CASE REPORT

A rare case of histoplasmosis in a renal allograft recipient

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ABSTRACT

Histoplasmosis is a very rare disease in non-endemic areas. It is a granulomatous disease caused by a saprophytic dimorphic fungus *Histoplasma capsulatum*. In immunocompromised individuals, it may cause a progressive and potentially fatal disseminated disease. Isolated cutaneous histoplasmosis is rare in renal transplant recipients. We report a case of a 45-year-old male who presented with skin lesions 4 years after renal transplantation. Histopathological examination of biopsy taken from the skin lesions showed intracellular budding yeast cells and was confirmed by culture of the biopsy sample. The patient was treated with Itraconazole and responded well with disappearance of all skin lesions.

Key words: Allograft, cutaneous lesions, histoplasmosis, renal transplant

INTRODUCTION

Histoplasmosis is a chronic granulomatous fungal infection worldwide in distribution, caused by a saprophytic dimorphic fungus *Histoplasma capsulatum*. Histoplasmosis may manifest as (1) acute pulmonary histoplasmosis, (2) chronic pulmonary histoplasmosis, (3) cutaneous, mucocutaneous histoplasmosis and (4) disseminated histoplasmosis. Infections caused by histoplasma are reported from the Gangetic plains in India. Histoplasma infection in renal allograft recipient is rare, and the incidence of post-organ transplant histoplasmosis is 1 case per 1000 person-years.[1] We report a case of isolated subcutaneous histoplasmosis in a renal allograft recipient in a non-endemic area with no documented exposure.

CASE REPORT

A 45-year-old businessman presented with swellings of the left forearm, right thigh, left foot and history of fever off and on of 4-month duration. The patient underwent renal transplantation 4 years back and has been on triple immunosuppressant therapy since then.

On examination, ill-defined swellings were found on the left forearm (4 cm × 5 cm), right thigh (3 cm × 3 cm) and left foot (5 cm × 6 cm). Skin over the swellings was erythematous and tender. On palpation, swellings were variable in consistency, margins were ill defined and not fixed to the skin or underlying structures (muscle or bone).

Laboratory investigations were normal except for raised serum creatinine (4.2 mg/dl) and mildly elevated total leucocyte count (10,000/cumm). X-ray chest was normal, Doppler imaging of the graft kidney was normal. Magnetic resonance imaging of limbs showed subcutaneous abscesses extending to intermuscular planes. Blood and urine culture did not grow any bacterium.

Before presenting to our centre, he received multiple antibiotics fine needle aspiration cytology of the swellings which was inconclusive. Biopsy specimen of one of the swellings was sent for bacterial and fungal cultures and histopathological examination. Histopathology showed

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intracellular budding yeast cells. Biopsy sample was inoculated onto Sabouraud's dextrose agar and incubated at 25°C. White and fluffy colonies were seen after 3 weeks of incubation [Figure 1]. Lactophenol cotton blue mount showed tuberculate macroconidia on long conidiophores [Figure 2]. Dimorphism was demonstrated by inoculating colonies onto brain heart infusion agar and incubated at 37°C. *H. capsulatum* was confirmed by a positive urease test.

Treatment was initiated with Itraconazole, and the dose of immunosuppressants was de-escalated. The patient responded dramatically, and all swellings disappeared. His serum creatinine level decreased to 1.6 mg/dl.

DISCUSSION

Histoplasmosis is caused by *H. capsulatum var. capsulatum* and *H. capsulatum var. duboisii*. Histoplasmosis occurs either after primary infection or after reactivation of latent infection, and its incidence among immunocompromised persons is on the raise. Despite of its worldwide distribution, histoplasmosis is uncommonly reported from India.[2] This might be due to poor diagnostic facilities and lower index of suspicion. Histoplasmosis is rare in renal transplant recipients. It does not appear to be related to the degree of immunosuppression and occurs any time after renal transplantation.[2] Primary histoplasmosis is acquired by inhalation of dust particles contaminated with bird or bat droppings that contain microconidia or by direct inoculation into skin or through an infected allograft.[3] The outbreak of histoplasmosis has been reported in cave explorers.[4] Immunosuppressed patients may develop disseminated disease, septic shock and carries a high mortality up to 23%. The largest Indian study of 24 cases reported that histoplasmosis is common in patients with diabetes and HIV-positive patients.[5] Additional case reports from India have reported pulmonary,[6] cutaneous[7] and genitourinary histoplasmosis.[8] Disseminated histoplasmosis was reported in the past. Skin lesions in histoplasmosis range from papulae and plaques with or without crusts, pustules and nodules to mucosal ulcers and erosions, molluscum contagiosum-like lesions, acneiform eruptions, erythematous papules and keratotic plaques. In non-endemic areas, due to the remarkable morphologic variation and non-specific appearance of skin lesions in histoplasmosis, a high index of suspicion is needed to make an early diagnosis. The prognosis of disseminated disease is worse than in cutaneous form, and a significant delay in diagnosis is reported in the cutaneous form. Limaye *et al.* reported disseminated histoplasmosis in two recipients from a single donor, who lived in an area where histoplasmosis is highly endemic. The donor was asymptomatic and died in a motor vehicle accident. Treatment with Amphotericin B was effective in both patients.[9] Wong and Allen reported funguria in a renal allograft recipient during her early post-operative period and later she also developed cutaneous manifestations.[10] Rathod *et al.* reported primary cutaneous histoplasmosis in a male patient who presented with skin lesions on face 14 months following renal transplantation.[7]

In our case, the patient presented with multiple subcutaneous lesions 4 years after the renal transplantation. He is on regular follow-up. There is no recurrence of cutaneous lesions.

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Conflicts of interest
There are no conflicts of interest.

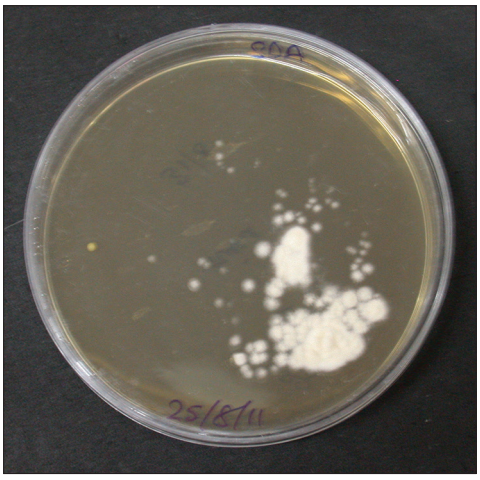

Figure 1: Culture on Sabouraud's dextrose agar showing white cottony colonies

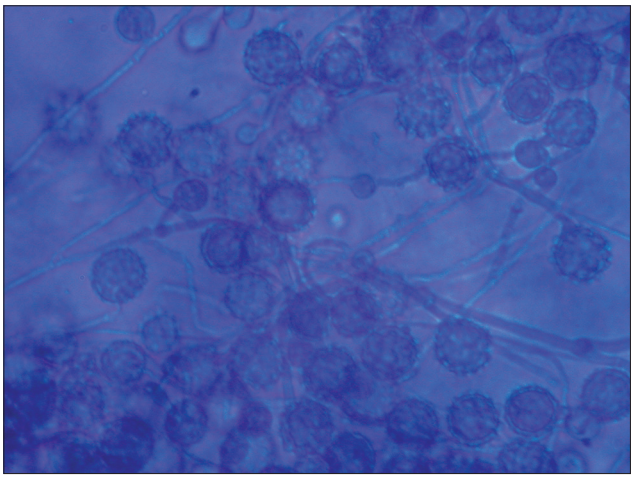

Figure 2: Large, single-celled tuberculate macroconidia borne on short conidiophores (lactophenol cotton blue, ×10)

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