

Discoid Lupus Erythematosus Leading to Squamous Cell Carcinoma Over Dorsum Of Left Foot.

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Abstract: DLE/CCLE is a benign disorder of skin, most frequently involving face and characterised by well defined, red scaly patches of variable size which heal with atrophy, scarring & pigmentary changes. The histology is characteristic. Squamous cell carcinoma and less commonly basal cell carcinoma occasionally occur in scars of DLE particularly over scalp, ears, lips & nose. In one study by Millard et al the incidence was 3.3% among 120 white patients with CCLE. We report a case of squamous cell carcinoma (SCC) developing over lesions of disseminated DLE other than scalp and lip in an Indian patient. A 40-year-old male patient came with a large erythematous ulcerated plaque over dorsum of left foot of size 5cm x 4 cm in size, over it he had 5-6 cauliflower like masses. Histopathological examination showed presence of keratin pearls suggestive of squamous cell carcinoma.

Keywords: Discoid lupus erythematosus(DLE)/Chronic cutaneous lupus erythematosus(CCLE), Squamous cell carcinoma(SCC), Keratin pearls.

I. Introduction

Discoid lupus erythematosus (DLE) is a benign disorder of the skin, clinically characterized by red scaly patches which heal with atrophy, scarring and pigmentary changes, and histopathologically by vacuolar degeneration of basal cell layer of epidermis and patchy dermal lymphocytic infiltrate. DLE is subdivided into a localized form in which lesions are confined to the face and neck or a disseminated form in which lesions also occur elsewhere on the body.[1] Malignant transformation is a rare complication of this condition and The scalp is the most common site involved followed by the lips. We report a case of squamous cell carcinoma (SCC) developing over lesions of disseminated DLE other than scalp and lip.

II. Case History

A 40-year-old male patient farmer by occupation came with a large erythematous ulcerated plaque over dorsum of left foot of size 5cm x 4 cm in size, over it he had 5-6 cauliflower like masses [figure 2]. 8 years ago, he developed a papule over dorsum of left foot which grew in size and ruptured to form erythematous plaque over 2-3 yrs. Then 5 years ago he noticed a fungating mass developing over the plaque which increased in size and number.

On examination, there were multiple, asymmetrically distributed, erythematous plaques of varying sizes over the extensor aspect of both forearms, dorsum of both hands and few lesions over left shoulder [figure 1]. Some of the plaques had adherent scaling, depigmentation and atrophy. An erythematous ulcerated plaque over dorsum of left foot of size 5cm x 4 cm in size, over it he had 5-6 cauliflower like masses largest being 2x2cm which was friable and bleeds easily on touch. [figure 2]. Left inguinal lymph nodes were enlarged. Systemic examination was normal. Hematological investigations were within normal limit. Antinuclear antibodies (ANA) was positive. Chest x ray was normal.

III. Results

The skin biopsy from a lesion over the forearm was performed and sent for histopathological. Histopathology showed epidermal hyperplasia with irregular hypergranulosis dermal perivascular lymphocytic infiltrate consistent with DLE [Figure 3]. Excisional biopsy of the fungating mass over foot lesion was done and sent for histopathological examination which showed features of well differentiated SCC, such as hyperkeratosis, dense superficial and deep perivascular lymphocytic infiltrate, foci of individual cell keratinisation keratin pearls formation can be seen and malignant cells invading dermis. [Figure 4,5&6]. The patient was treated with oral hydroxychloroquine, topical steroid, and sunscreen agent. SCC was managed by surgeons by performing wide excision of the tumour followed by skin grafting.

IV. Discussion

In disseminated DLE, characteristic lesions occur in widespread pattern over trunk and limbs. It is most commonly seen in women, tends to be persistent, resistant to therapy, and may be associated with severe

psychological upset.[1] The risk of a patient with DLE developing systemic lupus erythematosus (SLE) is small. It varies from 1.3% to about 6.5%. The risk is higher with disseminated DLE (22%) than in DLE confined to head and neck (1.2%).[1] The presence of laboratory abnormalities in DLE does not itself appear to predispose to the development of SLE.[1] This patient did not have any features of SLE, despite having disseminated DLE for more than 7 years. DLE patients showing signs of nephropathy, presence of arthralgias and elevated ANA titers (> or=1:320) should be carefully monitored, because they may be at risk of developing systemic LE.[2]

Squamous cell carcinoma (SCC) and, less commonly, basal cell carcinoma (BCC) are the most feared complications of DLE/CLE. In one study by Millard *et al.*, the incidence was 3.3% among 120 white patients with DLE/CLE.[3]The scalp is the most common site involved, followed by the lips.[3] . Malignancy in our patient may be related to his occupation as farmer necessitating frequent and prolonged exposure to sunlight. Heavy smoking could also be a contributing factor.

Squamous cell carcinoma usually arises in skin damaged by actinic rays. Exposure to chemicals such as coal tar, soot, arsenic and a variety of oils and distillation products is also implicated in its pathogenesis. It occasionally occurs in scars following inflammatory or degenerative processes. It is an end-stage complication of a wide array of inflammatory skin conditions.[4] Due to mutation in p53 tumor suppressor gene, there will be defect in apoptosis of keratinocytes that have sustained UV-radiation-induced DNA damage which ultimately lead to SCC.[5] Usually SCC in DLE develops after about two decades[1] but earlier onset has also been reported.[6,7] Cutaneous squamous cell carcinomas that arise secondary to inflammatory and degenerative processes have a much higher rate of metastasis than those developing in sun damaged skin. The interval between development of DLE and SCC has varied from 4 to 20 years.[4,6,10] Precipitating factors for SCC are age more than 40 years, male sex, sun/ultraviolet ray exposure, skin pigmentation, and chronic inflammatory processes. There is an inverse relation between skin pigmentation and development of SCC because of the protective effect of melanin.[5]

There have been sporadic reports of neoplastic change in DLE which range from SCC and basal cell carcinoma to malignant fibrous histiocytoma and atypical fibroxanthoma.[9]

The long-term prognosis of such cases is varied. SCC arising in DLE is regarded as a locally aggressive but low-grade carcinoma with recurrences. One study reported local recurrences in about 20% and metastasis in 30% cases.[11] Death has also been reported from multiple metastases.[12]

V. Conclusion

Neoplastic change occasionally occur in scars of DLE, particularly on scalp, ears, lips & nose. In white patients incidence noted where 3.3%, black people with DLE may also develop carcinoma especially of lip. Our patient had disseminated DLE with neoplastic change over uncommon site. To conclude SCC in a patient with DLE over dorsum of foot is rare in Indian patients.

References

- [1]. Goodfield MJ, Jones SK, Veale DJ. The connective tissue diseases (Discoid Lupus Erythematosus) In: Burns T, Breathnach S, Cox N, Griffiths C, editors. Rook's Textbook of Dermatology. 7th ed. Oxford: Blackwell Science Ltd; 2004. pp. 5–24.
- [2]. Tebbe B, Mansmann U, Wollina U, Auer-Grumbach P, Licht-Mbalyohere A, Arensmeier M, et al. Markers in cutaneous lupus erythematosus indicating systemic involvement. A multicenter study on 296 patients. *Acta Derm Venereol.* 1997;77:305–8. [PubMed]
- [3]. Millard LG, Barker DJ. . Development of squamous cell carcinoma in chronic discoid lupus erythematosus. *Clin Exp Dermatol.* 1978;3:161–6. [PubMed]
- [4]. Kar BR, Nair V, Ebenezer G, Job CK. Squamous cell carcinoma of the scalp arising from chronic cutaneous lupus erythematosus: report of two Indian patients. *Indian J Dermatol Venereol Leprol.* 2004;70:236–8. [PubMed]
- [5]. Grossman D, Leffell DJ. Squamous Cell Carcinoma. In: Wolff K, Goldsmith LA, Gilchrist BA, Paller AS, editors. Fitzpatrick's Dermatology in general medicine. 7th ed. New York: McGrawHill; 2008. pp. 1028–36.
- [6]. Nair VL, Chacko M. Disseminated discoid lupus erythematosus with squamous cell carcinoma. *Indian J dermatol Venereol Leprol.* 1991;57:196–7.
- [7]. Dawn G, Kanwar AJ, Dhar S, Nanda R. Squamous Cell Carcinoma over Disseminated Discoid Lupus Erythematosus on Non-photoexposed Skin. *Ind J Dermatol Venereol Leprol.* 1994;60:217–8.
- [8]. Kirkham N. Tumors and cysts of epidermis. In: Elder DE, Elenitsas R, Johnson BL, Murphy GF, editors. Lever's Histopathology of the skin. 10th ed. Philadelphia: Wolters-Lippincott; 2009. pp. 791–849.
- [9]. Matsushita S, Ishihara T, Kageshita T, Egawa K, Miyake T, Ono T. Multiple squamous cell carcinomas arising in lesions of discoid lupus erythematosus. *J Dermatol.* 2004;31:73–5. [PubMed]
- [10]. Pandhi RK, Gupta R, Kumar SA, Bhutani LK. Discoid Lupus Erythematosus in Northern India. *Ind J Dermatol Venereol Leprol.* 1984;50:97–9.
- [11]. Sulica VI, Kao GF. Squamous-cell carcinoma of the scalp arising in lesions of discoid lupus erythematosus. *Am J Dermatopathol.* 1988;10:137–41. [PubMed]
- [12]. Martin S, Rosen T, Locker E. Metastatic squamous cell carcinoma of the lip. Occurrence in blacks with discoid lupus erythematosus. *Arch Dermatol.* 1979;115:1214. [PubMed]



Figure 1 Lesions of DLE over dorsum of right hand.



Figure 2 Showing Fungating masses(SCC) over dorsum of left foot.

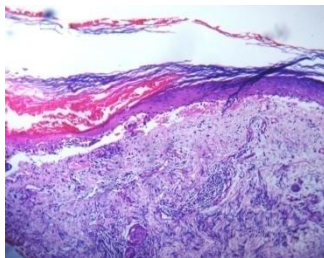


Figure 3 Histopathology of lesion over the forearm (hematoxylin and eosine staining, original magnification 10X)

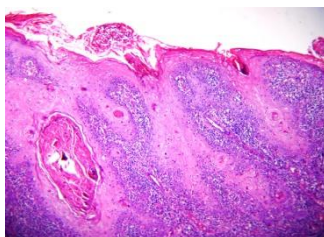


Figure 4 Histopathology of fungating mass (hematoxylin and eosine staining, original magnification 10X)

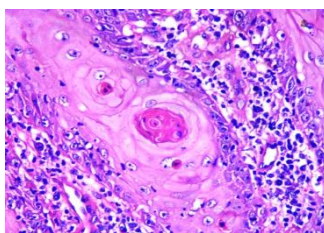


Figure 5 Histopathology of SCC showing keratin pearls under high magnification (hematoxylin and eosine staining, original magnification 40X)

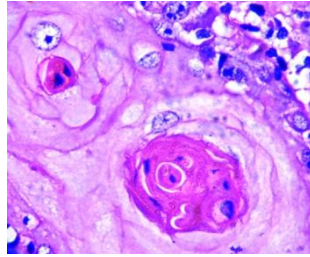


Figure 6 Histopathology of SCC showing keratin pearls under high magnification (hematoxylin and eosine staining, original magnification 100X)