intense pruritus. remissions and relapses and histopathologically no spongiosis or vesiculation was observed. Final diagnosis of naevoid psoriasis was

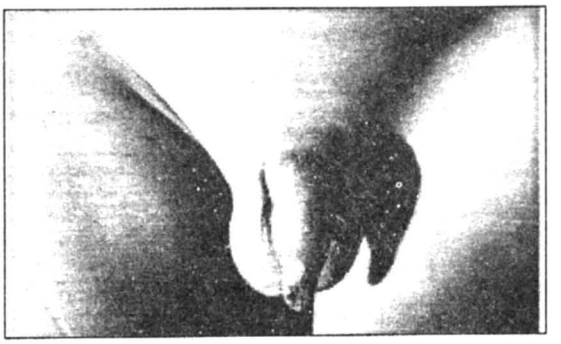

Fig.1. Showing 3 scaly erythematous linear bands of psoriasis on the penis.

established by typical histopathology of psoriasis, its presence since birth, persistent nature and absence of pruritus.

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CHROMOBLASTOMYCOSIS MASQUERADING AS PALMO-PLANTAR PSORIASIS

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A 8-year-old boy presented with scaly plaques of both soles and left palm of 4 year duration. The plaques were well defined scaly, fissured and hyperkeratotic resembling palmo-plantar psoriasis. KOH preparation of the scrapings revealed round, brown, thick-walled bodies with planate division. Grey black, velvety folded colonies were seen in culture on Sabouraud's dextrose agar. Lacto phenol cotton blue preparation revealed Fonsecaea pedrosoi *as the cause of chromoblastomycosis.*

Key Words : Chromoblastomycosis, Fonsecaea pedrosoi

Introduction

Chromoblastomycosis is a chronic fungal infection of the skin and subcutaneous tissues caused by *Phialophora verrucosa, Fonsecaea pedrosoi, F. compactum, Wangiella dermatitidis* and *Cladosporium carrionii.*[1] Patients with chromoblastomycosis have suppressed non-specific cell-mediated immunity for some antigens (skin allografts, DNCB, fungal antigens), while reactivity to bacterial and mycobacterial antigens is not

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impaired.[2] Adult male agricultural workers are most often affected. The infection usually results from trauma such as puncture from woodsplinter. The causative fungi have been cultured from patient's lesion and from the tree branch responsible for trauma.[3] Clinically it presents as a warty papule or brownish warty plaque on exposed site, particularly on feet and legs, arms, face and neck. Occasionally it may manifest as a tumour.[4] Rarely it may manifest as mycetoma.[5] Cerebral chromomycosis is most commonly caused by *Cladosporium trichoides*. Most cases of cerebral chromomycosis have occurred in immunocompetent hosts. Recent experimental evidence suggests that host immunosuppression may predispose patients to the disease.[6]

Case Report

A 8-year-old boy from Latur was brought to our hospital with complaints of painful fissures and localised thickening of both soles and left palm of 4 years duration. Lesions started as fissures on forefoot of right sole and it later involved the instep of left sole and left palm. The lesions were associated with itching. There was history of yellowish discharge tinged with blood during the course of the disease. Cutaneous examination revealed well-defined scaly, hyperkeratotic fissured plaques over medial aspect of plantar surface of right sole, instep of both soles and centre of left palm. Most of the fissures were superficial. Scaling was present. Hair, nails and mucous membranes were normal.

He was clinically diagnosed as palmoplantar psoriasis and was investigated further. Scrapings taken from plaques in KOH mount showed round brown thick-walled bodies with planate division. Culture on Sabouraud's agar yielded growth of *Fonsecaea pedrosoi*. Biopsy of skin from plantar plaque showed the features of lichen simplex chronicus.

Discussion

Chromoblastomycosis presenting like psoriasis is rare. It is also rare for chromoblastomycosis to involve both palms and soles together. To the best of our knowledge this is the first report of its kind. The reported case presented with bilateral palmoplantar scaly dermatosis. Clinically he was diagnosed as palmoplantar psoriasis. The differential diagnosis considered include tinea pedis, hyperkeratotic eczema, erythrokeratolysis, and contact allergic dermatitis. The diagnosis of chromoblastomycosis was not entertained as the case did not have any of the features of it. It is only the KOH preparation of the scrapings and the culture that helped to diagnose the case as chromoblastomycosis. Hence, it is mandatory to do KOH preparation, and culture from the scrapings in all cases of palmoplantar scaly dermatosis, so that an early diagnosis of chromoblastomycosis can be made. Histopathology did not corraborate the diagnosis of chromoblastomycosis. The reason for it may be that the infection being in early stage may not have gone in to granuloma formation.

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