Case Presentation

Lupus erythematosus panniculitis in a 77-year-old woman

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Abstract

Lupus erythematosus panniculitis (LEP) is an uncommon variant of lupus erythematosus, characterized by subcutaneous indurated nodules or plaques that often heal with lipoatrophy or scarring. We report a 77-year-old woman who presented with a 4-month history of painful subcutaneous nodules and erythematous plaques. She had a history of discoid lupus erythematosus in her 30s, which was successfully managed with topical treatments and had since resolved. Deep incisional biopsy of a plaque on her back confirmed the diagnosis of LEP. The patient improved with treatment including methotrexate, folic acid, and prednisone. This case highlights a unique clinical presentation of LEP, both in terms of patient age and lesion distribution, and underscores the critical role of histopathology in excluding subcutaneous panniculitis–like T-cell lymphoma.

Introduction

Lupus erythematosus panniculitis (LEP) is a rare form of chronic cutaneous lupus erythematosus characterized by inflammation of subcutaneous fat. LEP typically presents as firm, tender, indurated nodules or plaques in fatty areas, including the face, arms, shoulders, trunk, and buttocks. The disease often follows a relapsing–remitting course, with lesions frequently healing with scarring and lipoatrophy.[1] A recent retrospective review of 61 patients with LEP found that 21% had a concomitant diagnosis of systemic lupus erythematosus (SLE) and 28% had discoid lupus erythematosus (DLE).[2] LEP predominantly affects women, with female-to-male ratios in case series ranging from 4.5:1 to 8.5:1 and an average age of onset in the 30s to 40s.[2,3] Diagnosis can be challenging because of overlapping clinical features with subcutaneous panniculitis–like T-cell lymphoma (SPTCL) and morphea profunda.[4,5] Early recognition and intervention are critical to prevent extensive, irreversible atrophy and disfigurement.

Case Synopsis

A 77-year-old woman with a remote history of DLE in her 30s, which had resolved with topical treatments, presented with 4 months of progressively painful lesions on her lower back and thighs. Physical examination revealed firm subcutaneous nodules with overlying atrophy and erythematous, reticulated patches and plaques on the left upper arm, back, right buttock, and medial thighs (**Figure 1**). Review of systems was notable for fatigue and weight loss. Laboratory work-up was unremarkable, with negative antinuclear, anti-Smith, and anti-SSA/SSB antibodies.

Incisional biopsy of the back demonstrated suppurative inflammation, dystrophic calcification, fibrinoid collagen degeneration with lymphocytic and plasma cell infiltrates, and adipose hyalinization, consistent with LEP (**Figure 2**). Alcian blue staining revealed increased interstitial dermal mucin. Immunohistochemistry showed T-cell populations (CD3+, CD5+, CD7+) with a predominance of CD4+ over CD8+ cells, B-cell nodules of CD20+ cells, and low Ki-67 proliferation, effectively excluding SPTCL. Treatment with methotrexate, folic acid, and prednisone led to clinical improvement.

Case Discussion

We present a case of LEP that was atypical in terms of lesion distribution, appearance, and patient age. Although LEP typically affects fatty areas such as the proximal limbs, trunk, and buttocks, our patient's most symptomatic and extensively involved site was the lower back, a region not traditionally considered fat-dense; additional lesions were located on the thighs and upper arms. Violaceous, reticulate patches reminiscent of livedo reticularis or retiform purpura were observed overlying the subcutaneous nodules in this area.

LEP is most frequently reported in individuals in their 30s to 40s, yet our patient presented in her 70s. Physi-

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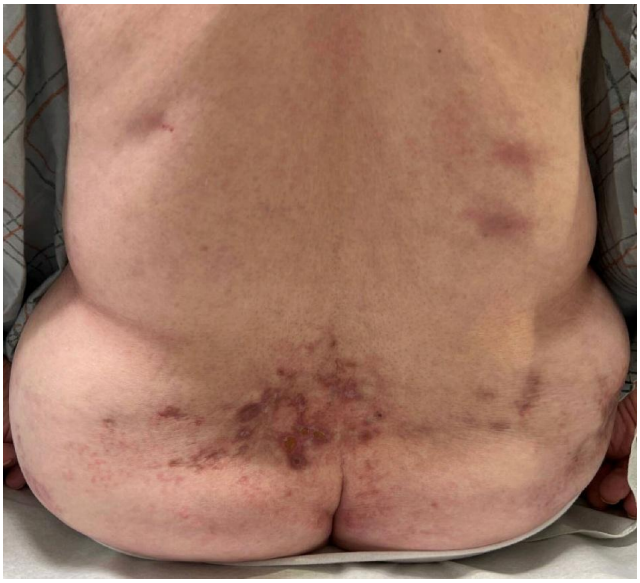

Figure 1. *Firm subcutaneous nodules with overlying atrophy and erythematous reticulated patches on the left upper arm, back, right buttocks, and medial thighs*

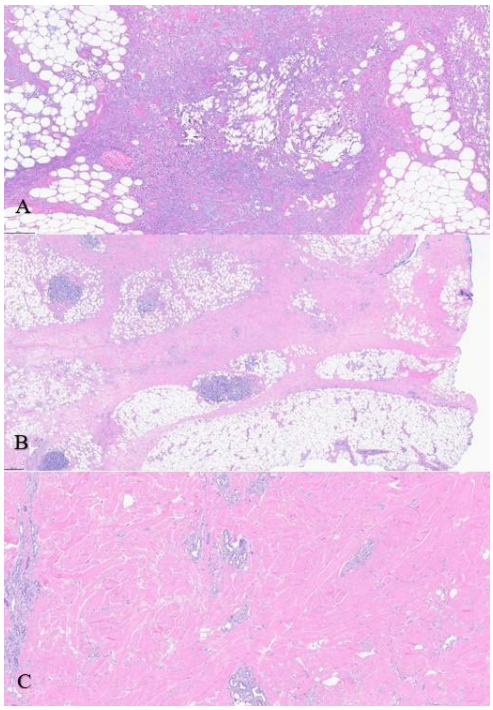

Figure 2. *Hyalinization of adipose tissue, foci of suppurative inflammation, dystrophic calcification, fibrinoid collagen degeneration in the fibrous septa with an associated lymphocytic and plasma cell infiltrate. Hematoxylin-eosin, original magnification:* ***(A)*** *×100,* ***(B)*** *×40,* ***(C)*** *×40.*

cians should maintain a high index of suspicion in patients of all ages presenting with painful subcutaneous nodules, particularly if changes of DLE or lipoatrophy are observed. Given her recent weight loss, there was initial concern for SPTCL. Careful clinical evaluation and histopathologic examination are essential to differentiate LEP from SPTCL, as both involve inflammation of subcutaneous tissue and may present with indurated nodules or plaques (Table 1).[4,5]

Clinically, LEP lesions are more likely to heal with lipoatrophy, present with overlying erythema, and display classic DLE findings such as scaling, depigmentation, or ulceration. In contrast, SPTCL more commonly presents with nodular skin changes on the extremities and trunk, often accompanied by constitutional symptoms. Histopathologically, LEP is characterized by lobular lymphocytic infiltrate, hyaline fat necrosis, and pathological changes of DLE, including calcification, mucin deposition, and basement membrane degeneration, all observed in our patient.[11] LEP is less likely to demonstrate the rimming of atypical cells around fat cells seen in SPTCL.[1] SPTCL is further distinguished by Ki-67 hotspots, CD8+ T-cell predominance, and T-cell receptor gene rearrangement, none of which were present in our patient. Immunostaining in our case demonstrated characteristic LEP findings, including lymphoid follicles and a mixture of CD4+ and CD8+ T-cells.[4]

Treatment with methotrexate, folic acid, and prednisone resulted in clinical improvement. Other therapeutic options with reported efficacy include antimalarials, thalidomide, dapsone, immunosuppressants such as cyclosporine and cyclophosphamide, intravenous immunoglobulin, and rituximab.[5] This case underscores the importance of considering LEP in patients of all ages to ensure prompt treatment and prevent disfiguring sequelae such as lipoatrophy, scarring, and ulceration.

Conclusion

LEP should be considered in the differential diagnosis for patients of all ages presenting with painful subcutaneous nodules or plaques, particularly in those with a history of or concurrent signs of lupus. Histopathologic evaluation is essential for confirming the diagnosis. Early recognition and appropriate treatment can help prevent sequelae such as lipoatrophy, scarring, and ulceration. Given the association of LEP with SLE, close monitoring for systemic involvement is recommended. Patients who are treatment-resistant should be carefully evaluated to exclude SPTCL.

Potential conflicts of interest

The authors declare no conflicts of interest.

Table 1. *Comparative Clinicopathologic Features Between LEP and SPTCL.*

Feature	LEP	SPTCL
Onset age	Most commonly presents in young to middle-aged adults, typically between 30 and 40 years.[2,3]	Most commonly presents in young adults, typically between 20 and 30 years.[6,7]
Gender	Predominantly affects women, with reported female-to-male ratios ranging from 4.5:1 to 8.5:1.[2,3]	Predominantly affects women, with a reported female-to-male ratio of 2:1.[8]
Lesion morphology	Painful, firm, deep subcutaneous nodules or plaques. Lesions frequently heal with lipoatrophy.[2] Overlying skin changes may include features of DLE, such as erythema, scaling, depigmentation, or ulceration.[2,4]	Tender subcutaneous nodules or indurated plaques frequently associated with systemic symptoms such as fever, malaise, or weight loss.[4,7]
Location	Commonly involves areas with abundant subcutaneous fat, including the upper arms, thighs, trunk, buttocks, and breasts.[2]	Commonly involves the trunk and extremities, particularly the lower limbs.[4,7]
Associated disease	May occur as an isolated manifestation or develop before or after the onset of SLE or DLE.[9]	Associated with autoimmune diseases, particularly lupus erythematosus, and linked to viral infections such as Epstein-Barr virus and human immunodeficiency virus, as well as other lymphomas including cutaneous T-cell lymphoma and mycosis fungoides.[7,8,10]
Pathology	Lobular lymphocytic infiltrate, hyaline fat necrosis, and histopathologic features of DLE, including calcification, mucin deposition, and basement membrane degeneration.[11]	Lobular lymphocytic infiltrate with atypical cytotoxic T cells that may rim adipocytes. Higher Ki-67 proliferation index compared with LEP, $CD8^+$ T-cell predominance, and TCR gene rearrangement.[1]
Immunostains	Infiltrate contains $CD4^+$ and $CD8^+$ T cells, $CD20^+$ B-cell aggregates, abundant $CD123^+$ plasmacytoid dendritic cells, and a low Ki-67 proliferation index without hotspots.[4]	Atypical lymphocytes express CD3, CD8, TIA-1, and β-F1, with an inverted CD4:CD8 ratio and frequent Ki-67 hotspots more than 30%. $CD20^+$ B cells may be present at the periphery.[4]
Treatment	First-line therapy includes antimalarials such as hydroxychloroquine, chloroquine, and quinacrine, as well as corticosteroids. Other options include dapsone, thalidomide, methotrexate, azathioprine, and cyclophosphamide.[5]	Treatment options include corticosteroids, immunosuppressive agents such as cyclosporine or methotrexate, bexarotene, and chemotherapy.[12]
Prognosis	Prognosis is generally favorable with appropriate management. However, the disease often follows a chronic, relapsing course, and more than 2/3 of patients may experience sequelae such as lipoatrophy and hypopigmentation.[3,13]	Prognosis is generally favorable, with a 5-year overall survival rate of approximately 90%.[14]

Abbreviations: DLE, discoid lupus erythematosus; LEP, lupus erythematosus panniculiti; SLE, systemic lupus erythematosus; SPTCL, subcutaneous panniculitis-like T-cell lymphoma.

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