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Topical Treatments



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Dermatophytoses—What is New?

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ABSTRACT

There is a recently observed ominous shift in the status of dermatophytosis worldwide, especially in India owing to rise in overall prevalence as well as emergence of chronic, recurrent, recalcitrant and resistant dermatophytosis necessitating changes in treatment protocol and diagnostic modalities. The observed change in clinico-epidemiological trend can be attributed to several factors like overcrowding, rapid urbanization, poor hygiene, rise in number of immunosuppressed hosts due to diabetes, human immunodeficiency virus (HIV), immunosuppressive medications and most importantly rampant misuse of topical steroids and freely available unholy combination of topical corticosteroid-antibacterial-antifungal. While previously *Trichophyton rubrum* was known to be the most common species causing dermatophytosis, recent studies have suggested the shift from *T. rubrum* to *Trichophyton mentagrophytes* as the most common causative organism of dermatophytosis. This is coupled with changes in clinical presentation with rise in proportion of atypical presentations.

There are also newer insights into the pathogenesis of dermatophytosis suggesting a role of T helper 17 (Th17) pathway in immunity against dermatophytosis and role of biofilms in resistance against antifungals. Diagnostic and treatment modalities have evolved with introduction of molecular methods like polymerase chain reaction (PCR) in diagnosis and newer treatment modalities like photodynamic therapy and lasers in treatment.

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INTRODUCTION

Superficial fungal infections are estimated to affect 20–25% of population of world in their lifetime.[1] Dermatophytosis is a subtype of superficial fungal infection caused by invasion of keratinized tissue like skin, hair and nails by dermatophytes usually of the genera *Trichophyton*, *Epidermophyton*, or *Microsporum*. In a study from south India, dermatophytosis accounted for approximately 30–40% of patient load of dermatology outpatient departments of various medical colleges.[2]

The extent of dermatophytosis may range from mild or localized to severe or extensive depending upon the host's immune response to the microorganism and other environmental factors. Previously 80% of patients developing acute dermatophytosis were known to respond to standard antifungal treatment while about 20% developed chronic dermatophytosis exhibiting poor response to antifungal agents.[3] However, the scenario of dermatophytosis in India and worldwide is changing rapidly owing to major epidemiological shifts and emergence of recalcitrant and resistant dermatophytosis necessitating changes in treatment protocol and diagnostic modalities.[4] In this article, we will be discussing the recent trends in epidemiology, newer insights into pathogenesis and clinical features of dermatophytosis as well as newer developments in diagnosis and treatment.

RECENT TRENDS IN EPIDEMIOLOGY

A significant shift in the epidemiology of dermatophytosis is being observed recently with increase in the overall prevalence of dermatophytosis along with a substantial surge in the recalcitrant, recurrent and extensive dermatophytosis (Figure 1). Worldwide *T. rubrum* was found to be the most common cause of dermatophytic infections followed by *T. mentagrophytes*.[5] However, recent studies from India have detected the shift of most common causative pathogen of dermatophytosis from *T. rubrum* to *T. mentagrophytes* especially in past 3–4 years.[4] The factors contributing to the higher incidence of dermatophytosis and recent shift in clinico-epidemiology include overcrowding, poverty, poor hygiene, contact with animals,[6] hot and humid climate,[1] occupations which require working in hot and humid conditions,[7] recent changes in attire and dressing sense like increased use of tight-fitting garments of synthetic fibers and occlusive footwear and rise in number of immunosuppressed hosts due to diabetes, HIV, immunosuppressive medications. Another major factor contributing to this epidemic is the rampant misuse of topical steroids and freely available combination of topical corticosteroid-antibacterial-antifungal which is being prescribed as panacea for all the dermatological conditions by general practitioners, alternative medicine practitioners and "chemists" and also being self-medicated by the patients.[8]

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Figure 1: Extensive tinea corporis and cruris.

Rise in Incidence of Chronic and Recurrent Dermatophytosis

Although the terms chronic dermatophytosis and recurrent dermatophytosis are used very commonly, consensus regarding standard definition for these terms are lacking. These terms have been defined on the basis of duration of disease or according to the response to treatment. Chronic dermatophytosis was defined by Hay et al.[9] in 1982 as "dermatophytosis which has not responded to topical or systemic antifungal therapy and runs a chronic course with episodic periods of remission".[9] On the basis of duration, chronic dermatophytosis has been defined arbitrarily as "patients who have suffered from the disease for more than 6 months to 12 months, with or without recurrence, despite adequate treatment".[10] The term "recurrent dermatophytosis" has been defined as "dermatophytic infection which runs a protracted course with phases of remission and exacerbation".[11] Recurrent or chronic dermatophytosis has emerged as a challenging health problem in India in last few years having significant financial and psychosocial implications on the patient and the society.[12] Exact prevalence of chronic and recurrent dermatophytosis in India is not known, but it was found to vary between 5 and 10% in different studies.[10] The prevalence of recurrent dermatophytosis was found to be 9.3% in a recent study by Pathania et al.[13] The major recent epidemiological studies on dermatophytosis including recurrent and chronic dermatophytosis have been summarized in Table 1.

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Table 1: Major Recent Indian Studies on Dermatophytosis including Recurrent and Chronic Dermatophytosis

Author (year)	Type of study	Location	Most common causative species	Most common clinical type	KOH positivity	Culture positivity	Other salient findings
Sharma et al. (2017)[14]	Clinico-epidemiological study on recurrent dermatophytosis	Sikkim, India	*T. mentagrophytes* (40%), *T. schoenleinii* (33.3%), *T. tonsurans* (16.6%), *T. rubrum* (6.6%)	Tinea corporis (54.16%) followed by tinea unguium (15.63%)	55.2%	63.5%	Recurrence rate—34.3% Clinical cure rate—58.3%
Mahajan et al. (2017)[15]	Clinico-mycological study on dermatophytosis	Varanasi, India	*T. mentagrophytes* (75.9%), *T. rubrum* (21.9%), *T. tonsurans* (0.7%)	Tinea corporis and cruris (27.2%)	79.6%	52.4%	Itraconazole found to be the most effective drug, followed by ketoconazole, terbinafine and fluconazole. Griseofulvin—least effective drug
Dabas et al. (2017)[16]	Clinico-mycological and molecular (species distribution and susceptibility pattern) study on dermatophytosis	New Delhi, India	*T. interdigitale* (*mentagrophytes*)—56%, *T. tonsurans* (25.7%), *T. rubrum* (7.5%)	Tinea cruris (37.9%) followed by tinea corporis (29%)	74.1%	53.2%	High MIC values were observed for *T. tonsurans* and *T. rubrum* to terbinafine and griseofulvin
Rudramurthy et al. (2017)[17]	Mutation study evaluating drug resistance	Chandigarh, India	*T. interdigitale* (66.1%), *T. rubrum* (26.3%), *T. tonsurans* (3%), *M. gypseum* (3%)	Tinea corporis (30.2%), tinea cruris (28.2%)	-	-	T1189C mutation in squalene epoxide gene resulting in Phe397Leu substitution in almost one-quarter of isolates of the terbinafine having high MICs suggests it as one of the probable mechanisms of terbinafine resistance
Pathania et al. (2018)[13]	Clinico-epidemiological study on recurrent dermatophytosis	Chandigarh, India	*T. mentagrophytes* (40%) followed by *T. rubrum* (32.2%)	Mixed (64.6%) followed by tinea cruris (17.3%), tinea corporis (18%)	98.7%	60%	MIC was lowest for itraconazole followed by terbinafine, fluconazole and griseofulvin
Nenoff et al. (2018)[4]	Molecular study	India—multicentric	*T. mentagrophytes* (93.2%) followed by *T. rubrum* (6.8%)	Tinea corporis (65.6%), tinea cruris (28.8%)	-	74.1%	Marked and dramatic shift of causative species of dermatophytosis from *T. rubrum* to *T. mentagrophytes*

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Rise in Incidence of Deep Dermatophytosis

Deep dermatophytosis is seen due to deeper invasion by dermatophytic strains, usually presenting clinically as nodular asymptomatic lesions with occasional overlying skin ulceration. Physical barriers like epidermal keratin, rapid turnover of keratinocytes, skin pH, fatty acids secreted by sebaceous glands generally prevents the deeper penetration of dermatophytes. Deep dermatophytosis is seen usually in individuals who are either immunosuppressed or have impaired physical barrier. There has been increase in the incidence of deep dermatophytosis as well owing to the rising population of immunocompromised hosts. In a report by Lanternier et al.,[18] it was observed that *CARD9* gene mutation was associated with deep dermatophytosis in a North African family.[18]

NEWER INSIGHTS INTO PATHOGENESIS OF DERMATOPHYTOSIS

Role of Th17 Pathway in Pathogenesis of Dermatophytosis

Contrary to the previous knowledge that Th1 immune pathway is solely responsible for immunity against dermatophytosis, newer studies have pointed toward the involvement of Th17 pathway in immunity against dermatophytosis.[19] A mouse model study by Nakamura et al.[20] found that application of *Trichophyton* antigen known as trichophytin leads to induction of both Th1 and Th17 cells. Sakuragi et al.[21] also observed increased Th17 cells in peripheral blood of a patient suffering from tinea capitis further suggesting the role of Th17 pathway in immunity against dermatophytosis. Loss of Th17 cell function has also been found to be associated with higher chances of dermatophytosis.[19] In their study on mouse model, Burstein et al.[22] also found the role of Th17 pathway and neutrophil recruitment in dermatophytosis limited to epidermis. They also concluded that absence of a functional interleukin-17 (IL-17) pathway leads to colonization of epidermis by dermatophytes.[22]

Pathogenesis of Chronic and Recurrent Dermatophytosis

There is paucity of literature regarding the pathogenesis of chronic and resistant dermatophytosis. Different studies on chronic dermatophytosis have attributed defective Th2 immunity and failure in patients to mount adequate cell-mediated immune response as factors leading to development of chronic dermatophytosis. In a recent study by De Sousa et al.,[23] patients suffering from chronic and widespread *T. rubrum* infection were found to have immunological defects involving impaired release of free radicals and nitric oxide along with dysfunctional macrophage

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killing mechanisms.[23,24] Rand et al.[25] found that atopic patients are more susceptible to chronic dermatophytosis probably owing to antagonizing actions of cell-mediated immunity and humoral immunity.[9,25] Patients suffering from chronic and recurrent dermatophytosis are also usually found to be resistant to standard antifungal treatment. Also, inadequate doses or duration of antifungal drugs may lead to improper eradication of infection further leading to chronic or recurrent infections.[10]

EMERGING DRUG RESISTANCE IN DERMATOPHYTOSIS

Several host and microbial factors like indiscriminate use of antifungals especially in inadequate doses or duration, use of topical steroids, rise in the number of immunosuppressed hosts, genetic mutation in dermatophytes, structural alteration of target protein, decreased uptake and increased efflux of drug play a role in emerging antifungal drug resistance.[26]

Clinical resistance is defined as the failure to cure a fungal infection despite the administration of adequate doses of antifungal drug with in vitro activity against the fungus. Microbiological resistance refers to nonsusceptibility of a fungus in in vitro susceptibility testing to a particular antifungal agent when minimum inhibitory concentration (MIC) of the drug exceeds the susceptibility breakpoint for that organism.[26] MIC values of antifungal drugs were found to be higher in Indian patients as compared to western data in a cross-sectional study by Sardana et al.[27] in 40 patients with recalcitrant dermatophytosis. The major mechanisms of resistance to various antifungal agents have been summarized in Table 2.

Biofilms are being recognized as a new mechanism adopted by dermatophytes for development of antifungal drug resistance. Biofilms are formed by the secretion of extracellular matrix encasing entire microbial community after adhering to a foreign substrate. Toukabri et al.[29] assessed the in vitro ability of dermatophyte species causing foot mycosis in forming biofilms and the ability of antifungal agents to prevent its formation. While all dermatophyte species possessed the ability to form biofilm, terbinafine was the most effective agent in prevention of biofilm formation.[29] Brilhante et al.[30] found that *T. rubrum, Microsporum gypseum* and *Trichophyton tonsurans* possessed the ability to form strong biofilm while *Microsporum canis* was found to produce weakest biofilm.[30]

Another factor contributing to the problem of drug resistance is the rampant use of substandard formulations of systemic and topical antifungal agents in India due to lack of strict regulation and quality control regarding the manufacturing of drugs. Many of the antifungal brands available commercially do not adhere to the guidelines regarding the manufacturing of drugs, but are nevertheless sold rampantly due to dubious marketing among the chemists and clinicians both. Most of the times, chemists substitute the authentic prescribed brands by the

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Table 2: Mechanisms of Resistance of Different Antifungal Drugs[28]

Drugs	Mechanism of resistance
Terbinafine	• Modification of target enzyme by mutation • Increased drug efflux • Stress adaptation • Overexpression of salicylate monooxygenase
Fluconazole	• Increased drug efflux • Stress adaptation
Itraconazole	• Increased drug efflux
Ketoconazole	• Increased drug efflux • Overexpression of lanosterol 14-demethylase
Imazalil	• Increased drug efflux
Tioconazole	• Increased drug efflux • Stress adaptation
Amphotericin B	• Increased drug efflux • Stress adaptation
Griseofulvin	• Increased drug efflux • Stress adaptation

clinicians with these substandard antifungal brands, a practice which is very common and unfortunately not recognized by uneducated or even well-educated patients. These poor quality antifungal brands with questionable pharmacokinetics further accentuating the risk of resistance.

CHANGES IN CLINICAL PRESENTATIONS

Apart from usual clinical presentations of dermatophytosis, there has been recent increase in the atypical presentations of dermatophytosis due to several factors. Several morphological features characterize atypical dermatophytosis which include severe inflammatory lesions with ill-demarcated borders, scaling in the center instead of central clearing, along with follicular papules, vesicles or pustules associated with induration or hyperpigmentation occasionally. Term "tinea incognito" was coined by Ive and Marks to represent lesions of dermatophytosis whose clinical presentation have been modified by the use of topical steroids.[31] Tinea pseudoimbricata[32] is a form of tinea incognito characterized clinically by two or rarely three concentric circles within a lesion of dermatophytosis. Presence of tinea pseudoimbricata is a clue pointing toward the application of potent topical corticosteroid preparations. It resembles tinea imbricata caused by *Trichophyton concentricum*, which is characterized by multiple concentric rings and generalized

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distribution as compared to pseudoimbricata. Familial forms of dermatophytosis are also being observed recently.[33] Tinea recidivans is seen due to initial response to treatment followed by relapse while on treatment or after stopping treatment. It is characterized by new lesions of dermatophytosis occurring at the periphery of the healing lesions (Figure 2).[34] Several other atypical morphological variants have been described in literature mimicking the lesions of other dermatosis such as erythema multiforme, seborrheic dermatitis, lupus erythematosus, dermatitis herpetiformis, rosacea (Figure 3), eczematous dermatitis (Figure 4), pityriasis rosea, impetigo and polymorphous light eruption.[1] Misuse of topical steroids is one of the major causes of atypical presentations of dermatophytosis along with other factors like resistance and immunosuppressed host.[35]



Figure 2: Tinea recidivans: New lesions of dermatophytosis occurring at the periphery of the healing lesions.



Figure 3: Rosacea-like tinea faciei.

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Figure 4: Eczema-like tinea manuum.

RECENT DEVELOPMENTS IN DIAGNOSTIC MODALITIES

Molecular Techniques in Diagnosis of Dermatophytosis

While traditional diagnostic modalities like potassium hydroxide (KOH) mount and culture have remained the backbone of diagnosis of dermatophytosis, molecular techniques have emerged as faster and more accurate diagnostic modality for dermatophytosis with a turnout time of 72 hours or lesser.[36] Molecular methods are also being used for identification of strains whenever identification by conventional methods is not possible. Commonly used molecular methods include PCR—restriction fragment length polymorphism (RFLP), real-time PCR, arbitrarily primed PCR, double-round PCR and PCR-direct sequencing. Deoxyribonucleic acid (DNA) of dermatophytes can also be detected directly in clinical samples. Although they are reliable methods for species identification and differentiation, differentiation of pathogenic from nonpathogenic species cannot be achieved by PCR. For rapid identification of dermatophyte species, matrix-assisted laser desorption/ionization time-of-flight mass spectrometry (MALDI-TOF MS) can also be used.[37] A recent study by Brillowska-Dabrowska et al.[38] assessed the specificity and sensitivity of PCR in identifying the 15 culture proven cases of *M. canis* out of 130 clinical samples. All the 15 cases were identified correctly without any false-positive or false-negative results. However, molecular modalities are expensive and not available widely. Nonetheless, owing to their accuracy, they have a huge potential and widespread use of these modalities are expected in future.

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RECENT DEVELOPMENTS IN TREATMENT MODALITIES

Recent Developments in Topical Treatment

Topical antifungal agents are being used both as monotherapy as well as adjuvant to systemic antifungal therapy in treatment of dermatophytosis in extensive disease. A meta-analysis including 65 studies was performed recently by Rotta et al.[39] to compare the efficacy of 14 topical antifungal agents in the treatment of dermatophytosis using a mixed treatment comparison model. No significant difference among the topical antifungal agents was observed with respect to the mycological cure at the end of the treatment. With respect to the sustained cure outcome, butenafine hydrochloride and terbinafine hydrochloride were found to be associated with better outcome as compared to clotrimazole, oxiconazole nitrate, and sertaconazole nitrate. Terbinafine was also found to demonstrate statistical better results as compared to ciclopirox (ciclopirox olamine), and better outcome was observed with naftifine hydrochloride as compared to oxiconazole.[39]

Efinaconazole 10% solution has been emerging as a promising topical modality for treatment of dermatophytosis. A report describing two phase III multicenter trials found mycological cure rate of 53.4% and 55.2% and complete cure rate of 15.2% and 17.8% with once daily application of 10% efinaconazole for 48 weeks in toenail onychomycosis.[40] The mycological cure rate was found to be comparable to oral itraconazole and terbinafine. In a recent study by Noguchi et al., topical 10% efinaconazole was also found to be effective treatment modality in onychomycosis.[41] Being a topical agent, the risk of systemic toxicities is minimal and it can be used in patients where systemic antifungal drugs are contraindicated.

Photodynamic Therapy as an Emerging Modality in Treatment of Dermatophytosis

Amidst the concerns regarding rising resistance to antifungal agents, photodynamic therapy (PDT) is emerging as potential treatment option for dermatophytosis.[42] In a comprehensive review by Smijs et al.,[43] several in vitro and ex vivo studies regarding the use of PDT against all the three genera of dermatophytes have been summarized. Various photosensitizers which included 5-aminolevulinic acid, porphyrins, methylene blue, proflavine hemisulfate, etc. were used in these studies. The light sources used in the studies included ultraviolet A (UVA) black light, Oriel solar simulator (Oriel Instruments, Newport Corp, Stratford, CT, USA), and the Paterson lamp (Photo therapeutics, Altrincham, Cheshire, UK). In vitro and ex vivo fungicidal effects were observed with various light sources and photosensitizers.[43] Ex vivo data suggested PDT to be fungicidal against both mature fungal forms and fungal spores. PDT has

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been used in various reports to treat different forms of dermatophytosis which include onychomycosis, tinea cruris, tinea pedis, etc. caused by different species such as *T. rubrum* or *T. mentagrophytes*. One to four PDT sessions were required usually to clear the infection.[42] Complete mycological cure was achieved in 31 of 59 patients (53%) of dermatophytosis treated with PDT. The treatment refractoriness of onychomycosis to antifungal therapy often requires prolonged systemic therapy with antifungal drugs thus putting the patient at risk to the increased toxicity of antifungal drugs. PDT is a promising therapeutic option especially in patients of onychomycosis in whom prolonged systemic antifungal therapy is not feasible.

Laser Therapy as an Emerging Modality in Treatment of Dermatophytosis

Nonablative laser therapy was recognized as an alternative therapeutic option in the treatment of onychomycosis almost a decade ago. The 1064-nm neodymium:yttrium-aluminum-garnet (Nd:YAG) laser was approved by the Food and Drug Administration (FDA) as a therapeutic modality for treatment of onychomycosis in 2010[44] and currently four types of laser are FDA-approved for the same indication.[45] However, there is still a scarcity of evidence supporting the efficacy of lasers for treatment of onychomycosis.[44] Most of the studies assessing the role of lasers in onychomycosis are small pilot studies or case series/reports. Carney et al. did not observe any improvement in onychomycosis severity score in an in vivo study analyzing the efficacy of five treatments of submillisecond 1064-nm Nd:YAG laser in eight patients with toenail onychomycosis.[46] However, a recent retrospective analysis of 56 patients found 1064-nm diode laser to be an effective treatment modality in the treatment of onychomycosis with positive correlation of treatment efficacy with number of treatment sessions.[44] Nonetheless, role of lasers in onychomycosis treatment still remains controversial.

FUTURE PERSPECTIVES

Retinoids in Dermatophytosis

Ardeshna et al.[47] reported successful treatment of recurrent dermatophytosis in three patients with a combination of isotretinoin and itraconazole. It was hypothesized by the authors that modulation of epidermal growth and cell turnover by retinoids may help in stopping the growth of dermatophytic infection in the epidermis. Currently a randomized controlled trial is going on in a tertiary care center at Chandigarh comparing the combination of isotretinoin and itraconazole/terbinafine with itraconazole/terbinafine in recurrent dermatophytosis.[48]

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Vaccines in Dermatophytosis

Although vaccines for immune prophylaxis of dermatophytosis in animals including cattle, horse, dog and cat are available with ongoing attempts to further increase its efficacy,[49] no vaccines for humans exist till date. Immunotherapy and immune prophylaxis against dermatophytosis in humans may be field of interest in future especially in view of recent rise in its incidence and surge in resistant, chronic cases, which are recalcitrant to available treatment modalities.

RECOMMENDATIONS FROM THE RECENT EXPERT CONSENSUS IN INDIA REGARDING THE DIAGNOSIS AND MANAGEMENT OF DERMATOPHYTOSIS[50]

Recently an expert consensus was formulated by 11 experts in the field of dermatology and mycology from India with the help of modified Delphi process comprised of two workshops along with five rounds of questionnaires. The consensus recommended KOH mount and microscopy as the point-of-care test in dermatophytosis while fungal culture should be performed in recurrent, chronic, recalcitrant and extensive dermatophytosis. MALDI-TOF MS was recognized as a promising experimental diagnostic technique but not practical for a real world scenario as it cannot be directly performed on clinical specimens, requiring a mandatory prerequisite of culture. Topical antifungal therapy was recommended for localized cases of naïve tinea cruris and corporis while a combination of systemic and topical antifungal agents was advocated for naïve and recalcitrant lesions of tinea pedis, extensive tinea corporis and recalcitrant tinea cruris and corporis. Topical azoles should be preferred owing to its broad-spectrum activity along with its anti-inflammatory and antibacterial properties. Terbinafine and itraconazole were recommended as the preferred systemic antifungal agents. The recommended minimum duration of treatment was 2–4 weeks in naïve tinea cases and more than 4 weeks in recalcitrant tinea cases. There was a uniform consensus among experts discouraging strongly the use of topical corticosteroids in management of dermatophytosis. Treatment with topical agents was recommended to be continued till 2 weeks after cure while systemic agents after cure should be continued only in recalcitrant cases. Doubling of terbinafine dose (500 mg/day) can be done in cases of recalcitrant tinea while uniform consensus regarding the doubling of itraconazole dose could not be reached. Fluconazole and terbinafine were recommended as the preferred systemic drugs in the pediatric population. Systemic antifungal agents should be avoided in pregnancy and only topical agents should be used.

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CONCLUSION

The incidence of dermatophytosis has reached almost to an epidemic level in the recent past owing to factors like overcrowding, rapid urbanization, increase in the number of immunosuppressed hosts, rampant misuse of topical steroids and resistance to antifungal agents. There has also been increase in the cases of chronic, recurrent and recalcitrant cases of dermatophytosis and dermatophytosis presenting with extensive and atypical lesions. The clinicians are also facing the challenge of emerging drug resistance in patients of dermatophytosis due to use of topical steroids and availability of substandard systemic and oral formulations of antifungal agents. Molecular methods like PCR for diagnosis of dermatophytosis are emerging as an accurate and reliable modality, although its use is limited to the experimental setting. PDT and lasers are also emerging as alternative treatment modalities in the treatment of dermatophytosis especially in refractory cases or in cases where systemic antifungal drugs are contraindicated.

REFERENCES

1. Sahoo AK, Mahajan R. Management of tinea corporis, tinea cruris, and tinea pedis: a comprehensive review. *Indian Dermatol Online J*. 2016;7:77-86.
2. Poluri LV, Indugula JP, Kondapaneni SL. Clinicomycological study of dermatophytosis in south India. *J Lab Physicians*. 2015;7:84-9.
3. Blutfield MS, Lohre JM, Pawich DA, Vlahovic TC. The immunologic response to *Trichophyton rubrum* in lower extremity fungal infections. *J Fungi*. 2015;1:130-7.
4. Nenoff P, Verma SB, Vasani R, et al. The current Indian epidemic of superficial dermatophytosis due to *Trichophyton mentagrophytes*: a molecular study. *Mycoses*. 2018.
5. Singh S, Beena P. Profile of dermatophyte infections in Baroda. *Indian J Dermatol Venereol Leprol*. 2003;69:281-3.
6. Qadim HH, Golforoushan F, Azimi H, Goldust M. Factors leading to dermatophytosis. *Ann Parasitol*. 2013;59:99-102.
7. Gurcan S, Tikvesli M, Eskiocak M, Kilic H, Otkun M. Investigation of the agents and risk factors of dermatophytosis: a hospital-based study. *Mikrobiyol Bul*. 2008;42:95-102.
8. Bishnoi A, Narang T, Handa S, et al. Paraneoplastic bullous pemphigoid associated with penile squamous cell carcinoma. *J Eur Acad Dermatol Venereol*. 2018;32:e140-e1.
9. Hay RJ. Chronic dermatophyte infections. I. Clinical and mycological features. *Br J Dermatol*. 1982;106:1-7.
10. Dogra S, Uprety S. The menace of chronic and recurrent dermatophytosis in India: is the problem deeper than we perceive? *Indian Dermatol Online J*. 2016;7:73-6.
11. Sentamilselvi G, Kamalam A, Ajithadas K, Janaki C, Thambiah AS. Scenario of chronic dermatophytosis: an Indian study. *Mycopathologia*. 1997;140:129-35.
12. Ranganathan S, Menon T, Selvi SG, Kamalam A. Effect of socio-economic status on the prevalence of dermatophytosis in Madras. *Indian J Dermatol Venereol Leprol*. 1995;61:16-8.
13. Pathania S, Rudramurthy SM, Narang T, Saikia UN, Dogra S. A prospective study of the epidemiological and clinical patterns of recurrent dermatophytosis at a tertiary care hospital in India. *Indian J Dermatol Venereol Leprol*. 2018;84:678-84.
14. Sharma R, Adhikari L, Sharma RL. Recurrent dermatophytosis: a rising problem in Sikkim, a Himalayan state of India. *Indian J Pathol Microbiol*. 2017;60:541-5.

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