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**LETTER TO THE EDITOR**

# Panniculitis-like presentation as the initial manifestation of peripheral T-cell lymphoma, not otherwise specified

## *Apresentação semelhante a paniculite como manifestação inicial de linfoma de células T periféricas, sem outra especificação*

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Dear Editor,

Panniculitis encompasses a heterogeneous group of inflammatory disorders of subcutaneous adipose tissue, including infectious, immune-mediated, and neoplastic conditions. Fever associated with an acute inflammatory plaque usually suggests infection; however, lymphoid malignancies may produce a clinically indistinguishable panniculitis-like presentation.

A 58-year-old female with type 2 diabetes, hypertension, and dyslipidemia presented with a 1-month history of fever, night sweats, and a painful inflammatory lesion on the posterior right thigh. The lesion had appeared 3 days after hiking in a rural area near Zurich, with no recalled insect bite, animal contact, or trauma. An infectious etiology was initially suspected. Before our evaluation, the patient was treated sequentially with amoxicillin–clavulanate, ciprofloxacin, ceftriaxone, and clindamycin, but the lesion kept progressing. Examination showed a large, infiltrated, erythematous-violaceous plaque with edema, central crusting, and marked local inflammatory signs (Fig. 1).

Laboratory testing revealed microcytic anemia (hemoglobin 11.3 g/dL; mean corpuscular volume 74 fL), leukopenia (1.41 × $10^9$/L), aspartate aminotransferase 1298 U/L, alanine aminotransferase 659 U/L, lactate dehydrogenase 1979 U/L, creatine kinase 666 U/L, and C-reactive protein 100 mg/L. Thigh computed tomography (CT) demonstrated diffuse skin thickening extending over 12 cm, with two nodular areas of deeper tissue densification. Initial thoraco-abdominopelvic CT identified several right inguinal lymph nodes.

Blood and tissue investigations for bacterial, mycobacterial, fungal, and vector-borne infections were negative, including studies for *Borrelia*, *Rickettsia*, *Coxiella burnetii,* and *Babesia*. Autoimmune investigation showed normal immunoglobulins and complement, no monoclonal peak, negative anti-double-stranded DNA, antineutrophil cytoplasmic antibodies, antimitochondrial, and anti-smooth muscle antibodies, and an antinuclear antibody titer of 1:160.

A deep incisional biopsy revealed a predominantly subcutaneous lymphoid infiltrate composed of atypical medium-to-large cells, associated with lipogranulomas and areas of necrobiosis (Fig. 2A and B). The atypical cells expressed CD2 and CD3 (Fig. 2C), with focal CD30 expression (Fig. 2D), and lacked CD5, CD7, CD8, and CD20. Epstein–Barr virus-encoded RNA was negative. Flow cytometry of the skin biopsy did not identify a $\gamma\delta$ T-cell receptor population, and microbiological examination of the tissue was negative.

The absence of a typical lobular adipocyte-rimming pattern and of the characteristic CD8-positive $\alpha\beta$ cytotoxic phenotype argued against subcutaneous panniculitis-like

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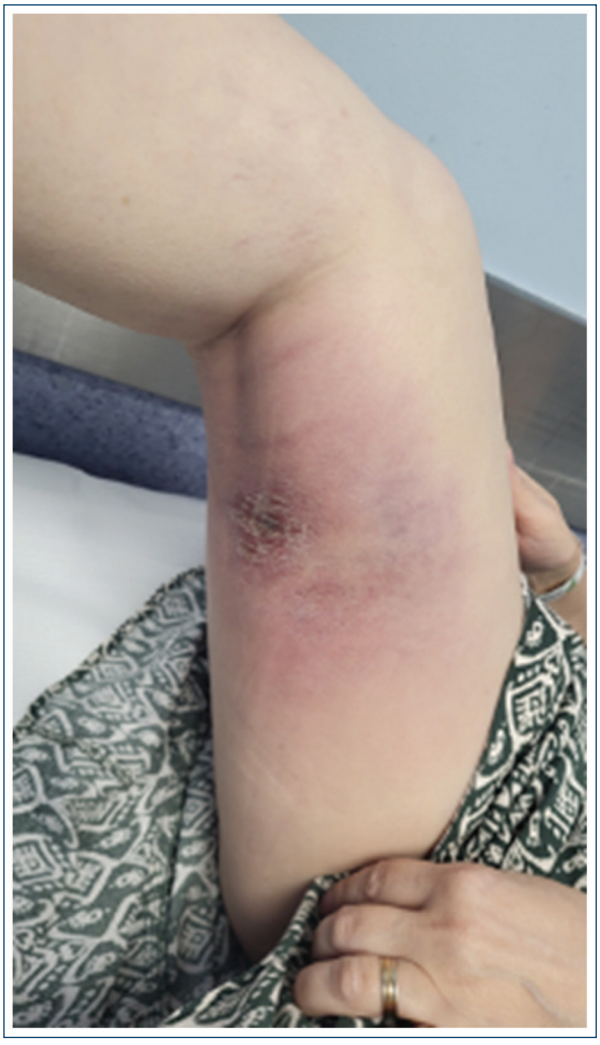


**Figure 1.** Infiltrated erythematous-violaceous plaque with edema and central crusting on the posterior right thigh at presentation.

T-cell lymphoma (SPTCL). The negative γδ T-cell receptor study did not support primary cutaneous γδ T-cell lymphoma. The lack of epidermotropism and only focal CD30 expression also argued against mycosis fungoides and anaplastic large-cell lymphoma, respectively. Integration of the clinical, morphological, and immunophenotypic findings supported peripheral T-cell lymphoma, not otherwise specified (PTCL-NOS), a diagnosis of exclusion according to current classifications.[1,2]

Subsequent staging with fluorodeoxyglucose (FDG) positron emission tomography (PET)/CT demonstrated metabolically active lymphadenopathy involving the para-aortic chains, right internal and external iliac, and right inguinal. Ultrasound-guided lymph node biopsy showed reactive changes; however, a limited amount of tissue was obtained. Peripheral-blood immunophenotyping was unremarkable, whereas bone-marrow and liver biopsies showed no evidence of lymphoma. Given the systemic symptoms, rapid unfavorable clinical progression, and distribution and intensity of nodal FDG uptake on PET-CT, the disease was considered a systemic PTCL-NOS, stage IVB, with cutaneous involvement presenting as panniculitis. The patient was treated with cyclophosphamide, doxorubicin, vincristine, etoposide, and prednisone (CHOEP) and was subsequently followed by the hematology team.

Primary cutaneous lymphomas with predominantly subcutaneous involvement represent the principal pathological differential diagnosis.[3-5] Importantly, a multicenter series of SPTCL and related adipotropic lymphoproliferative disorders found no nodal or visceral tumoral progression in confirmed SPTCL, supporting reconsideration of this diagnosis when distant lymphadenopathy is present.[6]

Cutaneous involvement in systemic PTCL-NOS has been documented, including presentations with multiple nodules, plaques, erythroderma, or ulcerated lesions.[7-9] Nevertheless, an acute solitary plaque mimicking infectious panniculitis appears exceptional. In the present case, the epidemiological context and inflammatory appearance initially reinforced an infectious hypothesis. Failure of multiple antimicrobial regimens, systemic abnormalities, and early deep biopsy were decisive in redirecting the investigation.

The initial combination of persistent fever, cytopenias, marked transaminase and lactate dehydrogenase elevation, and systemic inflammation also raised suspicion of secondary hemophagocytic lymphohistiocytosis, a recognized complication of peripheral T-cell lymphomas.[4-6]

PTCL-NOS is generally associated with an aggressive clinical course and poor outcomes, with prognosis influenced by clinical risk factors and response to treatment; retrospective evidence suggests poorer survival in patients with multifocal lesions and concurrent cutaneous and systemic involvement.[10] Prognostic assessment should incorporate age, performance status, serum lactate dehydrogenase, disease stage, and bone marrow involvement, using established indices such as the international prognostic index (IPI) or prognostic index for peripheral T-cell lymphoma (PIT).[11] For systemic PTCL-NOS, CHOP, or CHOEP remains a recommended first-line approach, whereas consolidative autologous stem-cell transplantation may be considered in eligible responding patients with advanced or high-risk disease; radiotherapy may complement systemic treatment in selected localized presentations.[12]

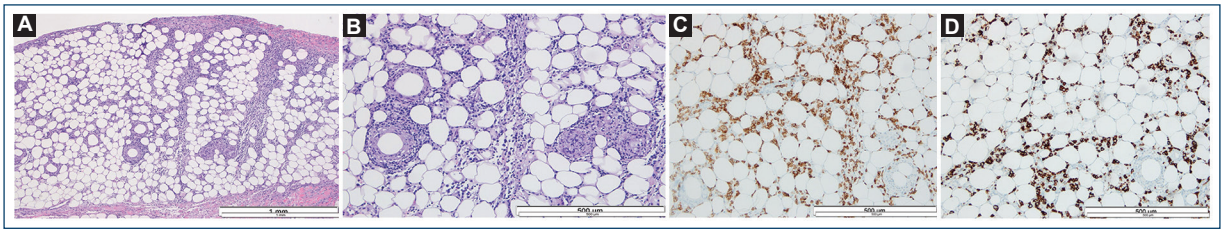



**Figure 2.** Histopathological and immunohistochemical findings. **A:** predominantly subcutaneous lymphoid infiltrate (H&E, scale bar 1 mm). **B:** atypical medium-to-large lymphoid cells and lipogranulomas (H&E, scale bar 500-1000 μm). **C:** CD3 expression by the atypical infiltrate. **D:** focal CD30 expression.

Persistent febrile panniculitis that fails to respond to appropriate antimicrobial treatment should therefore prompt deep tissue sampling, parallel microbiological investigation, and assessment for lymphoproliferative disease. Close integration of dermatological, hematological, radiological, and dermatopathological findings is essential to distinguish systemic peripheral T-cell lymphoma with cutaneous involvement from primary cutaneous T-cell lymphomas, as this distinction has major therapeutic and prognostic implications.[1-7,10-12]

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None.

## Conflicts of interest

None.

## Ethical considerations

**Protection of human subjects and animals.** The authors declare that no experiments on humans or animals were performed for this research.

**Confidentiality, informed consent, and ethical approval.** The authors have followed their institution's confidentiality protocols, obtained informed consent from all patients, and secured approval from the Ethics Committee. SAGER guidelines have been followed as applicable to the nature of the study.

**Declaration on the use of artificial intelligence.** The authors declare that no generative artificial intelligence was used in the writing or creation of the content of this manuscript.

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