

A case of chronic lichenoid dermatitis manifesting as hypopigmented, flat-topped papules

Ann Mazor Reed, DO,* Jennifer David, DO,** Stanley Skopit, DO, MSE, FAOCD***

* Dermatology resident, 2nd year, NSU-COM/Larkin Community Hospital, Miami, FL

** Dermatology resident, 1st year, NSU-COM/Larkin Community Hospital, Miami, FL

*** Program Director, Dermatology Residency, NSU-COM/Larkin Community Hospital, Miami, FL

Abstract

We report a case of a 65-year-old African American female with chronic lichenoid dermatitis that manifested as hypopigmented, flat-topped papules. The lesions were initially thought to be flat warts based on their clinical appearance. Histology of a lesion revealed resolving lichenoid dermatitis with some features suggesting a possible histological differential diagnosis of cutaneous T-cell lymphoma. However, molecular studies showed oligoclonal results, which was insufficient to meet the criteria of a clonal process. We present a possible new subtype of lichen planus, the hypopigmented variant. After previous therapy with topical corticosteroid cream, our patient improved with clobetasol ointment twice daily, an intramuscular triamcinolone injection and narrowband UVB (NB-UVB) three times a week for six weeks.

Introduction

Lichen planus (LP) is an idiopathic inflammatory disease of the skin, nails, hair, and mucous membranes.¹ The pathogenesis and etiology of LP is uncertain, although many believe it occurs secondary to T-cell-mediated autoimmune damage. Classical LP is characterized by small, pruritic, violaceous, flat-topped papules that favor the flexor surfaces of the extremities.² Typically, the lesions are symmetric and bilateral.¹ LP is a common inflammatory cause of hyperpigmentation.³ The hypopigmented variant of lichen planus has been hypothesized as a new subtype of lichen planus.

Case Report

A 65-year-old African American female presented to our outpatient dermatology clinic complaining of asymptomatic “white bumps” on her hands and feet that began three years prior to presentation. She stated that these lesions first developed on the dorsal surface of her hands and gradually spread to her forearms and elbows, eventually affecting her ankles as well. The patient had been under the care of another dermatologist for the past three years. Per patient history, prior treatment

for this condition included a “steroid cream” with no improvement. The patient’s past medical and surgical history were non-contributory. She had no known drug or environmental allergies and denied taking any medications, including over-the-counter medications.

The findings on physical exam included multiple, white, hypopigmented, flat-topped papules on the bilateral dorsal hands, forearms, and lateral ankles (**Figure 1**).

The patient had no oral or nail lesions nor did she exhibit any lymphadenopathy.

A shave biopsy was obtained from a papule on her right elbow and sent for histologic examination.

Analysis of the biopsy revealed a focally lichenoid infiltrate with thinning of the epidermis, some irregular, jagged epidermal retia, and prominent fibrosis in the papillary dermis (**Figure 2**). Immunohistochemical studies were performed to further characterize the process. Lesional cells showed a CD3+, CD5+ and CD7+ phenotype (**Figures 3, 4**). The CD4 to CD8 ratio was approximately 1:1. CD20 staining was very focal and CD30 staining essentially negative. Melan-A staining highlighted focal loss of melanocytes at the dermoepidermal junction (**Figure 5**). Some

features raised the possibility of a histological differential diagnosis of cutaneous T-cell lymphoma. However, molecular studies showed oligoclonal results, which was insufficient to meet the criteria of a clonal process. The patient’s lipid panel, complete metabolic panel, and complete blood count were all within normal limits. These findings along with her clinical presentation were highly suggestive of a long-standing and resolving lichenoid inflammatory process such as resolving lichen planus.

The treatment plan included clobetasol 0.05% ointment twice daily to affected areas on hands, forearms and ankles for two weeks per month, an intramuscular injection of 40 mg triamcinolone, and narrow-band UVB three times a week for six weeks. On two-month follow-up, the patient admitted to improvement of her skin condition.

Discussion

Lichen planus has numerous variants, including actinic LP, acute LP, annular LP, atrophic LP, bullous LP, LP pemphigoides, hypertrophic LP, inverse LP, LP pigmentosus, lichen planopilaris, linear LP, LP-lupus erythematosus overlap syndrome, nail LP, oral LP, ulcerative LP,



Figure 1. Numerous scattered, hypopigmented, flat-topped papules involving the dorsal hand (A), extensor surface of forearm and elbows (B), and lateral ankle (C).

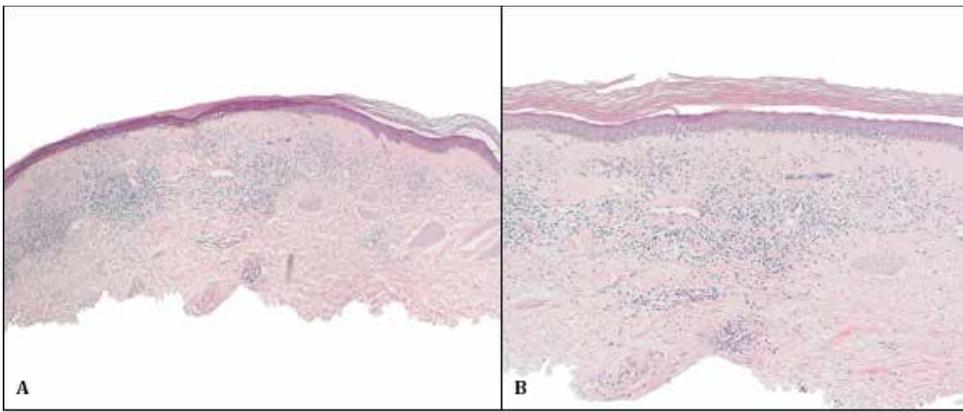


Figure 2. H&E (4x): Histopathology of lesional skin of right elbow demonstrating a focal lichenoid infiltrate with thinning of the epidermis and some irregular, jagged epidermal retia (A). Higher-power magnification (10x) visualizes the prominent fibrosis in the papillary dermis (B).

vulvovaginal LP, and lichenoid drug eruption.³ However, a hypopigmented variant has not been described. There is a subset of vitiligo patients that have some “lichenoid characteristics,” including analogous locations and, in the initial phase, pathological features similar to those of LP.⁴ This subtype of vitiligo is termed “lichen planus depigmentosus” and is considered by some to be the counterpart of lichen planus pigmentosus.⁴ Due to the normal melan-A stain and clinical findings, we do not believe our patient had lichen planus depigmentosus.

Although the pathogenesis and etiology of LP remain unknown, it is thought to be related to an autoimmune process in which CD8+ T lymphocytes attack basal keratinocytes.⁵ There are various triggers thought to be associated with the initiation of lichen planus. These include but are not limited to infections, vaccines, drugs, and contact allergens.³ Exposure to these triggers can initiate an autoimmune cascade and the generation of effector T cells with cytotoxic potential.³

There are many drugs commonly implicated in lichenoid drug eruptions. The most common ones include ACE-inhibitors, thiazide diuretics, antimalarials, quinidine and gold.

In its classical form, LP is characterized by small, polygonal, violaceous, flat-topped papules that favor the flexor surfaces of the extremities.² Because lichen planus most commonly presents as hyperpigmented or violaceous papules, other diagnostic possibilities were considered for our patient as well. Part of the differential diagnosis for hypopigmented flat-topped papules includes flat warts. However, because the histopathology did not reveal characteristics consistent with a wart, hypopigmented lichen planus was favored as our diagnosis.

Treatment

Formulating a treatment plan for lichen planus can be difficult, as there are some cases that remit spontaneously and others that are resistant to treatment. Additionally, it is important to rule out a drug-induced form, as withdrawal of the offending agent may lead to resolution of the

lesions without additional treatment. Topical corticosteroids remain the first-line therapy for localized LP.³ For severe cases, immunomodulating therapies such as cyclosporine may be effective. In our case, the patient improved

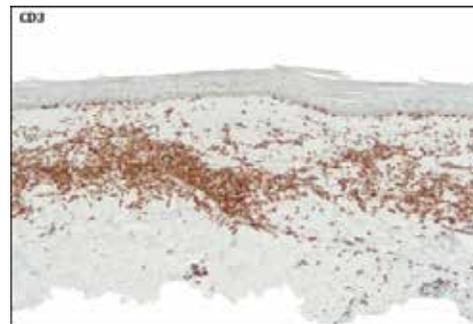


Figure 3. Immunohistochemical stain showing CD3+ phenotype.

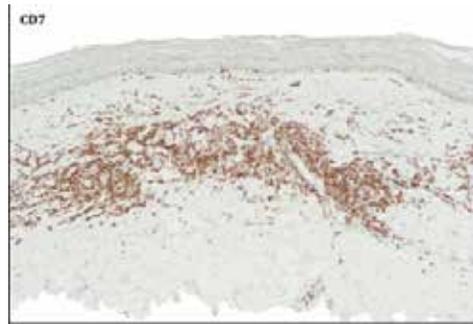


Figure 4. Immunohistochemical stain showing CD7+ phenotype.

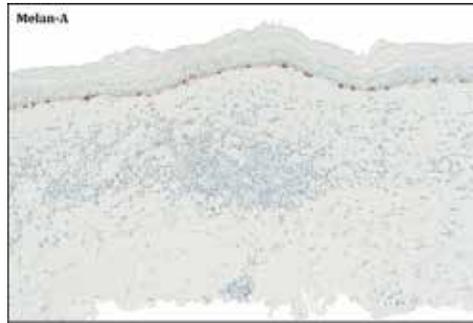


Figure 5. Melan-A staining highlights focal loss of melanocytes at the dermoepidermal junction.

with topical clobetasol ointment twice daily for two weeks per month, one intramuscular corticosteroid injection, and NB-UVB therapy three times a week for six weeks.

Conclusion

Lichen planus has many variants, but a hypopigmented variant has not been described. The numerous subtypes of LP are differentiated from classic LP by distribution and morphology. The morphology of the lesions in our patient was hypopigmented, whereas the most commonly described lesions of LP are violaceous or hyperpigmented. The clinical differential for the described lesions included flat warts, but there were no koilocytes on histology. In our patient, clinical findings and histopathologic clues pointed to a case of hypopigmented LP.

References

1. Gorouhi F, Davari P, Fazel N. Cutaneous and Mucosal Lichen Planus: A Comprehensive Review of Clinical Subtypes, Risk Factors, Diagnosis, and Prognosis. *ScientificWorldJournal*, c2014; 2014, Article ID 742826, 22 pages.
2. Boyd AS, Neldner KH. Lichen planus. *J Am Acad Dermatol*. 1991;25:593-619.
3. Bologna JL, Jorizzo JL, Rapini RP. *Dermatology*. 2nd ed. Vol. 1. Spain: Elsevier; c2008. Chapter 12, Lichen planus and lichenoid dermatoses; p. 159-180.
4. Patel AB, Kubba R, Kubba A. Clinicopathological correlation of acquired hypopigmentary disorders. *Indian J Dermatol Venereol Leprol*. 2013 May 1;79 (3):376-382.
5. Wagner, G, Rose, C, Sachse, MM. Clinical variants of lichen planus. *J Dtsch Dermatol Ges*. 2013 April ;11(4):309-319.

Correspondence: Ann Mazor Reed, DO; 4970 W Atlantic Blvd., Margate, FL 33063; Ph: 954-977-0270; F: 954-977-6824; Amazorreed@larkinhospital.com