

# Squamous cell carcinoma in black patients with discoid lupus erythematosus

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## Summary

Squamous cell carcinoma has rarely been reported in black African people, with only 11 cases reported in the world literature to date. We report on 2 further cases, the first to be reported in southern Africa, of squamous cell carcinoma in lesions of discoid lupus erythematosus

Lupus erythematosus is thought by clinicians to be common in Cape Town, particularly in people of mixed ancestry, although no accurate epidemiological figures are available to confirm this impression. A diagnosis of discoid lupus erythematosus (DLE) can frequently be made on clinical grounds.<sup>1</sup> Where the diagnosis is in doubt, histological confirmation is sought, but biopsy is not a routine procedure in clinically characteristic cases. Cutaneous squamous cell carcinoma (SCC) is seen in some of these patients, as a complication of this chronic inflammatory skin disease, but has not previously been reported in southern Africa. We report on 2 black African women with SCC occurring in longstanding DLE lesions.

## Case 1

A 40-year-old black South African woman with DLE, a heavy smoker and chronic alcohol abuser, had previously been seen at the Dermatology Clinic at Groote Schuur Hospital in 2001. At the time she had been given oral chloroquine phosphate and topical betamethasone valerate for 1 month. According to the usual practice in this dermatology clinic, biopsy confirmation was not sought as a confident clinical diagnosis was made. Thereafter, she had been lost to follow-up, with no further treatment. She presented again in September 2004 with a large ulcerative lesion on the upper lip, present for 4 months. The oral commissures were uninvolved. Biopsy showed moderately differentiated SCC. Her DLE was clinically inactive at this stage and review of the results of histological examination of skin adjacent to her tumour showed epidermal atrophy and follicular plugging, compatible with late DLE. She underwent wide local excision of the tumour and bilateral neck dissections. Reconstruction of the upper lip defect was done using the Abbey lip switch technique. The left neck dissection

specimen had 1 positive node out of 27 and the right neck dissection had 2 positive nodes out of 18. The patient is currently undergoing radiotherapy.

## Case 2

A 45-year-old woman of mixed ancestry (Fig. 1) had previously been treated at the Dermatology Clinic at Groote Schuur Hospital between 1982 and 1989 for DLE, with oral chloroquine phosphate and topical betamethasone valerate. She presented to the clinic again in 2004, with a 2 x 2 cm ulcerative lesion of the lower lip, present for 2 months and involving the vermilion border close to the right commissure. She was a heavy smoker and drinker. Biopsy showed SCC. The original diagnosis of DLE was based on classic clinical findings, in line with current practice in this clinic. Review of the results of histological examination of the skin adjacent to the tumour showed mild focal interface changes and a lymphocytic infiltrate in the upper dermis and periadnexal areas. This is compatible with, but not diagnostic of underlying DLE. Wide local excision of the tumour and a right neck dissection were performed. Reconstruction of the lip defect was done using Benard-Webster local advancement flaps. Histological examination revealed that the right neck dissection specimen was pathologically not involved.



Fig. 1. Patient with DLE on the lips, nose and malar area and SCC of the lower lip.

**TABLE I. CLINICAL FEATURES OF 10 AFRICAN-AMERICAN AND AFRICAN PATIENTS WITH DISCOID LUPUS ERYTHEMATOSUS AND SQUAMOUS CELL CARCINOMA**

Patient	Author	Year	Age/sex	Duration of DLE	Location of CA	Mets	Prior cytotoxic drugs for DLE	Treatment	Length of follow-up	Outcome
1	Jacobson and Annamunthodo <sup>6</sup>	1958	42/F	7	Cheek	No	No data	Excision	6 wks	No recurrence
2	Martin <i>et al.</i> <sup>7</sup>	1979	62/M	14	Upper lip	Regional and distant	None	Excision	1 yr	Tumor recurred, died
3	Martin <i>et al.</i> <sup>7</sup>	1979	54/M	18	Lower lip	Regional	None	Excision, neck dissection, radiation	No data	Alive at time of report
4	Keith <i>et al.</i> <sup>7</sup>	1980	46/M	18	Upper lip	No	None	Excision	1 month	Alive at time of report
5	Keith <i>et al.</i> <sup>8</sup>	1980	53/F	14	Scalp	No	None	Excision	4.5 yrs	No recurrence
6	Presser <i>et al.</i> <sup>8</sup>	1981	50/M	7	Elbow, pinna forearm	No	Nitrogen, mustard cyclophosphamide	Excision, radical neck dissection	No data	Alive at time of report
7	Mora and Perniciaro <sup>10</sup>	1981	35/M	30	Infranasal, upper lip	No	None	Excision, radical neck dissection	4 yrs	No recurrence
8	Dabski <i>et al.</i> <sup>5</sup>	1986	47/F	10	Cheek	Regional and distant	None	Excision, neck dissection, chemotherapy	2 yrs	Tumor recurred, died
9	Caruso <i>et al.</i> <sup>4</sup>	1987	27/F	18	Lower lip	Regional	Azathioprine	Excision, radical neck dissection, chemotherapy	1.8 yrs	Tumor recurred, died
10	Sulica and Kao <sup>11</sup>	1989	78/F	30	Scalp	Regional	None	Excision, methotrexate	1.8 yrs	Tumor recurred, died
11	Sherman <i>et al.</i> <sup>2</sup>	1991	43/M	20	Scalp, arm	No	None	Excision	2 yrs	Alive at time of report
12	Present case	Case 1	40/F	5	Upper lip	Regional	None	Excision, radical neck dissection, radiation	5 months	Alive
13	Present case	Case 2	45/F	20	Lower lip	No	None	Excision, radical neck dissection	5 months	Alive

DLE = discoid lupus erythematosus.

## Discussion

SCC comprises approximately 20% of all skin cancer.<sup>2</sup> Skin cancer is rare in black people, occasionally occurring as a complication of chronic inflammatory skin disease, in sinus tracts and in scars.<sup>3</sup> The occurrence of SCC in black people with DLE is reported infrequently, although this may not reflect the actual incidence. It has been observed in several previous patients in Cape Town, but has not been reported in the literature.

In 1993 Sherman *et al.*<sup>2</sup> analysed 10 patients who had been reported in the literature and including most of those who had previously been reported by Caruso *et al.*<sup>4</sup> in 1987. They failed to include 1 patient reported by Dabski *et al.*<sup>5</sup> in 1986. In total, 11 black people with SCC and DLE have been reported in the world literature and all have been African-Americans in the USA and Jamaica. To the best of our knowledge, our patients are the first 2 black patients to be reported from southern Africa. The clinical features of cases reported in the world literature are summarised in Table I. The number of cases is still too small to allow for statistical analysis of the data.

The initial reports suggested a male predominance.<sup>2,4</sup> However as more cases are being reported there appears to be a female predominance, possibly reflecting the gender ratio seen in DLE.

The geographical location (sunbelt areas) of the patients in Table I and location of the SCC lesions in sun-exposed areas of the body support the role of sunshine as an aetiological factor. The mechanism of ultraviolet (UV)-induced photocarcinogenesis appears to involve inactivation of the p53 tumour suppressor gene and multiple exposures are believed to be needed to inactivate p53 and initiate and promote the development of SCC.<sup>12</sup>

The development of SCC in black patients with DLE may be related to the lack of protective melanin pigment. This exposes the affected areas to the UVB component of sunlight involved in cutaneous carcinogenesis. An additional factor is likely to be the presence of chronic inflammation in DLE, as in chronic ulcers or burn wounds (Marjolin's ulcer).

Seven of the 13 cases reported in Table I involved the lips. The frequency of DLE-associated SCC at this site may reflect the level of UV exposure. A study in Beijing<sup>13</sup> identified DLE as a co-factor in 10 of 181 people with SCC of the lip (5.5%).

## Conclusion

We report on 2 cases of SCC in black patients with DLE, thought to be the first reported in southern Africa. This report serves to emphasise that DLE may be an additional background factor in the development of SCC of the skin. As black patients with DLE in sun-exposed areas of the body may be at risk of developing SCC in their skin lesions, every effort should be made to prevent direct sun exposure to the affected areas of the body, with behaviour modification and the daily use of high-factor UVA/UVB sunscreens. Regular follow-up, with prompt biopsy of any suspicious lesions, should be encouraged.<sup>14</sup>

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