

Panniculitis-like T-cell Lymphoma: Two Different Diseases and Same Imitator

Ferdi ÖZTÜRK^{1*}, Yunus TOSO¹, Fazıl Çağrı HUNUTLU², Şaduman BALABAN ADIM³ and Hayriye SARICAOĞLU¹

¹Department of Dermatology, Bursa Uludağ University Faculty of Medicine, Bursa, Turkey, ²Department of Hematology, Bursa Uludağ University Faculty of Medicine, Bursa, Turkey, and ³Department of Pathology, Bursa Uludağ University Faculty of Medicine, Bursa, Turkey. Email: ferdiozturk@uludag.edu.tr

Submitted Feb 23, 2026. Accepted after revision Aug 27, 2026

Published Sept 8, 2026. DOI: 10.2340/actadv.v106.adv-2026-0439 *Acta Derm Venereol* 2026; 106: adv-2026-0439.

Subcutaneous panniculitis-like T-cell lymphoma (SPTCL) and primary cutaneous gamma/delta T-cell lymphoma (PCGD-TCL) are rare entities that present as indurated erythematous nodules and plaques (1, 2). Both conditions share common histopathological features, such as lobular panniculitis and rimming of individual fat cells by cytotoxic T-cell infiltrates. However, their clinical behaviour and prognosis differ significantly. On the other hand, lupus erythematosus panniculitis (LEP) lesions appear clinically identical but are unusual in distal lower extremities and the presence of lymphoid follicles, a mixed inflammatory infiltration including plasma cells and involvement of epidermis or dermis histopathology support LEP (3). Here, we present 2 cases that were initially misdiagnosed as LEP.

CASE REPORT

A 42-year-old female presented to our clinic with haemophagocytic syndrome (HPS). Eight months prior, she had been evaluated at a secondary care centre for erythematous discoid macular and plaque rashes on the extremities (Fig. 1A and B). She was treated with steroids with cutaneous lupus erythematosus diagnosis. Six months later, she developed fever, pancytopenia, elevated transaminases, hyperferritinaemia and hypofibrinogenaemia, consistent with HPS. A bone marrow biopsy confirmed hemophagocytosis. She was referred to our centre for further evaluation. At our institution, cutaneous biopsy demonstrated epidermal atrophy with orthokeratosis, diffuse vacuolar basal degeneration, increased myxoid matrix in the dermis. Marked subcutaneous lymphocytic infiltration and necrosis were also noted, findings consistent with LEP. However, ANA and ANA profile tests were negative, and the patient did not meet the criteria for systemic lupus erythematosus. Immunohistochemistry revealed CD3+, CD7-, CD30-, CD8-, CD4-, CD56+, perforin+, granzyme+, TIA-1+, TCR-delta+infiltration, confirming a diagnosis of PCGD-TCL (Fig. 1C–H). Epstein-Barr virus-encoded RNA (EBER) was negative. The patient achieved clinical remission with CHOP-E chemotherapy. Despite this, control PET-CT revealed diffusely increased metabolic activity with areas of heterogeneous density. New lesions subsequently developed. She had autologous stem cell transplantation after BEAM

chemotherapy. Subsequently, the patient exhibited steroid-resistant HPS features and was treated with tocilizumab, but she did not respond. While being prepared for allogeneic stem cell transplantation, the patient's haemodynamic condition deteriorated, and she passed away.

A 59-year-old male presented to our clinic with asymptomatic, scattered ulcerating indurated plaques and nodules, predominantly on the face and scalp (Fig. 2A and B). Cutaneous biopsy had suggested findings including basal vacuolar degeneration compatible with LEP. PET/CT imaging revealed multiple cervical and thoracic lymphadenopathies and subcutaneous heterogeneous nodular opacities (Fig. 2I and J). An excisional biopsy of a cervical lymph node showed nonsuppurative granulomatous lymphadenitis, raising suspicion for Kikuchi disease. Immunohistochemistry revealed that both in cutaneous and lymph node biopsies CD3+, CD7+, CD4-, CD56-, CD8+, perforin+, granzyme+ and TIA-1+T cells (Fig. 2E–H), TCR-beta clonal rearrangement was positive, and EBER was negative. Based on these findings, the definitive diagnosis of SPTCL was established. The patient was started on cyclosporine. Within 2 months, the lesions regressed, leaving behind significant atrophy, telangiectasia and central protuberant crusts with a yellow hue (Fig. 2C and D). The patient followed 2 years with low-dose (3.5 mg/kg/day) cyclosporine without progression.

DISCUSSION

Clinical and histopathologic presentation of both cases initially was compatible with LEP. Overlapping histological features of LEP and STPCL or PCGD-TCL include vacuolar interface dermatitis and lobular panniculitis (3). Magro et al. hypothesized that LEP and SPTCL form a spectrum and presented cases with a new entity called atypical lymphocytic lobular panniculitis to define cases in between (3). Presence of lymphoid follicles, mixed inflammatory infiltration support LEP (3). While clonal T-cell populations may be observed in LEP, clonality is more frequent in SPTCL and PCGD-TCL (4, 5). Our cases support that definitive diagnosis requires a combination of clinical presentation, histopathology (e.g. cell morphology, atypia, and infiltration patterns), immunohistochemical

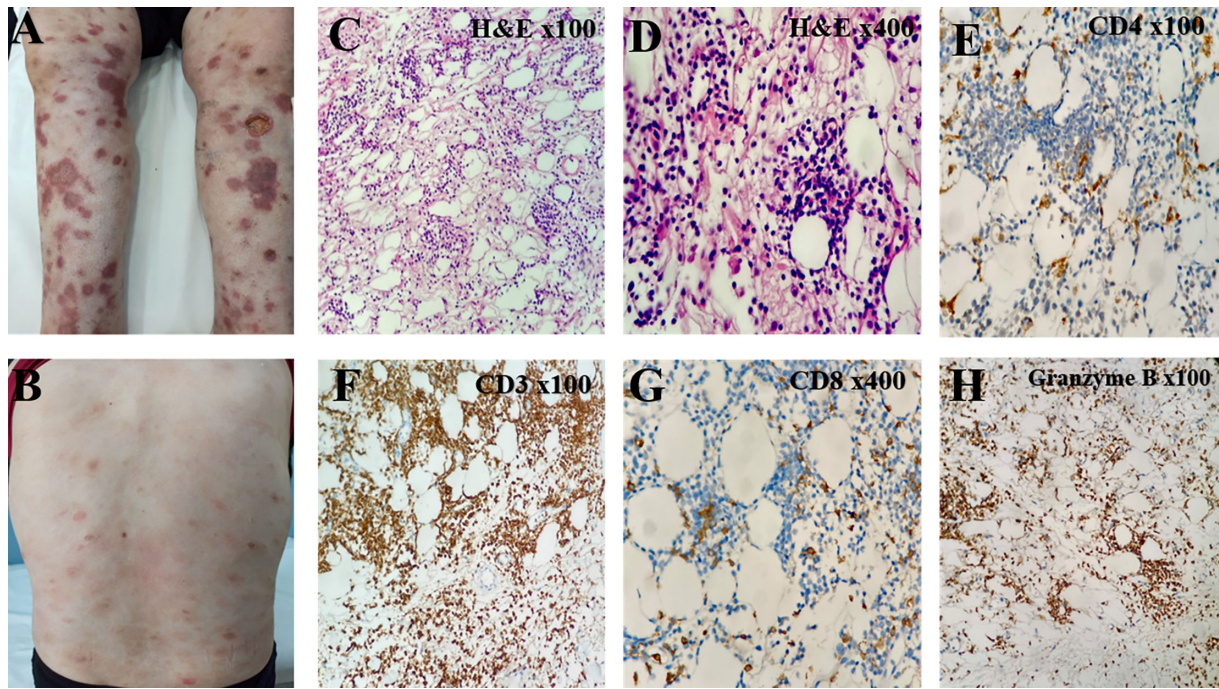


Fig. 1. Clinical and histological findings (Case 1). (A,B) Erythematous discoid macular and plaque rashes on the lower extremities and trunk. Dense atypical lymphoid infiltration in subcutaneous fat tissue (C) H&E 100x magnification, (D) H&E 400x magnification, (E) negative CD4 staining 100x magnification, (F) positive CD3 staining 100x magnification, (G) CD8 staining 400x magnification, (H) granzyme B staining 100x magnification.

staining (e.g. T-cell receptor subtype, CD8, CD56, cytotoxic proteins), clonality analysis and imaging (e.g. MRI, CT, PET) (3–5). Importantly, PET imaging revealed subcutaneous tissue infiltrations in both cases. In the first case, it was predictive of recurrence.

SPTCL has a favourable prognosis, with a 5-year survival rate exceeding 80% (1). Studies have shown no significant difference in response rates between immunosuppressive agents and chemotherapy in SPTCL

(1, 2). While HLH affects the prognosis in SPTCL cases, PCGD-TCL has a poor prognosis regardless of HLH or treatment type (1, 2). Combination chemotherapy is the initial treatment for PCGD-TCL (1, 5). Stem cell transplantation is the only possible cure. Despite the whole current treatment options, for PCGD-TCL recurrence is still almost inevitable.

SPTCL and PCGD-TCL are now recognized as distinct disease entities, although they were previously

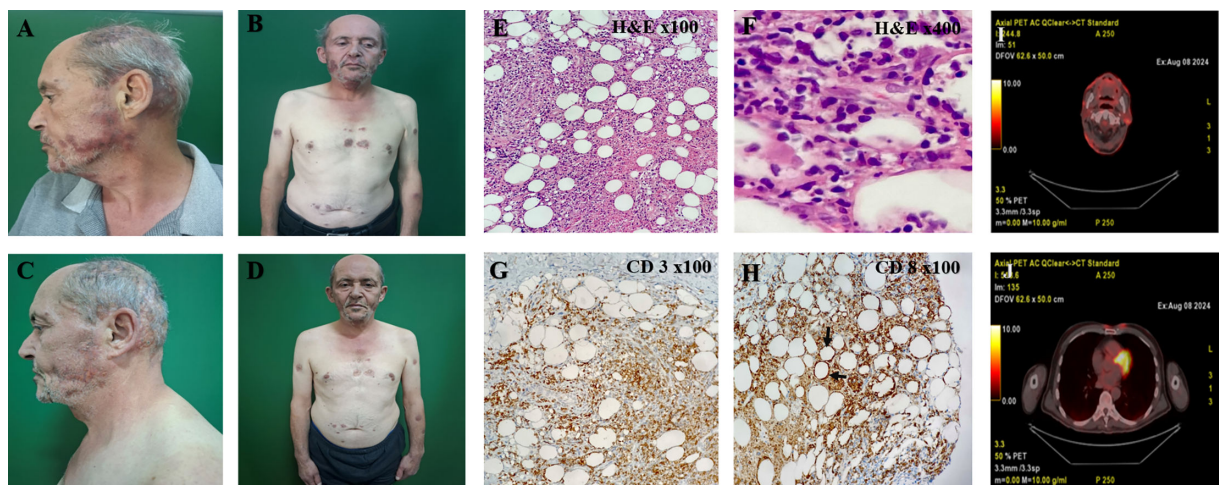


Fig. 2. Clinical and histological findings (Case 2). (A,B) Scattered indurated plaques and nodules, predominantly on the face and scalp. (C,D) Depressive alopecia in broad, irregular areas of the scalp and beard following treatment, consistent with recovery from the disease and post-inflammatory changes. Atypical lymphoid infiltration causing necrosis in subcutaneous fat tissue. (E) H&E 100x magnification, (F) H&E 400x magnification, (G) CD3 staining 100x magnification, (H) CD8 staining 100x magnification. Arrows show rimming of adipocytes by lymphocytes. (I,J) PET/CT imaging shows nodular lesions with FDG uptake in subcutaneous tissue.

considered phenotypic variations of the same condition. In this report, we presented 2 cases with overlapping clinical and histological features suggestive of LEP. Despite advancements in our understanding over the years, the diagnosis of these lymphomas remains challenging, and current treatment options are not universally effective. Both SPTCL and PCGD-TCL exhibit significant heterogeneity in their clinical presentation and progression. The existing classification is insufficient to capture the complexity of all cases. Further research is essential to improve our understanding of these diseases and to develop more effective diagnostic and therapeutic strategies.

ACKNOWLEDGEMENTS

Data availability statement: The data that support the findings of this study are available from the corresponding author upon reasonable request.

Ethics: Written informed consent for publication was obtained from the patients.

The authors have no conflicts of interest to declare.

REFERENCES

1. Willemze R, Jansen PM, Cerroni L, Berti E, Santucci M, Assaf C, et al. Subcutaneous panniculitis-like T-cell lymphoma: definition, classification, and prognostic factors: an EORTC Cutaneous Lymphoma Group Study of 83 cases. *Blood* 2008; 111: 838–845. <https://doi.org/10.1182/blood-2007-04-087288>
2. Wolff K, Goldsmith L, Katz S, Gilchrest B, Paller AS, Leffell D. *Fitzpatrick's Dermatology in General Medicine*. 8th Edition. McGraw-Hill; 2011: 2086–2088 pp.
3. Magro CM, Kalomeris T, Dillard A. Panniculitic primary cutaneous gamma delta T-cell lymphoma with concomitant features of autoimmune disease emphasizing a pathophysiologic continuum of lupus profundus with the panniculitic T cell lymphomas. *Clin Dermatol* 2023; 41: 680–691. <https://doi.org/10.1016/j.clindermatol.2023.09.008>
4. Magro CM, Wang X. Indolent primary cutaneous γ/δ T-cell lymphoma localized to the subcutaneous panniculus and its association with atypical lymphocytic lobular panniculitis. *Am J Clin Pathol* 2012; 138: 50–56. <https://doi.org/10.1309/AJCPQGVLTZQ77VFF>
5. Guitart J, Mangold AR, Martinez-Escala ME, Walker CJ, Comfere NI, Pulitzer M, et al. Clinical and pathological characteristics and outcomes among patients with subcutaneous panniculitis-like T-cell lymphoma and related adipotropic lymphoproliferative disorders. *JAMA Dermatol* 2022; 158: 1167–1174. <https://doi.org/10.1001/jamadermatol.2022.3347>