



## A SYSTEMATIC REVIEW OF DIAGNOSIS AND TREATMENT OPTIONS FOR TINEA IMBRICATA

**RANA ABDULAZEEM AL-BASSAM<sup>1</sup>, BASMAH SALEM AL AFARI <sup>1</sup> AND MANAL HASSAN MOHAMED SALEM<sup>2\*</sup>**

<sup>1</sup>*Intern doctor, Dar Al Uloom University Riyadh, Saudi Arabia ,*

<sup>2</sup>*Mater degree of dermatology,venereology and andrology , Doctor Abdulazeem Albassam Medical Group, Department of Dermatology, Riyadh, Saudi Arabia*

### ABSTRACT

Tinea imbricata is a cutaneous fungal disease and sometimes called (Tokelau). The causative agent is a dermatophyte known as *Trichophyton concentricum*. It is an endemic in developing countries particularly in South Pacific, India, Central and South America, as well as Mexico. It is generally observed in people with poor living conditions and poor personal hygiene. Predisposing factors are hot weather, humidity, and host immunity in addition to genetic factors. The patients usually presented with concentric or lamellar skin lesions. The aim of this review is to highlight important information about microbial, clinical and therapeutic aspects of tinea imbricata. In this review, we search the literature to identify articles talking different aspects of tinea imbricata. The electronic search was performed in four databases to identify eligible articles in the literature. Electronic databases were searched including MEDLINE and EMBASE using PubMed search engine. In addition, Cochrane library and ovid was searched. The titles and abstracts of the resulted articles were screened to identify eligible studies. Based on the primary screening results the irrelevant studies, duplicated and reviews were excluded. Tinea imbricata is found to be endemic in 3 main geographical regions, Southwest Pacific, Southeast Asia, and Central and South America. There is an autosomal recessive genetic factor which increases the vulnerability to tinea imbricata infection. The levels of both general and specific IgE class antibodies were higher than normal values. Usual clinical findings of tinea imbricata are multiple annular, concentric, squamous sores and may be associated with erythema. The infection usually starts in young people on the facial region and disseminate to the trunk, arms or legs. The eradication of the disease has not been possible yet, thus preventative procedures should be adopted as a strategy of disease control.

**KEYWORDS:** *Tinea, Ringworm, Dermatophytosis, Epidermophytosis*



**MANAL HASSAN MOHAMED SALEM**

*Mater degree of Dermatology,venereology and andrology , Doctor Abdulazeem Albassam Medical Group, Department of Dermatology, Riyadh, Saudi Arabia*

**Received on:** 26-08-2019

**Revised and Accepted on:** 09-10-2019

**DOI:** <http://dx.doi.org/10.22376/ijpbs/lpr.2019.9.4.L28-33>

## INTRODUCTION

Tinea imbricata (TI) is a slowly-developed superficial mycosis caused by the dermatophyte *Trichophyton concentricum*, which is commonly found in developing countries<sup>1</sup>. Lesions are characterized by small, brownish, pruritic macules and papules which later on develop into concentric rings of macular patches<sup>2</sup>. The infection typically starts in childhood, and breakthroughs slowly with an increase in age<sup>3</sup>. The majority of patients with TI have lesions involving more than 50% of the skin surface area<sup>4</sup>. The sores are fairly pruritic, and also the pruritus is worsened by warm weather<sup>5</sup>. Skin lesions are characterized by areas of lichenification which is created after chronic excoriation. Like other fungal superficial infections, patients do not have associated systemic symptoms<sup>6</sup>. The risk factors linked to TI are mainly found in people with similar origins with endemic population since travellers do not show the symptoms of diseases even after long and close stay with patients. Additionally, low socioeconomic class, poverty, low level of education and poor wellness are predisposing factors<sup>7-9</sup>. Women are a lot more commonly affected by TI in the grown-up population<sup>10</sup>, while this sex difference is turned around in children<sup>11</sup>. Direct transmission is not commonly reported with the TI infection. A T-cell defect triggered by an autosomal recessive top quality has actually been suggested, yet not confirmed<sup>12</sup>. The available evidence regarding the epidemiological aspects of TI is not sufficient. Thus, the aim of this review is to highlight important information about microbial, clinical and therapeutic aspects of tinea imbricta.

## METHODS

The electronic search was performed in four databases to identify eligible articles in the literature. Electronic databases were searched including MEDLINE and EMBASE using PubMed search engine. In addition, Cochrane library and ovid was searched.. We used 13 different search terms for each engine such as “*Trichophyton concentricum*”, “*Tinea*”, “*imbricata*”, “*chronic*”, “*mycosis*”, “*superficial*”, “*T. Concentricum*”. The studies were screened for presence of eligibility criteria such as addressing TI epidemiology, laboratory or clinical features, etiology, host immune response, or management. In purpose to make the review more comprehensive, any available study design was included in this

review (including case reports and case series). The full-text of eligible studies was retrieved to allow for extraction of data related to TI. No language or date limits have been used and only human studies were included. The results of the search were written in a logical flow of information with highlights on disease definition, etiology, geographical distribution, clinical distribution, and treatment.

## RESULTS AND DISCUSSION

Tinea imbricata was first defined in 1686 by the English explorer William Dampier throughout his journeys in the Philippines<sup>3,8,13</sup>. In 1878, Manson reported the first clinical description of the TI disease<sup>3,8</sup>. In 1940, the disease was detected in Guatemala<sup>3,8</sup> and in 1945 cases was reported in Mexico<sup>3,8</sup>. Tinea imbricata is known by a variety of names, amongst which is Tokelau the most common synonyms used in the South Pacific region<sup>3,8</sup>. Other common names of TI include bakwa, gogo, cacapash, elegant tinea, chimbere, Indian tinea, Chinesetinea, circinate tinea, concentric tinea, Gilbertese disease, grille, human ring worm, lace tinea, half-cracked tinea, and also shishiyot<sup>3,8</sup>. TI is brought on by the anthropophilic dermatophyte *T. concentricum* (Blanchard 1895). It is instead just like *T. mentagrophytes*<sup>3,8</sup>, *T. concentricum* provides with quick, septate hyphae, many chlamydospores, as well as no arthroconidia<sup>3,8,13</sup>. The culture is made in media such as Sabouraud dextrose or Sabouraud with certain antibiotics such as cycloheximide and chloramphenicol, and sometimes in sugar agar. The nests of TI developed in 1-3 weeks at room temperature (25°). They are creamy colored, waxy, crateriform or cerebriform with the brown facility and also white fine-grained edge. The bottom is amber in shade<sup>3,8,15</sup>. Sabouraud peptone agar with certain antibiotics was used to suppress bacterial growth in the culture where some strains needed for enhancement of thiamine<sup>3,8,13</sup>. One would be thermo sensitive, with growth at 20-25 ° C, and also one would be thermo-forgiving, with growth at 28-30 ° C<sup>3,8,13</sup>. A study validated PCR boosting and also sequencing of the inner transcribed spacer-rDNA areas<sup>14</sup>. TI is endemic in 3 main geographical regions, Southwest Pacific, Southeast Asia, and Central and South America. Endemic areas in Southwest Pacific region are Fiji<sup>3,8,13</sup>, Samoa<sup>3,8,13,13</sup>, Solomon Islands<sup>8,14,15</sup>, Tahiti, Tokelau, Papua New Guinea<sup>16</sup>, Indonesia<sup>17,18</sup>, and New Zealand<sup>3,8,13,19</sup>. Endemic areas in Southeast Asia are India<sup>3,8,13</sup>, China,

Thailand<sup>20</sup>, Malaysia<sup>8</sup>, and Philippines<sup>32</sup>. Endemic areas in Central and South America<sup>3,8,13,21,22</sup> are Guatemala, El Salvador, Panama, Colombia and Brazil<sup>3,8,13</sup>. TI occurs in warm tropical and subtropical climates along a slightly cold weather at an elevation of 1.000-2.500 m over the water level. However, both climates share a really high humidity rate (80 %)<sup>3,8,13</sup>. TI impacts topics residing in bad locations<sup>3,8,14</sup>. Malnutrition, iron shortage, poor health, and poor housing are important risk factors. TI would be somewhat extra common in adult girls as well as male children<sup>15,22</sup>. Some writers rejected sex and age distinctions<sup>3,8</sup>. Based on some studies<sup>23, 24</sup>, there is an autosomal recessive genetic factor which increases the vulnerability to TI infection. On the other hand, a study found a dominant autosomal inheritance pattern; however, there is no strong evidence supports this hypothesis<sup>22</sup>. The immunity role in TI infection was investigated by several studies. About 78 % of patients had antibody against *T. concentricum*, while patients with TI have increased levels of immunoglobulin (Ig) against *T. concentricum*. The levels of both general and specific IgE class antibodies were higher than normal values<sup>14</sup>. Usual clinical findings of TI are multiple annular, concentric, squamous sores and may be associated with erythema. The infection usually starts in young people on the facial region and disseminates to the trunk, arms or legs<sup>3,8,13,14</sup>. The transmission of TI to the children can be enhanced by close contact with an infected mother<sup>8</sup>. Palmo-plantar surfaces, as well as the scalp, can be affected. The temple, groin, and sometimes axillae are usually saved<sup>15,31</sup>, although a youngster with unique involvement of the temple was explained with more details in recent reports<sup>15</sup>. Seven various clinical presentation of TI were proposed such as annular, concentric, lamellar, lichenified, plaque-like, palmar-plantar and onychomycosis. Seborrhea-like sores on the scalp as well as hyperchromic/ hypochromic sores ultimately consisted of<sup>3,8</sup>. The nail lesions are occasionally involved<sup>3,8</sup>. Toenail participation is medically comparable from that activated by *T. mentagrophytes* as well as *T. rubrum*. A important clinical presentation of TI is distal subungual onychomycosis<sup>3,8,15</sup>. Pruritus might be missing out on<sup>25</sup> or severe or moderate<sup>3,15,22</sup>. In the last situation, chronic damaging reasons lichenification. Some writers observed that patients staying in cooler environments experience much less pruritus, which aggregated when the environment becomes warm and damp<sup>3,8</sup>. Clinical diagnosis of TI is really

usually simple. Differential diagnosis consists of different tineas induced by *Epidermophyton floccosum*, *T. mentagrophytes*<sup>26,27</sup>, *T. tonsurans*<sup>28</sup>, and *Microsporum audouinii*<sup>29</sup>, "pityriasis versicolor" "imbricata", second syphilis<sup>30</sup>, yaws<sup>13</sup>, erythema annulare centrifugum<sup>30</sup>, sarcoidosis<sup>31</sup>, and erythema gyratum repens<sup>3</sup>. The clinical presentation of TI is relentless and spontaneous renovation is incredibly uncommon<sup>3</sup>. In the 1950s, TI was managed by griseofulvin<sup>3,8,13</sup>. A study compared the efficiency 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 completed in the terbinafine and griseofulvin treated patients, continued as long as 8 weeks after completion of the treatment. The fluconazole treated patients did not report significant remission. The regime was of short duration in the itraconazole treated patients and the outcomes were not significant<sup>18</sup>. A study disclosed the absence of prophylactic activity of griseofulvin against *T. concentricum* infection. A double-blind, randomized study compared the effectiveness of terbinafine with itraconazole. Forty-three patients treated with terbinafine (250 mg/day), as well as 40 patients treated with itraconazole (100 mg/day) for a period of 4 weeks. Only four patients from the itraconazole group did not respond to the treatment. The remaining 68 patients were clinically and mycologically treated. Terbinafine was analyzed as having a significant clinical and mycological impact after 4 weeks. After 13 weeks of observation, terbinafine supplied a significantly lowered effect of reinfection/relapse in comparison to itraconazole ( $P < 0.001$ ). The writers validated the amazing efficacy of terbinafine for the fungicidal task and the lengthy determination in the skin<sup>25</sup>. Ketoconazole was likewise made use of<sup>17</sup>. Griseofulvin, at the dosage of 1g/day for 4-6weeks, or terbinafine, at a dosage of 250 mg/day (125 mg/day in children) for 4 weeks, is currently thought about as the most reliable medicine in TI. The Whitfield's cream (10 % benzoic acid and 10 % salicylic acid in vaseline and lanolin) is useful to get rid of squamous and also hyperkeratotic sores<sup>3,8,13</sup>. The Topical haloprogin was likewise utilized<sup>21</sup>. Reinfections and regressions are extremely common<sup>3,8,13</sup>. Some sites in susceptible persons can be affected by the disease for their lifetime even after completeness of the treatment<sup>8</sup>. It has actually been defined that individuals not genetically related to certain ethnic groups rarely obtain the infection, also after close and extended

contact with polluted individuals<sup>8</sup>. This statement is partly true, because of the fact that the examination of the literary works exposed that TI is incredibly uncommon in non-natives, but this

possibility exists<sup>32,33</sup>. Actually, from 1952, at least five instances were published as demonstrated in table1.

**Table 1**  
*Cases of TI in non-natives*

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

## CONCLUSION

It is necessary to keep in mind that the eradication of the disease has not been possible because of the recurrent nature of TI. The diseases commonly affect rural areas that are not easily accessible for control interventions. However, the incidence rate is decreasing, mainly due to adjustments in predisposing factors, such as climatological conditions, hygiene, and the migration of populaces to locations with greater genetic exchange. Preventative procedures are made complex, largely due to the fact that the commonly affected individuals have deep-rooted practices related to their life style, environmental and genetic characteristics. Sufficient health treatments as well

as using topical retardants in case of reoccurrence or reinfection ought to be stressed.

## AUTHORS STATEMENT

## CONTRIBUTION

The principal author, Al-Bassam, contributed in the design, conduction, and writing of this review. Other authors, Al afari, Alsharif , Albassam and Salem, contributed in conducting and writing of the study.

## CONFLICT OF INTEREST

Conflict of interest declared none.

## REFERENCES

- Parish LC. Reflections on Aldo Castellani and Tropical Dermatology. *Trans R Soc Trop Med Hyg.* 2018;112(4):155–7. DOI: 10.1093/trstmh/try045
- Bramono K. Chronic Recurrent Dermatophytosis in the Tropics: Studies on Tinea Imbricata in Indonesia. *Korean J Med Mycol.* 2012;17(1):1–7. DOI: 10.17966/kjmm.2012.17.1.1
- Bonifaz A, Archer-Dubon C, Saul A. Tinea imbricata or Tokelau. *Int J Dermatol.* 2004;43(7):506–10. DOI: 10.1111/j.1365-4632.2004.02171.x
- Mahajan R, Sahoo A. Management of tinea corporis, tinea cruris, and tinea pedis: A comprehensive review. *Indian Dermatol Online J.* 2016;7(2):77. DOI: 10.4103/2229-5178.178099
- Shrum JP, Millikan LE, Bataineh O. Superficial Fungal Infections in the Tropics. *Dermatol Clin.* 1994;12(4):687–93. DOI: 10.1016/s0733-8635(18)30131-1
- Hay RJ. Dermatophytoses and Other Superficial Mycoses. *Atlas of Infectious Diseases.* Current Medicine Group; 2000. p. 191–203. DOI: 10.1007/978-1-4757-9313-0\_13
- Kumar Patra S, Das S, Konar J. A Study On Dermatophytoses In A Tertiary Care Hospital Of West Bengal. *J Evol Med Dent Sci.* 2017;6(76):5415–8. DOI: 10.14260/jemds/2017/1174
- Bonifaz A, Vázquez-González D. Tinea imbricata in the Americas. *Curr Opin Infect Dis.* 2011;24(2):106–11. DOI: 10.1097/qco.0b013e328342cbc1

9. Panda S, Verma S. The menace of dermatophytosis in India: The evidence that we need. *Indian J Dermatology, Venereol Leprol.* 2017;83(3):281. DOI: 10.4103/ijdvl.ijdvl\_224\_17
10. Ndako J, Osemwiegie O, Spencer T, Olopade B, Yunusa G BJ. Prevalence of Dermatophytes and other associated Fungi among school children. *Glob Adv Res J Med Med.* 2012;1(3):49–56. Available from: <http://eprints.covenantuniversity.edu.ng/3460/>
11. García-Romero MT, Arenas R. New Insights into Genes, Immunity, and the Occurrence of Dermatophytosis. *J Invest Dermatol.* 2015;135(3):655–7. DOI: 10.1038/jid.2014.498
12. Abdel-Rahman SM. Genetic Predictors of Susceptibility to Dermatophytoses. *Mycopathologia.* 2017;182(1–2):67–76. DOI: 10.1007/s11046-016-0046-z
13. Satter EK. Tinea imbricata. *Cutis.* 2009;83(4):188–91. Available from: <https://europepmc.org/abstract/med/19445309>
14. Pihet M, Bourgeois H, Mazière J-Y, Berlioz-Arthaud A, Bouchara J-P, Chabasse D. Isolation of *Trichophyton concentricum* from chronic cutaneous lesions in patients from the Solomon Islands. *Trans R Soc Trop Med Hyg.* 2008;102(4):389–93. DOI: 10.1016/j.trstmh.2008.01.002
15. Maroñas Jiménez L, Monsálvez V, Gutiérrez García-Rodrigo C, Postigo Llorente C. Tinea Imbricata as a Clue to Occult Immunodeficiency. *Pediatr Dermatol.* 2014;31(6):e126–7. DOI: 10.1111/pde.12386
16. Meites E, McClenny NB, Baron EJ. Chronic Figurate Skin Lesions. *Clin Infect Dis.* 2004;39(4):582–3. DOI: 10.1086/422730
17. Budimulja U, Kuswadji K, Bramono S, Basuki J, Judanarso LS, Untung S. A double-blind, randomized, stratified controlled study of the treatment of tinea imbricata with oral terbinafine or itraconazole. *Br J Dermatol.* 1994;130(s43):29–31. DOI: 10.1111/j.1365-2133.1994.tb06091.x
18. Wingfield AB, Fernandez-Obregon AC, Wignall FS, Greer DL. Treatment of tinea imbricata: a randomized clinical trial using griseofulvin, terbinafine, itraconazole and fluconazole. *Br J Dermatol.* 2004;150(1):119–26. DOI: 10.1111/j.1365-2133.2004.05643.x
19. Patwardhan N, Dave R. Dermatomycosis in and around Aurangabad. *Indian J Pathol Microbiol.* 1999;42(4):455–62. Available from: <https://europepmc.org/abstract/med/11127378>
20. Hay RJ. Superficial mycoses. *Hunter's Tropical Medicine and Emerging Infectious Diseases.* Elsevier; 2000. 648–52 p. Available from: <https://www.us.elsevierhealth.com/hunters-tropical-medicine-and-emerging-infectious-diseases-9780323555128.html>
21. Arenas R. Dermatophytoses in Mexico. *Rev Iberoam Micol.* 2002;19(2):63–7. Available from: <http://reviberoammicol.com/2002-19/063067.pdf>
22. Bonifaz A, Araiza J, Koffman-Alfaro S, Paredes-Solis V, Cuevas-Covarrubias S, Rivera MR. Tinea imbricata: autosomal dominant pattern of susceptibility in a polygamous indigenous family of the Nahuatl zone in Mexico. *Mexiko. Mycoses.* 2004;47(7):288–91. DOI: 10.1111/j.1439-0507.2004.00989.x
23. Jazwinska LC, Bhatia K, Jenkias C, Serjeantson SW. HLA class II RFLP-typing in tinea imbricata patients from Papua New Guinea. *Tissue Antigens.* 1990;35(2):99–100. DOI: 10.1111/j.1399-0039.1990.tb01763.x
24. Hay RJ. Genetic susceptibility to dermatophytosis. *Eur J Epidemiol.* 1992;8(3):346–9. DOI: 10.1007/bf00158566
25. Taha M, Zaghloul AB. Superficial Fungal Infections. *Pigmented Ethnic Skin and Imported Dermatoses.* Springer International Publishing; 2018. p. 37–51. DOI: 10.1007/978-3-319-69422-1\_5
26. Batta K, Ramlogan D, Smith AG, Garrido MC, Moss C. “Tinea indecisiva” may mimic the concentric rings of tinea imbricata. *Br J Dermatol.* 2002;147(2):384. DOI: 10.1046/j.1365-2133.2002.04767.x
27. Rao A, Datta N. Tinea corporis due to *Trichophyton mentagrophytes* and *Trichophyton tonsurans* mimicking tinea imbricata. *Indian J Dermatology, Venereol Leprol.* 2013;79(4):554. DOI: 10.4103/0378-6323.113109
28. Hoque SR, Holden CA. *Trichophyton tonsurans* infection mimicking tinea imbricata. *Clin Exp Dermatol.* 2007;32(3):345–6.

DOI: 10.1111/j.1365-2230.2006.02337.x

29. Narang K, Pahwa M, Ramesh V. Tinea capitis in the form of concentric rings in an HIV positive adult on antiretroviral treatment. Indian J Dermatol. 2012;57(4):288.  
DOI: 10.4103/0019-5154.97672

30. Cotterman C, Eckert L, Ackerman L. Syphilis mimicking tinea imbricata and erythema annulare centrifugum in an immunocompromised patient. J Am Acad Dermatol. 2009;61(1):165–7.  
DOI: 10.1016/j.jaad.2008.10.027

31. Reddy R, Vitiello M, Kerdel F. Cutaneous sarcoid mimicking tinea imbricata. Int J Dermatol. 2011;50(9):1132–4.  
DOI: 10.1111/j.1365-4632.2010.04680.x

32. Veraldi S, Pontini P, Nazzaro G. A Case of Tinea Imbricata in an Italian Woman. Acta Derm Venereol. 2015;95(2):235–7.  
DOI: 10.2340/00015555-1887

33. Veraldi S, Giorgi R, Pontini P, Tadini G, Nazzaro G. Tinea Imbricata in an Italian Child and Review of the Literature. Mycopathologia. 2015;180(5–6):353–7.  
DOI: 10.1007/s11046-015-9930-1