Rapid and Sustained Effectiveness of Bimekizumab in Severe Palmoplantar Psoriasis: Real-World Outcomes Through 104 Weeks

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Keywords: Bimekizumab, biologic therapy, palmoplantar psoriasis, patient-reported outcomes, real-world evidence, treatment response.

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SUMMARY OF KEY RESEARCH FINDINGS

Palmoplantar psoriasis can be disproportionately disabling despite the relatively limited body surface area involved. Pain, fissuring, and pruritus may interfere directly with walking, manual activities, and work, while long-term real-world evidence remains limited, particularly for hyperkeratotic and overlapping pustular phenotypes.[1-3]

The authors evaluated 17 adults with severe palmoplantar psoriasis treated with bimekizumab in routine clinical practice and followed for up to 104 weeks.[4] Ten patients had hyperkeratotic palmoplantar psoriasis, and seven had overlapping hyperkeratotic/pustular disease. More than half had previously failed more than one biologic therapy.[4]

The response was rapid. Complete palmoplantar clearance, defined as modified Palmoplantar Psoriasis Area and Severity Index (m-PPPASI) of 0, was achieved by 8/17 patients (47.1%) at Week 4 and by 11/12 (91.7%) of those reaching Week 24. More importantly, this response was durable. At Week 52, 13/14 evaluable patients (92.9%) had complete clearance, with the remaining patient showing only minimal residual disease. The same level of disease control was maintained at Week 104.[4]

Clinical improvement was accompanied by sustained benefit from the patient perspective. At Week 104, mean Dermatology Life Quality Index (DLQI) was 0.14 and mean pruritus Visual Analogue Scale (VAS) was 0.5, while all evaluable patients reported complete resolution of pain.[4]

WHAT CHALLENGE DOES THIS ADDRESS?

Palmoplantar disease illustrates the limitations of assessing psoriasis severity primarily by extent of skin involvement. A relatively small affected area may still produce major functional disability and a considerable impact on quality of life.[1] The condition is also clinically heterogeneous. Hyperkeratotic, pustular, and overlapping phenotypes may represent different inflammatory patterns, and patients seen in daily practice frequently have long and complex treatment histories. The authors' cohort included both biologic-naïve and highly treatment-experienced patients, making these observations relevant to the population encountered outside clinical trials.[4]

RELEVANCE TO EUROPEAN PRACTICE

For clinicians, the important finding is not simply the rapid onset of response, but its persistence through 2 years. Sustained clearance was accompanied by near-normalisation of quality of

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life and resolution of pain, outcomes that are particularly important in palmoplantar disease.[2,4]

Safety events were generally manageable. Oropharyngeal candidiasis occurred in six patients and was successfully treated without permanent treatment withdrawal. Three patients discontinued bimekizumab: one because of worsening pre-existing atopic dermatitis with conjunctivitis, one because of primary lack of efficacy, and one because of secondary loss of response.[4]

Two observations also raise a broader clinical question regarding the interface between psoriatic and eczematous inflammation. In addition to the patient with exacerbation of pre-existing atopic dermatitis, the primary non-responder had severe, highly treatment-refractory palmoplantar disease with prominent pruritus and fissuring. She had previously failed cyclosporine, methotrexate, and apremilast, as well as all major biologic classes, including bimekizumab. Following bimekizumab discontinuation, she achieved a marked response to upadacitinib.[4,5]

Treatment response alone cannot define the dominant inflammatory pathway. Nevertheless, this clinical pattern raises the possibility that, in selected patients, apparent palmoplantar psoriasis may include a mixed psoriatic-eczematous inflammatory component rather than representing a purely Th17-dominant phenotype.[5]

WHAT ARE THE NEXT STEPS FOR THE RESEARCH?

The study is limited by its small sample size, retrospective design, and single-centre setting, and these findings therefore require confirmation in larger prospective cohorts.

Future studies should move beyond clinical morphology alone. Detailed phenotyping, histopathology, and, where feasible, molecular profiling may help determine whether treatment-refractory palmoplantar disease comprises distinct inflammatory endotypes. Attention should be paid to patients with intense pruritus, fissuring, eczematous features, or repeated failure across mechanistically different therapies.

Identifying such endotypes may ultimately help explain why apparently similar palmoplantar phenotypes respond differently to targeted treatment and could support a more individualised therapeutic approach.

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