Letter

Successful treatment of severe acrodermatitis continua of Hallopeau with guselkumab

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To the Editor

Acrodermatitis continua of Hallopeau (ACH) is a very rare variant of pustular psoriasis with sterile pustules on the fingers and toes, including the nail bed.[1-3] It often leads to onychodystrophy and eventually nail loss.[2,4] Owing to the rarity of this disease, therapy recommendations are based only on case reports, case series, and a few clinical studies. We report on a patient with severe ACH who achieved clinical remission under treatment with guselkumab.

A 59-year-old male smoker presented with a two-year history of plantar erythrosquamous lesions, including involvement of the distal phalanges of both hands and feet with marked nail dystrophy. Local therapy with high potency topical corticosteroids only led to a short-term improvement. In addition, the patient suffered from joint pain in the small finger joints and morning stiffness. On suspicion of psoriatic arthritis, weekly oral methotrexate (15mg) was initiated. After 18 months, methotrexate was discontinued owing to lack of efficacy on the skin lesions.

On initial presentation in our clinic, he presented with plantar and subungual hyperkeratoses, and erythematous and erosive swellings of several distal phalanges with marked nail dystrophy (Figure 1A). In addition, there were sharply demarcated, erythrosquamous plaques on the back of the left hand.

Mycological diagnostics were negative. Erosions of several distal and proximal interphalangeal joints were detected radiologically. Ultrasound of the right foot revealed arthritis of the ankle and tenosynovitis. Rheumatoid factor and antibodies against citrullinated proteins were negative. We made the diagnosis of ACH, accompanied by a mild form of plaque psoriasis and psoriatic arthritis. An 8-month therapy with the interleukin-17A-directed antibody, ixekizumab, in the dosage approved for plaque psoriasis showed good efficacy for the joint symptoms but was discontinued owing to lack of efficacy on the skin lesions (Figure 1B). Subsequent therapy with the TNF-blocker, adalimumab (40mg every other week), led to complete resolution of the joint pain, albeit with persisting morning stiffness. The skin lesions also improved but were not resolved completely and the nail dystrophy also persisted (Figure 1C). After 15 months of therapy, pain occurred for the first time in the metatarsophalangeal joints of the feet. After 28 months of therapy, prostate carcinoma was diagnosed, whereupon adalimumab was discontinued. Within three months without therapy, symptoms deteriorated drastically. Plantar hyperkeratosis with severe inflammation and erosions as well as pustules that coalesced into pools of pus occurred. In addition, 9/20 fingers showed pronounced subungual hyperkeratosis, nail dystrophy, and even nail loss (Figure 2A). There was severe joint pain at rest and morning stiffness as well as erythema and swelling of several joints.

We initiated therapy with oral prednisolone 20mg per day and the IL-23-directed antibody, guselkumab (100mg at weeks zero, four, and then every 8 weeks). Prednisolone was tapered and discontinued. The skin lesions on the hands remitted after one month of therapy and those on the feet improved after three months of therapy (Figure 2B). After 5 months of therapy, lesions on the feet had almost cleared. The joint pain in the small finger and toe joints persisted.

After 8 months of therapy (Figure 2C), the joint pain had decreased. The psoriatic plaques on the dorsum of the left hand had resolved. There was only slight joint pain in the toe joints; the finger joints were pain-free. After one year of therapy, the only remaining lesions were nail dystrophy on both big toes.

This case report illustrates the long, painful, and distressing journey of a patient with ACH for whom several systemic therapies failed. Although there was a partial response to methotrexate, ixekizumab, and adalimumab, there was no remission. Excellent control was finally achieved with guselkumab.

Kromer et al, in a retrospective study with a relatively large number of cases (n=39), recommend biologics as second-line treatment after failure of conventional therapies.[5] Guselkumab showed the highest efficacy, although

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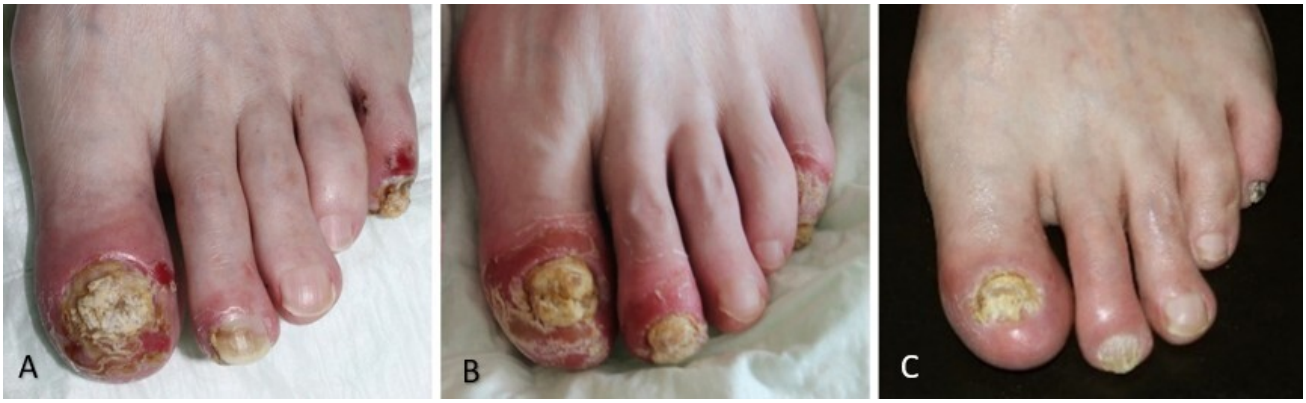

Figure 1. *Clinical images on initial presentation and during therapy with ixekizumab and adalimumab.* ***A)*** *On initial presentation, there were plantar and subungual hyperkeratoses, erythematous, and erosive swellings of several distal phalanges with marked nail dystrophy.* ***B)*** *Clinical picture after 8 months of therapy with ixekizumab showing a lack of efficacy on the skin lesions.* ***C)*** *Improvement in the skin findings without complete healing on the big toes and persistent nail dystrophy under 15 months of therapy with adalimumab.*

only a small number of cases were evaluated (n=2). High efficacy of guselkumab has also been described in other case reports.[6-8] Data on the efficacy of IL-17 antagonists and adalimumab in ACH are inconclusive,[9-13] highlighting both the heterogeneity of the disease and the significant therapeutic challenges associated with it. Further studies are warranted to investigate the efficacy of biologics in ACH.

Potential conflicts of interest

R. Mössner has been an advisor and/or received speakers' honoraria and/or received grants and/or participated in clinical trials of the following companies: Abbvie, Almirall, Biogen IDEC, Böhringer-Ingelheim, Celgene, Janssen-Cilag, Leo Pharma, Lilly, Moonlake, MSD SHARP & DOHME, Novartis Pharma, Pfizer, and UCB. M. P. Schön has been an advisor and/or received speakers' honoraria and/or received grants and/or participated in clinical trials of the following companies: Abbvie, Almirall, Biogen, BMS, Böhringer-Ingelheim, Janssen-Cilag, Leo, Lilly, Novartis, Scinai, and UCB.

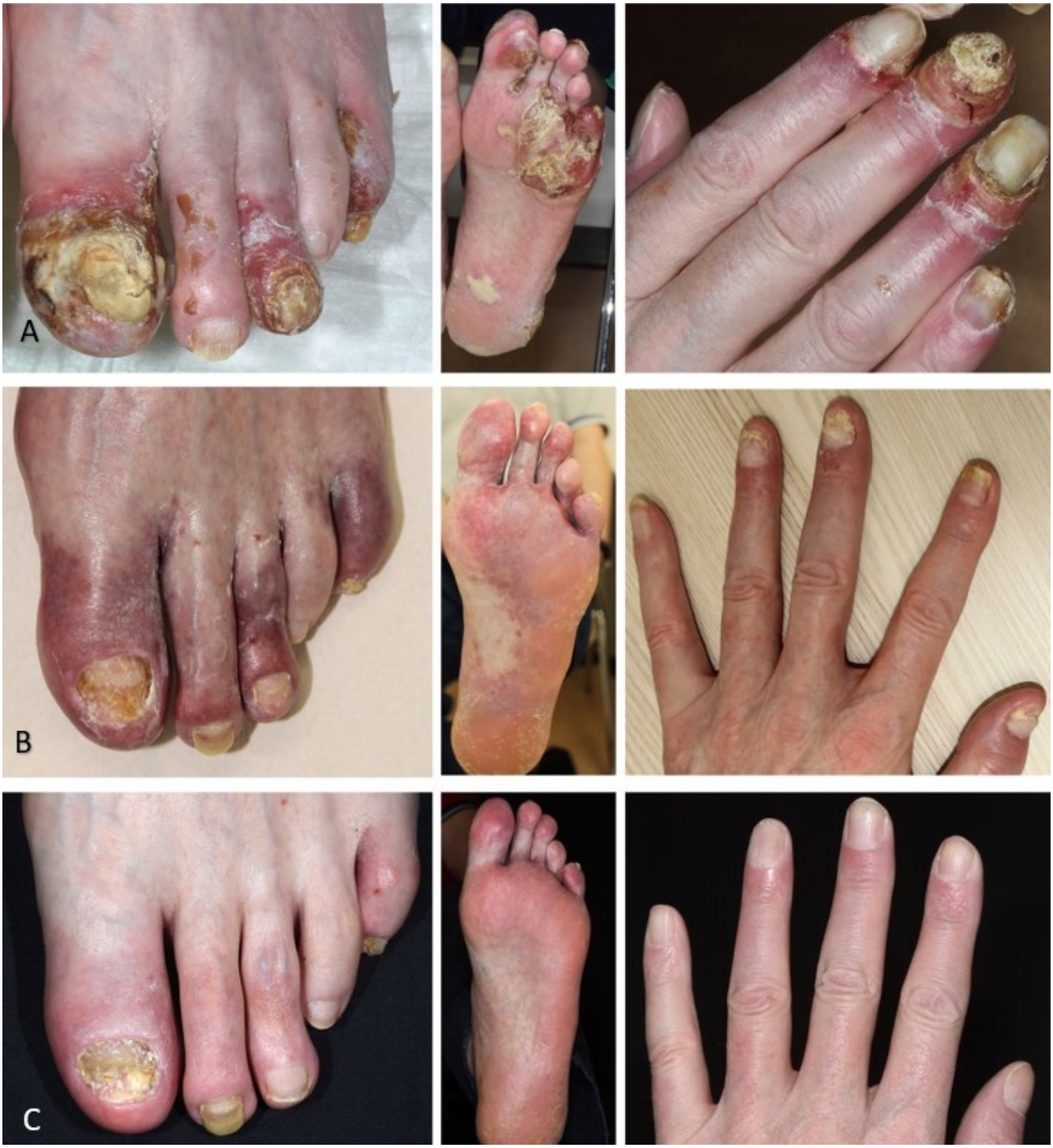

Figure 2. *Clinical images before and under therapy with guselkumab.* ***A)*** *Exacerbation of the skin lesions three months after discontinuation of adalimumab: plantar hyperkeratosis, severe inflammation, erosions, and coalescing pustules as well as pronounced subungual hyperkeratosis, nail dystrophy, and nail loss on several fingers and toes.* ***B)*** *Improvement after three months, and* ***C)*** *after 8 months of therapy with guselkumab. Nail dystrophy on both big toes is the only remaining symptom.*

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