etiology of cutaneous venous hypertension must always be alleviated at the time of spider vein treatment
Theories of Spider Vein Formation

• Sommer A, et al, described the O2 & CO2 difference between red and blue telangiectasia

• Their conclusion is that all telangiectasia are assumed to be in the capillary bed

• This would involve arterioles in continuity with dilated venous structures

• We do not see this on our histological exams

(Sommer, 1997)
Theories of Spider Vein Formation

- However, our biopsy specimens are limited to 1.5 mm diameter
- Another theory is the ‘Microshunt histology theory’
- Bihari et al, described a theory that many of the telangiectasia are associated with an AV microshunt
- The specimens for study were 2.5 cm by 1.5 cm

(Bihari, 1999)
Theories of Spider Vein Formation

• In a greater percentage of their patients using continuous wave doppler US they identified arteriole pulsations over spider telangiectasia.

• This is only subjective in that arterial pulsations can be heard transmitted from varying depths.

• In over 500 patients, we have never seen pulsatile flow using color flow doppler.
Theories of Spider Vein Formation

• However, we have heard multiple transmitted arterial signals that could not be accurately located

• When the etiology of the signals could be located it was usually at a considerable distance below the subdermal layer
Theories of Spider Vein Formation

• Mariani et al, 2000, proposed that all telangiectasia are related to a pathologic perforating vein located beneath the spider vein complex

• In essence, the authors are correct that there is a perforating vein but this is a branch that comes from a reticular vein and not a deeper source

(Mariani, 2000)
Theories of Spider Vein Formation

• Another interesting point is that the authors report in the 3-year follow-up, there were new spider telangiectasia in 59% of cases
• Only 5% recurred in the previously treated area

(Mariani, 2000)
Why Do Spider Veins Occur?

- What we know based on our clinical and histological studies is that spider veins are secondary to high venous pressure.
- The effect of high pressure causes dilatation of the smooth vessel wall of the venules in the reticular dermis.
- In all specimens examined the one constant finding in a spider telangiectasia is vessel wall hypertrophy.
Why Do Spider Veins Occur?

• This pressure must be transmitted from a distal source

• We know from US exams in over 500 patients with spider telangiectasia, that all reticular veins associated with spider telangiectasia demonstrate reflux
Why Do Spider Veins Occur?

• Conversely, we know that most spider veins are associated with a branch from a refluxing reticular vein

• I have never seen a valve in a reticular vein, although valves have been mentioned in the literature but without evidence

• On US, there is never segmental dilatation of reticular veins which would occur if a valve was present
Why Do Spider Veins Occur?

• Based on US and histological studies of branches and perforators in association with reticular veins, there is always an abnormal valve

• So what occurs is the following:

• For whatever reason; volume overload from pregnancy, genetic induced reflux, etc...there occurs transmission of high venous pressure to the reticular vein
Why Do Spider Veins Occur?

• The reticular veins are conduits of flow and pressure
• Due to incompetent valves in the draining branches, perforators, or volume overload from increased flow, there is a buildup of pressure in the reticular system
• This pressure is transmitted to reticular branches
Video: US of Telangiectasia
Viedo: Spider Vein Complex
Flow Characteristics

• A simplistic statement of fluid mechanics follows:

• According to Ohm’s Law, as applies to flow--
  \[ F(\text{blood flow}) = \frac{\Delta P}{R} = \frac{P_a-P_v}{R} \]

• Since flow and resistance are reciprocally related an increase in resistance decreases flow at any given \( \Delta P \)

• Also, an increase in resistance increases \( \Delta P \)
Flow Characteristics

• With reflux, increase flow occurs into the vessel as well as increased outflow resistance

• The response of the vessel wall to decreased outflow or increased inflow is written as Laplace Law

• \( T = \frac{F}{L} = \Delta P \times R \)

• Simply stated, to lower the intravessel pressure (in this case a vein) the vessel must dilate
Why Do Spider Veins Occur?

• The length of a spider vein is proportional to the amount of pressure it receives from the subcutaneous circulation
Case Study: Spider Veins From an Incompetent Branch of the GSV

(Bush, 2015)
Case Study: Spider Veins From an Incompetent Branch of the GSV

(Bush, 2015)
Case Study: Perforating Branch From Vein of Giacomini

(Bush, 2015)
Case Study: Perforating Branch From Vein of Giacomini

(Bush, 2015)
Unloading Cutaneous Venous Hypertension

- 4 Steps to Treating Spider Telangiectasia
- Unload the cutaneous venous hypertension
- Treat the spider vein
- Assess collateral flow
- Minimize sequelae
Unloading Cutaneous Venous Hypertension

- Identify the source
- Use US
- Trace from complex ➔ reticular ➔ source
  (Perforator, branch, GSV, AAGSV or SSV)
Unload the Cutaneous Venous Hypertension

• Surgical – Punch or Phlebectomy
• Chemical – Foam sclerotherapy – USGS or retrograde injection from complex
• Combination of chemical and surgical
Unloading the Cutaneous Hypertension With a Punch Biopsy

- A punch biopsy disconnects the cutaneous telangiectasia
- Also, the branch feeding the cutaneous pathology may also be removed at the same time

(Bush, 2015)
Post Treatment 2 Weeks of Lateral Reticular Complex

(Bush, 2015)
Ultrasound Spider Complex

- The following two videos will demonstrate the actual appearance of a spider complex by ultrasound.
- In this video, it was possible to see the branch from the incompetent vein that gives rise to the spider complex.
- There is usually a branch from the underlying reticular vein that gives rise to spider complexes.
Conclusion

• There is always a source for spider varicosities
• The source may be the GSV, AAGSV, SSV or a perforator
• Find the source and treat with either foam sclerotherapy or phlebectomy (In some cases, thermal ablation)
• Find the end point and unload the spider complex at this level (Where the spider vein branches out or begins)
Unloading the Cutaneous Venous Hypertension

- The white arrow points to the origin of the spider complex – Where the branch of the reticular becomes superficial
- A punch biopsy at this location unloads the cutaneous hypertension & interrupts the spider telangiectasia

(Bush, 2015)
Conclusion

• Disconnect collateral flow if present
• Treat the spider veins with the appropriate concentration
• If using foam, use very dilute concentrations for the spider veins and inform patient that staining could be present for a few months
References


Evaluation of Clinical and Histological Findings Using Varying Sclerosant Concentrations for the Treatment of Spider Telangiectasia

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Title: Evaluation of clinical and histological findings using varying sclerosant concentrations for the treatment of spider telangiectasia.

Authors

Objectives:

1. Evaluate histological changes with varying concentrations of sclerosants
2. Evaluate clinical responses to varying sclerosant concentrations
3. Determine the ideal sclerosant solution and concentration for spider telangiectasia

Methods: In 2014-2015, a clinical research study was completed on 22 patients. The participants were evaluated for histological changes after using varying concentrations of sclerosant solutions. Histological evaluation focused on inter-luminal and vessel wall alterations. In a separate group of 18 patients, clinical findings of 12 patients after sclerotherapy using Sotradecol (sodium tetradecyl sulphate) 0.05%, 0.1%, and 0.2% were evaluated for vessel clearance and post sclerotherapy sequelae. In six patients treated with Asclera (Polidocanol), 0.25% and 0.5%, clinical response was evaluated.

Results: Histology revealed changes in proportion to sclerosant concentration. Endothelial cell destruction: Sotradecol 0.05% (40%), 0.1% - 0.2% (100%). Polidocanol: 0.25% (50%), 0.5% (90%). Subintimal changes negative for Sotradecol 0.05% and Polidocanol 0.25% and minimal for 0.5%. Vessel wall alteration including fibrin deposition was only present in Sotradecol 0.1% and 0.2%. Intimal thrombosis and inflammatory cells only present in Sotradecol 0.2%.

Reticular veins measuring 2-3 mm demonstrated identical histological changes with both Sotradecol foam 0.1% and 0.2%. Clinically, Sotradecol 0.1% demonstrated 90% vessel clearance with 5% post sclerotherapy sequelae. Sotradecol 0.2%, revealed 90% vessel clearance with 50% post sclerotherapy sequelae. Polidocanol 0.25% and 0.5%, revealed less than 60% vessel clearance.

Conclusions: Sotradecol 0.1% both liquid and foam was determined to be the ideal sclerosant concentration based on histological findings correlated with clinical response.
Histology of Spider Veins After Treatment

• Desired functions of sclerosing agents are destruction of endothelial cells and exposure of subintimal layers to the sclerosant with eventual fibrotic occlusion of the vein

• Undesirable effects of sclerosants are vessel wall necrosis with extravasation of red cells

• The above extravasation leads to inflammatory changes and/or angiogenesis

(McAree, 2012) (Rao, 2005) (Green, 1998)
Sclerosants

• In this study, the sclerosing agents are Sotradecol® and Asclera® (Polidocanol)

• These are detergent agents that act by altering the surface tension around endothelial cells causing lysis

• The subintimal layer is exposed and depending on time of exposure and strength of solution, degradation of smooth muscle wall may occur
Sclerosants

• Evidence of muscle wall damage is visible on microscopic analysis of fibrin replacement of smooth muscle cells.
• This phenomenon is desirable if complete necrosis of wall does not occur.
• Sub intimal damage is manifested by a ragged appearance of the former lining of the vessel wall.
Sclerosants

• The ideal sclerosant will cause total lysis of endothelial cells and subintimal damage with minimal intraluminal debris such as red cells
Evaluation of Clinical and Histological Findings Using Varying Sclerosant Concentrations for the Treatment of Spider Telangiectasia

- This study was designed to correlate histologic findings and clinical results to determine the ideal sclerosant concentration using Sotradecol.
- Additional histologic determination of veins treated with Polidocanol were also done.
- There may be variability in clinical findings in the same patients using the same concentration for the same size vein.
• This variability exists due to the presence or absence of cutaneous venous hypertension
• In this study, all attempts were made to abolish cutaneous venous hypertension prior to treating the telangiectasia
• 1.5 mm punch biopsies were done at the time of phlebectomy and these biopsies were performed through the treatment site
Untreated Spider Telangiectasia

Histology of an Untreated spider vein – Copyright 2012 by www.veinexperts.org

(www.veinexperts.org, 2012)
Sotradecol 0.3%

- Note large amount of red cells in lumen
- Wall disruption
- Complete replacement of smooth muscle vessel wall with fibrin
- In this case, angiogenesis is possible

(www.veinexperts.org, 2015)
Sotradecol 0.2%

- 100% endothelial cell loss
- Subintimal changes
- Intraluminal debris
- Vein wall replacement with fibrin in some areas

(www.veinexperts.org, 2015)
Sotradecol 0.15%

- 100% endothelial cell loss
- Mild intracellular debris
- Mild subintimal changes
- Minimal vessel wall replacement with fibrin

(www.veinexperts.org, 2015)
Sotradecol 0.1%

- Complete loss of endothelial cells
- Intact vein wall with no fibrin replacement
- No intraluminal debris

(www.veinexperts.org, 2015)
Sotradecol 0.05%

- Incomplete endothelial loss
- No subintimal damage
- Very little histological findings

(www.veinexperts.org, 2015)
Polidocanol 0.5%

- 100% endothelial loss
- Vessel wall replacement with fibrin
- Intraluminal debris

(www.veinexperts.org, 2015)
Polidocanol 0.375%

- 100% endothelial cell loss
- Mild subintimal changes
- Minimal intraluminal debris

(www.veinexperts.org, 2015)
Polidocanol 0.31%

- Complete loss of endothelial cells
- Subintimal damage
- Vessel wall intact with no fibrin replacement
- No intraluminal debris
- The ideal concentration based on histology

([www.veinexperts.org](www.veinexperts.org), 2015)
Polidocanol 0.25%

• 50% endothelial loss in 1 mm vein
• No subintimal changes
• Can be tried initially for vessels < 0.5 mm
Clinical Findings in This Study

- 18 patients were treated with Sotradecol 0.2%
- Mild staining post treatment occurred in 50% of patients
- Using 0.1% Sotradecol, staining was visible in 10% of patients
- Vessel resolution was equal with 0.1% and 0.2% at about 80%
Conclusion

• Ideal concentration for a 1 mm vein is Sotradecol 0.1% - 0.15%

• This concentration range provides adequate treatment of telangiectasia, desired histological results and minimal post treatment sequelae

• The ideal concentration of Polidocanol for 1 mm spider telangiectasias is 0.3% based on histologic determination only
Conclusion

• Sotradecol 0.05% may also be used in veins < 0.5 mm with at least 80% response based on histological findings and limited clinical observations

• Polidocanol 0.25% can be used for vessels < 0.5 mm

• Histological findings with Sotradecol foam 0.1%, 0.2%, 0.3% and Polidocanol foam 0.3% are identical
Sotradecol 0.1% Foam in 2mm Reticular Vein

(Bush, 2015)
Conclusion

• This study provides evidence for appropriate concentrations of FDA approved sclerosants for treating spider telangiectasia's
Sclerosants

• Morrhuate Sodium (Detergent)
• Mixture of sodium salts and fatty acids of cod liver oil. Also contains Benzyl alcohol
• Numerous side effects- Most common being injection site sclerosis of skin
• Causes vessel wall irritation and eventual thrombosis
Sclerosants

• Dextrose (Osmotic) – Sclerodex
• Combination of dextrose & sodium chloride
• Weak sclerosing solution
• 10% saline with 25% dextrose
Sclerosants

• Glycerin produces mild endosclerosis
• Whereas the chromium atom is a potent coagulating factor that increases the sclerosing power of glycerin
• Chromium also helps reduce hematuria associated with glycerin use
• Widely used but, not approved by the FDA
Sclerosants

- Sotradecol and Polidocanol
- Detergent solutions
- Approved in US by FDA and marketed as Asclera and Sotradecol
- Sotradecol...C14 H30 O4S
- Polidocanol...C30 H62 O10
References


Venous Ulcers

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Venous Ulcers

• The ultimate expression of cutaneous venous hypertension is an ulcer
• Venous ulcers occur in 1% of the population (Evans, 1999)
• An increase in leg ulceration is directly related to post exercise venous pressure (Moyses, 1987)
• Leukocytes accumulate in the leg under conditions of high venous pressure; this activates a cascade of events at the micro-circulatory level that may lead to ulceration (Thomas, 1988)
Micro-Circulatory Events

• The micro-vascular leukocyte-trapping hypothesis has come from immuno-cytochemical and altered-structural studies that showed elevated numbers of macrophages, T lymphocytes and mast cells in skin biopsy specimens from lower limbs with CVI (Pappas, 1997)

• There is an increase expression of MMP; unrestrained MMP activity may contribute to the breakdown of the extracellular matrix (Herouy, 1999)

• Additionally, due to decreased perfusion related to probable capillary thrombosis from inflammation and higher venous pressures, skin breakdown is more likely
Objectives

• To heal an ulcer, the pressure must be reduced and the inflammatory state subside

• The goal of any treatment parameter is to effectively lower the venous pressure at the ankle and support wound healing
Treatment

• The standard of care is compression therapy still today.
• This is widely advocated, but in actuality archaic.
• Compression therapy with either elastic or inelastic dressings has been the historical treatment (Fletcher, 1997), (Erickson, 1995).
• As compliance increases, so do the healing rates. Even with compression alone, there is still a high recurrence rate (Erickson, 1995), (Scriven, 1998).
• The ESCHAR Study showed a lower reoccurrence rate of venous ulcers after stripping but not an increase in healing rates (Barwell, 2008).

(Fletcher, 1997), (Erickson, 1995)
(Scriven, 1998), (Barwell, 2008)
Treatment

• Recently, there have been scattered reports of increased healing rates using newer modalities of thermal ablation of the GSV or perforator (Poblete, 2009)

• Theoretically, any type of procedure that causes cessation of reflux from whatever source should allow normal healing processes to occur

• Sclerotherapy with Clarivein (MOCA), has shown much promise for the treatment of venous ulcer

• The advantage of Clarivein that it not only ablates the ulcer bed, but the lower GSV that is in association with the ulcer

(Poblete, 2009)
Treatment

• It may also ablate an associated perforator if there is one due to the fact that a branch may exist between the GSV and the posterior branch of the GSV where the perforator is located
• The disadvantage for Clarivein is that it is not reimbursed at this time
• This brings us to the treatment of choice at this time for venous ulcers which is initially foam sclerotherapy and many times will be the only treatment needed
• There are numerous reports describing the success of treating venous ulcers with foam sclerotherapy
Treatment

• Many of these reports describe injecting the GSV with foam, not realizing that the true mechanism was probably foam into the ulcer bed
• In 2010, I described a technique based on experience with 36 patients using a method to inject the ulcer bed alone, Terminal Interruption of Reflux Source (TIRS)
• In 2012, this technique was modified to allow delivery of foam using a percutaneous technique
• The percutaneous technique allows for the treatment of venous ulcers with or without the availability of an ultrasound
Treatment

• 3 ml of foam is injected using 1% of sclerosant
• If the patient is anticoagulated, use 3%
• There is rapid healing of the ulcer in most patients within 4-6 weeks
• Many times this is the only treatment that is necessary especially in patients with deep venous insufficiency without GSG reflux
• After treatment, only a 20 mmHg dressing is used
• In effect, you have created an internal compression dressing that is far more effective than any dressing applied externally
Treatment

• At time of initial consultation, I perform the percutaneous technique with a follow-up in 2-weeks

• If there are still patent vessels under the ulcer bed, repeat treatment is done

• If there are no refluxing vessels under the ulcer bed then this is not a venous ulcer

• Patients with RF Factor on Methotrexate or who have Calcinosis have a difficult and more prolonged time to heal
Treatment of Venous Ulcer by Percutaneous Technique
Treatment of Venous Ulcer by Percutaneous Technique
Treatment of Venous Ulcer by Percutaneous Technique
Treatment of Venous Ulcer by Percutaneous Technique
Treatment of Venous Ulcer by Percutaneous Technique
Summary

• If possible, I would prefer to do retrograde administration of sclerotherapy using the catheter developed by Clarivein (MOCA)

• This provides closure of the vein and sclerotherapy solution into the tributaries of the ulcer

• At the present time, Vascular Insights, is working on insurance approval through various carriers