Generalized "Sunbed Lentigines" in a Patient with Systemic Lupus Erythematosus

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Sir,

The development of melanocytic lesions following treatment with psoralen and ultraviolet A (PUVA therapy) is well documented. The first case of lentigines induced by cosmetic UVA tanning was reported in the 1980s (1), and another three cases have been published since then (2–5). We report the case of a woman with systemic lupus erythematosus (SLE) who developed generalized persistent melanocytic lesions while using UVA tanning beds.

CASE REPORT

A 45-year-old woman, phototype IV, with no family clinical history of interest was examined during consultation in October 2001. She presented generalized small asymptomatic brown spots on the trunk and limbs. The first lesions had appeared towards the end of 1994 on the frontal aspect of the legs and had progressively spread to the rest of the skin up until the time of consultation. Noteworthy among the personal history of the patient was the fact that, in August 1995, she had been diagnosed with SLE due to the presence of seronegative additive peripheral arthritis, with a duration of 2 years, nephropathy, oral aphthae, positive ANAs, positive antiDNAn and positive anti-Ro (SSa) and anti-La (SSb). Since 1995 she had been treated with low doses of oral corticoids, chloroquine and tramadol. Between 1994 and the diagnosis of SLE she had been treated with oral contraceptives and naproxen, the latter being used occasionally for her arthralgias.

Since May 1994 until the time of consultation, she for cosmetic reasons visited a tanning salon with a variable periodicity ranging between 4 times a month during the non-summer months and 12 times a month between May and September. The patient claimed to have used UVB sun filters and had not used topical products to speed up tanning. She had a light sunburn before the beginning of the tanning sessions. No inflammatory reactions, malar rash or any other cutaneous lupus lesions developed in the course of lentigines.

On exploration, the presence of generalized star-shaped brown maculae was observed on the trunk and limbs, with a tendency to respect skin creases (Fig. 1). The maculae ranged between 0.5 and 1.5 cm in diameter and were confluent in some zones. The rest of the examination was not noteworthy.

Histopathological study of the lesions showed an acanthotic epidermis at the expense of basal and parabasal type cells, with elongation of the epidermal crests and a modest increase in the number of melanocytes, with hyperpigmentation of the basal strata. Atypical melanocytes were observed focally. Numerous melanophages were visible in the dermis, without the presence of solar elastosis (Fig. 2). A lupus band test for IgG and C3 was positive in photoexposed (Fig. 3) and non-photoexposed skin.

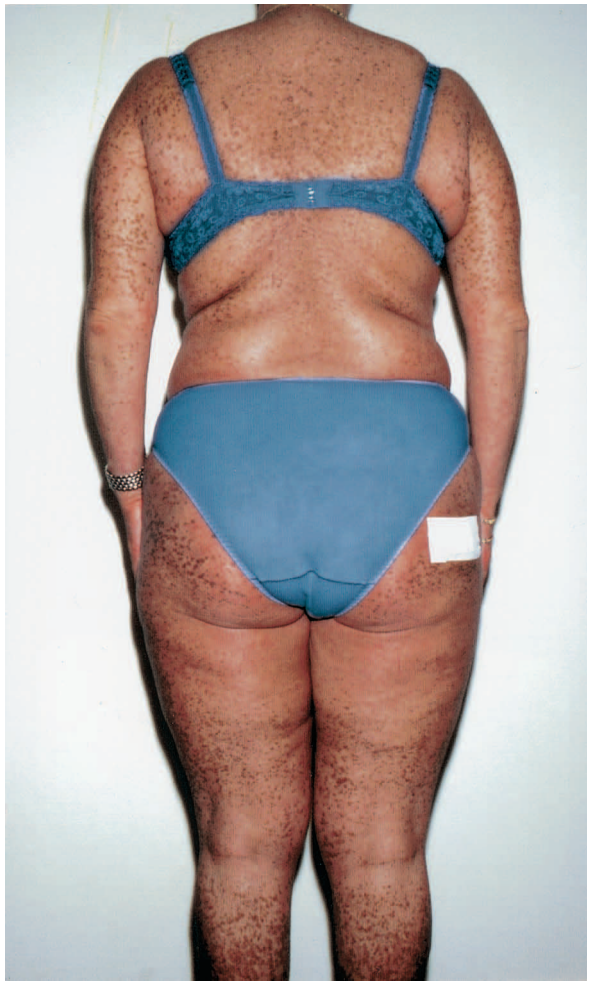

Fig. 1. Generalized star-shaped maculae with a tendency to respect skin creases.

DISCUSSION

PUVA-induced lentigines appear in between 2% and 53% of patients who undergo chronic treatment with photochemotherapy (1, 6). In 1987, Jones et al. (1) reported the first case of generalized lentigines induced by exposure to UVA in tanning beds. Subsequently, only three further cases have been reported of sunbed lentigines, all of these in women of fair phototype (2–5). The location and intensity of the lesions ranged from localized forms on the anterior aspect of the legs (2, 4) to generalized forms (1), as in the case reported here.

Histologically, both PUVA and sunbed lentigines are characterized by the presence of atypical melanocytes, except in the report by Kadunce et al. (5). Sunbed lentigines seem to lack the epidermal hypertrophy

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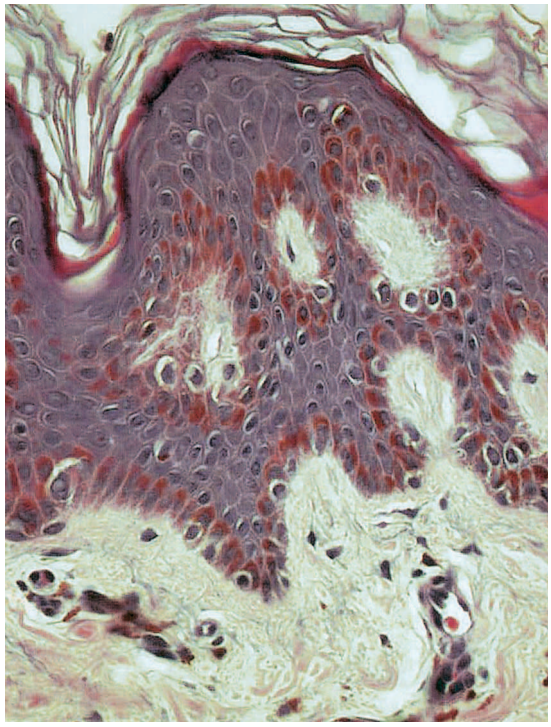

Fig. 2. Acanthotic epidermis with slight increase in the number of melanocytes, focally atypical (H&E ×400).

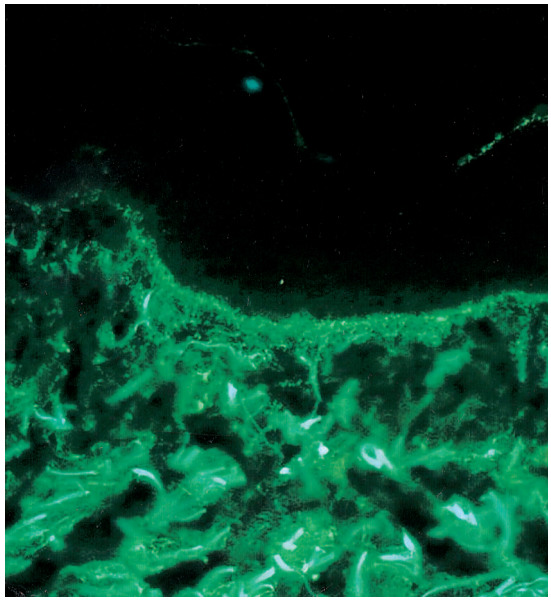

Fig. 3. Positive lupus band test for IgG in photoexposed skin.

observed in PUVA lentigines (5). The presence of atypical melanocytes probably increases the risk of a future development of malignant melanoma, why a stricter monitoring of patients should be established (7, 8). In our case, the histological findings were similar to those previously reported, except by the presence of acanthotic epidermis.

With regard to the aetiopathogenesis of sunbed lentigines, reference is made in two cases to the use of oral contraceptives (4) and hydrochlorothiazide (5). In our case there are three photosensitizing factors that might contribute to the patient's condition: SLE, naproxen and oral contraceptives. A history of photosensitivity has been reported in 57% to 73% of SLE patients, although its origin remains unknown (9). Our patient mentioned having mild sunburn before the beginning of sunbed sessions, and this disappeared later. No other inflammatory skin reactions had ever been noticed by the patient.

This is the first case reported where SLE might play an aetiologic role in the development of tanning-bed lentigines, although naproxen, oral contraceptives and individual susceptibility cannot be ruled out. In any case, the pathophysiology of sunbed and PUVA lentigines remains unknown (10) and further study is needed.

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