Lichenoid Dermatologic Toxicity Induced by Imatinib: A Case Report

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Introduction:
Chronic myeloid leukemia (CML) is a myeloproliferative neoplasm caused by the t(9;22) translocation, leading to the Philadelphia chromosome and BCR-ABL1 rearrangement. Tyrosine kinase inhibitors (such as Imatinib) are effective targeted therapies for this disease but are often associated with side effects, including cutaneous manifestations. Lichenoid drug eruption induced by Imatinib is rare and difficult to diagnose.

Case report:
A 59-year-old patient, with hypertension and atrial fibrillation under treatment for 2 years, was treated with Imatinib for CML. One month after starting the treatment, he developed generalized pruritus and hyperpigmented, infiltrated, and scaly macules that started on the trunk and then spread across the entire body. Dermoscopy revealed Wickham's striae, gray-brown pigmented dots, rosettes, and regular linear vessels. No mucosal signs were observed. Skin biopsy confirmed the diagnosis of lichenoid dermatologic toxicity with epidermal acanthosis, orthokeratotic hyperkeratosis, focal hypergranulosis, and mononuclear cells in the dermis. Imatinib was implicated by pharmacovigilance, and its discontinuation along with oral corticosteroid therapy (0.5 mg/kg) led to symptom improvement.

Discussion:
The patient, with no dermatological history and on antihypertensive and anticoagulant therapy for two years, started Imatinib one month before the appearance of skin lesions. The onset of this type of toxidermia varies from 1 to 12 months, with an average of 3.6 months. If we retrace the chronology of the symptoms, the appearance of lichenoid lesions one month after the start of Imatinib treatment corresponded to the diagnosis of lichenoid toxidermia, and the regression of the manifestations when treatment was stopped was suggestive.
In terms of semiology, no other drug-related explanation could justify this eruption, and there were no clinical or anatomopathological arguments in favor of another etiology.

Cutaneous reactions to Imatinib are common, but lichenoid drug eruption is rare, with only around thirty reported in the literature. These toxidermias manifest as psoriasiform, licheniform and eczematiform aspects.

Conclusion

Cutaneous reactions to Imatinib mainly include maculopapular exanthems, edema, pruritus, and sometimes purpura. More severe forms, such as Stevens-Johnson syndrome, have been described. In our patient, the study of imputability criteria and the improvement following cessation of treatment argue in favor of imatinib being responsible for the onset of lichenoid dermatologic toxicity.