Appearance of lentigines in psoriasis patient treated with guselkumab

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Abstract Development of lentigines in areas of resolving psoriatic plaques is a rare phenomenon that has been reported following various treatment modalities including phototherapy, topical therapies, and biologics. Although the exact mechanism is unknown, evidence suggests that the cause may be multifactorial, with factors such as skin type, sun exposure, inflammation, and immunologic cytokines all playing a potential role. Herein, we present the first reported case of a patient developing multiple lentigines following treatment of psoriasis with the IL-23 inhibitor guselkumab.

Keywords: psoriasis, guselkumab, lentigines

Introduction

Lentigines at the site of resolving psoriasis are a rare occurrence that was first reported as a sequela of phototherapy [1], but they have also occurred following topical [1] and biologic therapies [2]. There are currently no cases in the literature documenting lentigines following treatment with an IL-23 inhibitor. We present a patient with multiple lentigines in areas of resolving psoriasis following guselkumab treatment.

Case Synopsis

A 41-year-old man with Fitzpatrick skin type IV presented to the dermatology clinic with a 15-year history of psoriasis. He had previously tried topical corticosteroids, including LCD-fluocinonide, clobetasol, and topical calcitriol ointment. On exam, the patient had erythematous scaly plaques on the scalp and bilateral upper and lower extremities consistent with psoriasis. The patient was started on standard dosing of guselkumab.

Approximately two weeks later, he reported complete clearance, but noted development of dark spots in the areas of previous psoriatic plaques. On inspection, the patient had multiple small brown

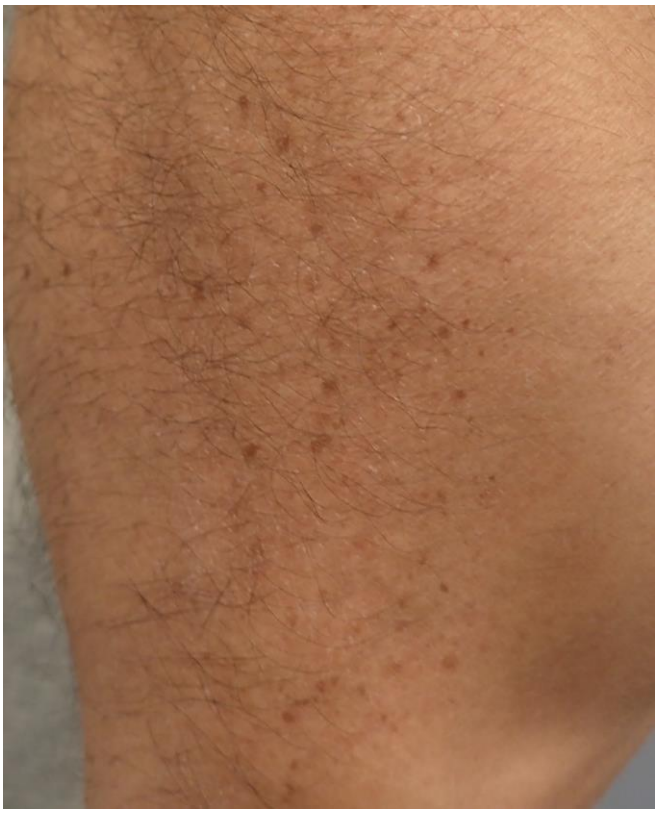

Figure 1. *Multiple brown macules on the lower leg consistent with lentigines in the distribution of resolved psoriatic plaques.*

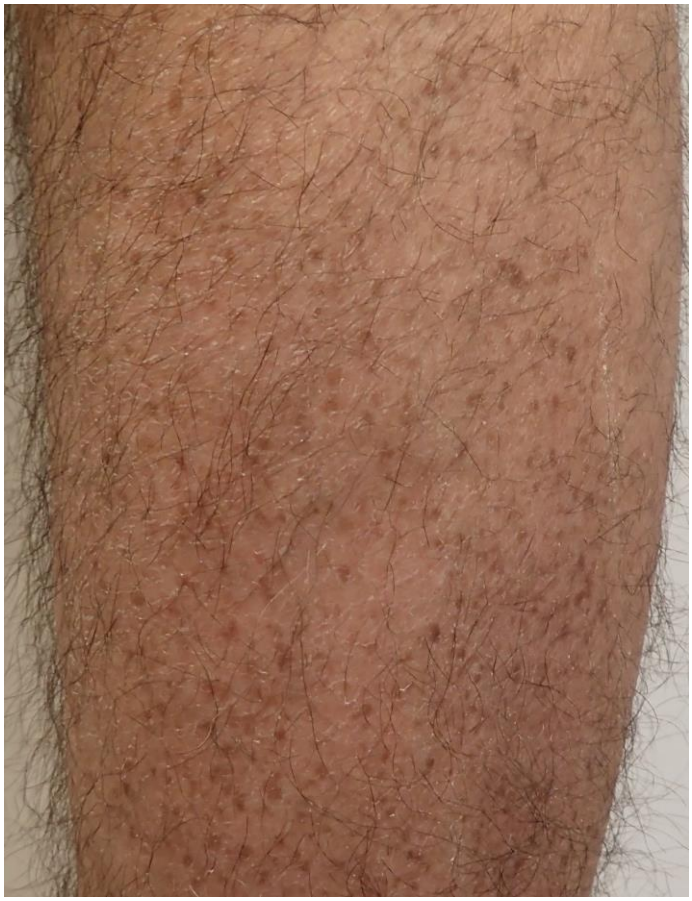

Figure 2. *Multiple brown macules consistent with lentigines on the extensor forearm in the distribution of resolved psoriatic plaques.*

macules on the extensor surfaces of the bilateral upper and lower extremities, primarily on the lower legs and forearms, consistent with lentigines. They have persisted for approximately one year without improvement (**Figures 1, 2**). No treatment for them has yet been initiated.

Case Discussion

Lentigines in resolving psoriatic plaques occur in conjunction with numerous treatments, including phototherapy [1], topical corticosteroids [1], topical calcipotriol, apremilast, etanercept, adalimumab, infliximab, secukinumab, and ustekinumab [2]. We are the first to report lentigines following guselkumab treatment to our knowledge. Lentigo development usually occurs within the first 6 months of treatment [2]. In most cases, patients had Fitzpatrick skin types of III or IV; however, there were two patients who were type II [3, 4]. This differs from solar lentigines, which commonly occur in patients with Fitzpatrick skin types I-III [5]. It may be that Caucasian patients with Fitzpatrick skin types III and IV have more active melanocyte synthesis systems [6], though further research is needed to verify this claim and whether it can be applied to other ethnicities.

This phenomenon was initially thought to be a result of an abnormal reaction to UV light [1], which may cause solar lentigines through photodamage resulting in melanocytic growth and proliferation [5]. However, lesion development in patients with no history of phototherapy and in areas that are not typically exposed to the sun [3] makes this improbable as the sole explanation. In this case, the lentigines may be a variant of post-inflammatory hyperpigmentation [7]. Suppression of psoriasis-related cytokines through biologic agents may play a role in melanogenesis and melanocytic proliferation [8]; however, lentigo development occurs in patients receiving non-biologic treatments as well. Furthermore, lentigo development has not been reported in association with biologic use in other autoimmune diseases with minimal skin involvement, such as rheumatoid arthritis or Crohn disease [1]. It is probable that the underlying cause is a function of the disease process of psoriasis rather than treatment-dependent [2]. LaRosa et al. speculate that the formation of lentigines may relate to the influence of increased expression of TNF and IL17 in psoriasis on melanocyte and melanin proliferation [3]. The cause may be multifactorial, with UV exposure, inflammation, genetic predisposition, and immunologic cytokines acting as causative and exacerbating factors.

Like lentigines of other etiologies, lentigines developing within resolving psoriatic plaques may be permanent, with one case reporting no improvement even after 5 years [2]. However, they have been partially treated with Q-switched ruby laser [9]. In most reported cases, it is unclear whether any treatment was initiated. Research determining whether typical treatments for solar lentigines would improve lentigines in resolving psoriatic lesions would be beneficial.

Conclusion

Although lentigines in areas of clearing psoriatic lesions is rare, physicians should be aware of this phenomenon. As an effective treatment is still unknown, encouraging sun protection in psoriatic patients may aid in preventing their formation.

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