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Overview of Diagnosis and Treatment Options for Tinea Imbricata

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Abstract: **Tinea imbricata (TI) or Tokelau is a shallow mycosis brought on by Trichophyton concentricum, an anthropophilic dermatophyte. It is endemic in some islands of the South Pacific (Polynesia), South-East Asia, Central and South America, and Mexico, and is usually seen in individuals residing in primitive and separated conditions. The skin lesions are typically concentric and lamellar (imbricata: in Latin, tiled) plaques of scale. Inclining conditions include humidity, inheritance, and immunologic elements. The diagnosis is generally made on clinical premises, supported by skin scrapings and culture. Tokelau is a extremely relapsing and persistent disease and, although no first-line treatment exists, finest results are obtained with oral griseofulvin and terbinafine and a topical mix of keratolytic lotions, such as Whitfield's. TI is a disease model that allows the connection of a series of ecological, hereditary, immunologic, and restorative conditions.**

Keywords: **Tinea imbricata (TI), Trichophyton concentricum.**

1. INTRODUCTION

Tinea imbricata is a gradually progressive, persistent, superficial fungal infection triggered by the dermatophyte Trychophyton concentricum, extremely endemic to locations of Asia, the South Pacific, and Central and South America.

Lesions start as little, brown, pruritic macules and papules and progress to concentric rings of scales. The infection usually begins in childhood, and advances slowly in time. Over 75% of those impacted will have lesions covering 50% or more of their skin surface. The lesions are quite pruritic, and the pruritus is aggravated by heat. Locations of lichenification develop after chronic excoriation. As this infection is shallow, patients do not have accompanying constitutional symptoms.

Danger aspects include sharing an ancestry with endemic populations (tourists do not appear to establish this condition even after long stays and close contact), low socioeconomic class, and bad health. Women are more commonly affected in the adult population; this sex ratio is reversed in children. Environmental direct exposures have not been connected to infection. A T-cell flaw caused by an autosomal recessive quality has been suggested, but not proven.

Tinea imbricata (TI) is a persistent shallow mycosis caused by the anthropophilic dermatophyte Tri-chophyton concentricum. TI is endemic in three geographical locations: Southwest Pacific, Southeast Asia, and Central and South America. TI is defined by prevalent, annular, concentric, squamous sores, typically accompanied by pruritus [1-41]

2. METHODOLOGY

Four databases were selected to ensure a comprehensive review of the literature: PubMed, EMBASE, Ovid, and the Cochrane Review. On January 25, 2014, a total of 13 different queries were used for each engine: (1) “Trichophyton concentricum ”, “Tinea ”, “imbricata ”, “chronic ”, “mycosis,” , “superficial ” , “T. Concentricum”. A hand search of the tables of contents of relevant journals published from January to December 2015 was then performed .

3. RESULTS AND DISCUSSION

Tinea imbricata was first described in 1686 by the English explorer William Dampier throughout his trips in Philippines [34, 37, 38] In 1878, Manson wrote the first clinical description of the disease [34, 38] In 1940, the infection was observed in Guatemala [34, 38] and in 1945 in Mexico [34, 38]

TI is known by a number of names, amongst which is Tokelau (the most pre-owned synonym, from the name of some islands in the South Pacific Ocean where the majority of the population is impacted by the disease) [34, 38] Other popular names of TI are bakwa, cacapash, chimbere´, Chinesetinea, circinatetinea, concentric tinea, stylish tinea, Gilbertese disease, gogo, grille´, Hanumarn ringworm, Indian tinea, lace tinea, ron˜a, flaky tinea, and shishiyotl [34, 38].

TI is caused by the anthropophilic dermatophyte T. concentricum (Blanchard 1895). It is rather much like T. mentagrophytes [34, 38] T. concentricum provides with brief, septate hyphae, many chlamydospores, and no arthroconidia [34, 37, 38] Some stress can provide with characteristic structures, the so-called favic chandeliers [38] On Sabouraud dextrose or glucose agar or on Sabouraud with chloramphenicol and cycloheximide, nests develop in 8-25 days at 25 ° C: They are whitish, waxy, cerebriform, umbilicated, or crateriform, with brownish center and white powdery edge. The underside is amber in color [34, 37, 38] In the past, Sabouraud peptone agar with actidione, penicillin, and streptomycin was also used [3] Some strains need the addition of thiamine [34, 37, 38] It was assumed that two stress of T. concentricum exist: One would be thermosensitive, with development at 20-25 ° C, and one would be thermo- tolerant, with growth at 28-30 ° C [34, 37, 38] Recognition of the stress was validated by PCR amplification and sequencing of the internal transcribed spacer-rDNA areas in only one research study [36].

TI is endemic in three particular geographical areas: Southwest Pacific (Fiji [3, 13, 34, 37, 38], Samoa [13, 34, 37, 38], Solomon Islands [36, 38, 39], Tahiti [4], Tokelau [34, 37, 38], Papua New Guinea [5, 16, 19, 21, 22, 26, 32, 34, 37, 38], Indonesia [29, 33], and New Zealand [18, 34, 37, 38]; Southeast Asia (India [30, 34, 37, 38], China [34, 37, 38], Thailand [6], Malaysia [1, 2, 38], and Philippines [7]; and Central and South America (Mexico [24, 31, 34, 35, 37, 38], Guatemala [34, 37, 38], El Salvador [34, 37, 38], Panama [34, 37, 38], Colombia [34, 37, 38], and Brazil [20, 34, 37, 38].

TI occurs in warm tropical and subtropical climates in addition to in cold environments at an elevation of 1.000-2.500 m above the water level. However, both climates share a very high humidity rate ([80 %) [34, 37, 38]

TI impacts topics living in poor locations [34, 36, 38] Malnutrition, iron deficiency, bad hygiene, and overcrowding are thought about as predisposing factors [34, 37, 38] TI would be somewhat more common in adult ladies and male children [22, 37] Some authors denied sex and age differences [2, 34, 38]

According to some authors [15, 16, 25, 27, 28], an autosomal recessive inheritance of vulnerability to TI exists. Inning accordance with other authors [35], the type of inheritance is autosomal dominant with incomplete penetrance.

Body immune system of patients with TI has been studied [21- 23, 25]: 78 % of patients had antibody to T. concentricum [21] In another study [23], patients with TI were found to have raised immunoglobulin (Ig) levels of all classes to T. concentricum antigen by an enzyme-linked immunosorbent assay. Both overall and particular IgE class antibodies were raised.

Common clinical discussions of TI are several, annular, concentric, squamous lesions, with or without erythema. The infection often starts in youth on the face and consequently involves the trunk and limbs [34, 36-38] Repetitive contacts of a contaminated mom with her kid are one of the most frequent modalities of infection [38] Palmo-plantar surfaces and the scalp can be affected; the pilosebaceous unit is never included [34, 37, 38] The forehead, groin, and axillae are generally spared [2, 37], although a kid with unique involvement of the forehead was just recently described [39].

Seven various clinical discussions of TI were proposed [22]: annular, concentric, lamellar, lichenified, plaque-like, palmar plantar and onychomycosis. Seborrhea-like lesions on the scalp and hyperchromic/ hypochromic sores were subsequently included [34, 38] The nails are occasionally included [34, 35, 38]: Nail involvement is medically equivalent from that triggered by T. mentagrophytes and T. rubrum. The clinical discussion is as distal subungual onychomycosis [34, 37, 38]

Pruritus may be missing [2] or severe or moderate [34, 36, 37]: In the latter case, chronic scratching causes lichenification [2, 36, 37] Some authors observed that patients residing in cooler climates experience less pruritus, which increases when the climate becomes hot and humid [34, 38].Clinical diagnosis of TI is very typically simple. Varyential diagnosis includes other

tineas (due to Epidermophyton floccosum [42], T. mentagrophytes [43, 44], T. tonsurans [43- 47], and Microsporum audouinii [48], pityriasis versicolor" imbricata" [49], secondary syphilis [50, 51], yaws [37], erythema annulare centrifugum [51], sarcoidosis [52], and erythema gyratum repens [34].The clinical course of TI is persistent. Spontaneous improvement is extremely uncommon [21, 38].

Since the 1950s, TI was treated with griseofulvin [3, 5-7,9-12, 19, 24, 26, 34, 37, 38] A research study compared the effectiveness of griseofulvin (1 g/day for 4 weeks), fluconazole (200 mg/week for 4 weeks),.

itraconazole (400 mg/day for 1 week), and terbinafine (250 mg/day for 4 weeks). Substantial remission was accomplished in the terbinafine and griseofulvin groups, lasting as much as 8 weeks after cessation of treatment. The fluconazole group experienced no considerable remission; the latter was of short duration in the itraconazole group [33] .A study revealed the lack of prophylactic action of griseofulvin against T. concentricum infection [53].

A double-blind, randomized, managed research study com- pared the efficacy of terbinafine with itraconazole. Forty-three patients received terbinafine (250 mg/day), and 40 received itraconazole (100 mg/day) for 4 weeks. A total of 72 patients were eligible. 4 patients coming from the itraconazole group did not respond either clinically or mycologically. All the remaining 68 patients were medically and mycologically treated. Terbinafine was examined as having a superior clinical and mycological cure rate after 4 weeks (P = 0.05). After 13 weeks of follow-up, terbinafine offered a significantly lowered rate of reinfection/relapse compared with itraconazole (P \ 0.001). The authors justified the remarkable efficacy of terbinafine for the fungicidal activity and the long perseverance in the skin [29] Ketoconazole was likewise utilized [24] .

Griseofulvin, at the dosage of 1g/day for 4-6weeks, or terbinafine, at the dose of 250 mg/day (125 mg/day in children) for 4 weeks, is currently thought about as the most effective drug in TI.

The Whitfield's lotion (10 % benzoic acid and 10 % salicylic acid in vaseline and lanoline) is practical to remove squamous and hyperkeratotic lesions [34, 37, 38] Topical haloprogin was also used [19] .

Reinfections and regressions are very common [21, 22, 34, 37, 38] Topics coming from a susceptible population can be affected by the disease for their life time even after sufficient therapy [38].

It has actually been specified that individuals not genetically related to particular ethnic groups really rarely obtain the infection, even after close and prolonged contact with contaminated persons [38] ·This statement is partly true, due to the fact that the evaluation of the literature exposed that TI is incredibly uncommon in non-natives, but this possibility exists. In fact, from 1952, at least five cases were published [1, 3, 4, 26, 40] (Table 1).

Table 1 Cases of TI in non-natives

# Cases [Ref.]	Countries
1. English officer [1]	Malaysia
2. Australian boy [3]	Fiji Islands
3. French soldier [4]	Tahiti
4. English nurse [26]	Papua New Guinea
5. Italian woman [40]	Tahiti, Samoa and Solomon Islands
6. Italian child	Solomon Islands

4. CONCLUSION

It is important to note that the removal of the disease has not been possible due to that it is extremely reoccurring which a lot of cases happen in separated rural areas that are challenging to access; however, the number of cases is reducing, primarily due to modifications in predisposing factors, such as climatologic conditions, hygiene, and the migration of populations to areas with greater genetic exchange.

Prophylactic measures are complicated, primarily because the affected people have extremely deep-rooted practices. Adequate health procedures and making use of topical rewardments in case of reoccurrence or reinfection should be stressed.

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