

Lichen planus pigmentosus

Charisse M. Orme, MD PhD, Randie H. Kim, MD PhD, Nooshin Brinster, MD, Nada Elbuluk, MD, Andrew G. Franks, Jr., MD

Ronald O. Perelman Department of Dermatology, NYU School of Medicine, NYU Langone Medical Center

Abstract

Lichen planus pigmentosus (LPP) is a type of lichenoid dermatitis with superficial dermal melanophages that presents as symmetrical, hyperpigmented macules and patches that are distributed over the forehead, temples, cheeks, and neck. The condition most often occurs in darker skinned individuals and is frequently resistant to treatment. Here we present a patient of Egyptian decent with a lacy reticulated LPP eruption on the face.



Figure 1. On the cheeks, temples, forehead, and mandible were lacy and reticular, dark grey, hyperpigmented macules and patches that coalesced towards the neck. Few scattered erythematous papulopustules were noted.

Case Presentation

PATIENT: 35-year-old-woman

DURATION: Three years

DISTRIBUTION: Face

HISTORY: A 35-year-old woman with a history of trichotillomania and depression presented to the Skin and Cancer Unit for evaluation of a dermatitis on the face. She first presented six months after the initial onset and presented again three years later. Several years prior to her facial cutaneous eruption, while living in Egypt, she took care to prevent darkening of her facial skin by using an occasional facial peel and hydroquinone. After moving to the United States, the patient noted some pink patches and hyperpigmentation on her cheeks. She also noted papules and pustules of the cheeks and perioral region, which she had periodically experienced throughout her life and attributed to acne. After moving to the United States, but before her facial eruption started, the patient started taking the fluvoxamine, which is an antidepressant. Initial workup for the facial eruption was started by an outside dermatologist and rheumatologist. The patient spent long hours working as a cashier and reported fatigue and knee pain at the end of the day, but a review of systems was negative.

On screening she was found to have an anti-nuclear antibody (ANA) titer of 1:80. An initial biopsy specimen was reported to show a resolving interface dermatitis with numerous melanophages that was consistent with possible drug photodermatitis or lupus erythematosus. She was first given hydrocortisone 2.5% cream for three weeks and then hydroxychloroquine for nine months but did not note any improvement in her facial hyperpigmentation. The hyperpigmentation on her face continued to progress, and a second

biopsy was performed two years after the initial onset that was consistent with a lichenoid dermatitis. The patient presented to Skin and Cancer Unit with worsening of her symmetrical facial hyperpigmentation. Our patient did not improve after nine months of hydroxychloroquine that was started by her original dermatologist and was started on topical tacrolimus 0.1% ointment at her last visit.

PHYSICAL EXAMINATION: On the cheeks, temples, forehead, and mandible were lacy and reticular, dark grey, hyperpigmented macules and patches that coalesced towards the neck. Few scattered erythematous papulopustules were noted.

LABORATORY: A complete blood count and comprehensive metabolic panel, thyroid function tests, cholesterol, and hemoglobin A1C were normal. An antinuclear antibody (ANA) titer was 1:80, with a nucleolar pattern. dsDNA, Smith, Ribonuclear protein, Ro, La, Scl-70, and centromere antibodies, rheumatoid factor, and cyclic citrullinated peptide were negative.

HISTOPATHOLOGY: There are vacuolar interface changes at the dermoepidermal junction and a superficial-to-mid dermal, perivascular, lymphocytic infiltrate. The epidermis is atrophic. Dyskeratotic cells are noted in the epidermis with colloid bodies in the upper dermis. Numerous melanophages are present in the papillary dermis. A colloidal iron stain does not demonstrate increased mucin deposition.

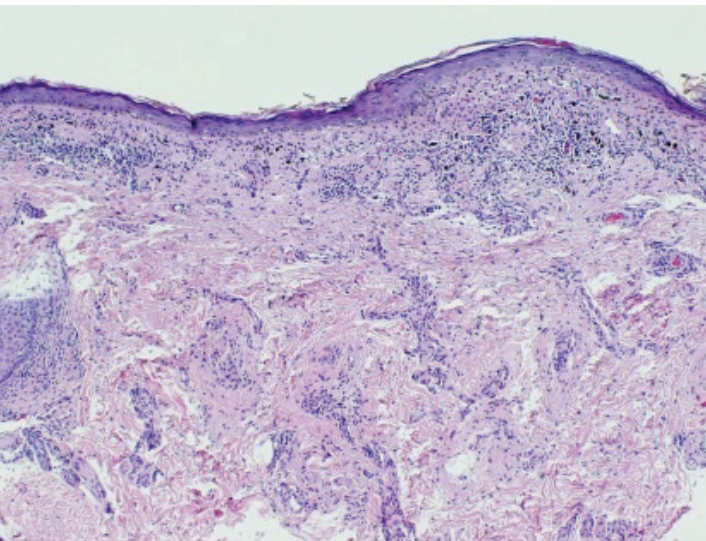


Figure 2. Vacuolar interface changes at the dermoepidermal junction and a superficial-to-mid dermal, perivascular, lymphocytic infiltrate. The epidermis is atrophic.

A periodic acid-Schiff-diastase stain does not demonstrate basement-membrane thickening.

DIAGNOSIS: Lichen planus pigmentosus

Discussion

Lichen planus pigmentosus (LPP) presents as a symmetrical, hyperpigmented, lichenoid dermatitis that predominantly affects the face and neck in skin types III to VI [1, 2]. It is thought to occur more frequently in young adult women. The hyperpigmentation may demonstrate a variety of patterns that include reticular, perifollicular, linear, and diffuse. LPP is considered a variant of lichen planus but is often placed in the family of pigmented dermatosis that include erythema dischromium perstans and lichen pigmentosus [1, 3-7]. The features of LPP on histopathologic examination include a vacuolar degeneration with perivascular or lichenoid infiltrate and superficial dermal melanophages [1, 8].

Lesions may be asymptomatic or have mild burning and pruritus [1, 3]. There is no definitive etiology, but some speculate that LPP may be precipitated by a photosensitizing agent that is applied to the facial skin. LPP was first described in an Indian cohort in 1974, and, since then, application of mustard oil and amla oil have been implicated [1, 9]. Our patient of Egyptian decent was unable to identify any agent that directly triggered her symptoms; however, she did have a history of using peels and hydroquinone on the face in the years prior to the appearance of the eruption. The patient's use of an oral antidepressant prior to onset of the condition did not correlate well as a trigger, and the dermatitis has continued to worsen long after its discontinuation, which makes a lichenoid drug eruption less likely.

The course of LPP is often chronic and difficult to treat. One open label trial suggested that topical tacrolimus ointment may play a beneficial role in treatment [8].

References

1. Kanwar AJ, et al. A study of 124 Indian patients with lichen planus pigmentosus. *Clin Exp Dermatol* 2003;28:481
2. Namazi MR. Lichen planus pigmentosus presenting as diffuse facial melanosis. *J Drugs Dermatol* 2004;3:436
3. Vega ME, et al. Ashy dermatosis and lichen planus pigmentosus: a clinicopathologic study of 31 cases. *Int J Dermatol* 1992;31:90
4. Rieder E, et al. Lichen planus pigmentosus. *Dermatol Online J* 2013;19:20713
5. Cutri FT, et al. Lichen planus pigmentosus-like ashy dermatosis. *Dermatol Rep* 2011;3:e46
6. Vega ME, et al. Ashy dermatosis versus lichen planus pigmentosus: a controversial matter. *Int J Dermatol* 1992;31:87
7. Bhutani LK. Ashy dermatosis or lichen planus pigmentosus: what is in a name? *Arch Dermatol* 1986;122:133
8. Al-Mutairi N, et al. Clinicopathological characteristics of lichen planus pigmentosus and its response to tacrolimus ointment: an open label, non-randomized, prospective study. *J Euro Acad Dermatol Venereol* 2010;24:535
9. Bhutani LK, et al. Lichen planus pigmentosus. *Dermatology* 1974; 149:43