SQUAMOUS CELL CARCINOMA IN DISCOID LUPUS ERYTHEMATOSUS
(A Case Report)

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Summary

A case of Squamous Cell Carcinoma arising in discoid lupus erythematosus of 10 years' duration is described. The constant exposure to U-V rays in sunlight, in addition to the chronic hypoxidosis in the scar resulting from chronic discoid lupus erythematosus, is assumed to induce malignant degeneration.

KEY WORDS: Squamous cell carcinoma, Discoid lupus erythematosus.

Introduction

Chronic Discoid lupus erythematosus (CDLE) shows a considerable amount of malignant degeneration. Squamous Cell – and less commonly basal cell carcinomata occasionally occur in scars of CDLE. An incidence of 3.3 percent has been reported in one series[1]. Malignant changes are said to be more common in middle aged males and especially in lesions of more than twenty years' duration[2]. We are here reporting a case of Squamous Cell Carcinoma in a patient with CDLE with a discussion of the possible pathogenesis of the malignant degeneration.

Case Report

A 49 year old farmer was admitted to the Dermatology section of Medical College Hospital, Calicut, in December 1981, with an ulcerated growth (Fig. 1) on the right side of mandible of three months' duration. The growth had started as a small nodule, rapidly increasing in size in the course of one month, breaking down to form a necrotic ulcer at the centre with everted irregular edges. At the time of admission, the growth measured 8 × 6 centimetres (Fig). It was firm in consistency and movable over the underlying muscles. The mucous membrane of mouth was clinically normal. Lymphnodes were not significantly enlarged. There were depigmented atrophic plaques with adherent scales and telangiectasia over the entire right cheek and right submandibular region. Two similar plaques of 4 centimetres size was noticed on the left side of scalp which had resulted in scarring alopecia.

In 1971, patient was admitted to the same hospital with ulceration and depigmented scaly plaques on the lower lips, which was diagnosed as discoid lupus erythematosus, after histopathological and other laboratory investigations. The lesion was satisfactorily

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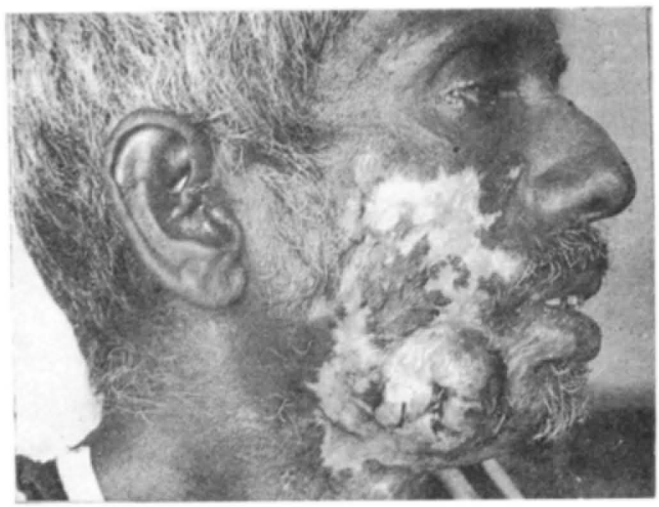

Fig. 1

Ulcer with evested edge

controlled with chloroquine tablets 250 mg t.i.d. Patient had stopped treatment after 45 days and was lost to follow-up for 10 years. During this period he had developed fresh lesions on the face. Systemic examination was non-contributory.

Laboratory Investigations:

Routine urine and blood tests gave normal results except a raised ESR of 45 mm in the 1st hour Tests for LE cells was negative. Liver function test, blood urea, X-ray of chest and ECG were normal. X-ray of mandible was also normal. Histopathological examination of specimen taken from the scaly plaque showed hyperkeratosis, atrophy of epidermis, liquefaction, degeneration of basal cells and periappendageal infiltrate with chronic inflammatory cells. Upper dermis showed vasodilatation. Specimen from the edge of the growth showed poorly differentiated Squamous cell carcinoma.

Simple wide excision of the growth and repair by skin grafting was done.

Discussion

The patient reported here had clinically and histologically typical lesions of CDLE. An ulcerated growth, which was histologically proved to be a poorly differentiated Squamous cell carcinoma, developed in one of the lesions approximately 10 years after the onset of the disease process. Malignant degeneration is well known in CDLE, and usually appears as solitary Squamous cell carcinoma. Based on information reported in literature, Lander et al[3] quoting Kappassan (1952) concludes that one out of every 50 chronic Discoid lupus erythematosis degenerates into a malignant tumour. Millard and Barker[1] report an incidence of 3.3 percent. Grana[4] states that the period from the onset of CDLE to the formation of the neoplasm is seldom only a few years, but may even take 30–40 years. Most reported malignancies are Squamous cell carcinomas. Granag reviewing literature found only five basal cell epitheliomas reported in CDLE. Squamous cell carcinoma and keratoacanthoma appearing in one patient has been reported[5].

External factors like trauma, ultraviolet light and X-rays are assumed to cause malignant degeneration in CDLE lesions. In our patient constant exposure to U-V rays of the sunlight might have accelerated the malignant degeneration.

The poorly vascularised cicatricial structures in CDLE lesion makes the epidermal cell system to suffer from a permanent hypoxidosis. Thus, the epidermal cells actually are continuously 'suffocated' and their physiologic degeneration accelerated, the keratinisation is increased (hyperkeratosis). Finally, malignant degeneration occurs, because, in the end cell elements or cell generations are formed to which the hypoxidosis is congenial[3].

One might range CDLE among facultative or precancerous lesions in a broad sense. Patients with CDLE should be kept under regular follow-up because of the above possibility of malignant transformation.

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