Dermatology Practical & Conceptual

Type C Lymphomatoid Papulosis Following Immune Checkpoint Inhibition in a Melanoma Patient: A Reactive Mimicker of Cutaneous CD30$^+$ Lymphoma

Alex Moscardi[1]*, Jacopo Tartaglia[1]*, Christian Ciolfi[1], Mauro Alaibac[1]

1 Unit of Dermatology, Department of Medicine, University of Padova, Italy

* These authors are equal contributors

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Corresponding Author: Mauro Alaibac, Dermatology Unit, Department of Medicine (DIMED), University of Padua; Via Gallucci, 4, 35128 Padua, Italy. E-mail: mauro.alaibac@unipd.it

Introduction

Lymphomatoid papulosis (LyP) is a chronic CD30$^+$ cutaneous lymphoproliferative disorder with a benign clinical course despite histopathologic resemblance to malignant lymphoma. Among its subtypes, Type C mimics primary cutaneous anaplastic large cell lymphoma (pcALCL), presenting clinically as ulcerated nodules composed of sheets of large atypical CD30$^+$ T cells [1]. We report a case of LyP Type C following immune checkpoint inhibitor therapy for metastatic melanoma, supporting the view that LyP is a reactive rather than neoplastic process, and illustrating a potential diagnostic pitfall in this context.

Case Presentation

A 62-year-old man with a history of two primary melanomas, one with a Breslow thickness of 1.3 mm on the left nipple in 2017 and a stage IIIB melanoma with axillary nodal involvement in 2021, received vemurafenib plus cobimetinib in early 2022. The treatment course was complicated by a purpuric rash requiring dose reduction. He subsequently received one year of adjuvant atezolizumab.

A few months after initiating immune checkpoint blockade, the patient presented with scattered erythematous to violaceous papulonodular lesions on the face and limbs, several of which showed central ulceration (Figure 1A, 1C). The eruption followed a relapsing-remitting course, and resolving lesions left residual hyperpigmented macules consistent with post-inflammatory dyspigmentation. Dermoscopy revealed a central violaceous to whitish structureless area, accompanied by diffuse scaling and peripheral uniform dotted vessels (Figure 1B, 1D).

A skin biopsy from the lower leg showed a dense dermal infiltrate of large CD30+ atypical lymphocytes with a mixed CD4+/CD8+ phenotype. No epidermotropism or

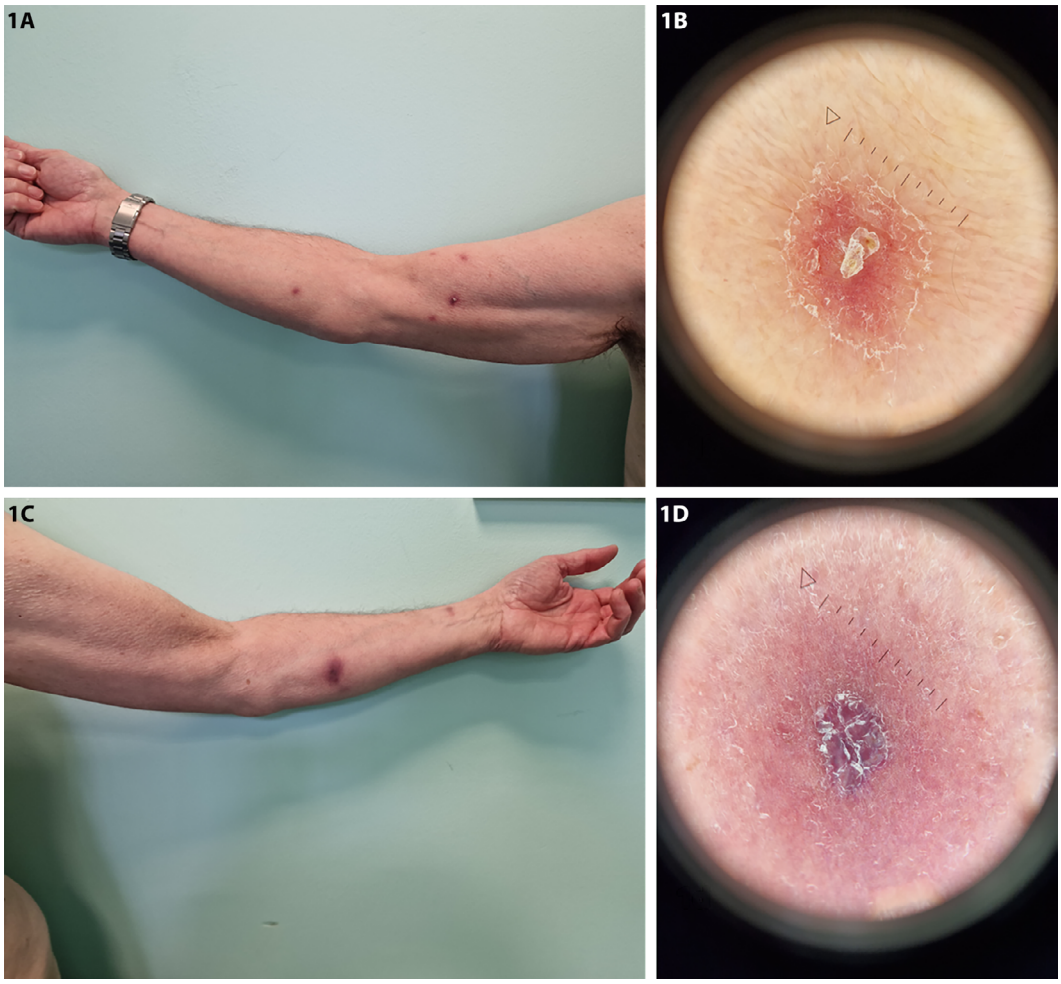

Figure 1. (A, C) Multiple erythematous to violaceous papulonodular lesions on the patient's arms. (B, D) Dermoscopic view revealing a violaceous to whitish structureless area with diffuse scaling and peripheral dotted vessels.

angioinvasion was observed. These findings supported a diagnosis of LyP Type C.

Discussion

Although histologically indistinguishable from pcALCL, LyP Type C lacks the aggressive course of true cutaneous CD30+ lymphomas, with spontaneous lesion regression and excellent prognosis. In this case, the onset shortly after checkpoint blockade suggests a delayed immune-mediated trigger. Immune checkpoint inhibitors are known to disrupt peripheral tolerance and enhance T-cell activation, frequently unmasking inflammatory dermatoses such as vitiligo, bullous pemphigoid, lichenoid eruptions, and granulomatous dermatitis [2].

LyP is exceptionally rare in immunosuppressed individuals, including transplant recipients, despite their elevated lymphoma risk [3]. Ferranti et al. argue against a causal link between immunosuppression and LyP, pointing out that large post-transplant registries report either no cases or isolated ones, which likely represent exceptions rather than evidence of association [4]. This suggests that LyP arises in settings of preserved or heightened immune surveillance. Our case supports the interpretation of LyP as a benign, reactive lymphoid process rather than a malignant one.

The dermoscopic features observed, including hyperkeratotic scaling, violaceous-whitish structureless areas, and peripheral dotted vessels, are consistent with previously reported patterns in LyP and may assist in supporting the diagnosis in clinically ambiguous presentations [5].

Conclusion

This case illustrates the potential for immune checkpoint inhibition to trigger reactive lymphoid proliferations mimicking aggressive lymphoma. Recognition of the benign nature of LyP, particularly Type C, is critical to avoid overtreatment, and awareness of its occurrence post-immunotherapy may assist in clinical decision-making.

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