Interferon-Induced Psoriasis in a Patient with Chronic Hepatitis C

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Although psoriasis represents an inflammatory disorder of uncertain etiology, several factors have been implicated in its development. Among the causes that may induce or worsen it, attention has recently been focused on interferon alfa-2b [1-5]. We describe the appearance of psoriasis in a patient treated for chronic hepatitis C with interferon alfa-2b. To our knowledge, this is the first such case reported.

Case Report

A 33-year-old man, formerly an intravenous drug addict, was diagnosed as having chronic hepatitis C and treated with recombinant interferon alfa-2b at a dosage of 5 million units administered subcutaneously 3 times a week for 12 months. He had no family history of psoriasis. Five months after beginning treatment, scaly erythematous lesions appeared on his elbows, forearms, back of hands, buttocks and scalp. Extensive biochemical and hematologic investigations were normal except for hepatic parameters. Enzyme-linked immunosorbent assays done at the beginning of treatment and 6 months later showed no evidence of infection with human immunodeficiency virus. Histopathological examination of a skin lesion showed marked acanthosis, long edematous papillae and hyperkeratosis with parakeratosis; large tortuous capillaries were seen in the papillary dermis. With a diagnosis of psoriasis vulgaris, interferon treatment was suspended, and we began treatment with emollients (petrolatum). Skin lesions disappeared within 2 months, and 6 months later the patient was asymptomatic.

Discussion

Although several cases of psoriasis induced or exacerbated by interferon alfa have been described in patients with malignant neoplasias [1, 3, 4], the lesions have not been reported in the setting of chronic hepatitis C. High doses of interferon alfa have been suspected in the development of psoriatic lesions in the former patients [5]. In our patient, cutaneous lesions slowly improved after interferon alfa therapy had been withdrawn. Interferon alfa may act as an inducing factor for psoriasis due to activation of the dermal dendrocytes which produce tumor necrosis factor α . This latter, in turn, induces the expression of adhesion molecules on keratinocytes and endothelial cells, as well as production of transforming growth factor α , which triggers the proliferation of keratinocytes [6]. Psoriatic lesions may appear because the

hyperproliferating keratinocytes escape the control mechanisms, due to genetic mutation; alternatively, self-reactive

T cell clones may be induced by superantigens, leading to the propagation of the psoriatic lesions [6]. In this regard, hepatitis C virus may act as a superantigen.

Psoriasis has not previously been described among the cutaneous lesions associated with infection by the hepatitis C virus. We believe that in our patient, both recombinant interferon alpha as well as the hepatitis C virus may be involved in the genesis of psoriasis.

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