Poorly differentiated squamous cell carcinoma arising within a lesion of discoid lupus erythematosus in an African-American woman

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INTRODUCTION

The incidence of skin cancer is rare in African Americans; however, there are several reports of patients with depigmented or hypopigmented lesions on sun-exposed areas, such as the scalp, who are at increased risk for squamous cell carcinoma (SCC). Decrease in melanin, immunosuppressive therapy, frequent ultraviolet exposure, human papillomavirus infection, chronic scarring, and inflammatory processes are factors associated with the development of SCC in black patients.[1] We present a rare case of a 55-year-old African-American woman who had SCC within a discoid lupus erythematosus (DLE) lesion.

Abbreviations used:

DLE: discoid lupus erythematosus
SCC: squamous cell carcinoma
SLE: systemic lupus erythematosus
UV: ultraviolet

CASE REPORT

A 55-year-old African-American woman with a 40-year history of systemic lupus erythematosus (SLE) presented with a cutaneous discoid lesion on her scalp. She had a several-month history of a large, tender, nonhealing wound within her discoid lupus lesion on her scalp. Her SLE was managed with prednisone for many years. Physical examination found a 12- × 18-cm depigmented, pink, atrophic plaque on the vertex of the scalp. In the center of the plaque was an 8- × 10-cm ulcerated, exudative, exophytic mass (Fig 1). Additional findings included anonychia, violaceous papules on the chest, and depigmented patches on the bilateral breasts, axillae, and inguinal creases. Two 4-mm punch biopsy specimens from the scalp showed a poorly differentiated pleomorphic spindle and epithelioid cell infiltrate containing numerous mitoses (Fig 2).

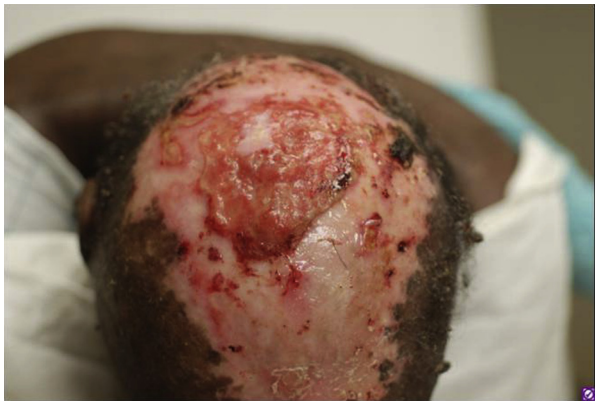

Fig 1. Clinical presentation of the SCC arising within DLE lesion on vertex of scalp. Depigmented, pink atrophic plaque with central ulcerated mass.

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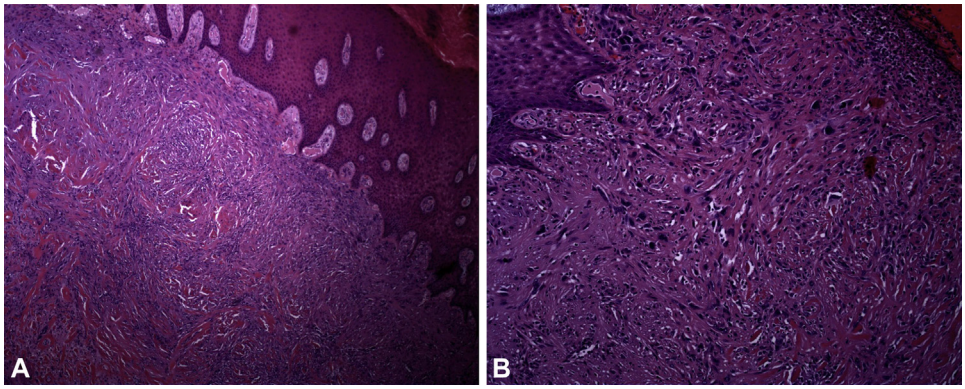

Fig 2. Histopathologic features of SCC. **A**, Punch biopsy from the scalp shows a poorly differentiated pleomorphic spindle and epithelioid cell infiltrate in association with dermal cicatrix. (Hematoxylin-eosin stain; original magnification: ×4.) **B**, Higher magnification image shows atypical spindled and epithelioid cells with enlarged pleomorphic and hyperchromatic nuclei and numerous mitoses. (Vimentin stain positive; original magnification: ×10.)

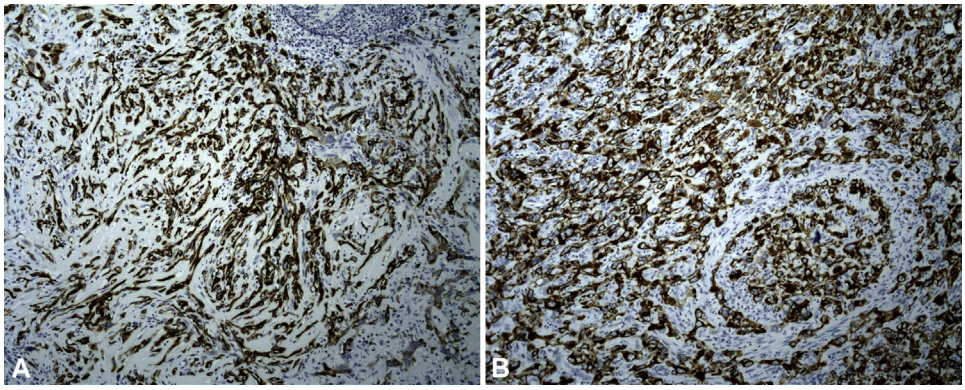

Fig 3. Immunoperoxidase study of SCC. **A**, Antibodies to pan cytokeratin show focal strong positive staining. (Cytokeratin 8 and 18 stain positive; original magnification: ×4.) **B**, Higher magnification of positive cytokeratin staining of many cells. (Cytokeratin 8 and 18 stain positive; original magnification: ×10.)

Initial and repeat immunoperoxidase studies with antibodies to vimentin were positive for spindle cells. Pan cytokeratin showed strong positive focal staining. S100, Melan A, CK7, Desmin, CD45, CD31, and CD34 immunostains were normal (Fig 3). The patient was referred to our multidisciplinary oncology clinic for further evaluation and treatment options. Positron emission tomography/computed tomography of the head was negative for cranial invasion or organ metastasis. The patient had a wide local excision of the tumor with local tissue rotation flap and full-thickness skin graft. The final diagnosis was poorly differentiated spindle and pleomorphic cell SCC with perineural invasion and extension into subcutaneous fat. Unfortunately, the patient was lost to follow-up, so we are unable to comment on her current status.

DISCUSSION

The association between DLE and SCC was reported as early as 1953.[2] The head and neck and the forearms are the most common locations for SCCs arising from DLE lesions. Risk factors for the development of SCC in lesions of DLE include the use of immunosuppressive therapy, chronic ultraviolet (UV) light exposure, human papillomavirus infection, and chronic scarring and inflammatory processes.[1] A recent study reported an almost 4-fold increased risk for nonmelanoma skin cancer in patients with DLE lesions.[3] SCC occurring in DLE lesions may also be more aggressive, as evidenced by increased rates of recurrence, metastasis, and mortalities, than other SCCs.[4]

SCC occurring in lesions of DLE is rare, but SCC occurring in DLE lesions in skin of color is even rarer. Fewer than 25 case reports have documented SCC arising within DLE in black patients. Black skin is

histologically found to contain larger, more melanized melanosomes, which allow it to filter almost twice the amount of UVB light as fair skin.[5] This serves as a protective mechanism against the ultraviolet induction of photocarcinogenesis.[6,7] Gervin et al[7] and Mulwafu et al[8] suggested that the frequent exposure of UVB light to areas lacking protection from melanin pigment causes the p53 tumor suppressor gene to be inactivated, which promotes the development of SCC in discoid lesions of black patients.[7,8]

The African-American population is at a lower risk for skin cancer, but when skin cancer does occur, SCC is the most common type found.[9] The limited evidence available suggests that having chronic DLE puts African Americans at increased risk for SCC. Additional cases of SCC in skin of color have been documented in preneoplastic dermatoses like albinism, xeroderma pigmentosum, and epidermodysplasia verruciformis.[10] For the black population, chronic inflammatory or scarring processes account for 20% to 40% of SCC cases and constitute the greatest predisposing factors for development of SCC. When SCC develops in chronic scarring processes in this population, the metastasis rate is 20% to 40%, compared with 1% to 4% when SCC develops from sun exposure in fair skin.[9]

We present this case to raise awareness of this potential complication in black patients. SCC occurring in lesions of DLE in black skin is thought to have a higher risk for metastasis than SCC occurring without known predisposing factors. Additionally, patients with skin of color in general are thought to be at an increased risk of poor outcomes from skin cancers, possibly because of delayed diagnosis. Early intervention and treatment are especially important to reduce the risk of morbidity and mortality when SCC occurs in DLE lesions in these patients. Patients with DLE should be advised to have frequent follow-up examinations, use broad-spectrum sunscreen regularly, and avoid direct sun exposure to discoid lesions.

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