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Topic: Inflammatory skin diseases

Eruptive pruritic papular porokeratosis successfully treated with bimekizumab

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Introduction

Porokeratoses are uncommon acquired disorders of keratinization that are characterized by the presence of a cornoid lamella. Clinically, they present as well-demarcated papules or plaques with a hyperkeratotic border and an atrophic center. These lesions may be isolated or disseminated, which allows for classification into several subtypes.
These include porokeratosis of Mibelli, disseminated superficial porokeratosis, disseminated superficial actinic porokeratosis, linear porokeratosis, porokeratosis ptychotropica, and eruptive disseminated porokeratosis (EDP). EDP includes eruptive pruritic papular porokeratosis (EPPP). Treatment remains challenging, particularly in disseminated forms.

Materials and Methods

We present the case of a 50-year-old man with a three-year history of pruritic, brownish, disseminated papules and plaques. The initial clinical diagnosis was eruptive pruritic papular porokeratosis. However, the histopathology results were inconclusive, initially suggesting psoriasis and subsequently Grover disease. Multiple treatments were attempted, including a combination of topical corticosteroids and calcipotriol, topical cholesterol and lovastatin, methotrexate, acitretin, phototherapy, roflumilast, and adalimumab, but there was no clinical response. A repeat skin biopsy confirmed the initial diagnosis of EPPP.

Results

Treatment with the dual anti-IL-17A/F monoclonal antibody bimekizumab was initiated and resulting in significant clinical improvement after 12 weeks and a marked reduction in pruritus, with flattening of the papular rim and resolution of hyperkeratosis. Residual post-inflammatory hyperpigmented macules remained.

Eruptive pruritic papular porokeratosis is considered a variant of EDP and, according to some authors, both entities may represent the same condition. Although the exact pathogenesis of porokeratosis remains unclear, eruptive and inflammatory forms are thought to represent an immune response against tumorigenic keratinocyte clones. Dysregulation of the mevalonate kinase (MVK) pathway has also been implicated, and recent studies have demonstrated upregulation of the IL-17 axis in cases of MVK alterations. In addition, histopathological findings in EPPP frequently reveal lymphocytic and eosinophilic infiltration, together with high expression of IL-31.

Conclusions

In this context, targeted therapies such as anti-IL-17 agents, JAK inhibitors, or anti-IL-31 agents may play a relevant role in managing these uncommon disorders, which significantly impair quality of life and self-perception. As EDP may be associated with underlying neoplasms or viral infections, appropriate screening is mandatory prior to initiating immunosuppressive therapies.