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CASE REPORT

Plasma Cell Predominance in Lichen Planus Pigmentosus: A Rare Case in a Young Female

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Abstract: Lichen planus pigmentosus (LPP), a rare variant of lichen planus, characterized by the insidious onset of dark brown to gray macules. In this report, we present a unique case of LPP in a 30-year-old female patient who developed diffuse hyperpigmented macules with nail dystrophy. Notably, histopathology revealed classic lichenoid interface dermatitis with predominant plasma cell infiltration in the papillary dermis, confirmed by strong CD38 and CD138 immunoreactivity. Laboratory evaluation revealed elevated serum Kappa light chain and thyroid stimulating hormone levels, raising the possibility of immune dysregulation. No evidence of plasma cell malignancy was found after further hematologic evaluation. To our knowledge, this may be the first reported case of plasma cell-rich LPP in a young female with concurrent nail involvement and isolated serum kappa light chain elevation without evidence of clonality. Awareness of this entity is essential for differentiating it from mimickers such as cutaneous plasmacytosis, multicentric Castleman disease, and secondary syphilis. This case highlights the need for heightened awareness of this variant and its possible systemic associations, and underscores the importance of integrating histopathology, immunophenotyping, and laboratory workup to avoid misdiagnosis and guide clinical care.

Keywords: lichen planus pigmentosus, pigmentary disorders, plasma cell, histopathology, immune dysregulation

Introduction

Lichen planus pigmentosus (LPP) is an uncommon variant of lichen planus (LP), characterized by dark brown to slate-gray macules primarily on sun-exposed or flexural areas, predominantly affecting individuals with Fitzpatrick skin types III to VI.[1] Although its pathogenesis remains incompletely understood, a CD8+ T cell–mediated cytotoxic response targeting basal keratinocytes is widely accepted as the initiating event, leading to vacuolar degeneration and pigment incontinence. Potential triggers such as hepatitis C infection, autoimmune conditions, and endocrinopathies, have been reported in association with LPP.[2,3] Histologically, LPP typically demonstrates a lymphocyte-predominant lichenoid interface dermatitis. However, recent studies have expanded the immunophenotypic spectrum, identifying the involvement of CD4+ T cells and Langerhans cells within the dermal infiltrate.[1] In contrast, plasma cell-predominant infiltrates in LPP are extremely rare. We report a histologically confirmed case of LPP with a predominant plasma cell infiltration, accompanied by nail dystrophy and serum kappa light chain elevation in a young female. To our knowledge, this clinicopathologic constellation has not been previously documented. This case highlights the diagnostic complexity and underscores the importance of recognizing atypical inflammatory variants of LPP.

Case Report

A 30-year-old female presented with a four-month history of asymptomatic hyperpigmented eruptions on the trunk and extremities, along with nail dystrophy. Her medical history was notable for hypothyroidism, managed with levothyroxine (100ug/day) for one year. The family history was unremarkable. She reported no personal history of hematologic or autoimmune

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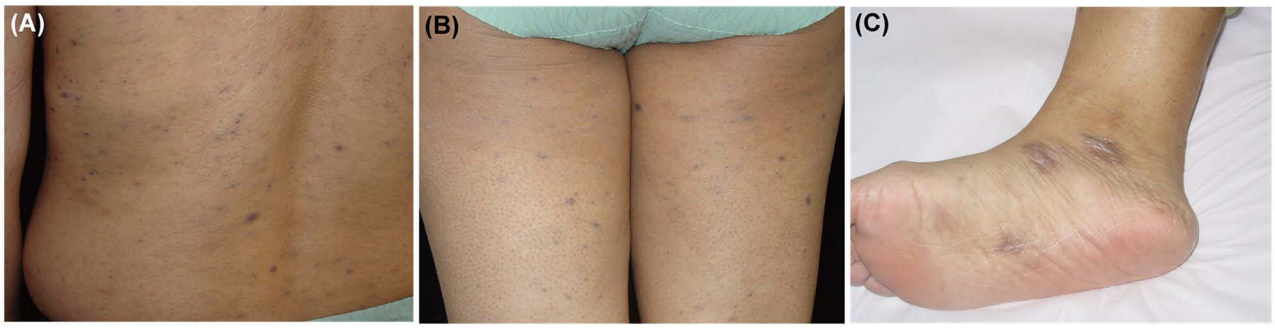

Figure 1 Clinical presentations: multiple well-demarcated, dark brown macules and patches were symmetrically distributed over the (**A**) back, (**B**) lower extremities and (**C**) right sole.

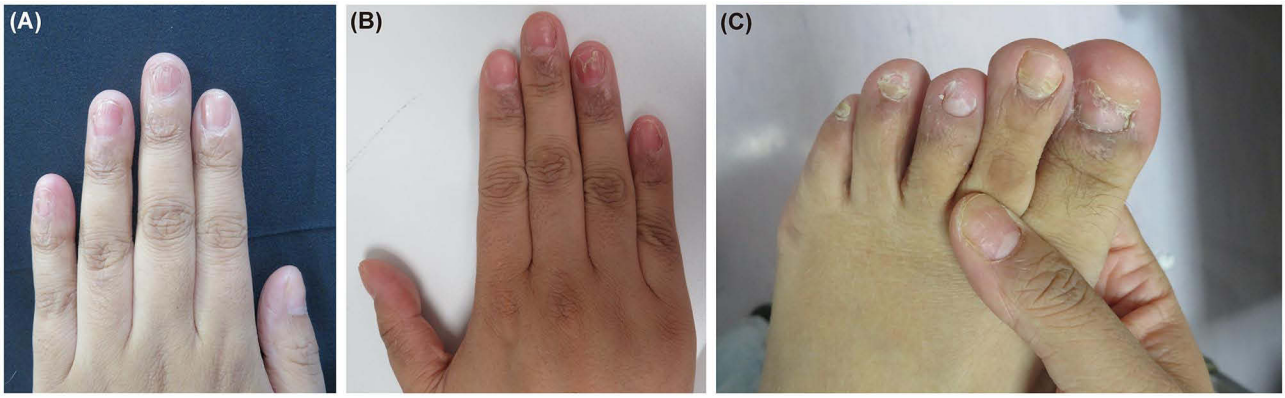

Figure 2 Nail changes: onycholysis, fissures and longitudinal striations were observed on the (**A**) left and (**B**) right fingernails. (**C**) The left toenails exhibit significant nail dystrophy with marked onycholysis, along with periungual erythema.

disorders. During the initial visit to our clinic, a skin examination revealed multiple well-demarcated, dark brown macules and patches symmetrically distributed over the trunk, limbs and soles (Figure 1A–C). Longitudinal streaks and partial onycholysis were observed on the fingernails and toenails (Figure 2A-C). No lesions were observed on the mucous membranes or scalp.

Laboratory investigations showed a mildly elevated Kappa light chain level at 25.04 mg/L (normal range: 6.70–22.40 mg/L), with a normal kappa/lambda ratio at 1.33 (0.31–1.56). Thyroid stimulating hormone (TSH) was elevated at 8.143 uIU/mL (0.75–5.60 uIU/mL). Other serologic tests including complete blood count, erythrocyte sedimentation rate (ESR), liver and kidney functions, hepatitis B, hepatitis C, treponemal antibody, and human immunodeficiency virus (HIV) were unremarkable. Results of antinuclear antibodies, extractable nuclear antigens, complement 3 (C3), C4, etc, were negative. Dermoscopy of the lesions revealed brown-grey dots arranged in circles and vertical striations on the affected nails (Figure 3A and B). A biopsy from the abdomen showed hyperkeratosis, vacuolar degeneration of the basal cell (Figure 3C), and a predominant infiltration of plasma cells in the papillary dermis, along with melanin incontinence (Figure 3D). Immunohistochemical markers were sent for analysis, and the results indicated immunoreactivity to CD38 and CD138 (Figure 3E and F), confirming the plasma cell nature of the infiltrate. To exclude plasma cell dyscrasia, flow cytometric immunophenotyping of peripheral blood was performed, showing no evidence of a clonal or aberrant plasma cell population. The clinicopathologic findings were consistent with lichen planus pigmentosus with predominant plasma cell infiltration. The patient was treated with topical betamethasone, resulting in gradual clinical improvement. No recurrence or progression was noted during an eight-month follow-up.

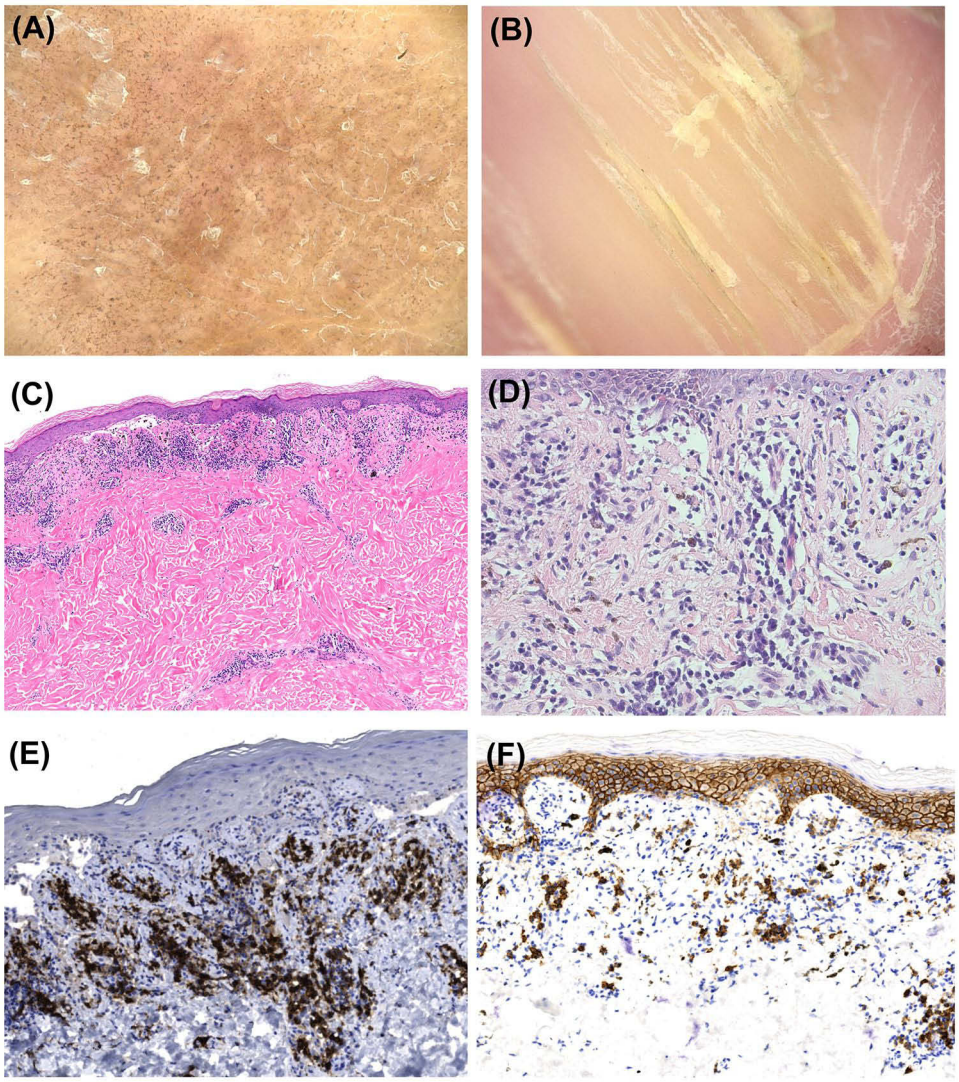

Figure 3 Dermoscopic, histopathological and immunohistochemical findings of this patient. Dermoscopy showed brown-grey dots and globules in circles of (**A**) the skin, and vertical stripes of (**B**) nails. (**C**) Histologically, lesions showed hyperkeratosis, irregular acanthosis and vacuolar degeneration of basal cells (H&E; original magnification x40). (**D**) In the papillary dermis, a band-like infiltrate of inflammatory cells, predominantly plasma cells with melanin incontinence were observed (H&E; original magnification x400). Immunohistochemical staining showed diffuse positivity for (**E**) CD38 and (**F**) CD138.

Discussion

Lichen planus pigmentosus, first described by Bhutani et al in 1974, is traditionally considered a T-cell–mediated disorder, with cytotoxic CD8+ T cells targeting basal keratinocytes.[4,5] Most cases show a lymphocyte-dominant infiltrate, consistent with its classification as a T-cell–driven condition.[6] However, a few cases of LP variants with plasma cell–rich infiltrates have been reported in the literature, particularly in classical, ulcerative, and planopilaris forms.[7–9] These cases predominantly affect middle-aged to elderly individuals, with a marked female predominance. Cutaneous manifestations often include hyperpigmented macules or violaceous plaques, particularly on the extremities, with nail dystrophy or anonychia being frequent.[9–11]

Histopathologically, these lesions maintain the classic features of LP but are distinguished by dense dermal infiltrates composed of >50% plasma cells, frequently showing polyclonal light chain expression. In contrast, plasma cell-predominant infiltrates in LPP, particularly in young individuals, remain undocumented to date. The present case appears to be the first report of plasma cell-rich LPP with concurrent nail involvement and serum kappa light chain elevation in a young female.

The exact etiology of plasma cell involvement in LPP remains unclear, but several factors have been proposed to contribute to its development. While LP is classically considered T-cell–mediated, plasma cells may contribute to chronic inflammation through autoantibody production and cytokine secretion. Recent studies suggest that persistent antigenic stimulation, which is potentially from viral triggers or drug exposure, could drive plasma cell differentiation.[9] Notably, Raybaud et al linked Epstein-Barr virus (EBV)–infected CD138+ plasma cells to erosive oral LP, suggesting viral triggers may amplify plasma cell recruitment.[12] Furthermore, the correlation between CD20 and CD138 expression in oral LP supports B-cell and plasma cell interplay, possibly driven by persistent antigenic stimulation.[13] In our case, the coexistence of hypothyroidism and elevated serum kappa light chains may reflect subclinical immune dysregulation, though no clonal plasma cell population was identified. This underscores the importance of considering systemic associations and potential triggers in plasma cell–rich lichenoid dermatoses.

The diagnostic evaluation of plasma cell-rich lichenoid dermatoses requires careful exclusion of neoplastic and infectious mimics, including cutaneous plasmacytoma, IgG4-related disease, secondary syphilis, and discoid lupus erythematosus. In our case, the absence of these features, together with a polyclonal plasma cell infiltrate and typical lichenoid pattern, supports the interpretation that the plasma cells represent an intrinsic component of the lichenoid reaction rather than a secondary or neoplastic process. However, the study is limited by its single-case design and the relatively short duration of follow-up.

Conclusions

LPP with prominent plasma cell infiltration is a rare histopathologic variant that warrants clinical attention due to its potential for diagnostic confusion with infectious, neoplastic, and systemic plasma cell disorders. Early recognition is crucial to avoid misdiagnosis and ensure appropriate treatment. This case expands the histologic spectrum of LPP and raises important questions regarding the immunologic role of plasma cells in its pathogenesis. Further research is needed to determine whether plasma cell-rich infiltration represents reactive immune activation or a distinct pathogenic subset. Additionally, long-term follow-up studies are warranted to better elucidate the natural history and prognostic outcomes of this condition. Increased awareness of this variant will help enhance diagnostic accuracy and may guide future approaches to treatment and classification in lichenoid dermatoses.

Ethics and Consent Statement

The written informed consent was obtained from the patient for the publication of the case details and images. No further institutional approval was required.

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Disclosure

The authors report no conflicts of interest in this work.

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