Multiple Lentigines Confined to Resolving Psoriatic Plaques in a Patient Treated with Adalimumab

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Hyperpigmented lesions confined to resolved psoriatic plaques have been rarely reported and have been previously described as lentigines, naevus spilus hyperpigmentation and speckled pigmentation [1–3]. We present the case of a 55-year-old woman with psoriasis who developed lentigines on resolving lesions while receiving adalimumab treatment.

Report of a Case
Our patient had suffered from chronic plaque psoriasis on the elbows and knees for more than 25 years and had been successfully treated with various topical treatments including steroids, tacalcitol and calcipotriol. She had been free of lesions while under treatment. In the 2 previous years she had also received methotrexate for psoriatic arthritis but with poor control of the disease. In August 2006, she started adalimumab 40 mg subcutaneously once every 15 days. At this time she presented psoriatic plaques on the elbows and knees. After 2 months of treatment, she was admitted to our service as the psoriatic plaques had disappeared on the elbows and knees. After 2 months of treatment, she was admitted to our service as the psoriatic plaques had disappeared and new lesions had appeared. On physical examination, she had light and brown regular lentigines over the previous sites of the psoriatic plaques (fig. 1), which were confirmed as such on histological examination. Treatment was continued, and no more lesions or changes in the morphology or colour of the lentigines were seen. Phototherapy was not performed in this patient.

Comment
Multiple large solar lentigines on the upper back have been described as clinical markers of past severe sunburn [4]. The majority of the lentigines in resolving psoriatic plaques are described following PUVA or UVB. In the few previous cases reported the underlying mechanism for this kind of lesion is unknown. This pigmentation can arise without phototherapy, and it has been suggested to be an unusual type of postinflammatory hyperpigmentation [5]. In our case the chronological association between the use of adalimumab and the development of the lesions may suggest that adalimumab is of aetiological relevance to their development and presumably has some effect on melanocyte biology. We do not know if the formation of lentigines could be: (a) drug induced, (b) the result of the immunosuppressive action of adalimumab, because systemic immunosuppressants are reported to cause an increase in melanocyte activity, or (c) might simply be a postinflammatory hyperpigmentation.

On the other hand, melanocytes may be involved in the psoriatic process in several ways: there have been several reports suggesting an association between chronic plaque psoriasis and vitiligo, and the absence of lentigines in psoriatic plaques has been presumed to be the result of selectively destroyed melanocytes by the psoriatic process [6].

The clinical implication of our finding is uncertain. Overall, careful surveillance will be required in those patients receiving biological treatments.

References