

Case Report

Keratoacanthomas Over Pre-existing Discoid Lupus Erythematosus: A Rare Case Report

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ABSTRACT

Discoid lupus erythematosus (DLE) is a chronic form of cutaneous lupus erythematosus, resulting in scarring and atrophy. Keratoacanthoma (KA) is a borderline cutaneous malignancy that is known to have a rapid evolution but spontaneous regression or good prognosis. The development of malignancies like squamous cell carcinoma over DLE plaque is known. The use of antimalarials for treatment with a better understanding of the disease has led to a decrease in the incidence of such a phenomenon. Thus, in this era, the occurrence of KA over DLE is extremely rare. Herein, we report a rare case of KA occurring over a DLE plaque in a 50-year-old patient. The patient was diagnosed based on clinico-histopathological correlation, and then started on acitretin, and referred to surgical oncology for excision of the tumour.

Keywords: Discoid lupus erythematosus, Keratoacanthoma, Lupus erythematosus, Neoplasm

INTRODUCTION

Discoid lupus erythematosus (DLE) is one of the most common forms of chronic cutaneous lupus erythematosus, which leads to scarring and atrophy. Keratoacanthoma (KA) is a low-grade neoplasm of the skin that is known to have a rapid evolution but spontaneous regression or good prognosis.^[1] The development of malignancies like squamous cell carcinoma (SCC) over a chronic inflammatory cutaneous lesion is known, but the development of KAs is rare.^[2] Development of a malignancy in DLE is mostly linked to chronic ultraviolet radiation exposure with altered immunity in a DLE plaque. Sometimes, the DLE disease itself can lead to a KA-like lesion, adding to the confusion in the diagnosis. Herein, we report a case of biopsy-proven KA occurring over a DLE plaque.

CASE REPORT

A man in his 50s, a farmer by occupation, presented with multiple erythematous depigmented atrophic plaques of varying sizes, predominantly over photo-exposed body areas such as forearms, scalp and face. The patient was diagnosed with DLE based on histopathology and antinuclear antibody titre positivity of 3+ in 1:100 dilution on HeLa cells.

He was on irregular therapy with hydroxychloroquine and methotrexate for the past 3–4 years. The patient also had multiple rapidly growing hyperkeratotic nodular plaques over the depigmented plaques on the forearm for the past 3 months [Figure 1]. No regional lymphadenopathy was present on clinical examination. The patient did not have any other systemic complaints.

Biopsy was taken from two sites: One from the active margin of the atrophic plaque with a provisional diagnosis of DLE and one from the hyperkeratotic plaque with differential diagnoses of

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SCC and KA. The rest of the investigations were within normal limits. Histopathology of atrophic plaque showed atrophic epidermis with lymphocyte exocytosis, interface dermatitis, perivascular lympho-histiocytic infiltrate and melanin incontinence in the dermis, suggestive of DLE [Figure 2]. Histopathology of hyperkeratotic plaque showed a crater with eosinophilic glassy cytoplasm with few mitotic figures and neutrophilic microabscesses with a dense lichenoid infiltrate of lymphocytes, suggestive of KA [Figure 3].^[1] The patient was started on acitretin and referred to the surgical oncology department for excision of the tumour.

DISCUSSION

KA is a borderline neoplasm assumed to originate from a hair follicle and has a good prognosis.^[1] It has been reported over

pre-existing skin lesions of psoriasis, seborrheic dermatitis and leprosy.^[2,3] Neoplastic changes over DLE were seen earlier due to the use of irradiation and irritants for treatment.^[4] This scenario has changed since the use of antimalarials and steroids for its treatment.^[4] Malignancy like SCC occurring over DLE plaques has been reported with an incidence of around 3.3%.^[5] However, the occurrence of KA over DLE is rare; and the list of reports of such a phenomenon has been tabulated [Table 1].^[2-4,6-8] Age >40 years; chronic occupational sunlight exposure; chronic inflammation, pigment loss and scarring due to DLE; and immunosuppression/altered immunity due to the medications given may have contributed to the development of KA over DLE in the present case. However, a KA-like lesion occurring due to the disease itself in DLE lesions has been described by Georgesen and Magro.^[9] In such cases, the presence of dermal mucin, lymphocyte-mediated interface dermatitis and basement membrane zone thickening can differentiate between the two disease processes.^[9]

KA is usually confused with SCC but has many peculiar clinical and histopathological differences from SCC. KA is known to have a triphasic evolution divided into a rapid proliferative phase, maturation phase and regression phase. It has a very low risk of metastasis and can spontaneously regress.^[1] Differentiating KA over SCC can be very difficult on histopathology, so the following features have been used to distinguish the two entities to a certain extent like our case.^[10,11]

1. KAs show symmetry and epithelial lipping and SCCs do not
2. SCCs often exhibit more cytologic atypia, anaplasia and pleomorphism than KAs
3. Ulceration and mitoses are in favour of SCC
4. KAs do not spread beyond the sweat glands whereas SCCs can
5. Clear distinction between proliferating tumour and stroma favour a diagnosis of KA
6. Intraepithelial microabscesses, intraepithelial elastic fibres and eosinophilic glassy cytoplasm of keratinocytes without keratin pearls may favour the diagnosis of KA.



Figure 1: (a and b) Multiple hyperkeratotic plaques of varying sizes with a crateriform appearance present over a background of hypopigmented to erythematous atrophic skin on the forearm.

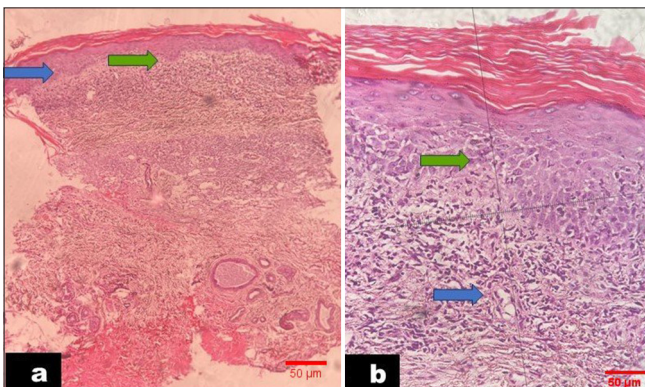


Figure 2: (a) Histopathology (haematoxylin and eosin [H&E], ×40) of the hypopigmented lesion shows shortening of rete ridges (blue arrow) with interface dermatitis (green arrow) in the dermis. (b) Histopathology (H&E, ×400) shows an atrophic epidermis with lymphocytic exocytosis and interface dermatitis (green arrow) with perivascular lymphocytic infiltrate (blue arrow).

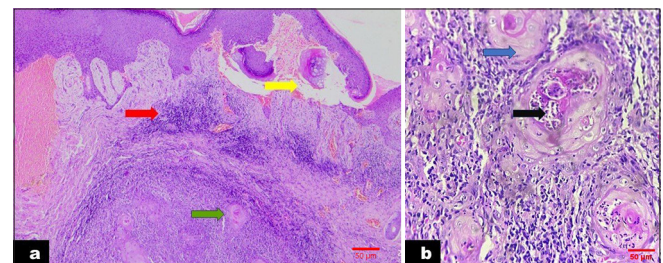


Figure 3: (a) Histopathology (haematoxylin and eosin [H&E], ×100) shows an epidermal crater (yellow arrow) with an aggregate of lymphocytes in the dermis (red arrow) and eosinophilic glassy cytoplasm of keratinocytes (green arrow) in the centre of the infiltrate. (b) Histopathology (H&E, ×400) shows a high-power view of eosinophilic glassy cytoplasm (blue arrow) with intraepithelial microabscesses (black arrow).

Table 1: Reported cases of KA in lupus erythematosus cases.

| Date of publication | Case details | Proposed cause | References |
|---------------------|---|---|--|
| 1966 | A 43-year-old female with DLE developed KA Treated with antimalarials, antimetabolites and intralesional steroids | UV radiation | Jolly and Carpenter ^[6] |
| 1974 | 1 out of 71 biopsies done for DLE had findings of KA | UV radiation | Farah <i>et al.</i> ^[4] |
| 1986 | A 62-year-old male with disseminated DLE developed multiple KAs | Concurrent HPV infection, loss of immunity and chronic inflammation | Mittal <i>et al.</i> ^[7] |
| 1989 | 31-year-old female with DLE developed multiple KAs Treated with etretinate at 1 mg/kg/day for 2 months with complete resolution | Chronic inflammation | Fanti <i>et al.</i> ^[2] |
| 1997 | A 23-year-old woman with SLE developed multiple KAs Treated with isotretinoin at 1 mg/kg/day for 8 weeks with complete clearance | Immunosuppression | Dessoukey <i>et al.</i> ^[3] |
| 2007 | A 14-year-boy with DLE had a crusted ulcer on the lower lip, diagnosed as KA on histopathology | UV radiation | Minicucci <i>et al.</i> ^[8] |

KA: Keratoacanthoma, UV: Ultraviolet, DLE: Discoid lupus erythematosus, HPV: Human papillomavirus, SLE: Systemic lupus erythematosus

CONCLUSION

This case report highlights the rare occurrence of KA over DLE, which should be considered while managing a hypertrophic lesion in a DLE patient, significantly differentiating it from SCC or hypertrophic DLE as the treatment and prognosis are different in all the diseases.

Ethical approval: Institutional Review Board approval is not required.

Declaration of patient consent: The authors certify that they have obtained all appropriate patient consent forms. In the form, the patients have given their consent for their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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