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SQUAMOUS CELL CARCINOMA IN DISCOID LUPUS ERYTHEMATOSUS

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Squamous cell carcinoma developing in a plaque of chronic discoid lupus erythematosus is reported in an elderly male.

Key words : Squamous cell carcinoma. Chronic discoid lupus erythematosus.

Squamous cell carcinoma of skin can develop in association with many chronic skin diseases especially those with severe scarring. Squamous cell carcinoma arising secondary to chronic discoid lupus erythematosus, though rare has occasionally been reported.[1-6] The incidence of carcinoma in DLE was found to be 3.40% in a recent study.[6] The period from the onset of DLE to the formation of neoplasm is seldom only a few years; it may be even 30 to 40 years. A case of squamous cell carcinoma which developed in a plaque of chronic DLE in an elderly male is reported.

Case Report

A 50-year-old male was seen in the cancer ward of this hospital with an ulcer on the right side of the face since 4 months. The patient had been having chronic disseminated discoid lupus erythematosus since 20 years. He had not received any specific treatment for his skin disease. Examination revealed multiple, bilateral, depigmented, atrophic, well-defined plaques with hyperpigmented borders on the face, nose, front of the chest (Fig. 1), left upper arm and the scalp. Most of the lesions had patulous hair follicles with adherent follicular scales, the removal of which revealed the carpet *tag* sign. Some of the lesions had telangiectasis also. On the right side of the face there was an ulcer, 3 × 4 cm in size, with raised everted irregular edges (Fig. 2). The base was indurated and the floor was covered with granulation tissue. Submental group of lymph glands on the right side were enlarged, hard and fixed to the underlying structures. General physical and systemic examination did not reveal any abnormality.

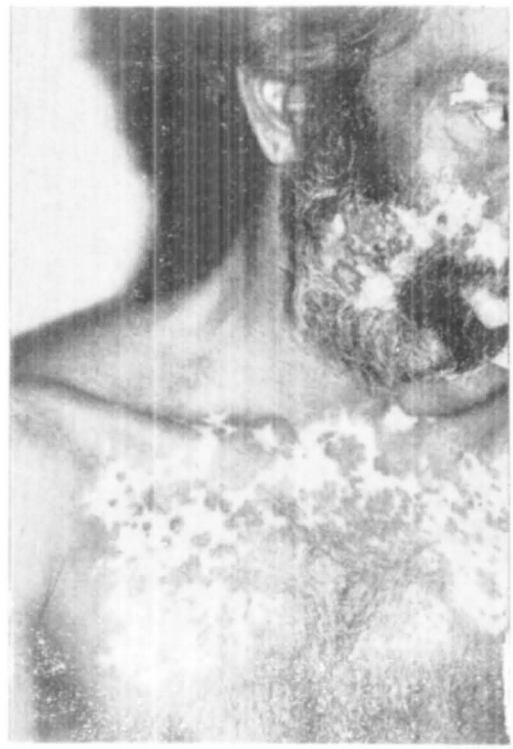

Fig. 1. DLE lesions on the front of the chest and face.

Routine laboratory tests on blood, urine and stools were normal. ESR was 12 mm. LE cell test and the VDRL test were negative. Blood urea, blood sugar, serum protein, bilirubin and alkaline phosphatase were all within normal limits. The skin biopsy taken from the chest

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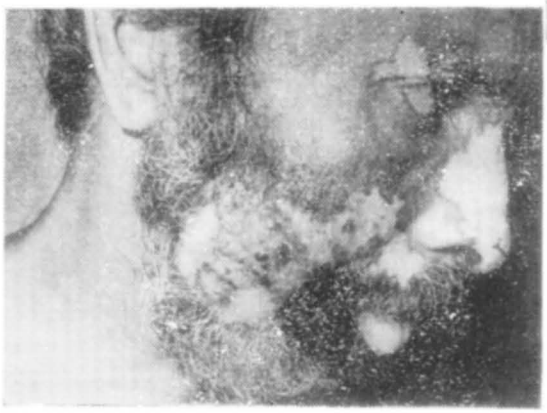

Fig. 2. Squamous cell carcinoma with ulceration in the DLE plaque.

lesion showed histopathological features typical of DLE. Biopsy of the ulcer showed histopathological features of undifferentiated squamous cell carcinoma. The patient was later treated in the cancer ward by local irradiation and oral chloroquine tablets.

The mechanism of malignant transformation in DLE is not well-understood. An external aetiological factor we now recognize in the production of carcinoma of the skin is chronic exposure to sunlight. Carcinogenesis following repeated exposures to sun and other external irritants is the end stage of a process of chronic destruction and repair. Incomplete and abnormal healing occurs after many insults accompanied by hyperplasia and loss of tissue resistance with eventual malignant change.[7] Not only the external factors, but the internal ones also must be playing their roles in the malignant transformation of a DLE lesion. In view of the current theories of SLE, it is attractive to assume that the sun-burned skin releases a UV-light altered DNA which serves as the antigen causing a locally destructive lesion. The immediate carcinogenic impulses are coming from the primary subepidermal changes and are of a more or less biologic nature; only their summation and torpid effect within a limited area are not physiologic. It is also suggested that the epidermal cell system in DLE suffers from permanent hypoxidosis due to poorly vascularised cicatricial structures in the dermis. The continuously suffocated epidermal cells undergo accelerated physiological degeneration (increased keratinisation) and finaly there will be malignant degeneration.[8]

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