

Implications of using Anti-Interleukin-23 Therapy in Treating Generalized Pustular Psoriasis: Commentary

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DESCRIPTION

The management of Generalized Pustular Psoriasis (GPP) poses significant challenges for dermatologists due to its rarity, unpredictable course, and potential life-threatening nature. The case report presented by Lee, et al. [1], in the Italian Journal of Dermatology sheds light on the promising implications of using anti-interleukin-23 (IL-23) therapy, specifically risankizumab, as a potential treatment strategy for this perplexing condition.

GPP is characterized by sterile pustules on a red background, often accompanied by systemic symptoms such as fever and elevated inflammatory markers like C-Reactive Protein (CRP)[2]. Traditional treatments like corticosteroids, phototherapy, and systemic medications have shown limited efficacy and often result in temporary relief with the risk of relapse. Biologic therapies, including anti-TNF α , anti-IL-17, and anti-IL-23 agents, have emerged as potential alternatives, albeit with varying degrees of success. While the focus has been on targeting IL-17 due to its critical role in inflammation and autoimmunity, the IL-23 pathway has also garnered attention. IL-23, a cytokine produced by dendritic cells, is instrumental in promoting the differentiation of Th17 cells, a key source of IL-17 [3]. These Th17 cells play a pivotal role in the pathogenesis of psoriasis and related conditions. The rationale for targeting IL-23 stems from its upstream position in the inflammatory cascade, impacting the initiation and perpetuation of inflammation through downstream cytokine signaling, including IL-17.

The case presented by Lee, et al. [1], demonstrates the potential efficacy of risankizumab, an anti-IL-23 therapy, in treating GPP. The response achieved with risankizumab, in fact, underscores the significance of IL-23 in the inflammatory processes underlying GPP. This case, although singular, raises important implications for clinical practice and further research. Firstly, it suggests that IL-23 inhibition can be a valuable therapeutic approach for GPP, even in the absence of direct IL-17 blockade. By targeting IL-23, the treatment might disrupt the cycle of inflammation at an earlier stage, potentially influencing downstream cytokines such as IL-17 and IL-22. This mechanism aligns with the complex interplay between different cytokines in

the psoriatic immune response. Secondly, the concept of maintenance therapy gains prominence. GPP is characterized by a waxing and waning course, with acute flares interspersed with periods of remission [4]. This unpredictability necessitates sustained management strategies. In fact, if it's true that the anti-IL-36 drug spesolimab showed good results in the treatment of GPP flares (single dose of 900 mg i.v., with the option of a 2nd dose if symptoms persisted on Day 8), there is, however, a need for a maintenance therapy to prevent new and potentially fatal flares [5]. The remission that could be achieved through risankizumab use post-spesolimab treatment highlights the potential for long-term control of GPP with targeted therapies. It is important to note, however, that the findings presented in the case reported by Lee, et al. [1] are based on a single patient, and extrapolation to a broader population requires caution. Rigorous clinical trials involving larger patient cohorts are essential to validate the effectiveness and safety of IL-23 inhibition in GPP.

CONCLUSION

The case report discussed here adds to the growing body of evidence supporting the therapeutic potential of anti-IL-23 agents in managing GPP. As research progresses, a better understanding of the interplay between IL-23 and other cytokines involved in GPP pathogenesis will likely contribute to the development of more targeted and effective therapeutic options for this challenging dermatological condition.

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