Eruptive lentiginosis in resolving psoriatic plaques

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INTRODUCTION

Eruptive lentiginosis confined to areas of resolving psoriatic plaques (ELRP) is a rare occurrence. A number of previous reports have described this phenomenon after the use of different treatment modalities to resolve psoriatic plaques, including topical, ultraviolet light, and biologic therapies. We present a case of ELRP after treatment with secukinumab. A review of the literature synthesizing all available reports describing lentiginous macules at the site of resolving psoriatic plaques was completed to describe the patient population, treatments, and clinical characteristics associated with this entity.

CASE REPORT

A 29-year-old man with Fitzpatrick skin type III-IV presented with a 6-year history of chronic plaque psoriasis. His psoriasis was not previously treated and he was not on any other medications. He had no other significant medical history. On physical examination, the patient had diffuse psoriatic plaques on the trunk and extremities with scalp and nail involvement covering roughly 20% body surface area. Treatment with secukinumab was initiated at a psoriasis-scheduled dose. Resolution of the psoriatic plaques started 3 weeks after initiation of secukinumab with complete clearance after 3 months. However, at 3 months, the patient presented with multiple 2- to 5-mm light to dark brown fairly symmetrical macules located on the upper extremities and trunk that were confined to previous sites of psoriatic plaques (Fig 1). Lentigines appeared in all areas of resolution; however, some areas had a higher density of lentigines relative to others. The patient did not have a history of lentigines, no phototherapy was performed, and the patient denied sun exposure on the affected areas during the treatment period. A punch biopsy of a macule found elongation of the rete ridges with mild acanthosis and hyperpigmentation of the basal layer compatible with lentigo (Fig 2). Treatment was continued, and follow-up of 3 months found no change in the macules.

Abbreviations used:

ELRP:	eruptive lentiginosis in resolving psoriatic plaques
IL:	interleukin
TNF:	tumor necrosis factor

DISCUSSION

Our case report describes ELRP after anti−interleukin (IL)-17 treatment and adds to the growing body of literature describing this phenomenon. A MEDLINE, EMBASE, and PubMed search and review of the references was conducted and found that ELRP has been described in 12 studies (10 case reports, 2 case series) for a total of 18 patients (Table I). Patients with a history of phototherapy were excluded. These lentiginous eruptions have been most commonly reported after treatment with biologics, which was the case for 6 reports representing a total of 7 patients (39%).

Biopsy results of the pigmented macules, when reported, were consistent with a diagnosis of lentigo. Based on the published reports, these lentigines appear within the first 6 months of treatment initiation, appearing as early as 3 months in some cases. All patients, with the exception of our patient

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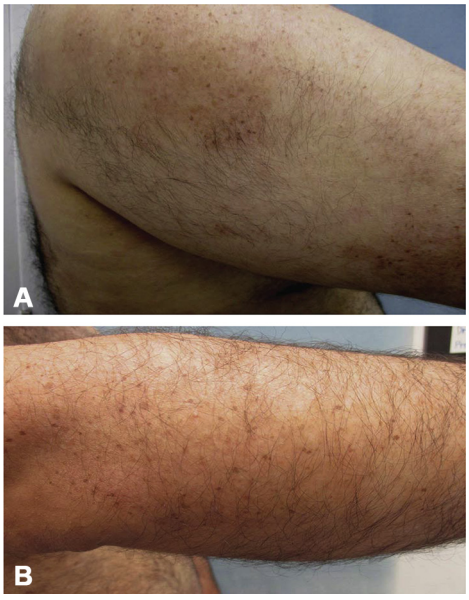

Fig 1. Eruptive lentiginosis in previous sites of psoriatic plaques on the right shoulder (**A**) and right arm (**B**).

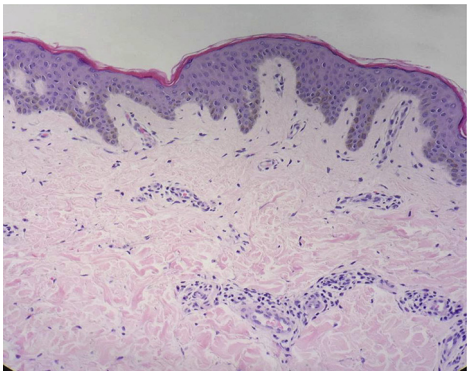

Fig 2. Punch biopsy of a macule found elongation of the rete ridges with mild acanthosis and hyperpigmentation of the basal layer compatible with lentigo.

described above, received prior treatment for their psoriasis that included 1 or a combination of topical and systemic therapies. Although follow-up was only reported for 4 patients, it seems as though the lentigines persist with little to no improvement for several years after onset. For instance, in 1 case report, minimal improvement was found in the lentigines over a period of 5 years. However, treatment with a Q-switched ruby laser has led to partial clearance of the pigmented lesions in one patient.[1]

The age of the patient population for which ELRP was described ranged from 7 to 74 years with an average age of 48. Two of these patients were younger than 18 years. Gender was reported for only 12 of the patients, of which, 7 were male and 5 were female. Furthermore, patients had a prolonged history of psoriasis ranging from 6 to 40 years before the onset of lentigines. This phenomenon was described in 2 patients with Fitzpatrick skin type 2 and in 8 patients with skin types 3 or 4. Skin type was not stated for the remaining 8 patients.

The pathophysiology of ELRP is not well understood. Since these pigmented lesions have appeared after a number of different treatment modalities, a common pathway affecting melanocytes is potentially implicated. A previous study by Wang et al[2] provides some insight into this mechanism. They found high levels of cytokines IL-17 and tumor necrosis factor (TNF) in psoriatic plaques. These cytokines, in addition to others, help stimulate melanocytic growth such that psoriatic lesions have almost twice as many melanocytes as nonlesional skin. The high levels of these cytokines also contribute to the suppression of those genes responsible for pigment production. Consequently, therapeutic neutralization of TNF and IL-17 with biologics reduced the inhibition of melanogenesis and led to a rapid recovery in pigment production in all patients that were treated with anti-TNF (etanercept) and anti—IL-17 (ixekizumab). The increased number of melanocytes combined with a recovery in pigment production led to an abundant production of melanin in resolving psoriatic plaques, potentially explaining the lentigines observed in this study. Although Wang et al[2] only focused on IL-17 and TNF, there may be several other cytokines and factors that help regulate melanogenesis[3] and melanocytic growth that are targeted by other biologics and treatments described in this study. Previous reports of eruptive lentiginosis after chemotherapy in cancer patients[4-6] provides further support that immune modulation may be responsible for these eruptions.

ELRP has only been reported in a small subset of patients. Perhaps ELRP represents a more exaggerated recovery in pigment production, associated with greater disease severity or greater inhibition of cytokines with treatment. Supporting this is the fact that in some patients, ELRP appeared after the resolution of thick psoriatic plaques and not thin ones. As well, it has been suggested that certain mutations in signaling proteins may predispose certain individuals to lentigines development, as immune modulation may be greater in these individuals.[5,7] Overall, it seems that the rapid clearance of psoriatic plaques with new targeted therapies may contribute to the appearance of ELRP.