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Dermatology

Metformin Induced Lichenoïd Drug Eruption

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Abstract **Case Report**

Cutaneous lichenoid drug eruptions are rare adverse drug reactions reminiscent of lichen planus. The most frequently reported culprit drugs are Checkpoint inhibitors, tyrosine kinase inhibitors and TNF-α inhibitors, but other drugs can also be responsible. This case-report is about a 49-year-old woman with a history of type 2 diabetes. She started taking metformin 160 weeks before the onset of a pruritic lichenoid rash on the trunk and limbs, of which the histopathology was in favor of an adverse cutaneous lichenoid drug eruption caracterized by the presence of eosinophilic polynuclei in the perivascular infiltrate. The patient benefited from oral corticosteroids with discontinuation of metformin which resulted in the resumption of the lichenoid rash.

Keywords: Cutaneous Lichenoid Drug Eruption, Metformin, Corticosteroids, Discontinuation

Abbreviations: Tumor Necrosis Factor (TNF).

INTRODUCTION

Cutaneous lichenoid drug eruptions are adverse drug reactions presenting as symmetric, violaceous and erythematous papules reminding of lichen planus. The most frequently reported culprit drugs are Checkpoint inhibitors followed by tyrosine kinase inhibitors and TNF-α inhibitors ; other less frequently reported drugs are metformin and calcium channel blockers (Halevy, 1993)

The latency between the initiation of the drug and clinical signs can vary from 0.1 to 208 weeks. Herein we report a rare case of metformin induced lichenoid drug eruption that occured 160 weeks after the introduction of metformin.

CASE REPORT

A 49-year-old woman with a history of type 2 diabetes on metformin for 03 years, and high blood pressure on amlodipine, which she took for 1 year before switching to perindopril 1 month prior to her consultation, presents with a lichenoid rash.

Her symptoms began 3 months ago, with a pruritic rash affecting the trunk (figure 1) and limbs (figure 2) but sparing the face, hands and feet, consisting of purplish macules and papules with Wickham striae on dermoscopy (figure 3). These lesions are small and associated with multiple papuloerosive lesions, covered with hemorrhagic crusts and a few pustules. Examination of the skin and mucous membranes revealed no abnormalities.

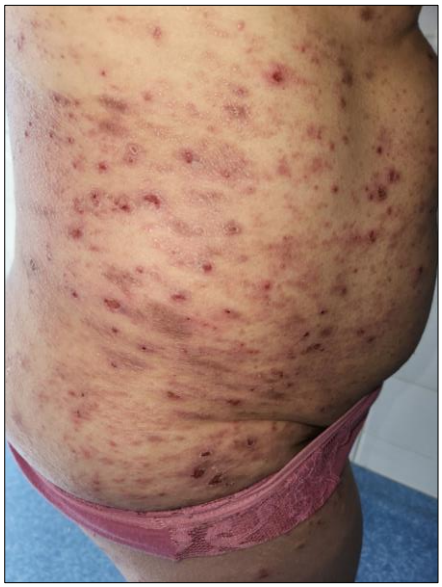

Figure 1: Lichenoid eruption distributed on the trunk

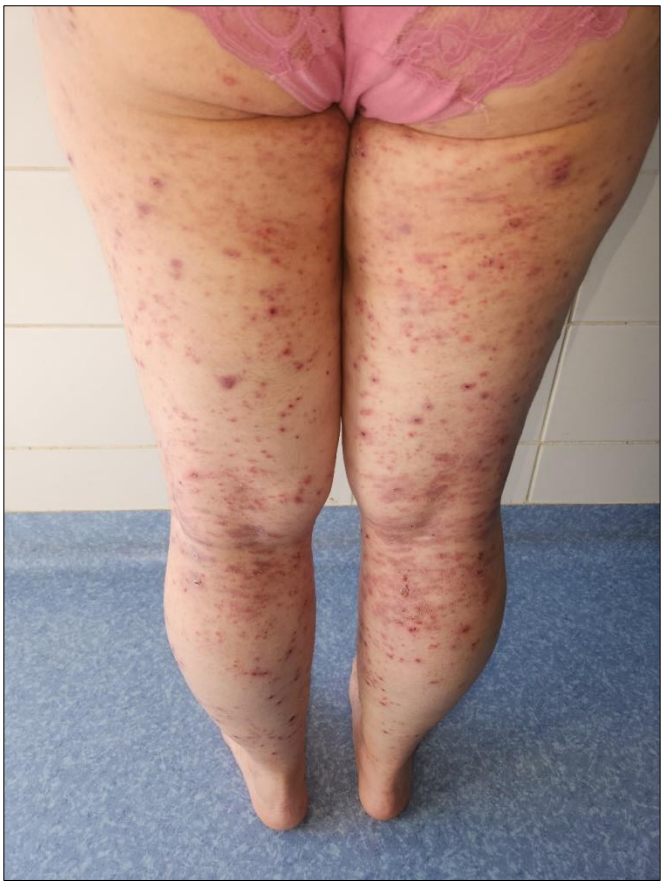

Figure 2: Lichenoid eruption distributed on the limbs

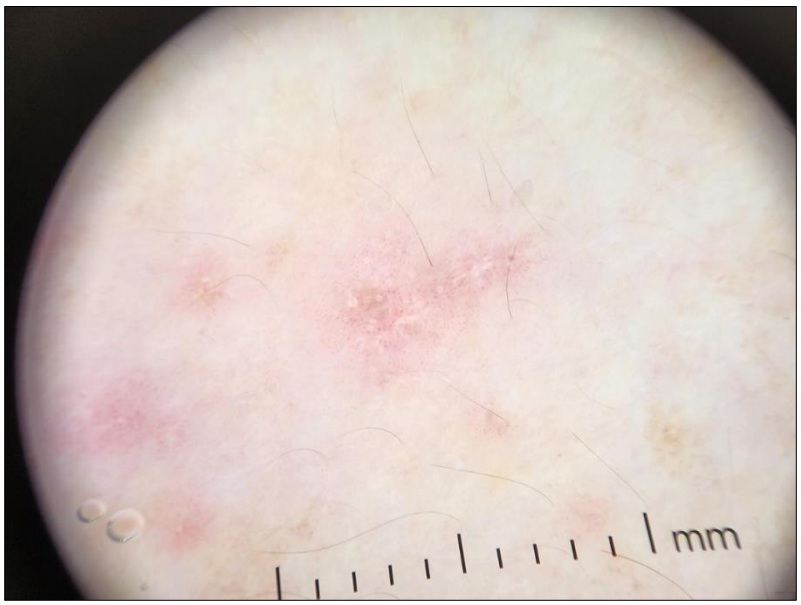

Figure 3: Dermoscopic image showing Wickham striae and dotted vessels on a lichenoid lesion

Biopsy revealed a regular, discreetly acanthosic epidermis, an orthokeratotic stratum corneum, and a fibrous underlying dermis with a predominantly lymphohistiocytic inflammatory infiltrate perivascularly arranged and beneath the epidermal basal layer, with eosinophilic polynuclei and marked pigment incontinence. Intra-epidermal apoptotic bodies are found in a tiered, confluent pattern, resulting in a bullous detachment with an intact roof. The contents of the bulla are represented by polymorphous, altered inflammatory elements (figure 4)

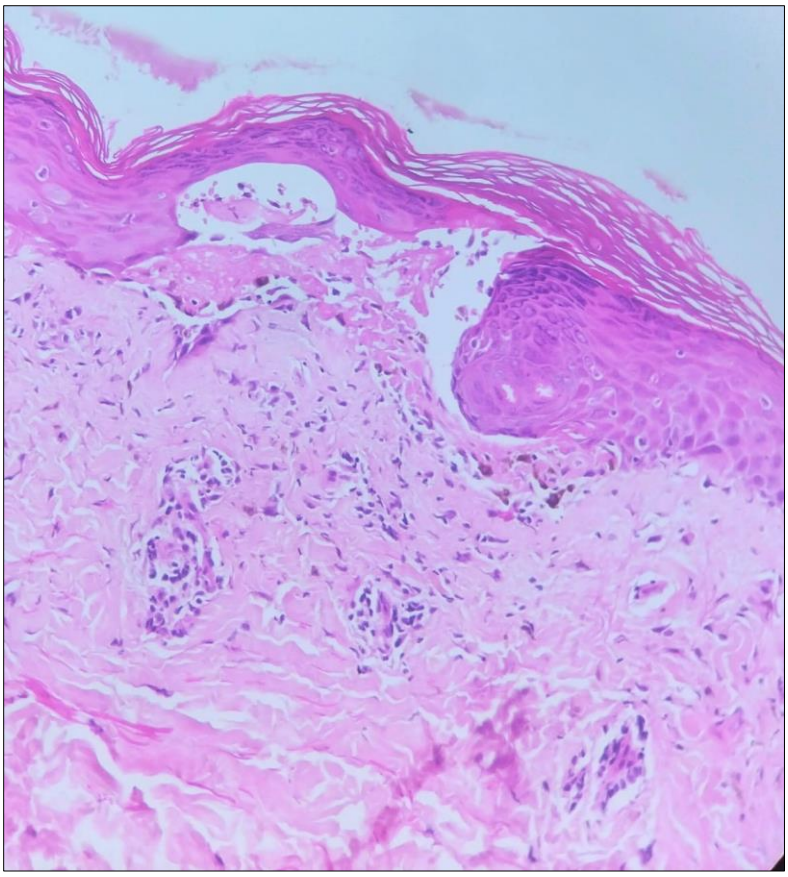

Figure 4: Histopathology revealing a bullous lichenoid pattern with eosinophilic polynuclei in the inflammatory infiltrate

An amlodipine-induced lichenoid drug eruption was initially suggested, the patient was put on oral corticosteroids at 60mg per day with progressive tapering and a decrease in clinical signs, but as soon as corticosteroid therapy was stopped, the rash reappeared, raising the possibility of metformin-induced cutaneous lichenoid drug eruption. Metformin was then discontinued with no further relapse of the rash.

DISCUSSION

Lichenoid drug eruptions are characterized by lichenoid lesions reminiscent of lichen planus. They appear on average at 58.5 years.

Identifying the causative drug remains a challenge, as the onset time is considerable - on average 15.7 weeks, but can be as long as 208 weeks - and symptoms resolve slowly, on average 14.2 weeks, but can be as long as 416 weeks.

Lichenoid toxidermia may resemble idiopathic lichen planus or differ from it in atypical presentation. Acral distribution is the most common, followed by distribution on the trunk, generalized involvement and, less frequently other locations.

Idiopathic lichen planus and cutaneous lichenoid drug eruptions share the same histological features and cannot be distinguished with certainty by histology alone, although a predominant parakeratosis and many eosinophils in the inflammatory infiltrate are characteristic of cutaneous lichenoid drug eruptions.

The drugs most often responsible are checkpoint inhibitors, tyrosine kinase inhibitors and anti-TNF alpha drugs(Maul *et al*., 2023), but other drugs such as calcium channel blockers and metformin(Boccardi *et al*., 2022) may also be involved.

Treatment includes dermocorticoids, systemic corticosteroids, discontinuation of the causative drug, antihistamines, phototherapy, menthol and camphor topicals, but other treatments are also possible.

A case of metformin-induced lichenoid drug eruption has been reported, with resumption of symptoms on resumption of metformin(Azzam *et al*., 2009)

CONCLUSION

Our patient is the second case reported in the literature of a metformin-induced lichenoid drug eruption which presented as a symmetrical pruritic lichenoid eruption of the trunk and limbs with lichenoid histology particular by the presence of eosinophilic polynuclei. Oral corticosteroid therapy followed by discontinuation of metformin resulted in resolution of the rash.

Acknowledgments: None

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