

## Psoriasis Exacerbation Induced by Interferon- $\alpha$ . Report of Two Cases

Sir,

Hepatitis C virus is an important cause of post-transfusion hepatitis and a common cause of acute sporadic hepatitis, which often leads to a chronic form. At present there is no therapy of established benefit for chronic hepatitis C. Recent studies have suggested that prolonged therapy with interferon- $\alpha$  may be useful in some patients with chronic hepatitis C (1).

We report the first 2 cases of psoriasis induced by interferon- $\alpha$  in patients treated for chronic hepatitis C.

### Case 1

A 60-year-old man, affected with chronic hepatitis C (hepatitis activity index, HAI: 7), diagnosed in June, 1992, received 3 million units (MU) of rIFN $\alpha$ 2b (recombinant interferon- $\alpha$ 2b) subcutaneously three times a week. Psoriasis, confirmed by a skin biopsy, had been diagnosed 1 year before the chronic hepatitis C diagnosis but had not required treatment. He had no family history of psoriasis. During the first 6 weeks of treatment the psoriatic lesions flared up, with generalized plaques (psoriasis area and severity index, PASI, increased from 4 to 7). Interferon treatment was interrupted after 4 weeks followed by substantial improvement of psoriasis which, without therapy, returned approximately to its previous state (PASI: 4.5) in 2 weeks.

### Case 2

A 42-year-old man developed a chronic hepatitis C (HAI: 4) in 1989. He was included in a clinical trial in which 3 MU of human lymphoblast interferon- $\alpha$  were injected subcutaneously three times a week. He had been affected with psoriasis, confirmed by skin biopsy, since 1980 and he had a positive family history of psoriasis (his father). Before treatment with interferon- $\alpha$ , psoriatic lesions were limited to elbows and scalp only (PASI: 0.8). Two weeks after the start of treatment with interferon he developed large plaques of psoriasis on elbows, knees, chest and scalp (PASI: 6.6). The patient continued treatment with interferon. The lesions were treated with topical drugs without further worsening.

### DISCUSSION

Interferons were originally expected to be a rational choice of therapy for psoriasis as well as tumors and viral infections because of their antiproliferative property (2-4). Recombinant human interferon- $\alpha$  has been used in the treatment of malignant metastatic carcinoid tumour, metastatic renal carcinoma, and more recently chronic hepatitis C and other diseases (1,5). Various cutaneous side-effects have been described after treatment with interferon- $\alpha$ , including itching, dryness and moderate hair-loss (5).

In 1986 Quesada & Gutterman reported the cases of 3 patients whose psoriasis was aggravated or induced during treatment with recombinant human interferon- $\alpha$  (3). Shiohara et al. described a rapid development of psoriasis on the warts of a hand after an intralesional injection of interferon- $\alpha$ . The presence of the Koebner phenomenon in the patient described was unlikely, because similar cutaneous lesions did not appear in the sites of dinitrochlorobenzene administration (2).

These reports suggest the possibility that interferons may participate in the pathophysiology of psoriasis. Interferon- $\gamma$  and also interferon- $\alpha$  activity was demonstrated in suction blister fluid obtained from psoriatic skin but not in the blister fluid of unaffected skin. Abnormal serum levels of interferon have also been found in the sera of psoriatic patients (6). Moreover, the activity of psoriatic lesions has been associated with a reduction in cAMP/cGMP (cyclic adenosine monophosphate/cyclic guanosine monophosphate) ratio, while it is known that interferon- $\alpha$  alters the intracellular content of these nucleotides, inducing an increase in cGMP concentration (3,5).

In our opinion our cases and the previously quoted reports (2-5, 8,9) suggest that interferon- $\alpha$  may play an aggravating role in psoriasis, while the mechanism through which this occurs is still unclear. Baker et al. (7) stressed that interferons involve those cells and mechanism that seem to be particularly altered in psoriatic lesions.

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