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Lentigines confined to the upper body plaques of a paraplegic psoriasis vulgaris patient

Paraplejik bir psoriasis vulgaris hastasında vücudun üst yarısındaki plaklara sınırlı lentijinler

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To Editor,

Studies show that the active inflammatory environment in psoriasis vulgaris (PsoV) plaques reduces melanin production. In patients with moderate to severe PsoV, when one or more inflammatory molecules are inhibited during phototherapy or monoclonal antibody treatment, lentigines may develop on the healing plaques[1,2]. However, the development of lentigines in psoriasis patients who are not receiving treatment has not been reported. In this article, a paraplegic PsoV patient without any systemic psoriasis treatment, who developed lentigines on stable psoriasis plaques on the upper half of his body and his non-lesional oral mucosa, will be discussed.

A 65-year-old male presented to the dermatology clinic for psoriasis lesions. The patient, who has been paraplegic and wheelchair-bound for 40 years due to an electric shock, had a history of plaque-type psoriasis for 10 years, prostate cancer surgery 2 years ago, type-2 diabetes, and hypertension. He had been taking a candesartan/amlodipine combination, empagliflozin, acetylsalicylic acid for ten years, and leuprolide acetate as a gonadotropin-releasing hormone receptor (GnRHR) agonist since prostate cancer surgery. There was no history of medication use other than intermittent topical clobetasol propionate ointment for psoriasis. In the physical examination of the patient, there were melanocytic macular lesions on the edges and inner aspect of the lower lip, and melanocytic macules limited to the psoriatic plaques on both elbows, dorsum of the hands and umbilicus. These macules were absent on the PsoV plaques on both knees (Figure 1). The patient's Fitzpatrick skin type was 4. The patient claimed that lentigines appeared after the diagnosis of prostate cancer. There was no relevant pathology in the fingernails and toenails. The punch biopsy from the lentiginous lesion on the right elbow revealed psoriasiform hyperplasia and increased pigmentation in the epidermis.

Today, it has been revealed that there is an increase in tumor necrosis factor-alpha and interleukin-17 signals in PsoV, which synergistically cause an increase in the melanocyte number and a decrease in the pigmentation signal on psoriasis plaques[1]. Therefore, therapeutic inhibition of one of these pathways may lead to the elimination of the inhibitory effect on melanocytes in psoriasis plaques and an increase in pigmentation[1,2]. The fact that this patient was not receiving any systemic PsoV treatment while the

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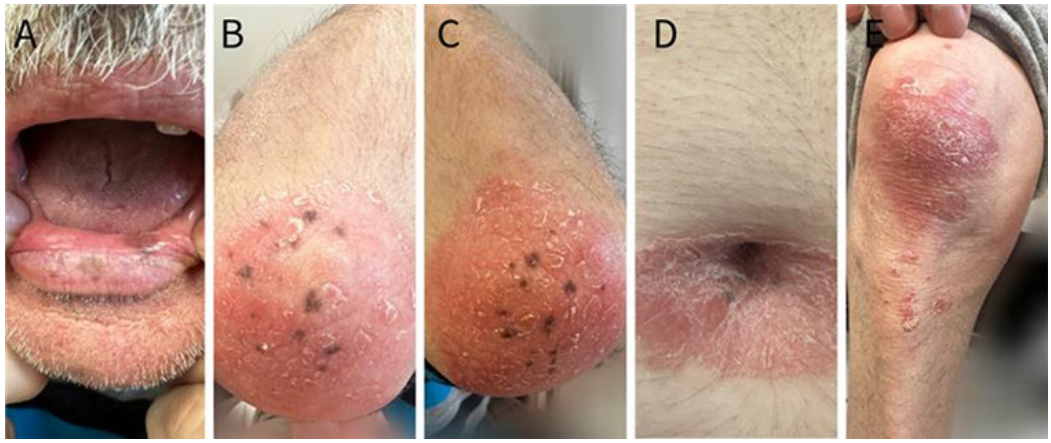

Figure 1. Clinical photographs of the patient. Lentigines on the lower lip **(A)**, lentigines confined to the psoriatic plaques on elbows and umbilicus **(B, C, D)**, the psoriasis plaque on the right knee without any lentiginous lesions **(E)**. (The backgrounds were blurred to hide the unnecessary details.)

lentigines appeared and that the lentigines formed on stable rather than healing plaques suggests that there may be additional triggering factors in the etiology.

Examining the patient's medication history, leuprolide acetate is a GnRHR agonist whose sex hormone-inhibiting effect is utilized in the treatment of prostate cancer[3]. In the literature, no increase in pigmentation has been reported in patients during GnRHR agonist treatments. Osuga et al.[4] reported no increase in skin pigmentation in 16 patients using GnRHR agonists. Therefore, it is unclear whether the leuprolide acetate was the trigger in our case. Besides, there are limited reports of amlodipine-associated pigmentation yet, and the reported hyperpigmentation was in a photo-distributed fashion, unlike the lentigines of our patient[5].

However, this sometimes, chronic application of topical immunomodulators might cause lentigines in chronic inflammatory plaque lesions, such as in the ILIAD phenomenon of atopic dermatitis[6]. Our patient's constant use of topical steroids might be the cause of lentiginosis. However, this does not explain the sparing of the lower body plaques perse.

Laugier-Hunziker syndrome is a sporadic lentiginosis of the oral mucosa and nails, which mostly appears in early and middle-aged adults, and is not associated with a systemic disease[7]. Such a condition might trigger the upper body PsoV plaques and lip pigmentation of our paraplegic patient; nevertheless, he had neither any nail pigmentation nor a history of neural, endocrine, or mesenchymal tumors, as in other precancerous lentiginosis syndromes[8].

Although there are not enough resources on how melanogenesis is affected in paraplegic patients, the fact that lentigines are not found on the lower extremity psoriatic plaques in this paraplegic patient suggest that neural factors may contribute. Further research is needed.

Ethics

Informed Consent: The patient's consent form including biopsy, photography, and scientific use was signed and kept in the Artvin Hopa State Hospital archives. The physical examination, biopsy, and construction of the article were all made by the author.

Footnotes

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