Atypical presentation of dermatophytosis: a presenting feature in three HIV patients

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Dear Editor,

Dermatophytosis is a superficial fungal infection of keratinized tissues like skin, hair, and nails [1]. Dermatophytosis manifests as an opportunistic infection being four times more prevalent in Acquired Immunodeficiency Syndrome (AIDS) patients [2,3]. In India, the incidence of superficial fungal infections in Human Immunodeficiency Virus (HIV)-positive individuals is variable, ranging from 8% to 22% [2,4].

Dermatological manifestations in HIV patients are atypical, more severe, and resistant to treatment compared to HIV-negative individuals [5]. The frequently isolated species causing dermatophytosis are *Trichophyton rubrum*, *Trichophyton mentagrophytes,* and *Epidermophyton floccosum*. Atypical, extensive, and anergic forms of dermatophytosis may at times constitute the presenting features of HIV-positive patients. Tinea imbricata or Tokelau is a fungal infection of the skin, manifesting as multiple concentric rings of erythematous papules, pustules, scaling, and crusting, caused by *Trichophyton concentricum*. Tinea pseudoimbricata or tinea indecisiva are the terms given for similar clinical presentations caused by fungal agents other than *T. concentricum* (e.g., *T. tonsurans*) [6].

Here, we report two male patients and one female patient who presented with tinea pseudoimbricata, subsequently detected to be HIV-positive.

Case 1: A 58-year-old transgender person reported to our clinic with the chief complaint of red, itchy, raised lesions over the lower abdomen, groins, and left leg for ten months.

On examination, multiple, well-demarcated, concentric rings of approximately 1.5×1.5 cm erythematous papules and hyperkeratotic plaques with scaling and crusting and no central clearing covering the lower abdomen, bilateral groins (Figure 1), and back of the left leg (Figure 2) were seen. Other mucocutaneous and systemic

Figure 1. Concentric plaques with scaling over the lower abdomen and bilateral groins.

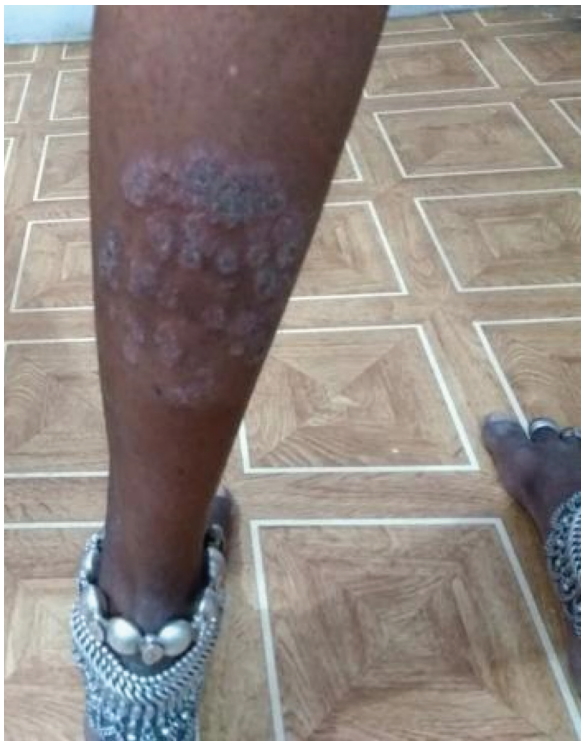

Figure 2. Concentric rings over the back of the left leg.

examinations were normal.

Case 2: A 49-year-old married male shop vendor reported to our clinic with the chief complaint of itchy reddish lesions with peeling of skin over the axilla, groin, and buttocks for six to eight months. On examination, erythematous hyperpigmented concentric rings of papules, plaques, and pustules with scaling and crusting (closely placed vesicles at the border of the plaque) were seen, extensively involving the groins, scrotum (Figure 3), buttocks (Figure 4), leg (Figure 5) and axilla (Figure 6).

Case 3: A 52-year-old housewife came to our clinic complaining of reddish, itchy lesions over the right foot (Figure 7), groin, and buttocks since eight to ten months beforehand.

On examination, well-defined erythematous plaque with ring-within-a-ring formation was seen on the dorsum of the right foot, front of the right ankle, and groin.

Figure 3. Erythematous plaque over groins and scrotum

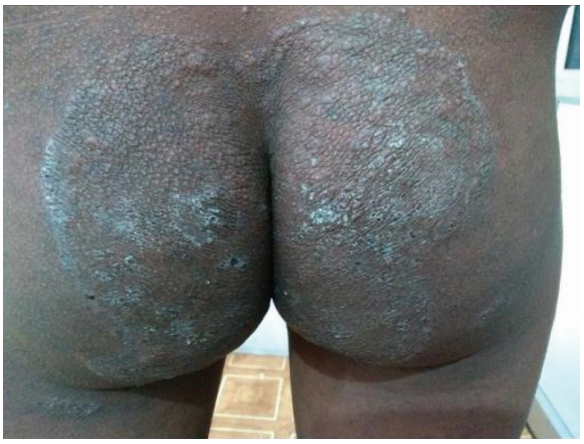

Figure 4. Large concentric rings with scaling over the buttocks.

Routine laboratory investigations were done for all three patients. Complete blood count and renal and liver function tests were within normal limits. Serum samples were detected positive for HIV-1 antibody according to National AIDS Control Organization (NACO) testing guidelines.

Skin scrapings were collected from multiple sites

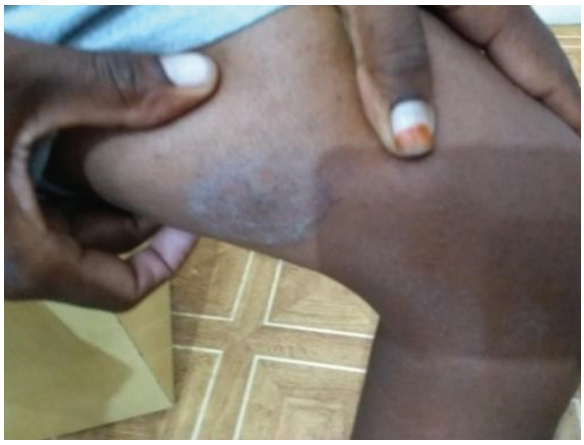

Figure 5. Erythematous plaque over the left leg.

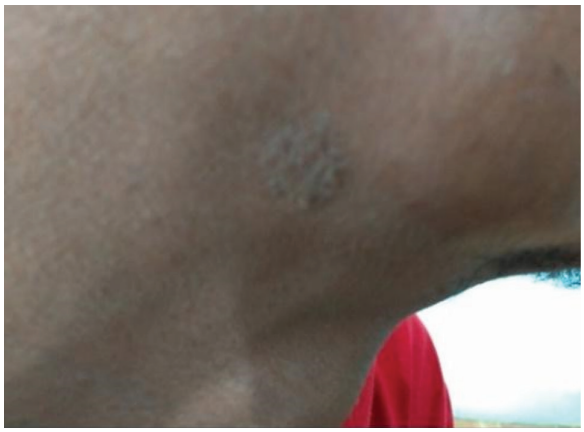

Figure 6. Concentric ring over the left axilla.

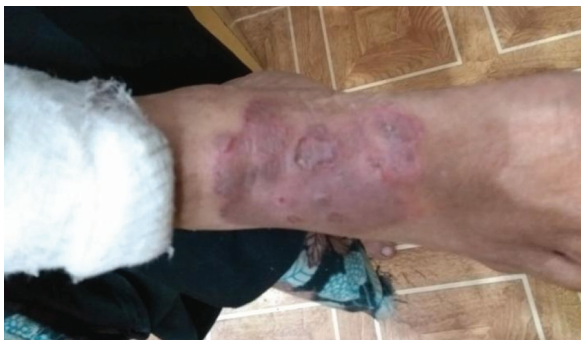

Figure 7. Erythematous concentric plaques on the right foot

along the margins of the lesion after cleaning with 70% alcohol. Direct mycological examination with 10% potassium hydroxide (KOH) revealed septate hyphae. Scrapings inoculated on Sabouraud's Dextrose Agar (SDA) with chloramphenicol (0.005%) and incubated at 25 °C revealed, creamy-white color, downy to glabrous colonies with a pale yellow-brown reverse pigment. After five days of incubation, growth of a flat spreading brown-colored powdery surface was observed. Lactophenol cotton blue preparation from colonies demonstrated teardrop-shaped (pyriform) microconidia attached to the hyphae at the narrow end. All findings were consistent with *Trichophyton rubrum* infection in all three patients.

Patients were treated with systemic itraconazole (200 mg per day) along with a topical antifungal (luliconazole) and oral antihistamines. All patients were counseled about the nature of the disease. As the patients were diagnosed with HIV infection for the first time, we referred the patients to an anti-retroviral therapy center for further management.

The term *imbricate* is derived from the Latin word *imbrex*, referring to overlapping roof tiles. Tinea imbricata is a distinct superficial mycosis caused by *T. concentricum* with a characteristic pattern of concentric or annular plaques of erythema and scales. The disease has a restricted geographical distribution in South-East Asia, South Pacific, Central, and South America [6]. Cases clinically simulating tinea imbricata but caused by species other than *T. concentricum* have been labeled as tinea pseudoimbricata or tinea indecisiva. Tinea pseudoimbricata has been reported to be caused by *T. tonsurans, T. rubrum, T. mentagrophytes, Microsporum audouinii,* and *Microsporum gypseum*. All our patients presented with multiple concentric or annular erythematous scaly plaques. Fungal culture in our patients revealed *T. rubrum*.

The exact etiopathogenesis is still unclear, but such cases are known to occur in patients with some degree of immunosuppression. The clinical pattern in Tinea imbricata has been explained by immunologic interaction between the host and pathogen. The majority of patients affected with tinea imbricata are anergic to the intradermal antigen of *T. concentricum*; the cellular immune response is impaired, with T-lymphocyte hyporeactivity and dominance of the humoral immune response. A similar hypothesis has been put forward to explain the concentric rings of tinea pseudoimbricata. Fungal invasion initially activates the host immune response leading to suppression of fungal cell growth. Topical application of a potent steroid causes local immunosuppression, which may diminish the host responses, specifically the cellular immune response against the fungus. This causes switching on of certain fungal genes and reactivation of fungal replication.

Beyond a certain time and threshold of fungal replication, host responses again become active, leading to inflammation. Injudicious intermittent therapy with topical steroids with or without antifungals repeats this cycle many times, leading to the appearance of concentric erythematous scaly rings.

Dermatophytes are aerobic fungi that produce proteases that digest keratin and allow colonization, invasion, and infection of the stratum corneum of the skin, hair shaft, and nails. Infection is generally cutaneous and restricted to the nonliving cornified layers because the fungi cannot penetrate the deeper tissues or organs of a healthy immunocompetent host. In the recent past, there has been an increase in patients presenting with atypical clinical types.

Dysregulation of the host immune system, increase in the minimum inhibitory concentration of antifungal drugs, atopy, climatic conditions or over-crowding, migration, urban clothing patterns, and poor hygiene are some predisposing factors in the Indian scenario that may be responsible for increasing atypical and unusual presentations [8]. Atypical clinical types include erythema multiforme-like, seborrheic dermatitis-like, lupus erythematosus-like, dermatitis herpetiformis-like, rosacea-like, psoriasis-like, eczematous dermatitis-like, impetigo-like, and polymorphous light eruption-like [8].

The human immunodeficiency virus (HIV) is acquired sexually, through blood or blood products, vertically, during birth, or through breastfeeding. HIV infection is accompanied by progressive immunodeficiency, which increases the risk of mycoses. The virus infects vital cells in the human immune system, such as helper T cells (CD4+ T cells), macrophages, and dendritic cells [9].

The dermatophytic clearance from the skin is secondary to activation of cell-mediated immunity and is TH1-dependent. Immune dysregulations with a skewed immune response toward TH2

cytokines may be responsible for these atypical, extensive, chronic, and unusual clinical forms [10]. T-cell hyporeactivity allows sequential waves of infection and immune response, leading to a ring-within-a-ring formation. The mechanism of T-cell hyporeactivity and lack of delayed-type hypersensitivity may lead to dermatophytic infection, mainly in patients with prolonged use of steroids and topical antifungal medications or inadequate treatment with antifungal agents leading to re-infection by the same pathogen and forming concentric rings [6].

In this study, we report three HIV-positive patients with tinea pseudoimbricata caused by *T. rubrum*, similar to the report of Narang *et al.* [6] Dermatophytic infections among HIV seropositive individuals can present in an atypical, extensive form and may be treatment-resistant. Familiarity with these atypical manifestations helps dermatologists in early diagnosis and management. In cases with the tinea pseudoimbricata type of lesions, we recommend taking a history of steroid application, serological tests for HIV, and a fungal culture to rule out *T. concentricum*.

Conflic of Interest: None declared.

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