IMAGES IN DERMATOLOGY

Lichen Planus: Typical presentation on the extremities

Carolina Narváez,* Javier Rosero,* Ernesto Rosero**



Figure 1(a-b). Multiple violaceous, polygonal, flat-topped, shiny papules.

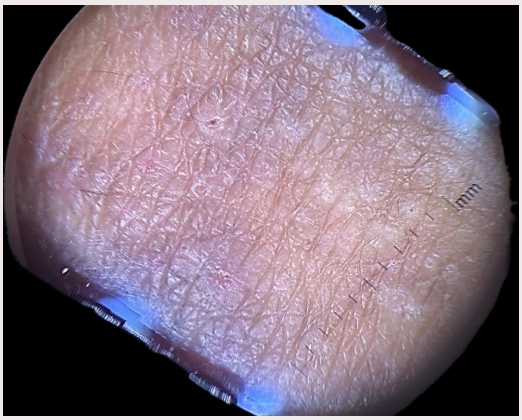


Figure 2. Dermoscopy: Fine whitish network (Wickham striae).

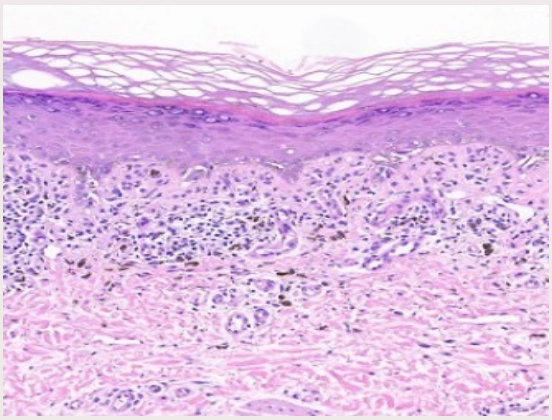


Figure 3. Orthokeratotic hyperkeratosis, underlying hypo-granulosis, and band-like lymphocytic infiltrate.

* Universidad Tecnológica Equinoccial. Quito, Ecuador.
** Independent Researcher. Quito, Ecuador

Correspondence:
Carolina Narváez
caro93narvaez@gmail.com
Quito-Ecuador.
Teléfono: 0987866885

A 26-year-old male with a history of chronic lymphocytic leukemia (CLL) diagnosed eight years ago, currently in remission, presented with chronic confluent papular lesions on the forearms. Physical examination revealed dermatosis localized to the anterior aspects of the forearms and wrists, characterized by multiple violaceous, polygonal, flat-topped, and shiny papules (Figure 1a and 1b). A fine whitish network, almost imperceptible, was also observed on the inner aspect of the right wrist and was better appreciated under dermoscopy (Figure 2).

The lesions exhibited the classic morphology of lichen planus, with well-demarcated papules tending to coalesce and form plaques with a reticulated appearance. The surface of the lesions showed the characteristic pearly sheen, and the fine whitish network corresponded to the so-called "Wickham striae," reticular whitish lines considered pathognomonic of this condition.[1]

Although the histopathological report described the presence of lichenoid interface dermatitis suggestive of a drug-induced lichenoid reaction (Figure 3), this diagnosis was ruled out because the patient was not taking any medications. Furthermore, drug-induced lichenoid eruptions typically present with a more eczematous, psoriasiform, or pityriasis rosea–like morphology, are often generalized, tend to spare the classic sites of lichen planus, and Wickham striae are infrequently observed.[2]

Ultimately, the case was classified as lichen planus, as the clinical presentation was definitive. Treatment with topical corticosteroids was initiated, with a favorable response. The patient remains under follow-up.

Lichen planus is a chronic immune-mediated inflammatory dermatosis affecting approximately 0.2–1% of the global population and may involve the skin, mucous membranes, nails, and hair.[3] Its etiopathogenesis remains incompletely understood; however, it is recognized as a biphasic autoimmune process primarily mediated by cytotoxic T-lymphocyte mechanisms in response to exogenous antigens or altered self-antigens.[4]

According to the literature, the histological pattern of lichen planus typically demonstrates hyperkeratosis without parakeratosis, underlying hypergranulosis, saw-tooth acanthosis of the epidermis, basal cell liquefaction, and direct contact between the epithelium and a dense band-like lymphocytic inflammatory infiltrate.[5]

Drug-induced lichenoid reactions may clinically and histologically mimic idiopathic lichen planus. To establish a correct diagnosis, clinicopathological correlation is essential. Chemotherapeutic agents, particularly tyrosine kinase inhibitors and conventional cytotoxic agents, are recognized causes of these reactions.[5]

Management of classic lichen planus typically involves high-potency topical corticosteroids as first-line therapy, whereas refractory cases may require systemic immunosuppressants or biologic therapies.[6]

INFORMED CONSENT

The patient included in this study signed an informed consent form authorizing the use of his images and clinical data exclusively for research and scientific publication purposes. It is guaranteed that no personal data were disclosed and that no photographs allowing identification were used.

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ORCID

Carolina Narváez *https://orcid.org/0009-0003-5405-1882*
Javier Rosero *https://orcid.org/0000-0002-3635-6653*
Ernesto Rosero *https://orcid.org/0009-0008-8228-7258*

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