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LETTER – THERAPY

Durvalumab-induced lichenoid eruption: expanding a rarely recognized adverse event and review of the literature☆

Dear Editor,

Immune Checkpoint Inhibitors (ICIs) have markedly improved the prognosis of several malignancies but are associated with a variety of immune-related Adverse Events (irAEs), many of which affect the skin.[1] The underlying pathophysiology involves loss of peripheral immune tolerance due to PD-L1 blockade, leading to overactivation of cytotoxic CD8+ T-cells and, in some cases, B-cell–mediated autoimmunity. This immune dysregulation results in T-cell infiltration and inflammation of the skin, manifesting as dermatologic irAEs.[2] A wide spectrum of skin irAEs has been described with anti–PD-L1 agents, including lichenoid dermatitis, bullous pemphigoid, psoriasiform or spongiotic dermatitis, vitiligo and even severe toxicities such as Stevens-Johnson syndrome and toxic epidermal necrolysis-like reactions.[1,2] Durvalumab, a fully human monoclonal antibody targeting PD-L1, is currently approved for unresectable stage III non-small cell lung cancer and advanced urothelial carcinoma. While its safety profile is consistent with that of other ICIs, cutaneous toxicities such as lichenoid reactions remain rare and likely underreported.[3,4]

We present the case of a 64-year-old man with stage IIIB lung adenocarcinoma treated with chemoradiotherapy followed by one year of maintenance durvalumab. Three months after initiating immunotherapy, he developed symmetrical erythematous-violaceous plaques with Wickham's striae on both forearms and dorsal hands (Fig. 1). The lesions were refractory to low-dose oral corticosteroids (10 mg/day) and potent topical corticosteroids. Histopathology revealed a lichenoid infiltrate with basal vacuolar degeneration, consistent with a drug-induced lichenoid reaction (Fig. 2). Treatment with calcipotriol/betamethasone and topical tacrolimus 0.1% led to partial improvement. As the lesions remained asymptomatic, durvalumab was continued. Complete resolution occurred following the completion of durvalumab therapy.

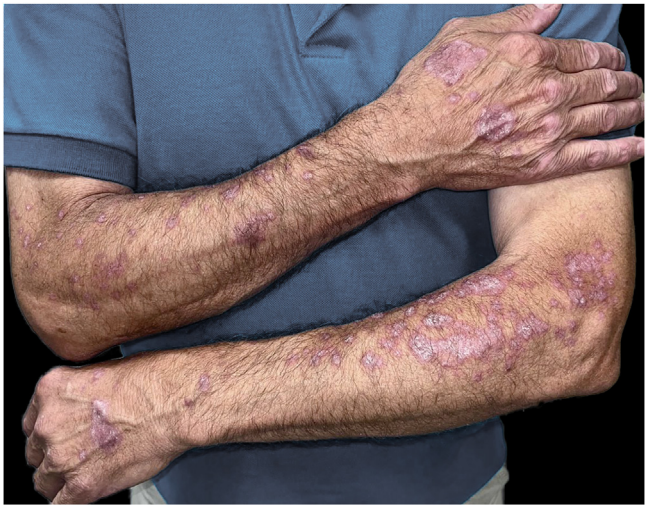

Fig. 1 Clinical appearance of erythematous-violaceous plaques on the dorsal hands and forearms, three months after initiating durvalumab therapy and prior to starting dermatologic treatment.

Lichenoid dermatitis is a recognized irAE of ICIs, especially PD-1 inhibitors such as nivolumab and pembrolizumab.[3,5,6] However, reports of this reaction under durvalumab are scarce. To our knowledge, only three previous cases of durvalumab-induced lichenoid eruptions have been documented.[7–9] These cases display heterogeneous clinical presentations ranging from hypertrophic variants mimicking squamous cell carcinoma to lichenoid reactions progressing to bullous forms or involving mucosal surfaces, including the esophagus. Table 1 summarizes the four reported cases, including the present one. Notably, our patient's presentation was purely cutaneous and non-bullous, managed entirely with topical therapy without the need for systemic immunosuppression or treatment interruption. The temporal relationship and complete resolution after durvalumab discontinuation further support causality.

Histopathologically, lichenoid eruptions induced by PD-1/PD-L1 inhibitors, such as durvalumab, may show greater

☆ Study conducted at the Hospital Universitario Virgen de las Nieves, Granada, Spain.

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Table 1 Comparison of reported cases of durvalumab-induced lichenoid eruption.

Feature	Myrdal et al. (2020)	Manko et al. (2021)	Mansilla-Polo et al. (2025)	García-Moronta et al, present case (2026)
Age, Sex	Female, 73-years	Female, 62-years	Male, 68-years	Male, 64-years
Underlying malignancy	Lung adenocarcinoma (stage IIIA)	Metastatic squamous cell carcinoma of unknown origin	Cholangiocarcinoma	Lung adenocarcinoma (stage IIIB)
Type of eruption	Hypertrophic LP	LP → LPP	Extensive LP	Skin-limited LP
Time of onset (after durvalumab)	2-months	2-years	6 cycles (∼3-months)	3-months
Lesion location	Lower limbs, forearms, chest	Trunk, limbs, face, oral mucosa	Limbs (palms included), oral and esophagus mucosa	Upper limbs
Histopathology	Epithelial proliferation with lichenoid inflammation	Lichenoid infiltrate → Subepidermal bullae, epidermal necrosis. IFD: IgG y C3	Lichenoid infiltrate	Lichenoid infiltrate with basal vacuolar degeneration
Treatment	Topical corticosteroids	Oral prednisone 60 mg/24 h, topical corticosteroids (durvalumab Paused)	Oral prednisone 45 mg/24 h + topical corticosteroids	Calcipotriol/ betamethasone + tacrolimus
Clinical course	Improved with topicals	Recurrent after durvalumab reintroduction	Resolved without durvalumab withdrawal	Resolved after durvalumab completion
Notable features	Mimicked squamous cell carcinoma	Phototherapy-triggered bullous transformation	Endoscopic-confirmed esophageal involvement	Exclusively cutaneous, asymptomatic after treatment

LP, Lichen Planus; LPP, Lichen Planus Pemphigoides.

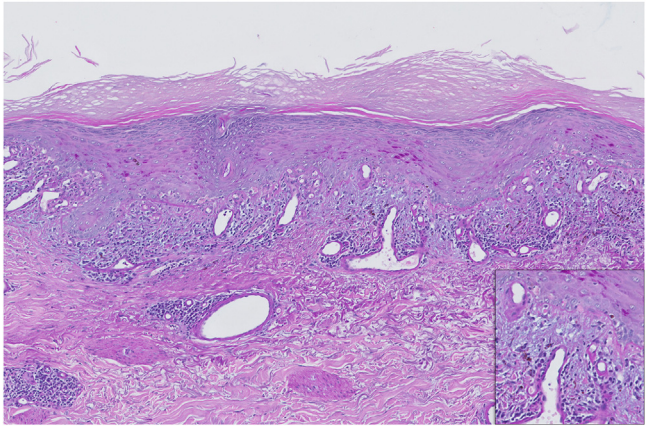

Fig. 2 PAS-stained section of an incisional biopsy from the patient, showing orthokeratotic hyperkeratosis with a thickened granular layer. At higher magnification, basal vacuolar degeneration with numerous apoptotic keratinocytes, blurred dermoepidermal junction, a band-like lymphoplasmacytic infiltrate, and abundant melanophages in the papillary dermis are evident. These findings are consistent with a lichenoid drug eruption.

spongiosis, epidermal necrosis, and a denser histiocytic infiltrate (CD163+) compared to idiopathic lichen planus. These features, although subtle, can support the diagnosis in the appropriate clinical context.[10]

This case broadens the phenotypic spectrum of durvalumab-induced lichenoid eruptions and highlights the importance of early recognition, as some cases may progress to bullous variants.[7] These reactions typically appear early after treatment initiation and often respond well to symptom-guided conservative therapy. Prompt diagnosis and management can allow the continuation of potentially life-prolonging oncologic treatment without the need for systemic immunosuppression or treatment interruption.[7–9] This case underscores the importance of conservative management, allowing continuation of potentially life-prolonging oncologic therapy when feasible.[1,7]

ORCID ID

Daniel Muñoz-Barba: 0009-0003-8823-5575
Francisco Javier León-Pérez: 0009-0001-6938-7237
Francisco Manuel Ramos-Pleguezuelos: 0009-0009-8829-7251

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Authors' contributions

Carmen García-Moronta: Study conception and planning; preparation and writing of the manuscript; critical literature review; approval of the final version of the manuscript.

Daniel Muñoz-Barba: Approval of the final version of the manuscript.

Francisco Javier León-Pérez: Approval of the final version of the manuscript.

Francisco Manuel Ramos-Pleguezuelos: Preparation and writing of the manuscript; approval of the final version of the manuscript.

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Research data availability

Does not apply.

Conflicts of interest

None declared.

Editor

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Carmen García-Moronta [a,d], Daniel Muñoz-Barba [a,d], Francisco Javier León-Pérez [a,d], Francisco Manuel Ramos-Pleguezuelos [b], Manuel Sánchez-Díaz [a,d,*], Salvador Arias-Santiago [a,c,d]

[a] *Department of Dermatology, Hospital Universitario Virgen de las Nieves, Granada, Spain*
[b] *Department of Pathology, Hospital Universitario Virgen de las Nieves, Granada, Spain*
[c] *Biosanitary Institute of Granada, Granada, Spain*
[d] *IBS.Granada Instituto de Investigación Biosanitaria de Granada. Granada, Spain*

* Corresponding author.
E-mail: manolo.94.sanchez@gmail.com, msanchezdiaz@ugr.es (M. Sánchez-Díaz).

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