Beltlike Lichen Planus Pigmentosus Complicated With Focal Amyloidosis

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To the Editor:
A 68-year-old man presented with slightly itchy macules on the waist and abdomen of approximately 2 years' duration. He reported that the initial lesions were dark red and subsequently coalesced to form a beltlike pigmentation on the abdomen. He denied any prior treatment, and the lesions did not spontaneously resolve. The patient was taking escitalopram oxalate, telmisartan, and aspirin for depression and cardiovascular disease that was diagnosed 3 years prior. He reported no exposure to UV radiation or a heat source. He denied use of any cosmetics on the body as well as a family history of similar symptoms.

Physical examination showed reticulate brown-purple macules with slight scale on the surface that had become confluent, forming a beltlike pigmentation on the waist and abdomen (Figure 1). Wickham striae were not seen. The oral mucosa and nails were not affected. Microscopic examination for fungal infections was negative.

Systematic physical and laboratory examinations revealed no abnormalities. A skin biopsy from a macule on the abdomen showed hyperkeratosis, thinned out stratum spinosum with flattening of rete ridges, hypergranulosis with vacuolar alteration of the basal cell layer, and bandlike infiltration of lymphocytes and melanophages with incontinence of pigment (Figure 2). Focalized purplish homogeneous deposits were observed in the upper dermis (Figure 3), of which positive crystal violet staining indicated amyloidosis (Figure 4). Congo red stain revealed amyloid deposition (Figure 5). Thus, the diagnosis of lichen planus pigmentosus (LPP) complicated with focal amyloidosis was made. The patient was treated with topical corticosteroids and tretinoin, and no notable therapeutic effects were observed at 3-month follow-up.

Lichen planus pigmentosus, a variant of lichen planus, is a condition of unknown etiology exhibiting dark brown macules and/or papules and a long clinical course. The face, neck, trunk, arms, and legs are the most common areas of presentation, whereas involvement of the scalp, nails, or oral mucosa is relatively rare.

The first clinicohistopathological study with a large sample size was documented by Bhutani et al in 1974 who termed the currently recognized entity lichen planus pigmentosus. Lichen planus pigmentosus is a frequently encountered hyperpigmentation disorder in Indians, whereas sporadic cases also are reported in other regions and ethnicities. In cases of LPP, the pigmentation is symmetrical, and its pattern most often is diffuse, then

PRACTICE POINTS
- Lichen planus pigmentosus can present in a unique beltlike distribution pattern.
- Focal amyloidosis due to chronic friction and scratching cannot be excluded from the differential diagnosis.

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reticular, blotchy, and perifollicular. Two unique patterns of LPP have been documented, including linear/blaschkoid LPP and zosteriform LPP. Our patient showed a unique beltlike distribution pattern.

The pathogenesis of LPP still is unclear, and several inciting factors such as mustard oil, gold therapy, and hepatitis C virus infection have been cited. Mancuso and Berdondini reported a case of LPP flaring immediately after relapse of nephrotic syndrome. It also has been considered as a paraneoplastic phenomenon. No exact cause was found in our patient after a series of relative examinations.

The histopathologic changes associated with LPP consist of atrophic epidermis; bandlike lymphocytic infiltrate with vacuolar degeneration of the basal layer in the epidermis; and prominent melanin incontinence in the upper dermis, which can be diverse depending on different sites of skin biopsy and the phase of LPP. Histopathologic findings in our patient were consistent with LPP. The differential diagnosis for the reticulate pattern of pigmentation seen in our patient included confluent and reticulated papillomatosis and poikilodermalike cutaneous amyloidosis, both easily excluded with histopathologic confirmation.

Local amyloidosis also was confirmed by crystal violet staining in our case and its etiology was uncertain. Generalized and local amyloidosis has been reported in association with lichen planus. The diagnosis of lichen planus was followed by the diagnosis of amyloidosis, and the typical skin lesions of these 2 conditions were able to be differentiated in these reported cases. However, beltlike pigmentation was the only manifestation for our patient and we could not separate the 2 conditions with the naked eye.

FIGURE 2. Histopathology showed hyperkeratosis, thinned out stratum spinosum with flattening of rete ridges, hypergranulosis with vacuolar alteration of the basal cell layer, and bandlike infiltration of lymphocytes and melanophages with incontinence of pigment (H&E, original magnification ×10).

FIGURE 3. Focalized purplish homogeneous deposits were observed in the upper dermis (H&E, original magnification ×40).

FIGURE 4. Crystal violet staining was positive for amyloidosis (original magnification ×40).

FIGURE 5. Congo red stain revealed amyloid deposition (original magnification ×40).
Chronic irritation to the skin resulting in excessive production of degenerate keratins and their subsequent conversion into amyloid deposits has been proposed to be an etiologic factor of amyloidosis. Because of the distribution pattern in our case, we believe focal amyloidosis could be attributed to chronic friction and scratching.

REFERENCES