

Erythrodermic Psoriasis Successfully Treated with Guselkumab

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To The Editor,

Erythrodermic psoriasis (EP) is severe and potentially life-threatening variant of psoriasis.¹ EP is defined as psoriasis involving more than 80% body surface area (BSA). In recent years the treatment options for EP have expanded; however international guidelines are lacking and the evidence-based treatment choices like conventional therapies and biologics are mainly from small case series or case report.²⁻³ Guselkumab is a fully human monoclonal antibody designed to bind to the p19 subunit of interleukin-23 (IL-23), that inhibits the inflammatory cascade in psoriatic skin. IL-23 plays a central role in T-helper (Th)17 cell stabilization and survival as well as production of IL-17A, a pro-inflammatory cytokine involved in the pathogenesis of psoriasis.⁴ Patients with EP typically exhibit accumulation of IL-17-producing cells in psoriatic skin lesions similar to plaque psoriasis. Selective IL-23 blockage by guselkumab may result in inhibiting pathogenic Th17 cells and consequently downstream production of multiple effector cytokines such as IL-17A, IL-22.^{3,5} Guselkumab has been recently approved only for the treatment of moderate to severe plaque psoriasis. Therefore, guselkumab use in EP, as the case of the other biological drugs, is still off label.

A 49 years-old male presented with generalized erythema and scales affecting virtually the entire body. He has been suffering from plaque-type psoriasis for 13 years. His disease showed a chronic remitting course, requiring topical therapies as well as several cycles of a conventional systemic drug such as metotrexate and biologic drugs such as infliximab and secukinumab with only partial and transitory disease improvement. Dermatological examination showed a generalized erythema of the entire skin body area with associated desquamation, expression of a EP (Psoriasis area and severity index [PASI]: 32 - Body surface area [BSA]: 91 %) (Figure 1a, 2a), associated with fever, tremors, asthenia and general malaise. His body mass index was 45.1 which indicates morbid obesity. His medical history was positive for diabetes mellitus, hypertension and grade 2 hepatosteatosis. Treatment with standard dose guselkumab (100 mg at Week 0 and 4, followed by a maintenance dose, every 8 weeks) led to achieve a complete and rapid remission (PASI100) at week 1 (fig. 1b, 2b). There was no washout period. The patient continued guselkumab treatment, still presenting PASI 100 response at the last follow-up (week 12), without experiencing any adverse events.



Figure 1: Patients at baseline (1a,2a) and after 1 week of guselkumab therapy (1b,2b)

Data from two Phase III, multicenter, randomized, double-blind clinical trials, VOYAGE 1 and VOYAGE 2 studies evaluated the efficacy and safety of guselkumab compared to placebo and adalimumab in patients with moderate-to-severe psoriasis.^{6,7} However, to date, data on EP treatment are very limited with no biologic being approved for its treatment. The only openlabel study, which led to approval for use of EP in Japan, included only 11 patients and recently showed guselkumab as an efficacious and well tolerated therapy up to 52 weeks (all patients achieved a mean absolute PASI of 3.9 with median percent improvement of 94.1% and mean baseline BSA was reduced from 86% to 7%).⁵ And the only case series published from Taiwan which included 13 EP patients and they suggested that for those patients with history of secondary failure to other biologic, guselkumab is still effective in most patients. PASI response at week 12.¹

To our knowledge, this is the first case report of a EP patient which showed rapid response with guselkumab, with PASI100 response reached at week 1 and maintained through week 12. This report suggest that selective blockade of IL-23 using guselkumab may be a viable treatment option in the management of EP. However, further studies are needed to confirm our data, with controlled trials specifically dedicated to EP.

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