

SHORT COMMUNICATION

A Case of *Tinea Imbricata* in an Italian Woman

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Tinea imbricata (TI) is a chronic superficial mycosis caused by *Trichophyton concentricum*. This mycosis is characterised by widespread, annular, concentric, squamous lesions, often accompanied by pruritus. TI is endemic in South-West Pacific, South-East Asia, and Central and South America (1–15).

We present a case of TI in an Italian woman who acquired the infection after a 3-month trip to Tahiti, Samoa and Solomon Islands. To our knowledge, this is the 5th case of TI in a visitor to an endemic area.

CASE REPORT

A 47-year-old Italian woman was admitted because of dermatitis located on the thighs and legs. The patient stated that she was in good general health and that she was not in therapy with systemic drugs. Clinical history revealed that the dermatitis appeared 7 months earlier, just a few days after a 3-month trip to Tahiti, Samoa and Solomon Islands. In Tahiti and Samoa the patient had sexual intercourse. She also stated that a previous diagnosis of contact dermatitis was made at other centres, for which she was treated, although unsuccessfully, for 7 months with topical and oral corticosteroids and oral antihistamines.

Dermatological examination revealed dermatitis located on the buttocks, thighs and legs. It was characterised by multiple, annular, concentric, erythematous-purpuric lesions (Fig. 1). In addition, 3 clinically similar lesions were present on the right breast and the abdomen. The patient complained of severe pruritus.

Patch tests were negative. Two biopsies were performed. The histopathological examination showed the presence of numerous hyphae within the stratum corneum. Direct immunofluorescence was negative. Mycological examination showed short, septate hyphae and several chlamydospores. Three cultures on Sabouraud dextrose agar medium added with chloramphenicol and cycloheximide were positive for *T. concentricum*. No PCR amplification study was performed.

All laboratory examinations, including immunological tests, were within normal ranges or negative, except for erythrocyte sedimentation rate (43 mm at the 1st hour; normal <25 mm).

The patient was treated with griseofulvin (1 g/day for 6 weeks) and 1% terbinafine cream (2 applications/day for 6 weeks). Almost complete remission of the clinical picture was observed 3 weeks later. However, culture was still positive, but the infection was cleared 6 weeks after starting the therapy. Follow-up one year later was negative.

It was possible to examine the mother and 2 sisters of the patient, but none of them had skin lesions.

DISCUSSION

TI is known by several names, among them Tokelau (the most used synonym, from the name of islands in the South Pacific where most of the population is af-



Fig. 1. Multiple, annular, concentric, erythematous-purpuric lesions on the legs.

ected by the disease) (12, 15). Other popular names of TI are bakwa, cacapash, chimberé, Chinese tinea, circinate tinea, concentric tinea, elegant tinea, Gilbertese disease, gogo, grillé, Hanumarn ringworm, Indian tinea, lace tinea, roña, scaly tinea and shishiyotl (12, 15).

The dermatophyte *T. concentricum* (Blanchard 1895) presents with short, septate hyphae, numerous chlamydospores and no arthroconidia (12, 15). Some strains can present with characteristic structures, the so-called “favic chandeliers” (15). On Sabouraud dextrose or glucose agar or on Sabouraud with cycloheximide and chloramphenicol, colonies develop in 8–25 days at 25°C (Fig. S1¹). The underside is amber in colour (12, 15). Some strains require the addition of thiamine (12, 15). It was hypothesised that 2 strains of *T. concentricum* exist: one which grows at 20–25°C, and one which grows at

¹<http://www.medicaljournals.se/acta/content/?doi=10.2340/00015555-1887>

28°–30°C; however, this has not been proved yet (12, 15). Identification of the strains was confirmed by PCR amplification and sequencing of the internal transcribed spacer-rDNA regions in only one study (14).

TI affects subjects living in isolated and poor areas (12, 14, 15). Poor hygiene and overcrowding are considered as predisposing factors (12, 15).

Genetic predisposition to TI has been studied. According to some authors (4, 5), an autosomal recessive inheritance of susceptibility to TI exists. According to other authors (13), the type of inheritance is autosomal dominant with incomplete penetrance.

Immunology of patients with TI has also been extensively studied (6–8). In a study by Hay et al. (6), 52% of patients had immediate-type hypersensitivity or negative responses (46% of patients) to intradermal trichophytin. The majority of patients failed to develop delayed-type hypersensitivity on skin testing or as assessed by leucocyte migration inhibition. Furthermore, 78% of patients had antibody to *T. concentricum*. In another study by Hay et al. (8), patients with TI were found to have raised immunoglobulin (Ig) levels to *T. concentricum* antigen. IgE class antibodies were raised.

TI is characterised by multiple, annular, concentric, squamous lesions, with or without erythema. The infection often begins in childhood on the face and subsequently involves the trunk and limbs (12, 14, 15). Repeated contacts of an infected mother with her child is one of the most frequent modality of infection (38). Palmo-plantar surfaces and the scalp can be affected, although the pilosebaceous unit is never involved (12, 15). The forehead, axillae and groin are usually spared. Hay et al. (7) proposed 7 different clinical patterns of TI: concentric, lamellar, lichenified, plaque-like, annular, palmar/plantar and onychomycosis, to which seborrhoea-like lesions on the scalp and hyper-hypochromic lesions have been subsequently added (12, 15). The nails are occasionally involved as distal subungual onychomycosis (12, 13, 15). Pruritus may be absent or mild or severe (12, 14); in the latter case, chronic scratching causes lichenification (14). Differential diagnosis of TI includes other tinea (due to *Epidermophyton floccosum*, *T. mentagrophytes*, *T. tonsurans* and *Microsporum audouinii*), pityriasis versicolor “imbricata”, secondary syphilis, yaws, erythema annulare centrifugum, sarcoidosis and erythema gyratum repens. The clinical course of TI is chronic. Spontaneous improvement is very rare (6, 15).

Since the 1950s, TI was treated with griseofulvin (2, 9, 12, 15). A study compared the efficacy of griseofulvin, fluconazole, itraconazole and terbinafine. Significant remission was achieved in the terbinafine and griseofulvin groups. The fluconazole group experienced no significant remission; and was of short duration in the itraconazole group (11). A double-blind, randomised, controlled study compared the efficacy of terbinafine with itraconazole. Terbinafine was assessed

as having a superior clinical and mycological cure rate after 4 weeks. After 13 weeks of follow-up, terbinafine provided a significantly reduced rate of relapse/reinfection compared with itraconazole (10). Relapse and reinfection are very common (6, 7, 12, 15). Subjects belonging to a susceptible population can carry the disease for their lifetime (15).

It was stated that people not genetically related to specific ethnic groups very rarely acquire the infection, even after prolonged and close contact with infected persons (15). This statement is true, because the review of the literature revealed that TI is exceptionally rare in non-natives, but this possibility exists. In fact, from 1952, at least 4 cases were published. The first patient was an English officer of the Malaysian police (1). The other cases were an Australian boy who acquired the infection in Fiji islands (2); a French soldier who was infected in Tahiti (3) and an English nurse who contracted TI in Papua New Guinea (9). It is possible that in our patient the infection was contracted because of sexual intercourse with natives. The clinical presentation of TI in our patient was atypical. This was likely due to the fact that the patient was treated for a long period of time with topical and oral corticosteroids.

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