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PRIMARY MALIGNANT AMELANOTIC MELANOMA ARISING FROM A VITILIGO PATCH OF AN AFRICAN TANZANIAN: CASE REPORT

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SUMMARY

Skin cancer is rare in people of African origin while vitiligo occurs worldwide. The occurrence of primary malignant melanoma and vitiligo together is very rare. We present a rare case of primary malignant amelanotic melanoma arising from a depigmented patch of a patient with vitiligo. It was completely excised and followed for one year. No recurrence or metastases was noted during the follow up period.

INTRODUCTION

Malignant melanoma is a rare tumour in people of African descent including those affected by albinism (1). The incidence of malignant melanoma in Africans is estimated to ranging from 0.5 to 1.5 per 100,000 people (2). Acrolentiginous melanoma is the most common type of melanoma in this population. Ultra violet radiation does not appear to be a significant risk factor for melanoma in blacks who tend to develop melanoma on acral, non-sun-exposed areas (2).

CASE REPORT

A 32 year old female presented with a four year

history of depigmented patches on the face, trunk and extremities. The depigmented patches were irregular, well demarcated and non-pruritic. Both upper and lower limbs were affected but sparing the hands and the feet. After three years of having depigmented patches, a small nodule appeared in the centre of a depigmented patch on the left leg. The nodule rapidly increased in size and became ulcerated. Within six months the tumour had grown to a size of four centimeters in diameter (Figure 1). Thorough examination of the left limb did not reveal any other growths. The inguinal lymph nodes were not enlarged. She had no personal or family history of melanoma.

Figure 1

Lateral and aerial view of a tumour arising from the centre of a vitiligo patch. The Brown coloration is due to povidone iodine



An excision was done with a one centimetre safety margin and deeper to sub-cutaneous tissue. The margins were examined histologically to confirm complete excision. Histologically, the tumour composed of nested atypical melanocyte (Figure 2). The tumour cells expressed S-100, Melan-A (Figure 3) and HMB-45. There were many mitotic figures and proliferation activity was high when assessed with MIB-1. At one year follow up there was complete healing without recurrence and no changes in the inguinal nodes.

Figure 2

Hematoxylin and Eosin staininig showing nested atypical melanocytes (X10)

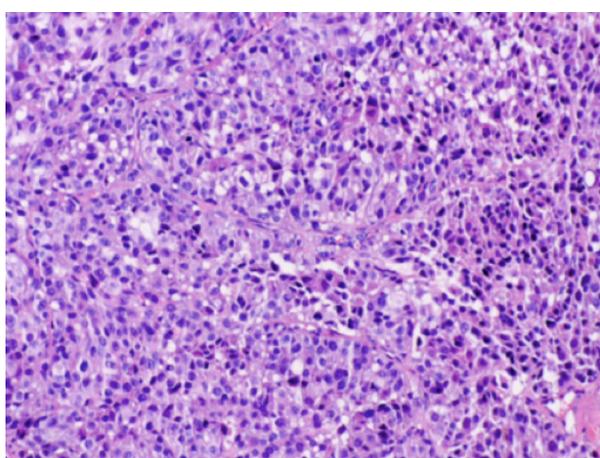
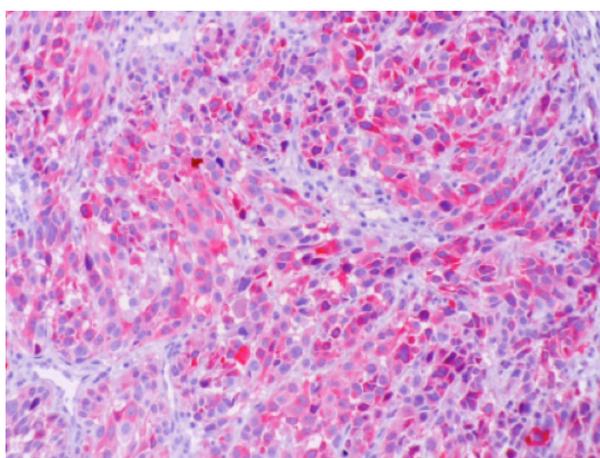


Figure 3

Tumour cells stained positive with Melan-A (X10)



DISCUSSION

The prevalence of vitiligo in the general population is estimated at 1% (3) while the prevalence of vitiligo in patients with malignant melanoma has been reported to range from 3-6% (4,5). Different depigmentary changes associated with melanoma have been reported. These pigmentary changes include halo

nevus, achromic patches on melanoma scars, depigmentation of injection site after immunotherapy and depigmentation associated with regressing malignant melanoma. The association of vitiligo with regressing melanoma has been widely reported in literature, however primary melanoma presenting initially with vitiligo is very rare. Lugo-Somolinus *et al* (6) reported two cases of vitiligo-like primary melanoma. We are reporting a similar case of primary melanoma arising in vitiliginous lesion.

The pathologic mechanism of vitiligo is not clearly known. Complex factors involving cellular and humoral immunity, toxic metabolites and genetic factors partly contribute to its pathogenesis. Ciu *et al* (7) demonstrated that antibodies to melanocytes were present in both vitiligo and melanoma patients and these antibodies were directed to similar melanocyte antigens. The implicated antigens are tyrosinase, melan A, gp100 and TRP-28. This association explains the simultaneous appearance of vitiligo and regressing melanoma, however it contradicts the occurrence of melanoma in vitiligo lesions.

Tobin *et al* (9) observed that melanocytes are never completely cleared from vitiligo lesions and that this melanocyte can recover their functionality. Similarly, Gottschalk *et al* (10) reported melanocyte survival in three cases of vitiligo which had been inactive for two to five years. Melanoma may develop from these melanocytes that have escaped immune destruction. The mechanisms of immune escape are not clearly known but Le Poole *et al* (11) hypothesised that the tumour escape may be due to early activation of T-cells secreting IFN- α which contribute to tumour escape and development of vitiligo. Vitiligo-like lesions are generally regarded as a good prognostic factor in melanoma patients (12,13). The survival rate of these patients has been shown to be better than would have been expected for a patient with a similar melanoma stage. This may be attributed to destruction of melanocyte by an enhanced immune response.

In conclusion, this is a rare presentation of a rapidly growing primary malignant amelanotic melanoma arising from vitiligo lesions without evidence of tumour elsewhere. The one year follow up period might be short to assess the recurrence.

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