SHORT COMMUNICATION

Mantle Cell Lymphoma Presenting as a Primary Cutaneous Mass: A Case Report

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Cutaneous involvement of mantle cell lymphoma (MCL) remains uncommon and can mimic inflammatory dermatoses or primary cutaneous B-cell lymphomas, delaying diagnosis and therapy. Non-classic (blastoid/pleomorphic) variants may present with rapidly enlarging plaques or nodules that ulcerate. We report a 73-year-old man whose first recognized manifestation of systemic MCL was multifocal ulcerating plaques of the thigh and upper back. The case underscores the diagnostic value of the characteristic immunophenotype and illustrates an early cutaneous response to zanubrutinib plus R-miniCHOP (1–3).

CASE REPORT

A 73-year-old man presented in May 2025 with a 2-month history of enlarging ulcerated plaques on the lateral left thigh and upper back. The initial thigh lesion began as an infiltrated erythema, the size of a fingernail, and evolved into a round, indurated plaque with central ulceration and black–brown crusts; a violaceous infiltrated rim developed at the periphery. A morphologically similar lesion then appeared on the upper back and ulcerated with bloody exudation. He reported no pain, pruritus, systemic B symptoms or relevant personal/family history. Examination showed firm, immobile, non-tender, erythematous-to-violaceous indurated plaques (2–5 cm) with central necrosis/ulceration and an erythematous rim on the upper back and left thigh (**Fig. 1**A and D).

A biopsy of the left-thigh lesion revealed diffuse sheets of small- to medium-sized lymphoid cells with effacement of follicular architecture (**Fig. 2**A and B). Immunohistochemistry showed CD20 positivity with co-expression of CD5 and cyclin D1; SOX11 was weakly positive, p53 was positive and the Ki-67 index was ~60%. Tumour cells were negative for CD3, BCL6, CD10 and MUM1(Fig. 2C–L). These findings supported non-classic MCL with blastic features. Staging was IIIA with cutaneous involvement (sMIPI 5, intermediate risk).

After diagnosis, the patient underwent local excision of the soft-tissue lesion (Fig. 1B and E). Postoperatively, the patient commenced zanubrutinib-augmented R-miniCHOP: oral zanubrutinib 160 mg twice daily plus rituximab 375 mg/m² (day 1), cyclophosphamide 400 mg/m² (day 1), doxorubicin 25 mg/m² (day 1), vincristine 1 mg (day 1) and prednisone 40 mg/m² (days 1–5), every 21 days. After the second cycle, the cutaneous lesions regressed markedly (Fig. 1C and F).

DISCUSSION

MCL is an aggressive B-cell non-Hodgkin lymphoma that usually involves lymph nodes and extranodal sites. Clinically evident skin disease remains uncommon and most often reflects secondary dissemination rather than a primary cutaneous lymphoma entity. Recognizing secondary cutaneous involvement is important because WHO-HAEM5 classifies MCL as a systemic lymphoma and clinically apparent skin lesions usually represent secondary disease; this affects staging and generally indicates haematology-led systemic therapy rather than skin-directed measures (1, 3, 4). Our patient's lesions – indurated plaques with central necrosis/ulceration – fit reported morphologies of secondary MCL in the skin and soft tissue (3, 5). The biopsy showed a monomorphic small-to-medium B-cell infiltrate with loss of follicular architecture; immunophenotyping (CD20+, CD5+, cyclin D1+, SOX11+, CD10−/BCL6−/MUM1−) and a high Ki-67 index supported the diagnosis of MCL and effectively excluded primary

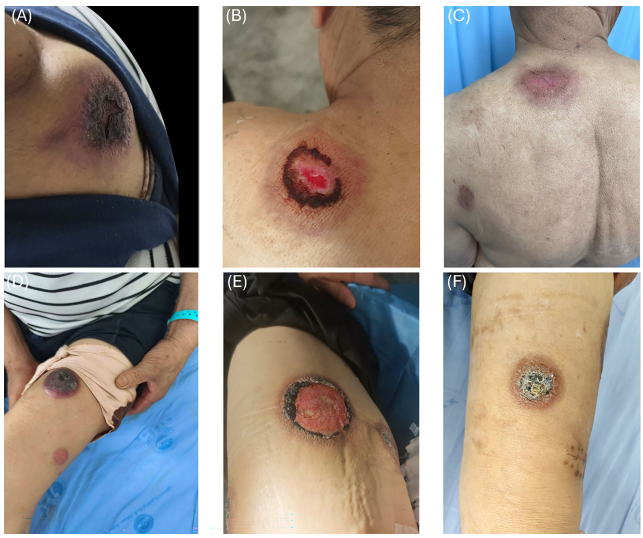

Fig. 1. Clinical evolution of the cutaneous lesions. Back: pre-treatment. (A), post soft-tissue excision (week 1) (B), and post cycle 2 (C). Left lateral thigh: pre-treatment (D), post soft-tissue excision (week 1) (E) and post cycle 2 (F).

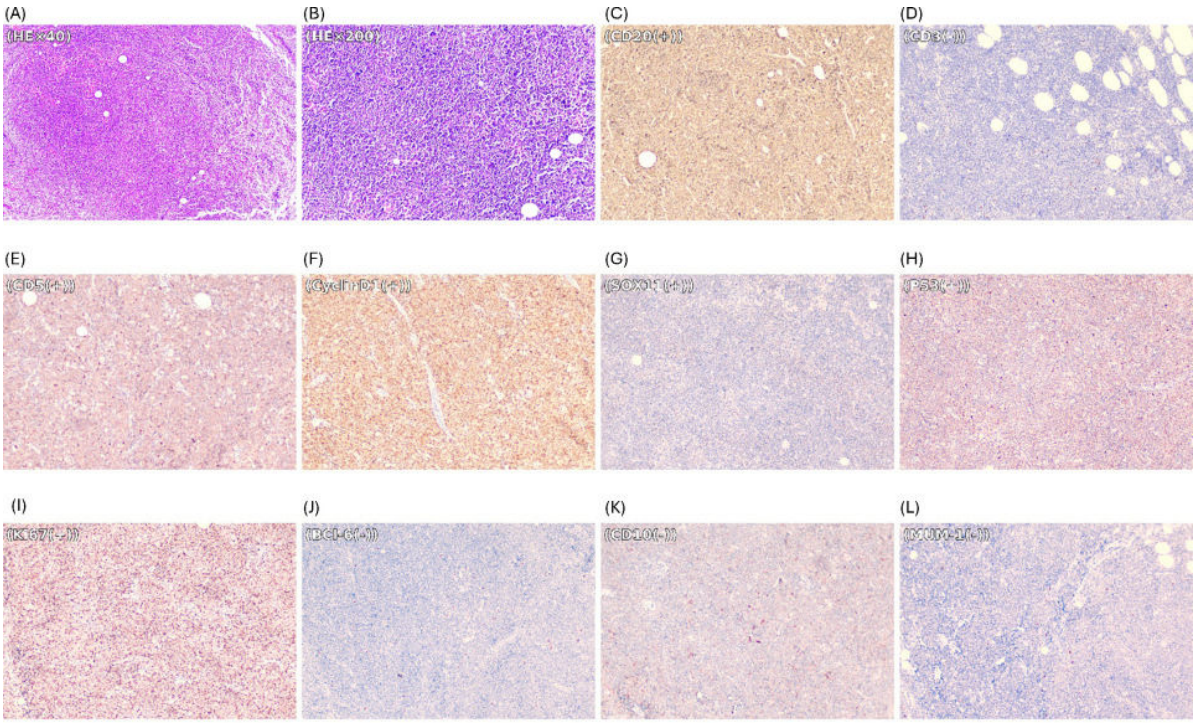

Fig. 2. Histopathology and immunohistochemistry of skin tissue from the left thigh. (A) HE×40, (B) HE×200, (C) CD20 positive, (D) CD3 negative, (E) CD5 positive, (F) cyclin D1 positive, (G) SOX11 positive, (H) P53 positive, (I) Ki67 positive (~60%), (J) BCl-6 negative, (K) CD10 negative (L) MUM-1 negative

cutaneous follicle centre lymphoma, marginal zone lymphoma, and diffuse large B-cell lymphoma (DLBCL) (1, 3, 6).

From a diagnostic perspective, SOX11 remains a useful marker when assessing cutaneous or blastoid-appearing B-cell proliferations, including cyclin D1-negative scenarios. Recent series reinforce its practical specificity for MCL among large/blastoid B-cell neoplasms and help avoid misclassification as DLBCL or high-grade B-cell lymphoma (6). WHO-HAEM5 maintains MCL as a unified entity with morphologic variants, while emphasizing the clinical impact of proliferative activity and TP53 status (1). A Ki-67 index of ≈60% and p53 positivity in our patient indicate elevated biological risk. Contemporary cohorts show that Mantle Cell Lymphoma International Prognostic Index (MIPI) and its combined variant, MIPI-c (which adds Ki-67 to MIPI) and p53 overexpression/TP53 alteration together define a high-risk subset with inferior outcomes across treatment backbones; even intensive strategies may not fully abrogate this risk (2, 7). Despite an intermediate sMIPI, the marked proliferative activity supports instituting systemic treatment and close surveillance.

From a therapeutic standpoint, Bruton tyrosine kinase (BTK)-directed approaches have reshaped MCL management across fitness levels. In older/unfit patients, SHINE trial showed adding ibrutinib to bendamustine–rituximab prolonged progression-free survival, validating frontline combinations (8). In younger/fit patients, TRIANGLE trial improved outcomes by layering ibrutinib onto intensive immunochemotherapy, with or without autologous transplant (9). Zanubrutinib achieves durable responses in relapsed/refractory disease and is being tested in combination regimens (2, 10). In our patient, early cutaneous improvement with zanubrutinib plus R-miniCHOP aligns with the robust BTK inhibitor activity observed in systemic disease and with current practice trends captured in guidelines (2, 4, 10). Soft-tissue/skin masses can respond rapidly to effective systemic therapy (5), but longer follow-up is required to determine the durability of response, particularly when the Ki-67 index is high or p53 is overexpressed (2, 7).

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

The authors have no conflicts of interest to declare.

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