“A CLINICAL AND MYCOLOGICAL PROFILE OF DERMATOPHYTOSIS IN KLES DR PRABHAKAR KORE HOSPITAL AND MEDICAL RESEARCH CENTRE, BELGAUM”

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LIST OF ABBREVIATIONS USED

^{0}C	-	Degree celcius
A.D.	-	Anno Domini
Ab	-	*Arthroderma benhamiae*
AIDS	-	Acquired immunodeficiency syndrome
CHSI	-	Chitin Synthase I
cm	-	Centimeter
DMSO	-	Dimethyl sulfoxide
DSO	-	*Distal Subungal Onychomycosis*
DTH	-	Delayed-type hypersensitivity
DTM	-	Dermatophyte test medium
E.	-	*Epidermophyton*
eg.	-	Example
etc	-	Etcetera
HIV	-	Human immunodeficiency virus
i.e.	-	That is
IFN-γ	-	Interferon-γ
IgE	-	Immunoglobulin E
IgG	-	Immunoglobulin G
KOH	-	*Potassium Hydroxide*
LCB	-	Lactophenol Cotton Blue
M.	-	*Mentagrophyte*
Mc	-	*Microsporum canis*
Mg	-	*Microsporum gypseum*

mg	-	Milligram
l	-	Litre
mm	-	Millimeter
n	-	Total number
NaOH	-	Sodium hydroxide
p	-	Probability
PAS	-	Periodic Acid Schiff
PCR	-	Polymerase chain reaction
PSO	-	*Proximal Subungal Onychomycosis*
SD	-	Standard deviation
T.	-	*Trichophyton*
TDO	-	*Total Dystrophic Onychomycosis*
Te	-	*Trichophyton equinum*
Tr	-	*Trichophyton rubrum*
Tt	-	*Trichophyton tonsurans*
Tv	-	*Trichophyton verrucosum*
WSO	-	*White Superficial Onychomycosis*

ABSTRACT

Background and objectives

Dermatophytosis, a group of taxonomically closely related keratinophilic fungi called dermatophytes varies with geographical area as well as climatic conditions and there is wide variation in the spectrum of dermatophytic isolates. This study was aimed to understand the clinical and mycological profile of dermatophytosis.

Methodology

The present one year cross sectional study from January 2013 to December 2013 was done in the Department of Dermatology, Venereology and Leprosy, KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belgaum A total of 125 patients presenting with dermatophyte skin infection were subjected to clinical examination and KOH examination for fungi and culture.

Results

Maximum cases were noted in the month of August (16%). The commonest clinical forms noted were Tinea corporis (52%) and Tinea cruris (43.2%). Multiple clinical forms were present in 23 cases and commonest clinical form was Tinea cruris with Tinea corporis (73.91%). Most of the patients were males (67.2%) (male to female ratio 2:1) and Tinea corporis was the commonest clinical diagnosis (48.81%). The commonest age group was 21 to 30 years (36%) and had Tinea corporis (56%) commonly. 36.8% of the patients had duration of > 35 weeks. The commonest morphological variant was noted as annular (37.6%).

The KOH examination was positive in 78.4% cases and culture was positive for fungus in 64.8% of the cases. In patients with positive culture, *T. mentagrophyte* was the commonest isolate (48.15%). The commonest dermatophyte isolated was *Trichophyton* (88.64%).

Conclusion

There is wide variation in the clinical and mycological profile of dermatophytosis. The detection of emerging organisms may be help in the treatment and adequacy of current pharmacologic regimens.

Key words:

Dermatophytosis; Mycological profile; Skin infection; Tinea corporis; Tinea cruris;

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INTRODUCTION

Dermatophytosis infection represents a superficial infection of the keratinized layers of hair, nail and skin cause by dermatophytic fungi. The Romans called this disease “Tinea” which means insect larva. This word “Tinea” is still used as generic term for superficial fungal infections. In India, this is very common infective condition. The clinical picture of this infection is a reflection of host parasite relationship and is therefore varied. There is a wide variation in the clinical picture with the causative species.

In clinical dermatology, dermatophytosis are classified according to their localization on different regions of body. In each region of body clinical picture varies with the causative species of fungus, the size of inoculums and the duration of infection. Thus Tinea corporis caused by *Trichophyton verrucosum* is more likely to be inflammatory, than the dry scaly lesion produced by *Trichophyton rubrum*. The anatomical region affected will also modify the course of disease and clinical pricure; for example Tinea cruris lesion are more macerated than the Tinea corporis lesions, while Tinea unguim and Tinea pedis are chronic infections which are difficult to eradicate. Other factors which may modify the course of the disease are: immune status of the host, intercurent diseases like diabetes mellitus and lymphoid malignancies, age, habits, race, genetic background of the patient. This wide diversity of clinical presentation prompted us to make a detailed clinical study of the dermatophytosis in the various regions of the body.

Dermatophytosis are caused by a group of taxonomically closely related keratinophilic fungi called dermatophytes. The dermatophytes are mould fungi

which produce long chains of cells-hyphae, and arthrospores in tissue. They are not part of normal skin flora. Dermatophytosis is, therefore exogenous infections from soil, animal or human sources. These are represented by 42 closely related species in three genera namely *Trichophyton, Mircosporum* and *Epidermophyton*. Only a few of these are pathogenic to human and most others are non pathogenic soil inhibiting keratinophiles. These have been classified ecologically as *zoophilic, geophilic* and *anthropophilic*. They show variations in their cultural characteristics and pathogenecity. Infections caused by the *zoophilic* species is often inflammatory than that caused by *anthropophilic* species. The degree of inflammation is also inversely related to likelihood of persistence and early relapse.

It is well known fact that common causative agent vary in different geographical locations for the same type of fungal infection. For example *Trichophyton violaceum* is a common cause of Tinea capitis in India, while *Trichophyton tonsurans* is the commonest causative agent in America. There is also much evidence to suggest that the common causative species in given geographical area may change with time. For example, in the 1940's *M. audounii* was the commonest causative agent of Tinea capitis in United States while today *T. tonsurans* is the main agent implicated.

Dermatophytosis in India, have received increasing attention in recent years from different parts of the country. In the present study we undertake to evaluate the clinico-mycological profile of dermatophytosis in and around Belgaum.

OBJECTIVES

The objectives of the present study were;

Primary objective

To study the clinical and mycological profile of dermatophytosis in tertiary care hospital.

Secondary objective:

- To correlate between site of involvement and causative agent.
- To study the relation of dermatophytosis with demographic data like age, sex and duration.

REVIEW OF LITERATURE

History

Terminology and clinical description

Superficial fungal infections or dermatophytosis have been noted and described from earliest historical times. The Greek's termed this disease "Herpes" and this term with modifications was later used as *Herpes circinatus or herpes desquamans*. The Romans, who thought that the disease was caused by insects, as it resembled circular holes in clothes eaten by moths, called it "Tinea" which means insect larva. The earliest written record of the word "Tinea" is attributed to John de Trevisa (1326 – 1412).[1] The use of this term continues till date in all text books of dermatology as well as mycology.

The earliest written descriptions corresponding to this disease have been found in Eber's papyri of Egypt. Scalp disease resembling Tinea capitis has been mentioned in the Torah – Levictus. Celsus in 300 A.D. described a suppurative infection of the scalp, known as the Kerion – Celsi. Favus was also described in Celsus de Medica as Porrigo. Two seventeenth century (1668) paintings in Seville, by the baroque painter Bartolome Murrillo show children affected by Tinea Capitis.[2]

The first observation and descriptions of Tinea unguium was one of the Mahon brothers in the early 19th Century. David Gruby in 1841 carefully described several different types of dermatophytic infections including favus and endothrix infections.[1,3] Malmstem in 1845 also made detailed description of Tinea of the scalp.[1]

In 1895, Adamson described his observations on method of infection and clinical picture of *Microsporum audouinii* infections.

Mycological history

Historically, the science of mycology is said to have originated with Hook in 1677,[4] followed in 1835 by Agostino Bassi's delineation of fungal origin of silk worm disease.[1,4] Soon Remak (1834, 1837) demonstrated fungal elements in favic crusts and named it Achiorion Schoenleinii. Schoenlein (1839) and Langenbach (1839) also made their contributions. It is generally agreed however, that, the development of medical mycology began truly, if not historically, with David Gruby.[4] In 1841, he published careful description of several dermatophytic infections. In subsequent studies (1841–44), he described the detailed microscopy of *Trichophyton schoenleinii*; discovered and named *Microsporum audouinii*, and infections caused by it; described and named Candida albicans and also studied *Trichophyton tonsurans* in detail.

In 1845, Malmstem, named the Genus *Trichophyton* and Robin, in 1847, described in detail *Trichophyton mentagrophytes*.[5,6] Grawitz and Duclaux (Paris) were the first to grow pure fungal cultures in 1836.[4]

Raymond Jaques Adrein Sabouraud, began his studies in Mycology in 1892. In 1910, he published his classical work Les Teignes, in which he not only established the multiplicity and diversity of dermatophytic species, but also classified them into 4 genera.[7] These were *Microsporum, Trichophyton, Epidermophyton* and *Achorion*. Sabourad, also established taxonomic criteria for identification of fungi. He also perfected and used on a large scale, X- Ray epilation

as a successful therapy for Tinea capitis.[2] He is thus rightly known as the "father" of Medical Mycology.

In 1927, Arturo Nannizzi, first reported the perfect state of *Microsporum gypseum* complex.[4]

In 1930 Langeron and Milochevitch proposed a classification of dermatophytes;[6] variants of which are still followed in certain parts of Europe, but it was Emmons in 1934, who with his co-workers laid the basis for the current taxonomic approach to the dermatophytes. He was also responsible for dropping Sabouraud's 4th Genus Achorion.[8]

The close collaboration between renowned dermatologist Joseph Hopkins (1882 – 1951) and Mycologist Rhoda Benham (1894-1957), made medical mycology a clinical science. Their students Chester Emmons, Lucille. K. George, Carrion and silva, with their systematic and scientific study of pathogenic fungi and the disease caused by them made many significant contributions.

In 1914, a breakthrough in Medical Mycology, of different nature was achieved by Norman Conant and his collegues when they wrote and Published "Manual of Clinical Mycology."[3] This book had profound influence on dermatologists and mycologists of recent times.

The rediscovery of a perfect state of *Microsporum gypseum* by Griffin in 1960, was followed quickly by that of many other dermatophytes. It soon became clear that *Microsporum* species were associated with Nannizia perfect state, and *Trichophyton* species with the arthroderma perfect state. These discoveries enabled Ajello L. (1968) to put forward his amended classification of dermatophytes. In this,

he placed first importance on the perfect state, and second importance to the character of the walls of the Macroconidia.[5]

Mycology

Botanical classification

Depending on the mode of reproduction, the fungi pathogenic to human beings are classified systematically into four classes by Emmomns (1977).[8]

Classification of fungi[8]

1. Phycomycetes consisting of
    a) Oomycete
    b) Zygomycetes
2. Ascomycetes
3. Basidiomycetes
4. Fungi Imperfecti (Deuteromycetes)

The dermatophytes are placed in "Fungi Imperfecti" because of their supposedly incomplete development cycles. Today, the perfect state of many dermatophytes have been reported and they are being reclassified accordingly.

Classification of dermatophytes[5]

The etiologic agents of the dermatophytosis are classified in three anamorphic (asexual or imperfect) genera, *Epidermophyton*, *Microsporum*, and *Trichophyton*, of anamorphic class *Hyphomycetes* of the *Deuteromycota* (Fungi Imperfecti). The descriptions of the genera essentially follow the classification

scheme of Emmons on the bases of conidial morphology and formation of conidia and are updated following the discovery of new species.

Dermatophytes are classified into 3 genera:[9]

1. *Trichophyton*
2. *Epidermophyton*
3. *Microsporum*

Each genus is further classified into many species. According to the recent available data there are 42 recognised species: 24 in genus *Trichophyton*, 16 in genus *Microsporum*, and 2 in genus *Epidermophyton*. These pathogenic dermatophytes, parasitise only the keratinized tissues of man and animals. For proper identification, fungal cultures must be done to see for microconidia/ macroconidia and hyphae.[9]

Trichophyton species usually infect skin, hair and nail. *Microsporum* species infect skin and hair. *Epidermophyton* species infect skin and nail.[9]

Trichophyton species

The genus *Trichophyton* is characterized by the development of smooth-walled micro as well as Macroconidia. Macroconidia are mostly borne directly on hyphae or on pedicels, and are thin or thick walled, ranging from 4-8 x 20-50 um in size. They may be few or absent in many species. Microconidia are pyriform, spherical, clavate or of irregular shape and range from 2-3 x 2-4 um in size.[9]

Microsporum species

Microsporum species form both macro and microconidia on short conidiophores. Macroconidia are hyaline, multiseptate, variable in form, fusiform, spindle-shaped to obovate, ranging from 7-20 x 30-160 um in size, with thin or thick echinulate to verrucose cell wall. Their shape, size and cell wall features are important characteristics for species identification. Microconidia are hyaline, single celled and pyriform to clavate, smooth walled, 2.5-3.5 x 4-7 um in size and are not diagnostic for any one species.[9]

Epidermophyton

Epidermophyton species show smooth, thin-walled macroconidia which are often produced in clusters growing directly from the hyphae. Numerous chlamydoconidia are formed. No microconidia are formed.[9]

Anamorph genera and species of organism[9]

Epidermophyton

- *E. Floccosum*

Microsporum

- *M. audouinii*
- *M. canis*
- *M. equinum*
- *M. ferrugineum*
- *M. fulvum*

- *M. gallinae*
- *M. gypseum*
- *M. nanum*
- *M. persicolor*
- *M. praecox*
- *M. racemosum*
- *M. vanbreuseghemii*

Trichophyton

- *T. concentricum*
- *T. equinum*
- *T. gourvilii*
- *T. kanei*
- *T. megninhi*
- *T. mentagrophytes*
- *T. raubitschekii*
- *T. rubrum*
- *T. schoenleinii*
- *T. simii*
- *T. soudanense*
- *T. tonsurans*
- *T. verrucosum*
- *T. violaceum*
- *T yaoundei*

Ecological Classification

Anthropophilic species

These have adapted away from soil or animal reservoir and infect human. They are often epidemic in nature. They generally cause a relatively non-inflammatory infection often located on covered areas of body (feet, groin). These infections are transmitted from human to human by direct contact or through fomites.[10]

Geophilic species

The sporadic infections caused by *geophilic* species are usually inflammatory. Although *M. gypseum* strains isolated from soil are of low virulence, those isolated from humans, are more virulent and account for epidemic spread of infections under more appropriate conditions.[11,12] Although many species of soil inhabiting keratinophiles have been isolated, most are non-pathogenic to man.

Zoophilic species

They cause highly inflammatory and suppurative infections in humans. Domestic animals and pets are becoming an increasing source of infection. Exposed area of the body are favoured sites of infection.

Anthropophilic species (area of endemicity)[9]

- *E. floccosum*
- *M. audouinii (Africa)*
- *M. ferrugineum (East Asia, East Europe)*

- *T. concentricum (Southeast Asia, Melanesia, Amazon area, Central America, Mexico)*
- *T. gourvilii (Central Africa)*
- *T. kanei*
- *T. megninii (Portugal, Sardinia)*
- *T. mentagrophytes (complex of two species)*
- *T. raubitschekii (Asia, Africa, Mediterranean)*
- *T. rubrum*
- *T. schoenleinii*
- *T. soudanense (Subsaharan Africa)*
- *T. tonsurans*
- *T. violaceum (North Africa, Middle East, Mediterranean)*
- *T. yaoundei (Central Africa)*

Zoophilic species (typical host)[9]

- *M. canis (cat, dog)*
- *M. equinum (horse)*
- *M. gallinae (fowl)*
- *M. persicolor (vole)*
- *T equinum (horse)*
- *T mentagrophytes (two sibling species and variants; rodents, rabbit, hedgehog)*
- *T. sarkisorii (Bactrian camel)*
- *T. simii (monkey, fowl)*

- *T. verrucosum (cattle, sheep, dromedary)*

Geophilic species[9]

- *E. stockdaleae*
- *M. amaonicum*
- *Microsporum anamorph of A. cookiellum*
- *M. boullardii*
- *M. cookei*
- *M. gypseum (complex of three species)*
- *M. nanum*
- *M. praecox*
- *M. racemosum*
- *M. ripariae*
- *M. vanbreuseghemii*
- *T ajelloi*
- *T. flavescens*
- *T gloriae, T. Iongifusum*
- *T phaseoliforme*
- *T terrestre (complex of three species)*
- *T vanbreuseghemii*

Clinical classification

Dermatophyte infections are classified based on site of involvement, morphology of lesion and anatomo-clinical basis.

1. Based on site of involvement

Clinical types Site of involvement

Tinea corporis (Glabrous skin)

Tinea cruris (Groin)

Tinea capitis (Hair and scalp)

Tinea faciei (Face)

Tinea manuum (Palm)

Tinea unguim (Nail)

Tinea pedis (Feet)

Tinea barbae (Beard area)

2. Based on morphology[13,14]

a) Tinea corporis

Non-inflammatory

- Tinea circinata
- Vesicular/eczematous
- Plaque type
- Tinea imbricata

Inflammatory

- Herpetiform
- Kerion of glabrous skin
- Majocchi's granuloma
- Nodular granulomatous perifolliculitis of leg
- Agminate folliculitis

- Subcutaneous abscess/Tinea profunda
- Mycetoma

b) Tinea cruris

- Papulosquamous
- Vesiculopustular

c) Tinea faciei

- Non-Inflammatory
    - Black dot
    - Grey patch
- Inflammatory
    - Kerion
    - Favus

d) Tinea barbae

- Inflammatory
- Superficial/ Sycosiform
- Circinate/ Spreading

e) Tinea manuum

- Non-Inflammatory hyperkeratotic
- Inflammatory Vesicular

f) Tinea pedis

- Chronic Intertriginous
- Chronic Hyperkeratotic
- Vesicular
- Acute Ulcerative

g) Onychomycosis

- Distal subungual Onychomycosis
- Proximal subungual Onychomycosis
- Superficial White Onychomycosis
- Total dystrophic Onychomycosis

3. Anatomo clinical classification of dermatophytes[15]

There are four groups:

a. Dermatophytoses limited to keratinized structure

- Tinea corporis, Tinea cruris, Tinea faciei, accompanied or not by non-perforating folliculitis
- Tinea of palms and soles
- Tinea capitis (excluding kerion and favus)
- Tinea unguim

b. Perforating Dermatophytosis:

- Chronic perforating folliculitis
- Kerion and sycosis
- Favus

c. Dermatophytosis actively invading dermal tissues (dermatophytic granuloma)

- Dermatophytic granuloma following invasion of hair follicles
- Majocchis granuloma

d. Generalized Dermatophytosis

- Dermatophytic disease

Epidemiology

The prevalence of dermatophytosis skin diseases in any community depends upon wide range of factors genetic, racial, nutritional status and age structure of the community, climatic factors, social and hygienic standards, customs and occupations, immunosuppression and perhaps some degree of inherited susceptibility. Improvements in living conditions have generally been associated with a decline in *zoophilic* dermatophyte and an increase in *anthropophilic* dermatophyte infections.

Environmental factors

The distribution of the dermatomycoses, their aetiological agents and the predominating anatomical infection patterns vary with geographical location and a wide range of environmental and cultural factors. Dermatophytes thrive at surface temperatures of 25–28^{0}C and infection of human skin is supported by warm and humid conditions. For these reasons, superficial fungal infections are relatively common in tropical countries and are exacerbated by the wearing of occlusive clothing.[16]

In addition, the frequency of dermatphytosis is greater in communities with low socioeconomic status: crowded living conditions provide multiple opportunities for skin-to-skin contact and close proximity to animals, while hygiene may be suboptimal.[16]

Host factors

Genetic

In households afflicted with *T. concentricum* and *T. rubrum*, relatives are more likely to be infected than conjugal partners, even with equal exposure to the fungus. Pedigree from families with chronic dematophytosis suggests an autosomal dominant pattern of susceptibility. This could be explained by the genetics position towards the development of immediate hypersensitivity responses and autosomal dominant difference in keratins which affect the ability of a fungus to attack and digest the protein.[17]

Race

All races are affected equally and their prevalence depends mainly on environmental factors.[18]

Age

The occurrence of dermatophytes infection varies among different age group mainly due to different rate of exposure and amount of sebum production and decreased immunity with old age.[19]

Tinea pedis: the incidence increases with age from adolescence. It occurs more frequently in people who wear occlusive shoes

Tinea corporis: It is more common in children than in adults and occurs most frequently in hot climates.

Tinea cruris: It is commonly seen in young men living in a warm climate.

Tinea capitis: This is a dermatophyte infection of the scalp and hair and tends to affect young children worldwide.

It has been noted that dermatophyte infections are more common in adolescents and adults. However, adults are generally less susceptible to skin infection than are children owing to the fungistatic properties of fatty acids in the sebum

Sex

In general dermatophyte infections are commonly seen among males and this could be attributed to there lifestyle and environmental exposure. Tinea cruris, Tinea pedis is more prevalent in males.[20]

Associated diseases

Increased incidence of dermatophyte infection is seen in association with immunocompromised states such as acquired immunodeficiency syndrome (AIDS), diabetes mellitus, cancer and organ transplantation.

The severity of infection is increased in human immunodeficiency virus (HIV) disease.[21,22]

Several host conditions such as atopy and epidermal barrier defects like ichthyosis are associated with an increased prevalence of dermatophytosis.

Protein deficiency has been described as predisposing factor.[23]

Agent factors

Dermatophytosis are infections of the skin, hair and nail caused as a result of colonization of the keratinized layers of the body. This colonization is brought about by the organisms belonging to the three genera namely *Trichophyton, Epidermophyton, Microsporum.*[24]

Aetiology

The genus *Trichophyton* includes 24 species. The colonies on agar media may have powdery, velvety or waxy appearance. The predominant spore type is micro conidia with sparse macroconidia. Reverse side pigmentation is characteristic of the species and is used for the identification of the species within the genus. The macroconidia are thin walled with smooth surface and variable shape. Some of the *Trichophyton* species are fastidious in their requirement for amino acid as nitrogen source. *Trichophyton tonsurans* requires ornithine, citrulline and Arginine whereas *Trichophyton mentagrophytes* requires methionine. This nutritional specificity has been used by many authors in the identification of the *Trichophyton* species.[24]

The genus *Microsporum* includes 16 species. The colony *morphology* of *Microsporum* species on agar surface is either velvety or powdery with white to brown pigmentation. Both macro and microconidia are produced but the predominant conidial structures are macroconidia. Microconidia are less abundant. The macroconidia are multi septate with thick wall and rough surface. Rarely some species produce neither micro nor macroconidia. They do not have any special nutritional requirements.[24]

The genus *Epidermophyton* includes only 2 species. The colonies are slow-growing, powdery and unique brownish yellow in colour. This genus is devoid of microconidia. Macroconidia are abundant and produced in clusters. These macroconidia are thin walled with smooth surface.[24]

Virulence factor

Virulence of the infecting organism and the ecological origin play an important role in dermatophyte infections.[25]

There may be observed strain to strain differences for the same dematophyte e.g: *T. mentagrophytes var mentagrophytes* is a *zoophilic* organism capable of producing marked inflammatory reaction in the human host and it produces granular colony on culture while *T. mentagrophytes var interdigitale* is *anthropophilic*, produces a non-inflammatory type of infection and colony are downy.[25]

The sequencing of seven dermatophyte genomes has recently been completed, and the sequence is available via the Broad Institute database. The Broad Institute sequenced and annotated the genomes of five dermatophyte species. *Trichophyton rubrum* (*Tr*) is *anthropophilic* and the most common cause of dermatophyte infections in humans worldwide.[25]

Trichophyton tonsurans (*Tt*) is also *anthropophilic* and is a common cause of Tinea capitis. *Microsporum canis (Mc)* is also zoophilic and is the most common cause of tinea capitis in Europe.[25]

Microsporum gypseum (*Mg*) is a *geophile* that is associated with gardener's ringworm.[25]

Genome sequence of remaining two species, the Phylogenetically related *Arthroderma benhamiae* (*Ab*, a teleomorph of *Trichophyton mentagrophytes*) and *Trichophyton verrucosum* (*Tv*).[25] These organisms cause a highly inflammatory infection in humans.

The disease caused by human-adapted organisms *Tr* and *Tt* tends to be chronic with low inflammation, whereas zoophiles (*Te*, *Mc*, *Ab*) and *geophiles* (*Mg*) generally cause an inflammatory infection.[25]

All seven genomes were found to encode high numbers of protease-encoding genes compared to related, nondermatophytic fungi. Dermatophytes have expanded sets of exopeptidases, endopeptidases, and secreted proteases.[25]

Pathogenesis

Dermatophytes are not endogenous pathogens. Transmission of dermatophytes to humans occurs via three sources, each resulting in typical features.[17]

Category

- *Anthropophilic*
- *Zoophillic*
- *Geophilic*

The possible route of entry for the dermatophytes into the host body is injured skin, scars and burns and increased hydration of skin and maceration.[17]

Infection is caused by arthrospores or conidia.[17]

Dermatophyte infections involve three main steps:[17]

a) Adherence to keratinocytes

b) Penetration through and between cells

c) Development of a host response

a. Adherence to keratinocytes

Superficial fungi overcome several obstacles in order for the arthroconidia, the infectious element, to adhere to keratinized tissue. Dermatophyte must resist the effects of competition from normal flora, ultraviolet light, variation in temperature and moisture, and sphingosines produced by keratinocytes.[26]

Trichophyton rubrum has ability to adhere to epithelial cells which has been attributed to carbohydrate specific *adhesins*, expressed on the surface of microconidia.[26]

T. mentagrophytes has morphological presentation of fibrillar projections during the adherence phase on the skin surface, fibrils (long and sparse) connect fungal arthroconidia to keratinocytes and to each other. On inner skin layers, newly formed arthroconidia show thin and short appendices covering their entire surface; the latter begin to vanish as a large contact area is established between conidia and skin tissue.[26]

b. Penetration

After adherence, spores germinate and penetrate the stratum corneum at a rate faster than desquamation. Penetration is accomplished by the secretion of

proteinases, lipases, and mucinolytic enzymes, which also provide nutrients to the fungi.[26]

Fungal mannans in the cell wall of dermatophytes also decrease the rate of keratinocyte proliferation. New defenses emerge once the deeper layers of epidermis are reached, including competition for iron by unsaturated transferrin and possibly inhibition of fungal growth by progesterone.[26]

c) Development of a host response

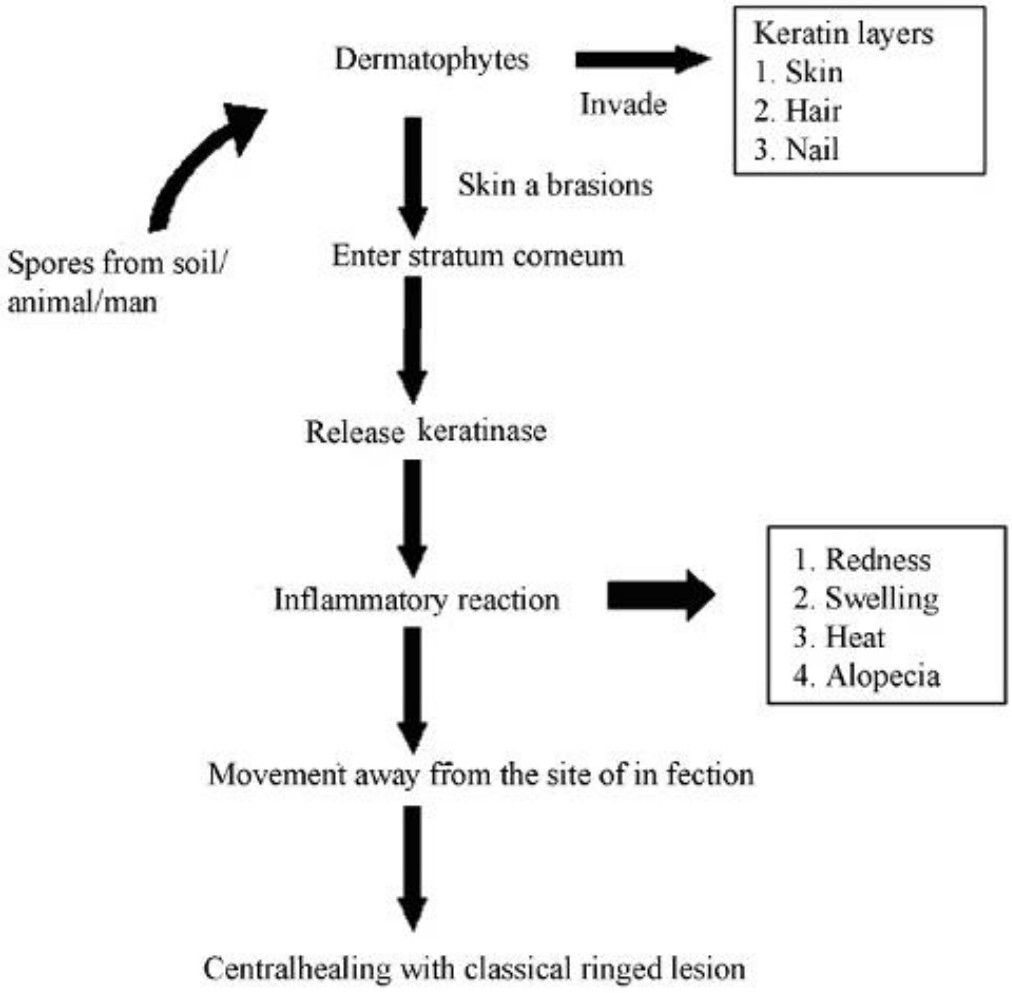


Figure 1. The schematic route of entry of dermatophytes into the host system and onset of immune response in the host in response to the pathogen entry[24]

Fungal metabolic products diffuse through the malphigian layer to cause vesicle, erythema, or even pustule formation along with pruritus. I*n vivo* activity is mainly restricted to newly differentiated keratin and Adamson's fringe within the hair shaft. Acute dermatophytosis is associated with a Delayed type hypersensitivity skin response towards them and while persistent disease corresponds to Immediate Hypersensitivity responses to the production of Th2 cytokines by mononuclear leukocytes, to high levels of IgE and IgG4 antibodies.[26]

Immunology

a) Acquired resistance

The efficient and protective response against dermatophytosis is a cell-mediated response of the DTH, characterized namely by the action of macrophages as effector cells, interferon-α secretion from type 1 T-helper lymphocytes and by some key cytokines like interferon-γ (IFN-γ). Immune detection and chemotaxis occur via low-molecular weight chemotactic factors or alternative complement pathway activation. However, the immune response that is raised, and especially the degree of inflammation, varies according to the dermatophyte species, the host species and the pathophysiological status of the host.[26]

In general, the *zoophilic* species cause more inflammatory infections which may heal spontaneously and result in relative resistance to reinfection. The *anthropophilic* species usually cause more chronic, less circumscribed infections which result in less resistance to reinfection.[26]

b) Hypersensitivity ("Trichophytin" Reaction)

The "trichophytin" reaction is the term used for cutaneous hypersensitivity to dermatophyte antigens injected intradermally in humans. Both immediate- and delayed-type reactions occur, but the latter is most often associated with infection.[26]

Trichophyton species can be isolated from patients with deep-seated trichophytosis in a liquid medium consisting of beef extract, peptone, and maltose. After 2 to 3 months at room temperature, the growth is ground and filtered.[26]

In patients with deep-seated trichophytosis, parenteral injection of "trichophytin" caused signs and symptoms analogous to those induced in tuberculous patients by injection of tuberculin: general toxic reactions including elevated temperature, perspiration, loss of appetite, headache, and pain in the joints. There was inflammation, formation of pustules, and burning at the injection site.[26]

Dermatophytid reactions (4–5% of patients) are inflammatory eczematous allergic skin reactions at sites distant from primary fungal infection. Being KOH and culture negative, it is associated with a DTH response to trichophytin test and may involve a local DTH response to systemically absorbed fungal antigen.[26]

c) Antibodies;

Antibody formation does not seem to be protective. The dermatophyte antigen is thought to be processed by epidermal Langerhans cells and presented in local lymph nodes to T lymphocytes which proliferate, migrate to the infected site, and produce inflammation. The epidermal barrier becomes permeable to transferring and migrating cells leading to spontaneous resolution of lesions.

Trichophytin skin test is now positive and clearing of second infection will be more rapid. Rivalier showed that a dermatophytic infection in humans results in a relative resistance to subsequent infection called *'le phenomene de la reaction acceleree'* or *'le phenomene de Bruno Bloch'*, mainly by the inflammatory forms (kerion), caused by *zoophilic* species, but not always follow the more chronic *anthropophilic* infections. Fungi which do not invade the hair follicle do not seem to give rise to an equivalent immunity when growing in the horny layer of the smooth skin. In contrast, a study could not demonstrate such acquired immunity in experimental *T. rubrum* infection of smooth skin.[26]

d) Non-Specific Resistance

Natural defenses against dermatophytes depend on immunological and nonimmunological mechanisms. Many nonspecific factors may account for natural resistance to infection. It is mainly related to the "serum factor," a fungistatic substance in serum of normal individuals and animals. This factor is believed to limit the growth of the dermatophytes to the keratinized layers, i.e., prevent their invasion of living tissues.[26]

Host factors that help limiting the infection to keratinized tissue include their preference for cooler skin temperatures than the normal body temperature, serum inhibitory factors(beta-globulins, ferritin and other metal chelators) binding to iron essential for growth of dermatophytes. Unsaturated transferrin inhibits the growth of dermatophytes by binding to the hyphae. A growth modifying, α2 macroglobulin keratin inhibitor, has also been identified in serum. The natural resistance of scalp to

T. capitis in adults may be due to post pubertal, fungistatic and fungicidal, long chain saturated fatty acids.[26]

Commensal Pityrosporum yeast aids lipolysis and increases pool of fatty acids available for inhibiting fungi.[26]

Humoral immunity has a minor role in acquired resistance to dermatophytosis.[26]

Tinea corporis

Syn: Tinea circinata, Tinea glabrosa, Ringworm of body.

Definition

Dermatophyte infection of the glaborous skin of the trunk and limbs with the exception of certain specific location like scalp, palms, soles and groin.

Etiology

All species of dermatophytes belonging to the genera, *Trichophyton*, *Microsporum* and *Epidermophyton* are capable of producing Tinea corporis. The three most common causatuive organisms are *T. rubrum, M. Canis* and *T. mentagrophytes*, variations may occur based on the existence of endemic species in specific to geographic area.[26] Tinea corporis is the most common infection caused by dermatophytes in India as reports by most workers and *Trichophyton rubrum* is the largest single etiological agent as reported from many parts of the country.[20] The lesions caused by *T. rubrum* are frequently seen over covered surface of the body, especially under tight clothing.

Epidemiology

The source of infection is usually an active lesion on any animal or another human, fomites like clothing, furniture etc, soil or spread from an existing localized infection.

A warm and humid climate is associated with more frequent and severe Tinea corporis.[27]

The infection is more common in adult males. Children appear to have an increased incidence of Tinea corporis caused by *zoophilic* organism.[13]

Tinea imbricata caused by T. concentricum is a geographically restricted variant of Tinea corporis. Susceptibility is probably determined through an autosomal recessive trait.[13]

Clinical features

Tinea corporis may be diverse in the clinical presentation. The characteristic lesion is annualr usually sharply marginated with a raised erythematous and sometimes vesicular border, commonly the centre of the lesion shows some clearing, but variation may occur. The degree of inflammation varies depending on the species of the fungus, the hosts immune response and the extent of follicular invasion. Pustules and vesicles are seen in inflammatory lesions, while in less inflammatory lesions scaling is the most prominent finding. Tinea corporis is generally less inflammatory than Tinea capitis or Tinea barbae. Central resolution is perhaps more frequent in inflammatory lesions although not confined to them. The

central skin may show post inflammatory pigmentation, a change of texture or residual erythematous dermal nodules.[19]

The various clinical expression of Tinea corporis infections are:[13]

1. Annular lesions, " Classical Ringworm"

The lesion begins usually as an erythematous papule, then enlarges to form an annular lesion with a relatively normal center and a sharply defined 'active' elevated, red, and infiltrated border. The lesions may be single or numerous. The main symptom is moderate pruritis.

2. Eczematous annular

The characteristic feature is the lack of central clearing, the lesions are round, but scaling, redness and slight infiltration are present throughout the lesions. Endothrix trichophyton species most frequently cause this type of infection.

3. Plaque type

Chronic extension results in the formation of large scaling dull red plaques with sharply defined borders. *T. rubrum* is the chief offender.

4. Herpetiform type

This is an inflammatory vesicular type of ringworm, usually due to a *zoophilic* species. The primary lesion is a mound of fused vesicles, which ruptures leaving a red, eroded base on which a crust may form. New vesicles may form in the

periphery, but the lesions do not enlarge more than about 2 cm and spontaneous resolution is possible.

5. Kerion type (Tinea profunda)

Kerion of the glabrous skin is usually due to a *zoophilic* organism *T. verrucosum* or *T. mentagrophytes*. The lesion is intensely inflamed, consisting of an elevated, sharply circumscribed, boggy tumor with a bright red, exuding granulating surface. Follicular pustules are a prominent feature. The suppuration is an evidence of reaction to the fungus itself, although secondary bacterial infection may occur later as the lesion ulcerates.

6. Majocchi's Granuloma

This variant is essentially a granulomatous folliculitis and perifoliculitis. The lesion develops in the form of non-tender, non-pruritic nodules or infiltrates with borders of a plaque of chronic low grade eczematous ringworm. The nodules become slowly absorbed or undergo necrosis and heal with a depressed scar. The etiologic agent is *T. rubrum*. Patients are more frequently women who shave their legs. Apparently a portion of the hair follicle becomes disrupted and a fragment of infected hair along with its accompanying fungal elements is displaced into the corium, there eliciting a foreign body response, which may be due to fungal cells or due to hair fragments.

7. Agminate folliculitis

Caused by *zoophilic* organisms present as well defined, erythematous plaques studded with perfollicular pustules.[13]

8. Mycetoma

Rarely subcutaneous masses with draining may be seen. Causative agents are *M. audouinni, T.verrucosum, T. mentagrophytes, T. violaceum, T. tonsurans, M. ferrugineum, M. canis* and *T. rubrum*.

9. Subcutaneous abscess

Inflammatory infection by the dermatophyte species *T. mentagrophytes, T.tonsurans, T.rubrum* or *M.audouinni* may rarely produce deep subcutaneous nodules. There is rarely lymph node involvement with associated hematogenous bone and cartilage dissemination.

There are few of deep dermatophytre infection caused by *T.rubrum, T.verrucosum, T.violaceum,* and *M.gypseum* disseminating to internal organs like liver, spleen, bone and brain in immunosupressed individuals.[28]

Tinea cruris

Syn: Ringworm of the groin, Dhobi's itch, Eczema marginatum, Jock's itch.

Definition

It is infection of groin by species of dermatophyte. It includes infection of genitalia, pubic area and perianal skin.[13]

Etiology

T. rubrum is the main cause. *T. mentagrophytes var interdigitale* and *E. floccosum* also account for some cases.[19]

Epidemiology

Tinea cruris is more prevalent in warm humid conditions. This condition has always been much more common in young men than in women.[29] The anatomical differences in male and females, where in there are greater areas of occlusive skin, scrotum in contact of thigh also contribute. Males also have higher incidence of Tinea pedis, which can serve as reservoir for auto infection from foot to the groin.

Besides direct human to human contact, transmission can also occur through fomites such as towel, clothing and bed linen.

Clinical features

Pruritus is the predominant symptom and lesions are usually bilateral but asymmetrical and are characterized by erythematous plaques, with raised margins extending from the groin down to thighs. Some central clearance is usually present, but is often incomplete. The scrotum may often appear completely normal. Secondary changes such as lichenification due to chronic scratching or secondary bacterial infection may supervene.

Lesions caused by *E.Floccosum* are well marginated, scaly patches which seldom extend beyond the genitocrural crease.

T. rubrum cases are classically chronic, of the dry desquamative type and often spread to involve adjacent skin over the pubis, lower abdomen, buttocks and lower back. The rarer *T.mentagrophytes var interdigitale* infection may be vesicular and inflammatory.[19,29]

Although rare in western countries, scrotal involvement is not uncommon in India, and the penis may be occasionally involved.[30]

Tinea capitis

Syn: Ringworm of the scalp, Tinea tonsurans

Definition

Tinea capitis is dermatophytosis of scalp and associated hair.

Etiology

Virtually any species of *microsporum* or *trichophyton* can cause Tinea capitis. Exception are *T. concentricum,* and *T. mentagrophytes var interdigitate*. The causative organisms can be classified according to their host preference (i.e. *anthropophilic, zoophilic, geophilic*) and according to whether they produce arthroconidia outside or just under the cuticle of the hair (ectothrix) or within the hair (endothrix).[13]

Ectothrix species

Microsporum (small spore ectothrix)

M.audounii	*Anthropophilic*
M.ferrugineum	*Anthropophilic*
M.canis	*zoophilic*

Microsporum (large spore ectothrix)

M.gypseum	*Geophilic*
M.fulvum	*Geophilic*
M.nanum	*Zoophilic*

Trichophyton (large spore ectothrix)

T.verrucosum	*Zoophilic*
T.mentagrophytes	*Zoophilic*
T.megninii	*Anthropophilic*
T.rubrum	*Anthropophilic*

Endothrix Species

T.violaceum	*Anthropophilic*
T.tonsurans	*Anthropophilic*
T.soudanense	*Anthropophilic*
T.gourvilli	*Anthropophilic*
T.yaourndei	*Anthropophilic*

Epidemiology

Children between the age of 4-14 years are generally affected although adult cases may be seen especially with *T.tonsurans* or *T.violaceum* infection.

Transmission is favoured by existence of overcrowding or poor personal hygiene. Protein deficiency along with vitamin A deficiency also act as predisposing factor.[23] The disease can be transmitted from child to child through exposure at school and also from fomites like brushes, comb, cap and pillow covers etc. Affected hair can harbor fungi for a year or so after they have been shed from the host. The existence of an asymptomatic carrier state in Tinea capitis has been documented.[13]

Clinical Features

The clinical appearance of Tinea capitis is most variable, depending on the species of causative agents and the degree of the host immune response. The cardinal features are partial hair loss with an inflammatory response of various intensity. There are four different clinical presentations:

1. Non-Inflammatory, or epidemic type of Tinea capitis

The lesion begins as a small erythematous papule surrounding the hair shaft. The lesion then spread centrifugally, involving all the hair in its path giving rise to circular patches of partial hair loss with scaling. The patches which are usually observed on occiput or posterior aspect of the neck, show numerous broken off hair that are grey and lusterless due to coating of arthroconidia “grey patch” ringworm. *M. audouninii* or *M. ferrugineum* are the common causative agent. Green fluorescence is seen under wood’s lamp.

2. Inflammatory Tinea capitis

The infections are most commonly caused by *zoophilic* organisms (eg. *M.canis*) or *geophilic* organisms (eg. *M.gypseum*). clinically a spectrum of

inflammatory changes may be seen ranging from pustular folliculitis to a kerion. Agmitate folliculitis is less inflammatory consisting of dull red plaques studded with follicular pustules, seen in *zoophilic* infections. Kerion is the most severe pattern of reaction, which presents as a painful, inflammatory boggy mass studded with broken hair discharging follicles and sinuses. Thick crusting with matting of adjacent hair is common. There may be associated fever and regional lymphadenopahty. Secondary bacterial infection is common. The lesions usually heal with scarring alopecia.

3. “Black dot” Tinea capitis

This is most often caused by endothrix organisms *T.tonsurans* and *T.violaceum*. There are multiple polygonal patches of alopecia with indistinct finger like margins. Because of the arthroconidia, the hair shaft is extremely brittle and breaks at the level of the scalp. The remnant of the hair left in the infected follicle appear as a black dot on clinical examination. There may be diffuse scaling with minimal hair loss or inflammation.

4. Faveus “Tinea favosa”

Favus (latin “honeycomb”) is typically a chronic infection that begins early in life and commonly extends into adulthood. *T.schoenleini* is the most common causative agent other being *T.violaceum* and *M.gypseum*. The classical picture is characterized by the presence of characteristic crust known as scutula. These concentrations of hyphae and keratinous debris take root at the opening of the hair follicle then gradually expand from a yellowish red papule to form a yellow cup shaped crust 1cm or more in diameter. The centre of the scutulum is often pierced by a single lusterless dry hair which frequently attain a normal length. They extend

peripherally to form large adherent mats of scutula and hair with "mousy" odour. The centre of the infected area becomes extensively scarred and almost totally devoid of hair.

Tinea barbae

Syn: Tinea sycosis: "Barbers itch" Ring worm of the beard

Definition

Ringworm of the beard and the moustache area of the face with invasion of coarse hairs seen with adult males.

Etiology

The common causative agents are mostly the *zoophilic* dermatophytes, *T.mentagrophytes* and *T.verrucosum*. These produce more inflammatory infections of the ectothrix type *M.canis* has been implicated in a few cases. In urban areas *arthrophilic* species *T.violaceum, T.rubrum, T.megninii* and *T.schoenleinii* are recognized as occasional causes. In such cases the disease is less inflammatory.

Epidemiology

Usually the infection is contracted by exposure to animals mostly cattle and dogs in a rural setting. Transmission from person to person is via contaminated barber's razors or clippers.

Clinical features

Three main clinical types are:

1. Inflammatory or kerion like

Most often caused by *T. mentagrophytes* and *T.verrucosum*. This variety is analogous to kerion formation in Tinea capitis. Nodular and boggy lesions associated with seropurulent discharge and crusting are present commonly on the chin, neck, maxillary and sub-maxillary areas. There is sparing of the upper lip area. The hair are lose and easily pluckable. Undermining and sinus tract formation can occur in severe cases. Scarring and alopecia are the end result.

2. Superficial or Sycosiform type

This type manifests to diffuse erythema with perifollicular papules and pustules and resembles bacterial folliculitis. The causative organisms are the relative non-inflammatory anthropophiles *T.violaceum* infection results in brittle lusterless hair due to endothrix infection.

3. Circinate or spreading type

This variant presents with lesions similar to those found in Tinea circinata of glabrous skin with a an active spreading vesiculopustular border and central scaling. There may be relative sparing of hair in this variant.

4. Atypical lesions

Atypical forms like granuloma annulare like or abscess like tumors with *M.canis* and verrucous granulomatous lesions with *E.floccosum* [verrucous epidermophyton] may be recognized.

Tinea faciei

Syn: Ring worm of the face. Tinea faciale.

Definition

Infection of the glabrous skin of the face (excluding the beard and moustache area of the adult male) with a dermatophyte function.

Etiology

T.mentagropytes and *T.rubrum* are most commonly isolated dermatophytes. *M.audouinii* and *M.canis* have also been isolated.

Epidemiology

Tinea faciei occurs either by direct inoculation of the facial skin by a dermatophyte fungus from an external source (e.g pet animal) or by secondary spread from pre existing infection of another body site.

Clinical Features

A substantial number of patients show annular or circinate lesion, with an indurated raised margin in half the number. Simple papular lesions and in some cases completely flat patches of erythema also occur. Vesicles and pustules are occasionally seen. Although erythema is prominent, scaling may not be evident in all cases. The patient complaints of itching, burning and exacerbation after sun exposure, the last symptom being frequent source of diagnostic error. Besides photosensitivity,[31] eczematous contact dermatitis, discoid lupus erythematosus and granuloma annulare are commonly imitated.[32]

Tinia pedis

Definition

Infection of the feet or toes with dermatophyte fungus.

Etiology

The 3 *anthropophilic* species: *T.rubrum, T.mentagrophytes var interdigitale* and *E.floccosum* are together responsible for the vast majority of the Tinea pedis cases. Double infection with any two of these species may sometimes occur.

Epidemiology

The condition is common in adult males. The mean age of onset was noted to be 15 years.[33] The wearing of shoes and the resultant maceration of toe cleft skin predisposes to this condition.

Clinical Features

Tinea pedis may be present as one of the 4 clinically accepted variants or as an overlap of one or more of these types.[13]

1. Chronic intertriginous type

This is most common type and is characterized by fissuring, scaling, maceration in the interdigital or subdigital area. The lateral toe webs are the most common sites of infection. From here the infection spreads to the sole or instep of the foot but seldom involves the dorsum. Hyperhidrosis may be an aggravating factor.

2. Chronic hyperkeratotic type:

This is usually bilateral and presents with minimal inflammation and a patchy and diffuse moccasin like scaling over the soles. *T.rubrum* and occasionally *T.mentagrophytes* are the usual causative organism. The hand as well as toe nail may be involved, occasionally the puzzling “one hand- two feet” may be seen with *T.rubrum* infection.

3. Vesicular or vesiculobullous type:

A vesiculobullous reaction is more likely to be caused by *T.mentagrophytes var interdigitale*. Small vesicles or vesiculopustules are seen near the instep and on the mid anterior plantar surface, vesicles may become pustules and when they rupture, collarette of scaling are left behind. The infection frequently recedes during the cooler months, only to recur during the warmer months.

4. Acute ulcerative type:

This type is commonly associated with maceration, weeping, denudation and ulceration of sizeable areas of the sole, white hyperkeratosis and a pungent odour are present. Secondary bacterial infections are common. The vesiculobullous type and acute ulcerative type of Tinea pedis may be associated with a vesicular allergic reaction (id) in the hands or on the lateral foot or toe area.

Tinea manuum

Syn: Ringworm of the hand

Definition

Ringworm of palmar skin and the interdigital skin of the hand.

Etiology

The three *anthropophilic* species involved in Tinea are also implicated in Tinea manuum. *T.rubrum* the most common followed by *E.floccosum* and *T.mentagrophytes var interdigitale*. *T.violaceum* and some *zoophilic* species may also infect palmar skin.

Epidemiology

In most cases apart from animal infection, there is pre existing foot infection. Poor peripheral circulation and palmar keratoderma are possible predisposing factors.

Clinical features

There are two main types:

a. Inflammatory vesicular

Vesicles usually multiloculated, occur in clusters principally on the palms but rarely on the dorsal surface. *T. mentagrophytes* is the most common causative organism.

b. Non-inflammatory squamous type / Hyperkeratotic

T.rubrum is most commonly implicated. The palm and the palmar aspect of the fingers are involved by a diffuse fine branny adherent scaling which is especially prominent in furrows. The underlying skin may seem normal, only slightly erythematous, but may become markedly thickened and hyperkeratotic with painful fissures. Usually one hand is involved nd the involvement of nails is variable.

Tinea unguium

Syn: Ringworm of nails

Definition

Tinea unguium is clinically defined as a dermatophytic infection of the nail plate. The term onychomycosis includes all infection of nail caused by any fungus including non-dermatophytes and yeasts.

Etiology

The most common dermatophytes causing Tinea unguium worldwide are *T.rubrum, T.mentagrophytes var interdigitale* and *E.floccosum*. Non-dermatophytic filamentary fungi account for 2-5% of all cases of onychomycosis, geographical location and climate being the predisposing factors.[34] The criteria to be fulfilled in order to implicate any of organism as pathogen are:[20]

1. If dermatophyte is isolated on culture, it is considered to be the pathogen.
2. If a mould or yeast is cultured, it is considered significant only if hyphae, spores or yeast cells are seen on microscopic examination.

3. Confirmation of an infection by a non-dermatophyte requires isolation of organism on atleast 5 out 20 inocula without concurrent isolation of a dermatophyte.

Epidemiology

In a majority of patients nail infections are associated with Tinea pedis or Tinea manuum. Most of the fungal nail infection are exclusively seen in adults. The faster nail growth in children protect them. Trauma of the nails, poor peripheral circulation, athletes foot and old age are other contributory factors.[35]

Onychomycosis has been divided into following types:

1. Distal Subungal Onychomycosis (DSO)

This is the most common type. It starts by invasion of the stratum corneum of the hyponychium of the distal nail bed or the lateral nail fold. Subsequently, the infection moves proximally in the nail bed and invades the ventral surface of the nail plate. Subungal hyperkeratosis results from a hyperproliferative reaction of the nail bed in response to infection.

2. White Superficial Onychomycosis (WSO)

This is primarily invasion of the dorsal surface of nail plate. It is usually caused by *T.mentagrophytes* and is characterized by well circumscribed powdery white patches away from the free edge of the nail. WSO appears as white, sharply outlined areas on the surface of toe nails. The finger nails are not affected.

3. Proximal Subungal Onychomycosis (PSO)

Most commonly seen in HIV – infected persons. Generally caused by *T. rubrum*. The first clinical sign is a whitish to brownish area on the proximal part of the nail plate.

4. Total Dystrophic Onychomycosis (TDO)

As the infection process of TDO continues, the invasion of nail plate results in gross and total destruction of nail.

Endonyx Onychomycosis

This variant is seen with infection caused by dermatophytes that cause endothrix scalp infections, notably *T.soundanese*. The nail plate is scarred with pits and lamellar splits. The fungus invades from the top surface but penetrates deeply into nail plate.[19]

Laboratory testing of specimens

1. Direct Microscopic Examination

Direct microscopy provides an early and reasonably reliable method of diagnosing or excluding fungal infections.

Potassium Hydroxide (KOH) preparation

Direct mounts are made by mixing a small portion of the material in 2-3 drops of 10% KOH on a microscope slide. A cover slip is placed over KOH specimen and the slide is gently heated. The slide is allowed to cool and 'ripen' for

few minutes before examination. The KOH 'cleans' the specimen by digesting proteinaceous debris, bleaching pigments and loosening sclerotic material without damaging fungus, making hyphal forms easier to see. The slides are examined under bright field microscope with low condenser, first under 10x and then under 40x. The hyphae stand out as highly refractile long undulating branched septate threads. At times these hyphae fragment into rounded or barrel shaped arthrospores. The arthrospores are outside the hair shaft in chains in a mosaic pattern or intrapilar depending on the species involved and whether it is endothrix or ectothrix. 20% KOH are used for nail samples. In case the nails do not soften satisfactorily, the slide may be kept in an incubator at 37^0C for 1 hour. Hair should be examined as soon as possible after mounting.

Some modifications of KOH preparation

- Addition of 5% glycerin to 25% KOH or NaOH prevents desiccation.
- 20% KOH dissolved in 40% DMSO helps in rapid penetration and maceration of tissue without resorting to heating.[36]
- Addition of Parker Superchrome Blue-black ink to KOH solution selectively colors the hyphae making them more prominent.
- Sodium sulphide may also be used as a clearing agent.
- Eosin 1% may be added to KOH to stain the keratin. It lends a pinkish background while fungal elements remain unstained.[37]
- Modified Parker's ink and 1% Eosin method: Eosin 1% is added to Parker's ink in 2:1 proportion. The mixture is painted over the affected site and

allowed to dry. Apply cellophane tape, gently press, remove it, stick over glass slide and observe under microscope. Background stains pink and fungal elements stain blue.

2. Calcofluor white stain

Calcofluor white is a fluorescent brightener which selectively binds to chitin and cellulose in the fungal cell wall. It fluoresces light blue color when exposed to ultraviolet light (346-365nm).

3. Acridine Orange[38]

A drop of 0.01% acridine orange may be added to KOH and observed under fluorescent microscope.

4. PAS (Periodic Acid Schiff)[38]

Nail clipping stained with PAS is more rewarding as compared to KOH wet mount. The polysaccharides of fungi are oxidized by periodic acid to form aldehyde groups that yield magenta coloured compound with Schiff's fuchsin sulfide.

5. Gomori Methenamine Silver Stain[38]

This stain works on the principle of liberation of aldehyde groups and their subsequent identification by reduced silver method. The aldehyde reduces methenamine silver nitrate complex resulting in brown black staining fungal cell wall due to deposition of reduced silver wherever aldehydes are located.

Culture

The most common media used for the isolation of dermatophytes is Sabourauds Dextrose agar with chloramphenicol and cycloheximide to inhibit bacterial and saprobic fungal contamination, incubated at three temperatures i.e., 25^0 C, 30^0C and 37^0C

Dermatophyte test medium (DTM) is used for the presumptive identification of dermatophytes. On incubation at 25^0C, the dermatophyte test media turns red due to change in color of the indicator phenol red by increased pH through their metabolic activity while most fungi do not.[38] Potato flakes agar amended with cycloheximide and chloramphenicol is available as Rapid Sporulating Medium to promote rapid conidiation and colony pigmentation.[39]

SDA with 1% thiamine can be used for sporulation. The media should be inoculated and kept at room temperature for minimum of weeks. Sporulation usually occurs in 7 – 10 days. Some stains like T.verrucosum may take longer and some stains of T.tonsurans grow better when incubated at 37^0 C.

Identification

Identification is based on

1. Colony characteristics in pure culture on SDA
2. Microscopic morphology

1. Colony characteristics[39]

In observing gross colony morphology, note the color of the surface and the

reverse of the colony, the texture of the surface (powdery, granular, wooly, cottony, velvety or glabrous) the topography (elevation, folding, margins, etc.,) and the rate of growth.

2. Microscopic morphology

The appearance and arrangement of the conidia and other structures may be determined by tease mounts or slide culture preparation mounted on lactophenol cotton blue. Sometimes special media like corn meal agar, potato glucose agar, lactrimel agar, rapid sporulation medium may be required to stimulate sporulation.

a. Tease mount (Lactophenol Cotton Blue)

For preparing a mount, a portion of fungal fragment is removed with a spud and is teased on a glass slide in a drop of LCB stain using 2 teasing needles. A cover slip is placed and examined under the microscope.

b. Slide culture

Microscopic structures are beautifully preserved for study in fine details. A microscopic slide is placed on a bent glass rod at the bottom of a petri dish along with 1-2 cover slips and a filter paper. Petri dishes are closed with their lid, wrapped with craft paper and sterilized using hot air oven. Block of 1x2cm of Sabourauds agar poured into petri dishes up to a depth of 4mm is cut using sterile scalpel blade. The block is transferred to the surface of the glass slide. The agar block is inoculated at four sides using the fungal strain to be identified. The inoculated block is covered with sterile cover slip and incubated at 250 C. A little sterile distilled water is added on the filter paper to avoid drying of agar. When growth appears, a drop of LCB is

placed on a slide and cover slip from block is placed over it. Likewise drop of stain is placed on glass slide of the slide culture after removing agar block; fresh cover slip is applied over it and is examined under the microscope.[38,40]

c. Scotch Tape Technique

A 4 cm strip of scotch tape No. 800 is looped back on itself with the adhesive side out and held between the thumb and index finger. The adhesive side is pressed firmly to the surface of the fungal colony. It is gently pulled and is placed in a small drop of LCB on a microscopic slide.

Physiological tests

1. In vitro hair perforation test

This is performed to differentiate between *T.mentagrophytes* and *T.rubrum* as well as *M.canis* and *M.equinum*. This test is taken positive when dermatophyte species show wedge shaped perforation in hair. It is positive in *T.mentagrophytes* and *M.canis*.

2. Urease Test

Is done on Christensen's medium. *T.mentagrophytes* hydrolyze urea thereby turns medium red while *T.rubrum* shows negative result.

3. Special nutritional requirements

The method employs a casamino acids basal medium i.e., vitamin free [Trichophyton agar, (T1)] and to which various vitamins are added i.e., inositol (T2), thiamine + inositol (T3), thiamine (T4), and nicotinic acid (T5). In addition, the

series includes an ammonium nitrate basal medium (T6) to which histidine is added (T7). After inoculation, incubated at room temperature or 37^0C (if *T.verrucosum* is suspected) and read after 7 and 14 days. The amount of growth is graded from 0-4+.

4. Growth on Polished Rice Grains

This is a useful test for differentiating *M.audouinii* from *M.canis* and from other dermatophytes that typically grow and sporulate on rice grains.

5. Temperature tolerance and temperance enhancement

Used for distinguishing *T.mentagrophytes* complex from *T.terrestre*, *T.mentagrophytes* from *M.persicolor* and *T.verrucosum* from *T.schoenleinii*. At 37^0 C, members of *T.mentagrophytes* complex show good result whereas *T.terrestre* does not grow and *M.persicolor* generally grows poorly or not at all; growth of *T.verrucosum* and *T.soudanense* is enhanced but that of *T.schoenleinii* and *M.ferruginum* is not.

6. Hair bait technique

This technique is used for the isolation of *geophilic* species like *M.gypseum* from soil.

Immunodiagnosis[38]

- Skin tests with dermatophyte antigen trichophytin are used for the diagnosis of dermatophytosis.
- Trichophytin is a crude extract from dermatophytes producing positive delayed type hypersensitivity in most of the adults.

- The patients without delayed type reaction are more susceptible to chronic dermatophytosis.

Serological tests

Various serological tests like immunodiffusion are done to establish the diagnosis of dermatophytosis.

Animal pathogenicity

It is done on guinea pigs. *M.canis, M.gypseum* and *T.mentagrophytes* may be established more readily in laboratory animals as compared to other species. It is done for studying nature of lesions and immunity produced by the organism.

Molecular Identification Techniques

Methods used are largely based on arbitrarily primed PCR or restriction fragment length polymorphism generated from PCR products. An oligonucleotide probe for *T.rubrum* has been developed. Nested PCR targeting Chitin Synthase I (CHSI) gene in skin and hair specimen of patients clinically suspected with dermatophytosis is used.[41]

METHODOLOGY

The present study of dermatophytosis was carried out in the Department of Dermatology Venereology and Leprosy , in Dr. Prabhakar Kore's, KLE Hospital and Medical Research Centre, Belgaum over a period of one year from January 2013 to December 2013.The study group comprised of 125 patients of clinically diagnosed superficial dermatophytosis attending the outpatient Department of Dermatology and Venerology.

Inclusion criteria

All new cases of dermatophytosis presenting to the Department of dermatology.

Exclusion Criteria

Patient who are on antifungal treatment.

Sample size

Considering the formula $n = (z^2 \times p \times q)\ d^2$ where, p is prevalence; q is 100-p; d is absolute error i.e 15 % total error of p; z for 95 % confidence is 1.96;

$= (1.96)^2 pq/d^2$

Where, p = 58.33%; q = 100-58.33 = 41.67; d=absolute error=15% of p= 8.749;

$n=(1.96)^2 \times 58.33 \times 41.67/(8.749X8.749)$

$=121.99 \sim 125$

Therefore sample size = 125

Sample size has been estimated with 95% confidence interval and 15% tolerable error

Ethical clearance

Prior to the commencement, the ethical clearance was obtained from Institutional Ethics Committee, Jawaharlal Nehru Medical College, Belgaum

Method of collection of data

Informed consent

Patients who fulfilled the selection criteria were briefed about the nature of study and a written informed consent was obtained (Annexure I).

History

A detailed history was taken regarding duration and progress of lesion in past and family history, age, sex, occupation etc in predesigned proforma (Annexure II).

Examination

A complete dermatological examination was done to assess type of lesion, morphology and distribution was done along with general physical examination. Relevant systemic examination was done and the findings were recorded.

Specimen Collection

From Skin

The affected area was first thoroughly cleaned with 70% alcohol to remove surface contaminants. After the alcohol dries, skin scraping was collected from the border of the active lesion using a blunt sterile scalpel, moving the scalpel perpendicular to the skin surface. Specimen was collected on sterile paper envelope.

From Nail

The affected nail was cleaned with 70% alcohol. The nail clipping was collected on sterile paper envelope.

From Scalp

Hair from scalp was epilated with a flame sterilized forcep and the active border area was scraped with a scalpel to collect epidermal scales on sterile paper envelope.

Direct Examination

Potassium Hydroxide (KOH) Preparation

The sample is placed on a clean sterilized glass slide and few drops of 10% KOH and 20% KOH for nail sample is added, then cover slip is placed. The slide was gently heated passing over flame. After 15-20 minutes, the specimen was examined first under low power microscope and then under high power with low condenser to look for presence of hyphae or arthrospores.

Culture

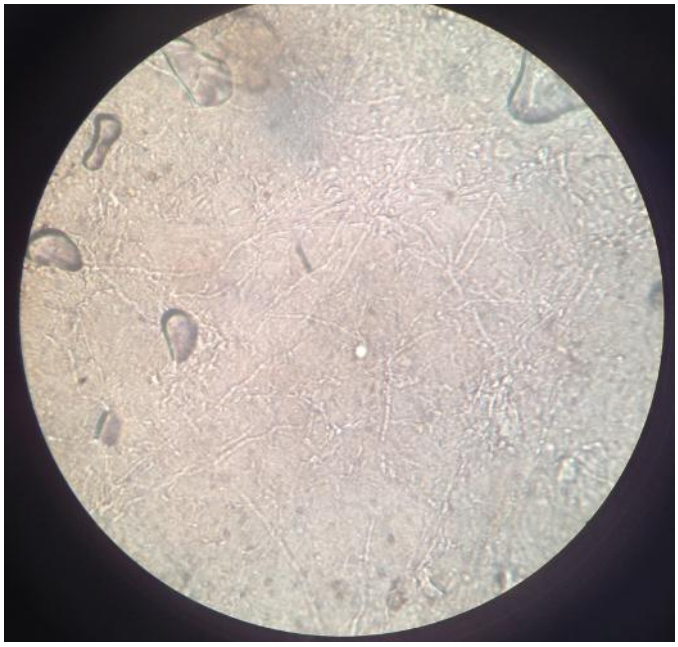
The specimen collected were inoculated on to sabourauds Dextrose Agar containing Chloramphenicol (50mg/l) and cycloheximide (500mg/l); irrespective of demonstration of fungal elements on KOH mount. Each sample was inoculated into pair of tubes. One tube with antibiotic and other without antibiotic and were incubated at 27^0C. The cultures were examined daily for a period of 4weeks.Slopes showing no growth for 4weeks were discarded. If growth was obtained on Sabourauds dextrose agar, identification was made on colony morphology, microscopic appearance.

Macroscopic Examination Of Culture

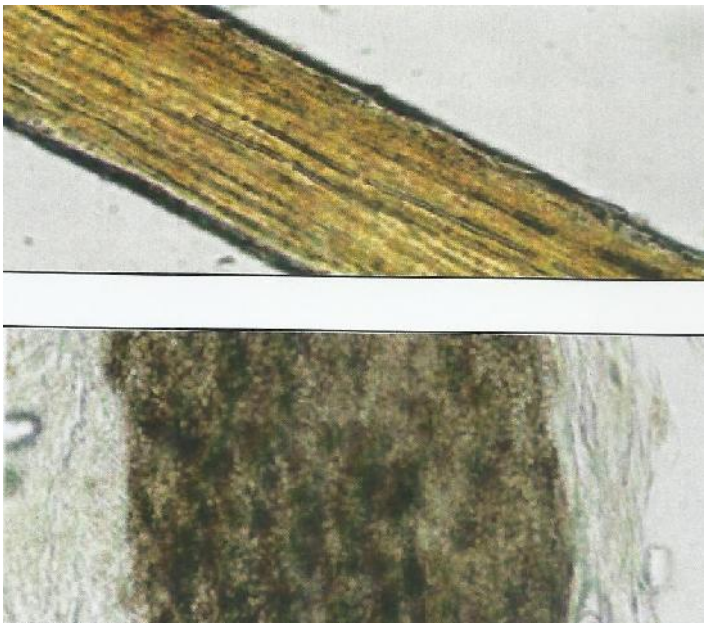
The growth on Sabourauds dextrose agar was observed to study the colony morphology, colour of the surface, texture of the surface, topography and rate of growth.

Microscopic Examination

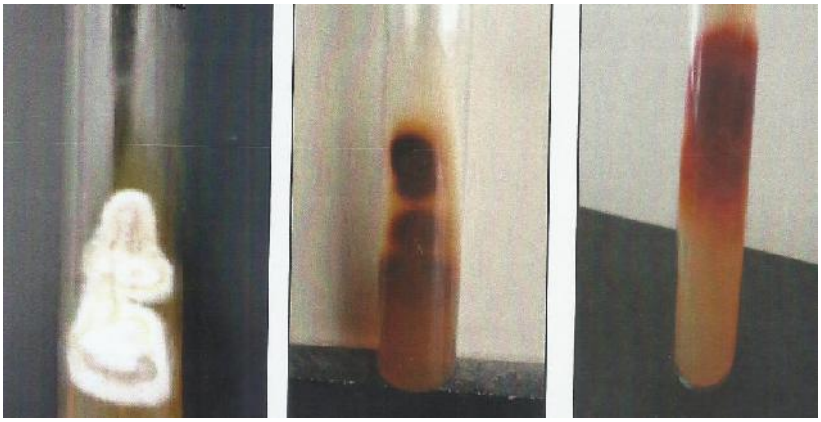
Tease Mount: For preparing a mount, a portion of fungal fragment was removed and was teased on a glass slide in a drop of Lactophenol Cotton Blue stain. A cover slip was placed and examined under microscope.



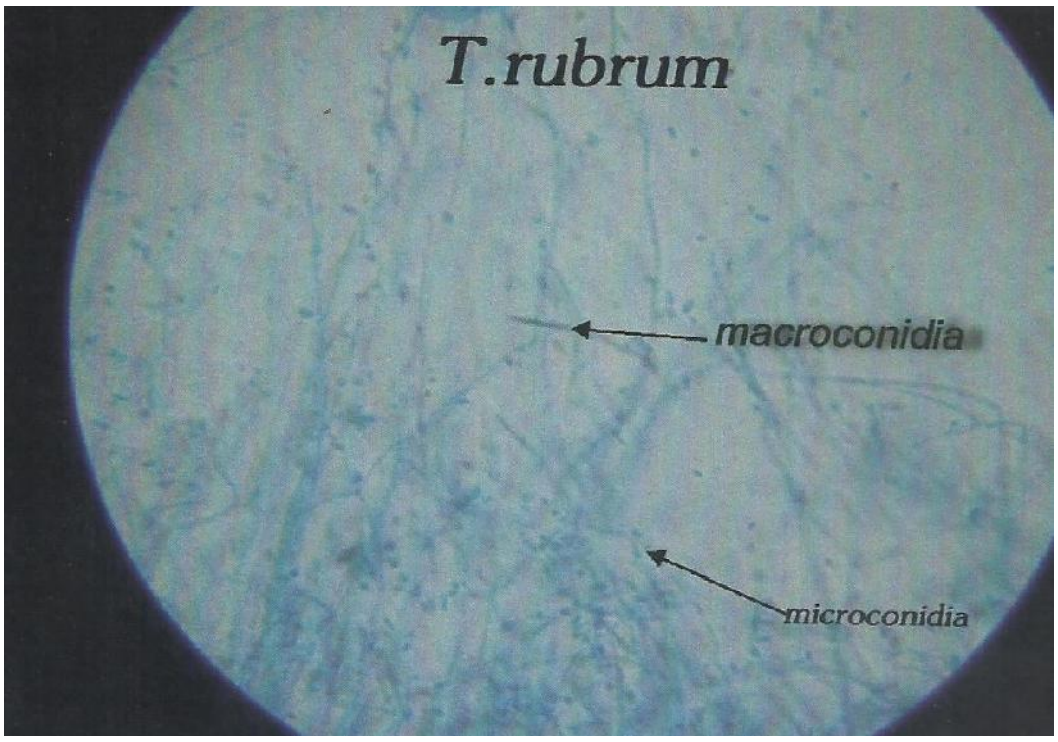
Photograph 1. KOH preparation



Photograph 2. KOH preparation for hair shaft

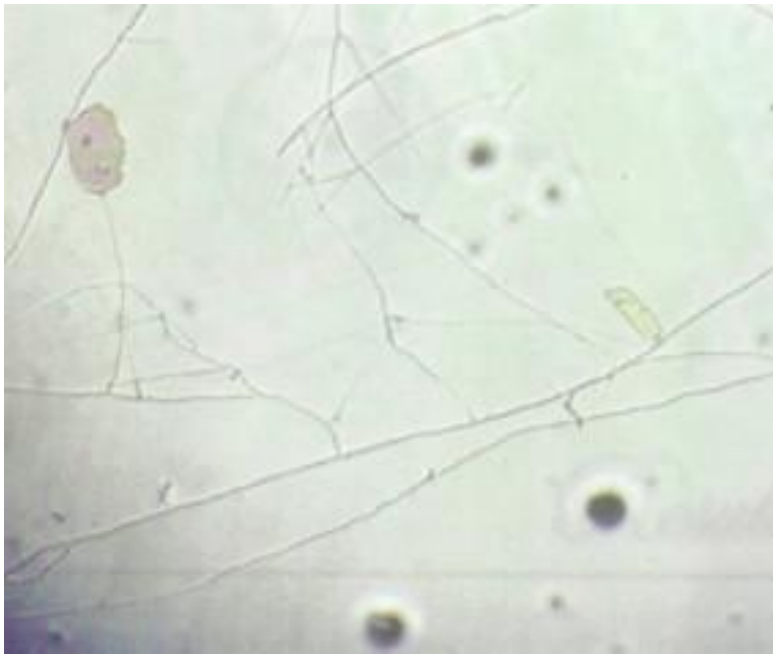


Photograph 3. *T. Rubrum*

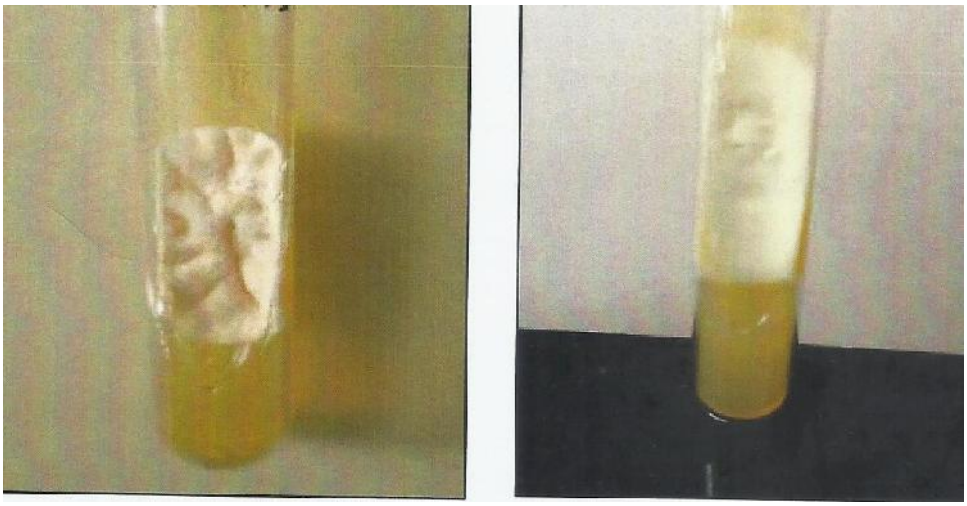


Photograph 4. LPCB preparation of *T. Rubrum*

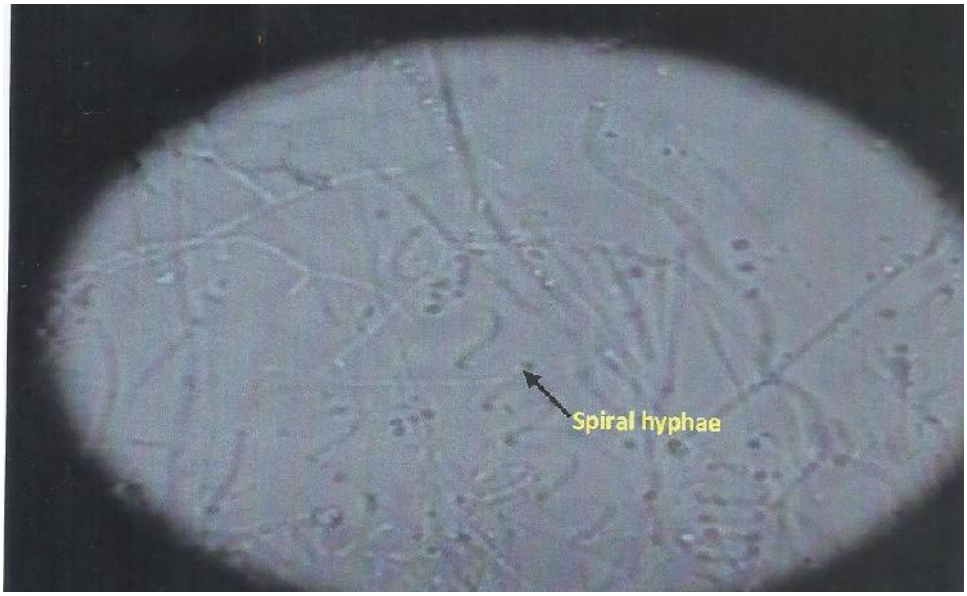
Photograph 5. *T. Soudanese* colony in SDA



Photograph 6. Reflexive branching seen in *T. Soudanese*

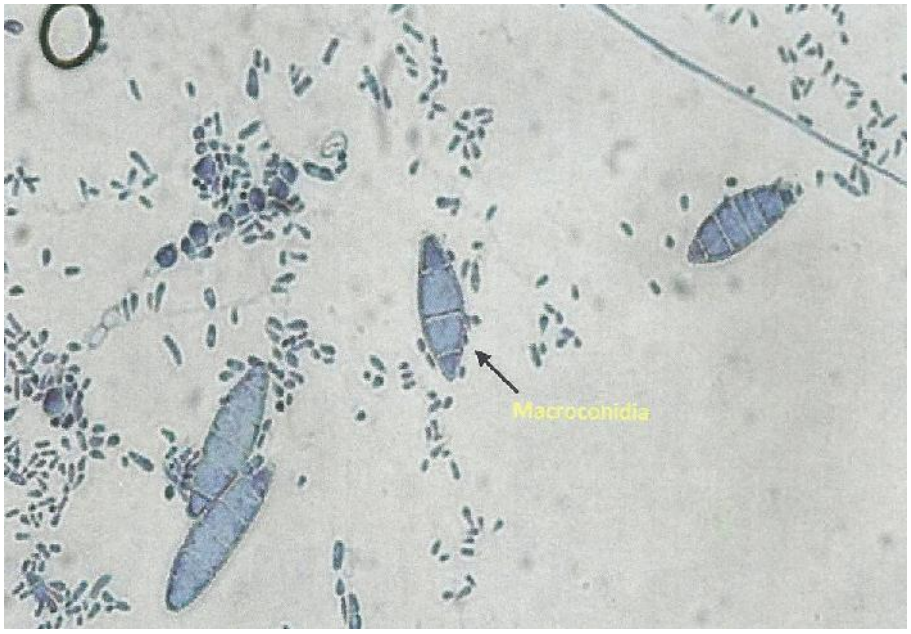


Photograph 7. *T. Mentagrophytes*



Photograph 8. LPCB preparation of *T. Mentagrophytes*

Photograph 9. *M. Gypseum*



Photograph 10. LPCB preparation of *M. Gypseum*

Statistical analysis

The data obtained was coded into Microsoft excel spreadsheet (Annexure III). The continuous data was expressed as mean ± standard deviation (SD). Categorical data was expressed in terms of rates, ratios and percentages and comparison was done using chi-square test. A probability value ('p' value) of less than or equal to 0.050 was considered as statistically significant.

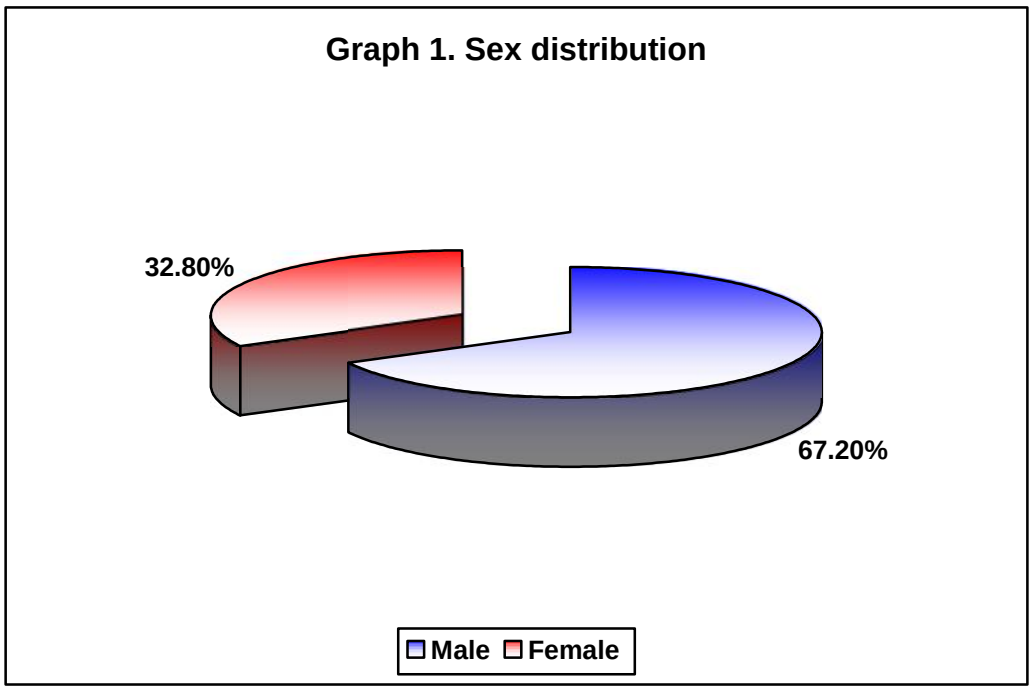
RESULTS

The present one year cross sectional study was conducted in the Department of Dermatology, Venereology and Leprosy, KLES Dr. Prabhakar Kore Hospital and Medical Research Centre attached to Jawaharlal Nehru Medical College, Belgaum during the period of January 2013 to December 2013.

The study comprised of 125 patients presenting with dermatophytosis. The data obtained was coded and entered into Microsoft excel spreadsheet and analysed. The results and observations are as below.

Table 1. Sex distribution

Sex	Distribution (n=125)	
	Number	**Percentage**
Male	84	67.20
Female	41	32.80
Total	**125**	**100.00**



In the present study 67.2% of the patients were males and 32.8% were females. The male to female ratio was 2:1.

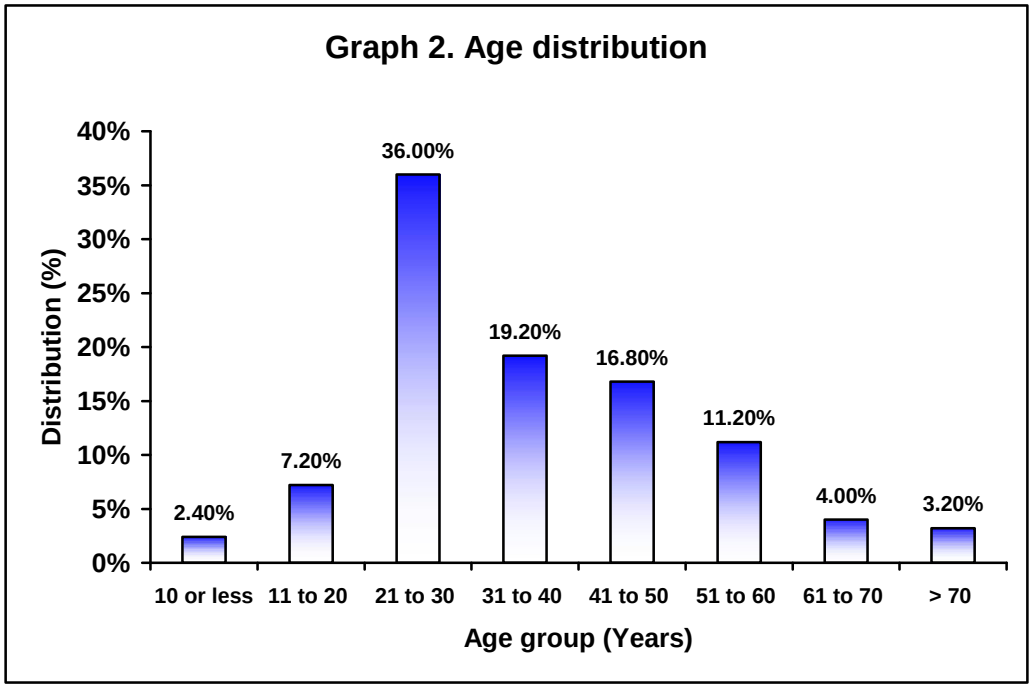
Table 2. Distribution of clinical types of dermatophysis in males and females

Clinical types of dermatophytosis	Sex			
	Male (n=84)		Female (n=41)	
	Number	Percentage	Number	Percentage
T. corporis	41	48.81	24	58.54
T cruris	41	48.81	13	31.71
T. pedis	9	10.71	3	7.32
T. unguim	3	3.57	6	14.63
T. capitis	2	2.38	1	2.44
T. mannum	2	2.38	0	0.00
T. barbae	1	1.19	0	0.00
T. faciei	1	1.19	1	2.44

In this study the commonest clinical type of dermatophytosis among males was T. corporis and T. cruris noted in 48.81% of the males each. In females the same dermatophytes were present among 58.54% and 31.71% respectively.

Table 3. Age distribution

Age group (Years)	Distribution (n=125)	
	Number	Percentage
10 or less	3	2.40
11 to 20	9	7.20
21 to 30	45	36.00
31 to 40	24	19.20
41 to 50	21	16.80
51 to 60	14	11.20
61 to 70	5	4.00
> 70	4	3.20
Total	**125**	**100.00**



In the present study the commonest age group was 21 to 30 years comprised of 36% of the patients. The distribution of other age groups is as shown in Table 3 and Graph 2.

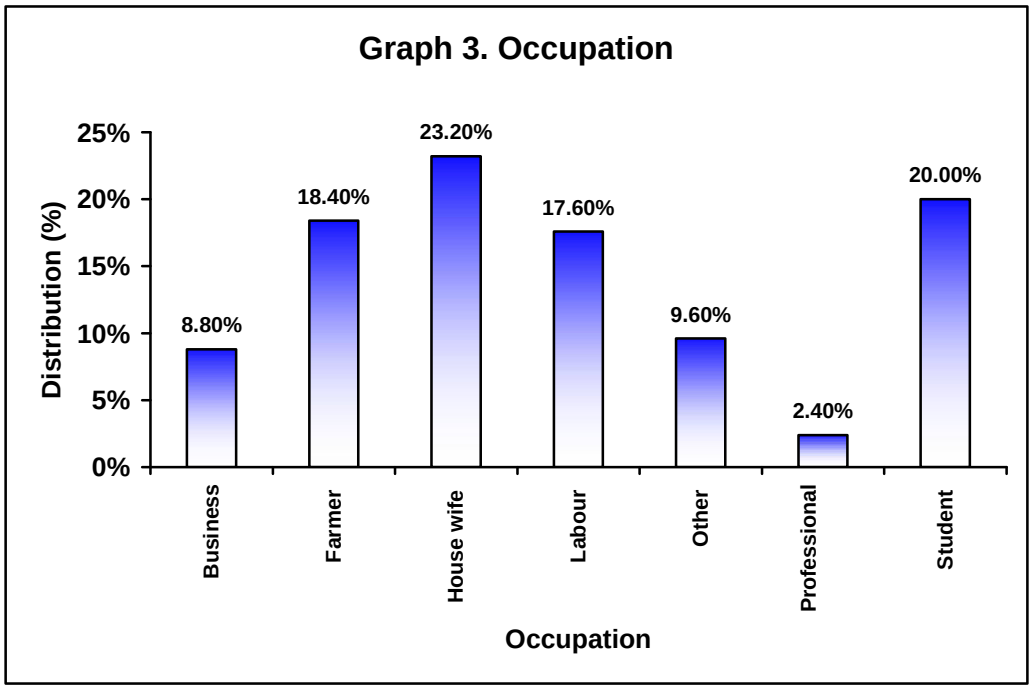
Table 4. Clinical diagnosis and age

Clinical types of dermatophytosis	Age group (Years)															
	10 or less (n=3)		11 to 20 (n=9)		21 to 30 (n=45)		31 to 40 (n=24)		41 to 50 (n=21)		51 to 60 (n=14)		61 to 70 (n=5)		>70 (n=4)	
	No	%	No	%	No	%	No	%	No	%	No	%	No	%	No	%
T. unguim	0	0	1	11	2	4	1	4	3	14	2	14	0	0	0	0
T. capitis	1	33	0	0	2	4	0	0	0	0	0	0	0	0	0	0
T. corporis	2	67	3	33	25	56	10	42	10	48	8	57	3	60	4	100
T. pedis	0	0	0	0	3	7	5	21	1	5	3	21	0	0	0	0
T cruris	0	0	4	44	8	18	13	54	12	57	3	21	4	80	0	0
T. barbae	0	0	0	0	0	0	0	0	1	5	0	0	0	0	0	0
T. mannum	0	0	0	0	2	4	0	0	0	0	0	0	0	0	0	0
T. faciei	0	0	1	11	0	0	0	0	0	0	1	7	0	0	0	0

In the present study commonest age group was 21 to 30 years comprised of 45 (36%) patients. Of these, 25 (56%) had T. corporis and 8 (18%) had T. cruris. Among the others 2 each (4%) had T. unguim, T. capitis and T. mannum. T. pedis was present in 3 (7%) cases.

Table 5. Occupation

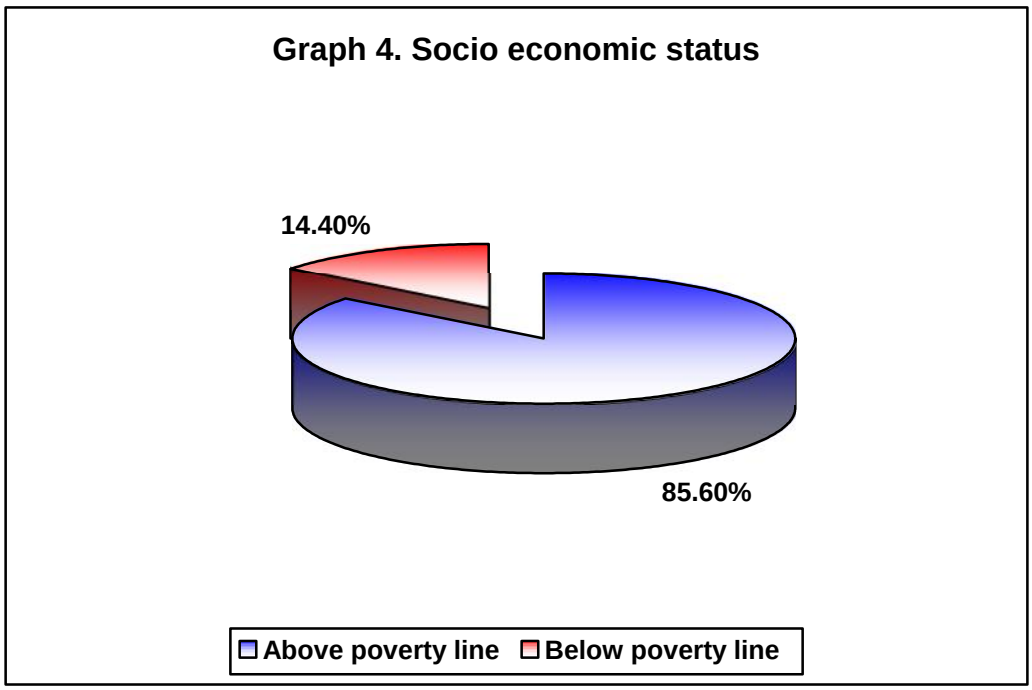
Occupation	Distribution (n=125)	
	Number	**Percentage**
Business	11	8.80
Farmer	23	18.40
House wife	29	23.20
Labour	22	17.60
Other	12	9.60
Professional	3	2.40
Student	25	20.00
Total	**125**	**100.00**



In the present study most of the patients (23.2%) were housewives followed students (20%). The occupation of other patients is as shown in table 5 and graph 3.

Table 6. Socio economic status

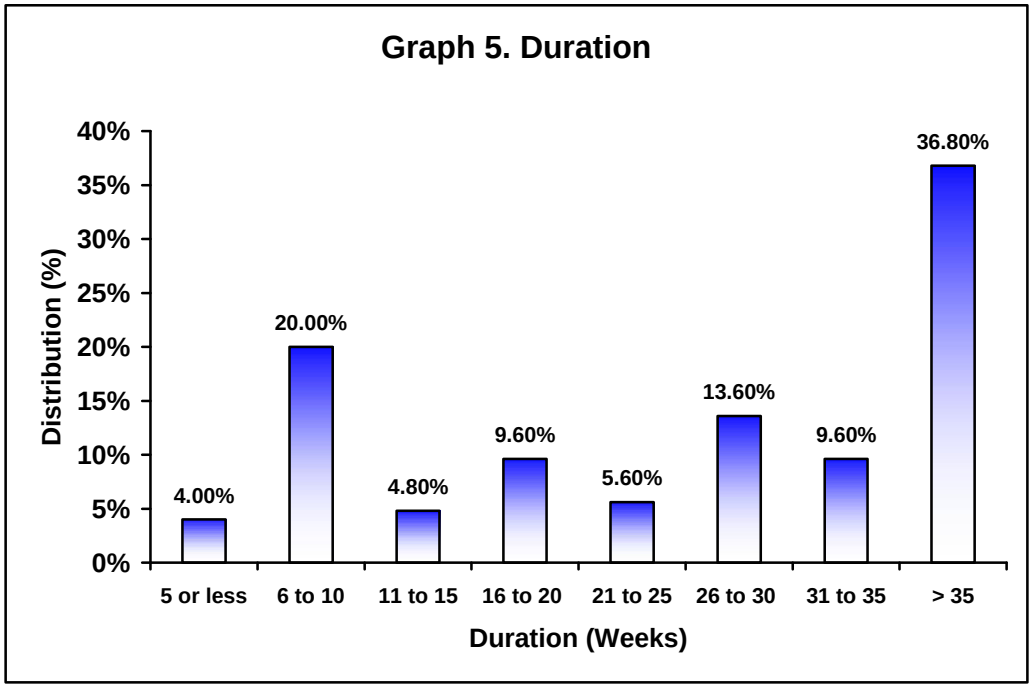
Socio economic status	Distribution (n=125)	
	Number	**Percentage**
Above poverty line	107	85.60
Below poverty line	18	14.40
Total	**125**	**100.00**



In this study majority of the patients were above poverty line (85.6%)

Table 7. Duration

Duration (weeks)	Distribution (n=125)	
	Number	**Percentage**
5 or less	5	4.00
6 to 10	25	20.00
11 to 15	6	4.80
16 to 20	12	9.60
21 to 25	7	5.60
26 to 30	17	13.60
31 to 35	12	9.60
> 35	46	36.80
Total	**130**	**104.00**



In the present study 36.8% of the patients reported duration of more than 35 weeks.

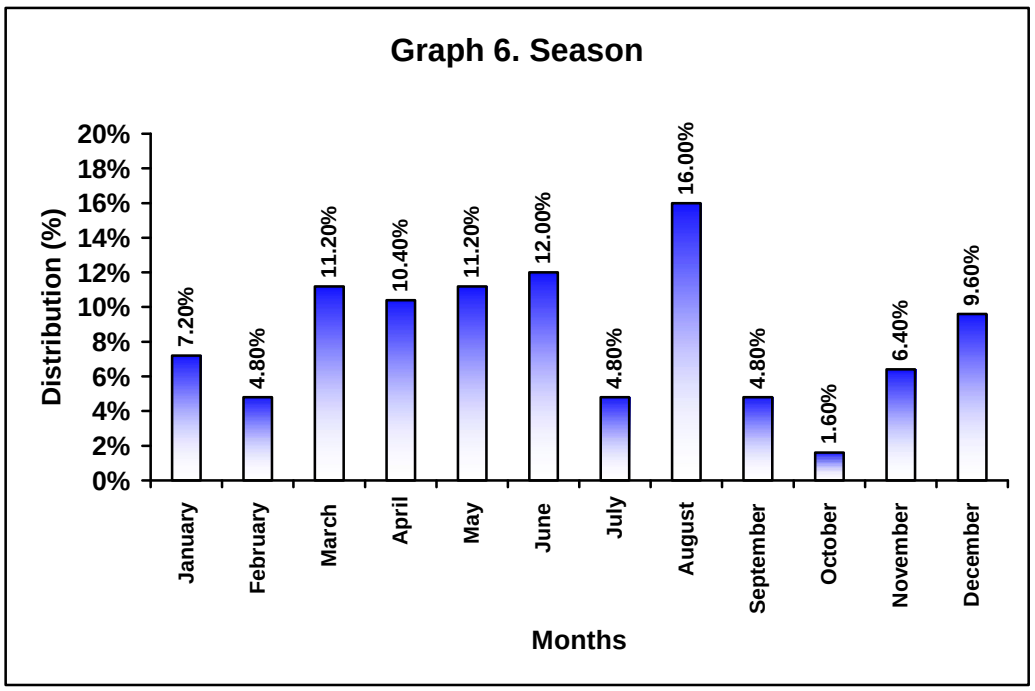
Table 8. Duration of disease and sex

Duration (Weeks)	Sex			
	Male (n=84)		Female (n=41)	
	Number	Percentage	Number	Percentage
5 or less	4	4.76	1	2.44
6 to 10	17	20.24	8	19.51
11 to 15	5	5.95	1	2.44
16 to 20	9	10.71	3	7.32
21 to 25	2	2.38	0	0.00
26 to 30	14	16.67	3	7.32
31 to 35	9	10.71	3	7.32
> 35	24	28.57	22	53.66

In this study most of the males (28.57%) and females (53.66%) reported duration of more than 35 weeks.

Table 9. Season

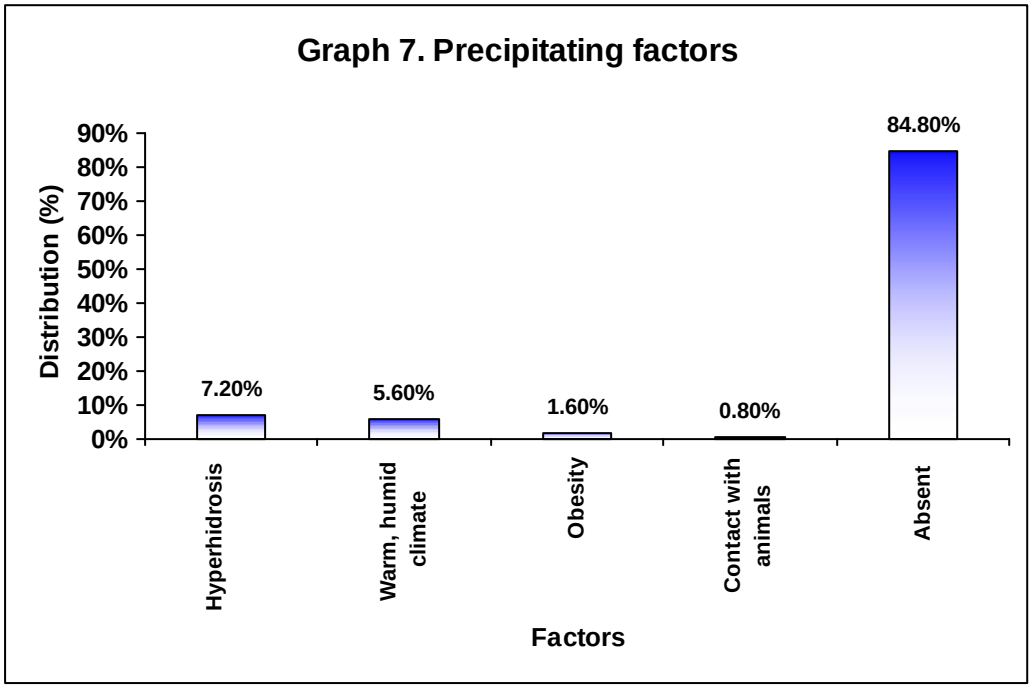
Month	Distribution (n=125)	
	Number	**Percentage**
January	9	7.20
February	6	4.80
March	14	11.20
April	13	10.40
May	14	11.20
June	15	12.00
July	6	4.80
August	20	16.00
September	6	4.80
October	2	1.60
November	8	6.40
December	12	9.60
Total	**125**	**100.00**



In this study maximum cases were noted in the month of August (16%).

Table 10. Precipitating factors

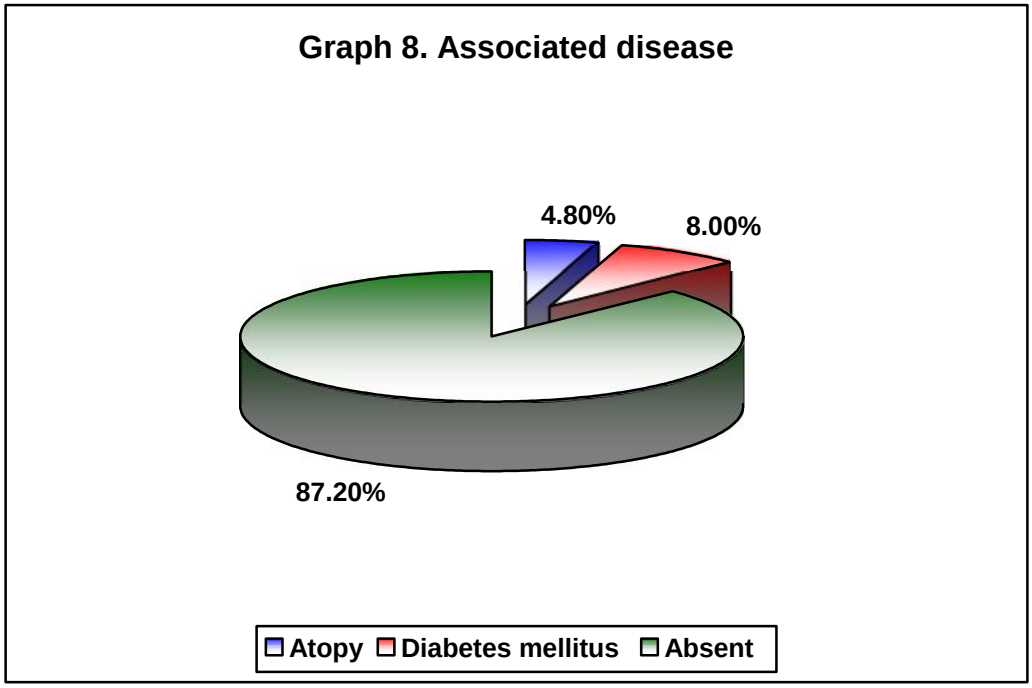
Findings	Distribution (n=125)	
	Number	**Percentage**
Hyperhidrosis	9	7.20
Warm, humid climate	7	5.60
Obesity	2	1.60
Contact with animals	1	0.80
Absent	106	84.80
Total	**125**	**100.00**



In the present study 84.8% of the patient did not report any precipitating factors. In the remaining, hyperhidrosis was the commonest precipitating factor noted in 7.2% cases.

Table 11. Associated disease

History	Distribution (n=125)	
	Number	**Percentage**
Atopy	6	4.80
Diabetes mellitus	10	8.00
Absent	109	87.20
Total	**125**	**100.00**

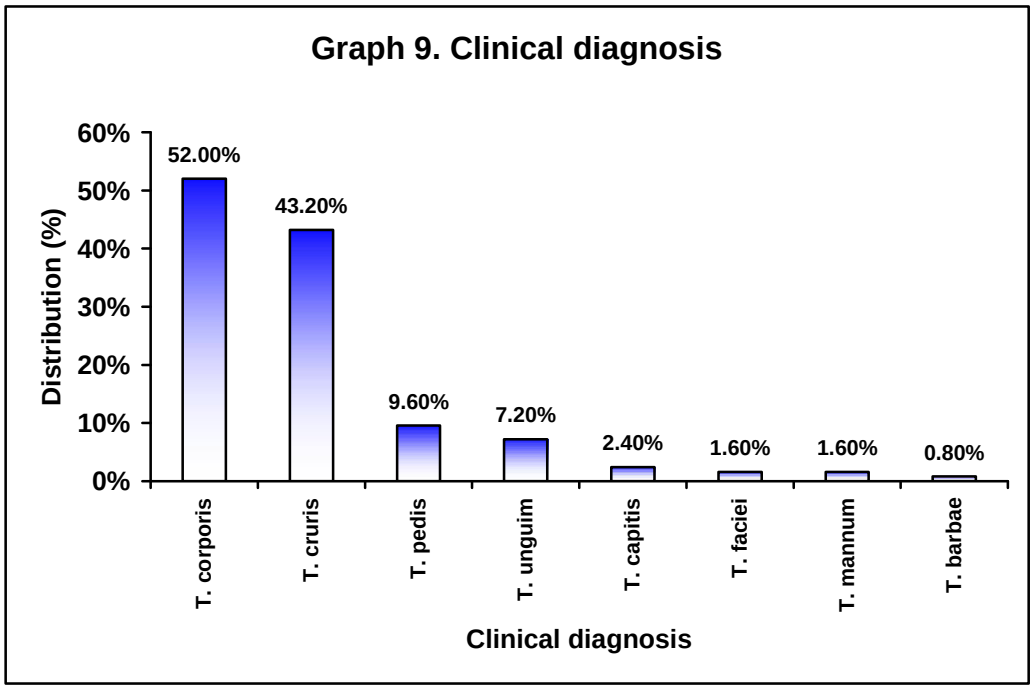


In the present study diabetes mellitus was the associated disease in 8% of the patients and atopy in 4.8%. However, 87.2% of the patients reported no associated disease.

Table 12. Clinical diagnosis

Type	Distribution (n=125)	
	Number	Percentage
T. corporis	65	52.00
T cruris	54	43.20
T. pedis	12	9.60
T. unguim	9	7.20
T. capitis	3	2.40
T. faciei	2	1.60
T. mannum	2	1.60
T. barbae	1	0.80

Multiple presentations hence total not shown



In this study the commonest clinical diagnosis was T. corporis noted in 52% followed by T. cruris present in 43.2% of the patients.

Table 13. Multiple clinical diagnosis

Diagnosis	Distribution (n=23)	
	Number	**Percentage**
T. cruris and T. corporis	17	73.91
T. corporis and T. pedis	3	13.04
Tinea unguim and T. corporis	1	4.35
T. cruris, T. pedis	1	4.35
T. cruris and T. barbae	1	4.35
Total	**23**	**100.00**

In the present study, multiple diagnosis were noted in 23 cases. Among these, T. cruris and T. corporis was noted in 17 (73.91%) patients and T. corporis and T. pedis in three (13.04%) cases.

Table 14. Morphological variants

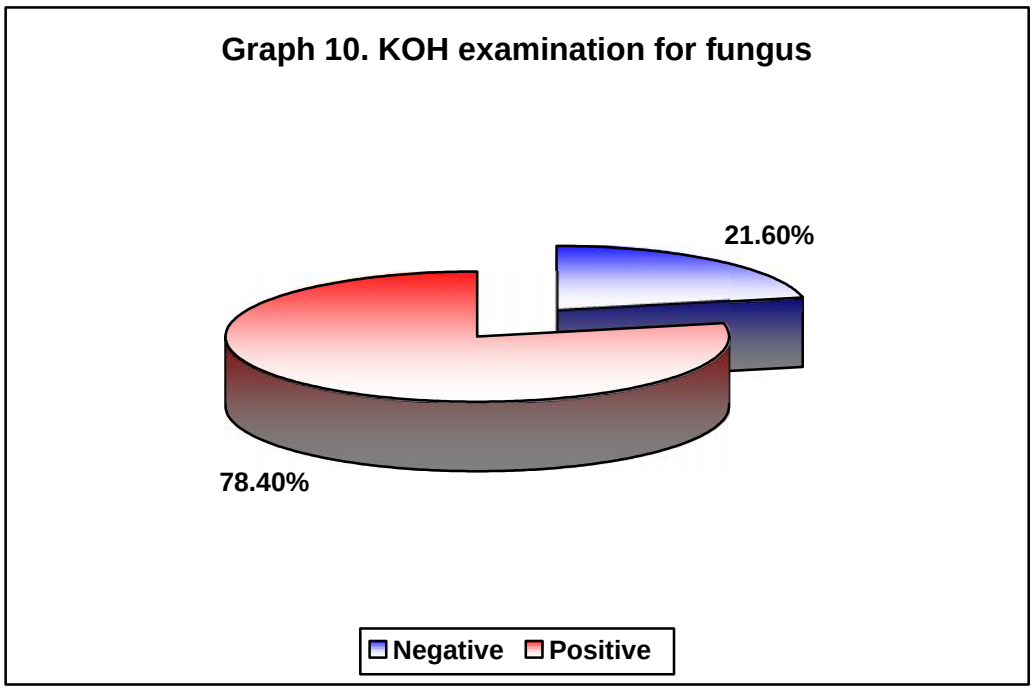
Variants	Distribution (n=125)	
	Number	**Percentage**
Annular	47	37.60
Plaque	24	19.20
Papulosqamous	24	19.20
Eczematous	14	11.20
Vesiculo pustular	9	7.20
Chronic Hyperkeratotic	8	6.40
Distal subungual onychomycosis	7	5.60
Total dystrophic onychomychosis	2	1.60
Vesicular	2	1.60
Chronic intertriginous	1	0.80
Grey patch	1	0.80
Kerion	1	0.80
Moccasin	1	0.80
Black dot	1	0.80

Multiple presentations hence total not shown

In this study the commonest morphological variant was noted as annular (37.6%) followed by plaque and papulosquamous (19.2% each). The other morphological variants are as shown in table 13.

Table 15. KOH examination for fungus

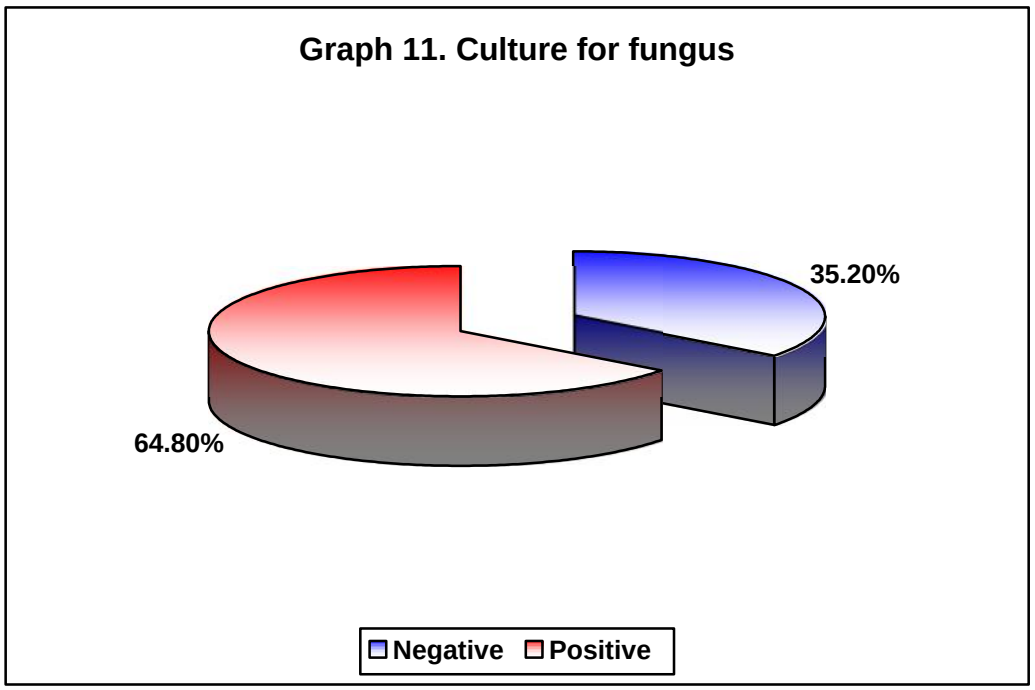
Findings	Distribution (n=125)	
	Number	**Percentage**
Positive	98	78.40
Negative	27	21.60
Total	**125**	**100.00**



In the present study KOH examination for fungus was positive in 78.4% of the cases.

Table 16. Culture for fungus

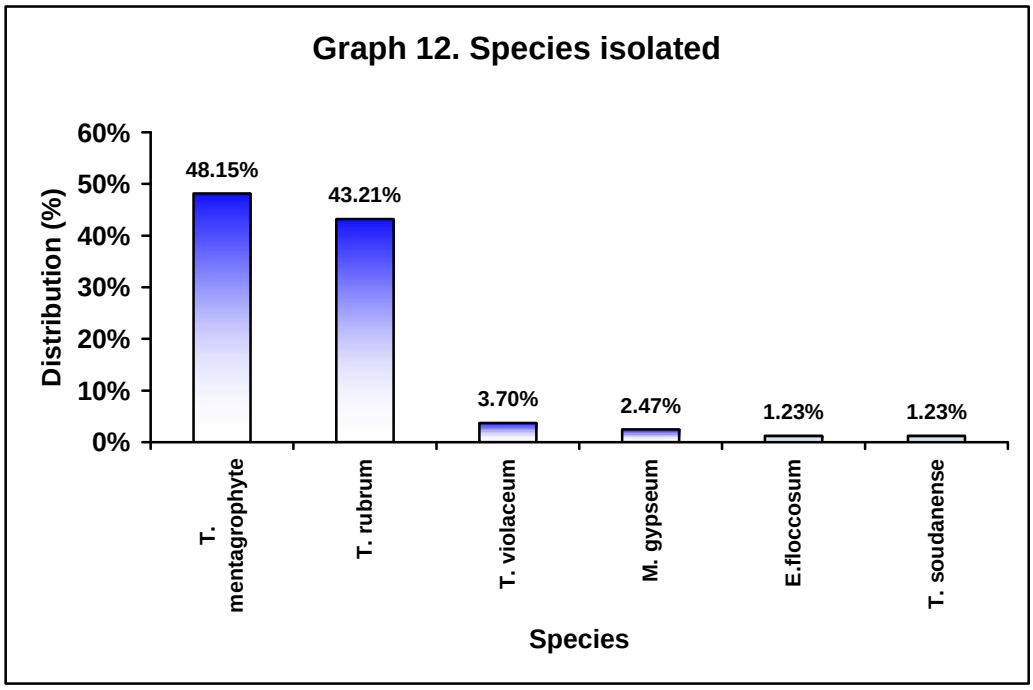
Findings	Distribution (n=125)	
	Number	**Percentage**
Positive	81	64.80
Negative	44	35.20
Total	**125**	**100.00**



In this study culture was positive for fungus in 64.8% of the cases.

Table 17. Species isolated

Species	Distribution (n=81)	
	Number	**Percentage**
T. mentagrophyte	39	48.15
T. rubrum	35	43.21
T. violaceum	3	3.70
M. gypseum	2	2.47
E. floccosum	1	1.23
T. soudanense	1	1.23
Total	**81**	**100.00**



In this study *T. mentagrophyte* was the commonest isolate noted in 48.15% of the patients followed by *T. rubrum* (43.21%).

Table 18. KOH and culture

KOH	Culture				Total	
	Positive		Negative			
	No	%	No	%	No	%
Positive	81	82.65	17	17.35	98	100.00
Negative	0	0.00	27	100.00	27	100.00
Total	**81**	**64.80**	**44**	**35.20**	**125**	**100.00**

p<0.001

In this study KOH examination for fungus was positive in 98 cases. Of these 81 (82.65%) cases had positive culture for fungi and 17 (17.35%) had negative culture. This difference was statistically significant (p<0.001).

Table 19. Comparison of KOH examination for fungus in different morphological variants

Variant	KOH for fungus			
	Negative (n=27)		Positive (n=98)	
	Number	Percentage	Number	Percentage
Annular	11	40.74	36	36.73
Plaque	5	18.52	19	19.39
Papulosqamous	5	18.52	19	19.39
Eczematous	1	3.70	13	13.27
Vesiculo pustular	4	14.81	5	5.10
Chronic Hyperkeratotic	2	7.41	6	6.12
Distal subungual onychomycosis	3	11.11	4	4.08
Total dystrophic onychomychosis	0	0.00	2	2.04
Vesicular	0	0.00	2	2.04
Chronic intertriginous	0	0.00	1	1.02
Black dot	0	0.00	1	1.02
Kerion	0	0.00	1	1.02
Moccasin	0	0.00	1	1.02
Grey patch	0	0.00	1	1.02

In this study commonest morphological variant was noted as annular noted in 47 cases (37.6%). Of these, 36 cases (36.73%) were positive for KOH and 11 (40.74%) were negative on KOH.

Table 20. Comparison of clinical diagnosis and culture

Clinical diagnosis	Culture			
	Negative (n=44)		Positive (n=81)	
	Number	Percentage	Number	Percentage
T. corporis	21	47.73	44	54.32
T. cruris	22	50.00	32	39.51
T. pedis	4	9.09	8	9.88
T. unguim	3	6.82	6	7.41
T. capitis	1	2.27	2	2.47
T. mannum	0	0.00	2	2.47
T. faciei	0	0.00	2	2.47
T. barbae	1	2.27	0	0.00

In this study the commonest clinical diagnosis was T. corporis noted in 65 (52%) cases. Among these 44 (54.32%) had positive culture and 21 (47.73%) had negative culture.

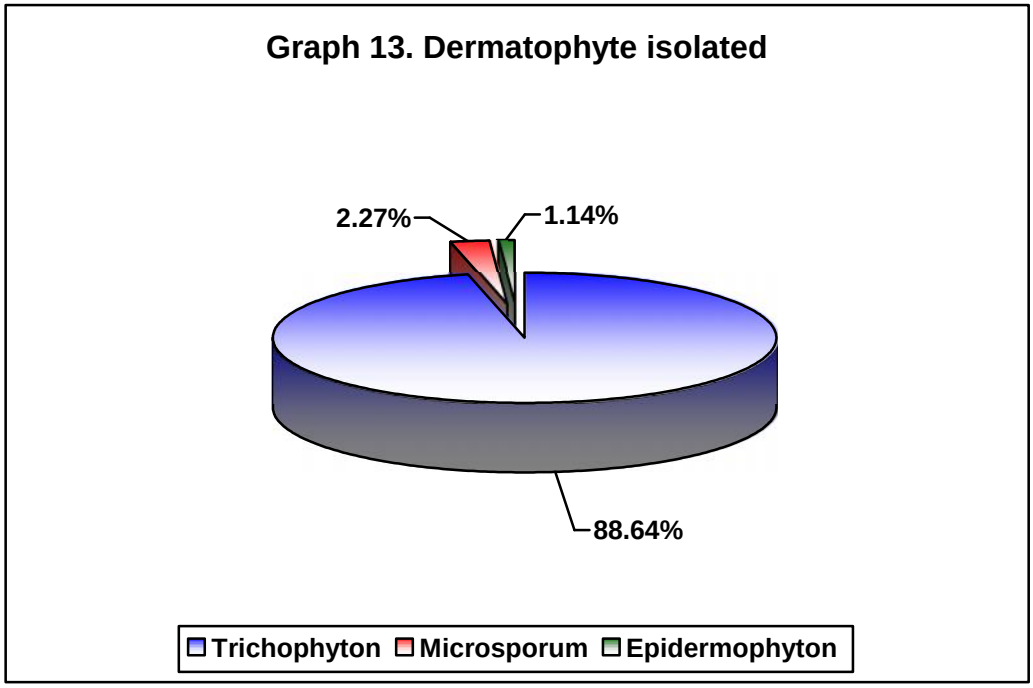
Table 21. Morphological variants in different clinical diagnosis

Morphological variants	Clinical diagnosis															
	T. unguim (n=9)		T. capitis (n=3)		T. corporis (n=65)		T. pedis (n=12)		T. cruris (n=54)		T. barbae (n=1)		T. mannum (n=2)		T. faciei (n=2)	
	No	%	No	%	No	%	No	%	No	%	No	%	No	%	No	%
Annular	0	0	0	0	45	69	0	0	13	24	0	0	0	0	2	100
Plaque	0	0	0	0	12	18	1	8	19	35	0	0	0	0	0	0
Vesiculo pustular	0	0	0	0	2	3	0	0	9	17	0	0	0	0	0	0
Black dot	0	0	1	33	0	0	0	0	0	0	0	0	0	0	0	0
Chronic Hyperkeratotic	0	0	0	0	2	3	7	58	0	0	0	0	1	50	0	0
Chronic intertriginous	0	0	0	0	0	0	1	8	0	0	0	0	0	0	0	0
Distal subungual onychomycosis	7	78	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Eczematous	0	0	0	0	14	22	1	8	4	7	0	0	0	0	0	0
Grey patch	0	0	1	33	0	0	0	0	0	0	0	0	0	0	0	0
Kerion	0	0	1	33	0	0	0	0	0	0	0	0	0	0	0	0
Moccasin	0	0	0	0	0	0	1	8	0	0	0	0	0	0	0	0
Papulosqamous	0	0	0	0	6	9	1	8	24	44	1	100	0	0	0	0
Total dystrophic onychomychosis	2	22	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Vesicular	0	0	0	0	0	0	1	8	0	0	0	0	1	50	0	0

In the present study the commonest clinical diagnosis was T. corporis noted in 65 cases (52%). Among these, annular variant was noted in 45 (69%) cases, eczematous in 14 (22%), plaque in 12 (18%), papulosquamous in 6 (9%) and chronic hyperkeratotic in 2 (3%).

Table 22. Dermatophytes isolated

Dermatophyte	Distribution (n=81)	
	Number	Percentage
Trichophyton	78	88.64
Microsporum	2	2.27
Epidermophyton	1	1.14
Total	**81**	**92.05**



In the present study the commonest dermatophyte isolated was noted as *Trichophyton* (88.64%).

Table 23. Clinical diagnosis and species isolated

Clinical diagnosis	Species											
	***E. floccosum* (n=1)**		***M. gypseum* (n=2)**		***T. mentagro-phyte* (n=39)**		***T. rubrum* (n=35)**		***T. souda-nense* (n=1)**		***T. violaceum* (n=3)**	
	No	**%**	**No**	**%**	**No**	**%**	**No**	**%**	**No**	**%**	**No**	**%**
T. unguim	0	0	0	0	5	13	1	3	0	0	0	0
T. capitis	0	0	1	50	0	0	0	0	0	0	1	33
T. corporis	1	100	1	50	22	56	18	51	1	100	1	33
T. pedis	0	0	0	0	4	10	3	9	0	0	1	33
T cruris	1	100	0	0	10	26	21	60	0	0	0	0
T. barbae	0	0	0	0	0	0	0	0	0	0	0	0
T. mannum	0	0	0	0	1	3	1	3	0	0	0	0
T. faciei	0	0	0	0	1	3	1	3	0	0	0	0

In the present study *T. mentagrophyte* was isolated in 39 cases. Among these, clinical diagnosis of T. corporis was noted in 22 (56%) cases, T. cruris in 10 (26%), T. unguim in 5 (13%), T. pedis in 4 (10%), T. mannum and T. faciei in each 1 case (3%).

Table 24. KOH and culture with clinical diagnosis

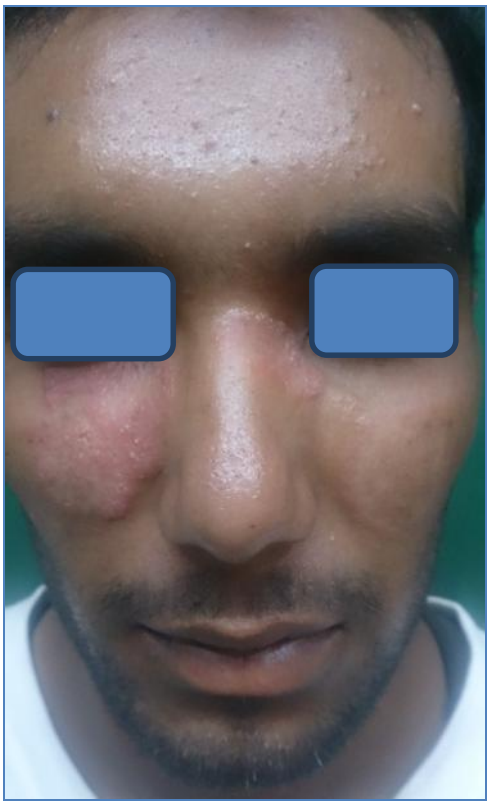
KOH	Culture	Clinical diagnosis															
		T. unguim (n=9)		T. capitis (n=3)		T. corporis (n=65)		T. pedis (n=12)		T. cruris (n=54)		T. barbae (n=1)		T. mannum (n=2)		T. faciei (n=2)	
		No	%	No	%	No	%	No	%	No	%	No	%	No	%	No	%
+	+	6	67	2	67	44	68	8	67	32	59	0	0	2	100	2	100
-	+	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
+	-	0	0	1	33	7	11	2	17	10	19	1	100	0	0	0	0
-	-	3	33	0	0	14	22	2	17	12	22	0	0	0	0	0	0

In the present study clinical diagnosis of T. corporis was noted in 65 patients. Of these, 44 (68%) had both KOH and culture positive and 14 (22%) had KOH and culture negative. However, in 7 patients (11%) KOH was positive and culture was negative.

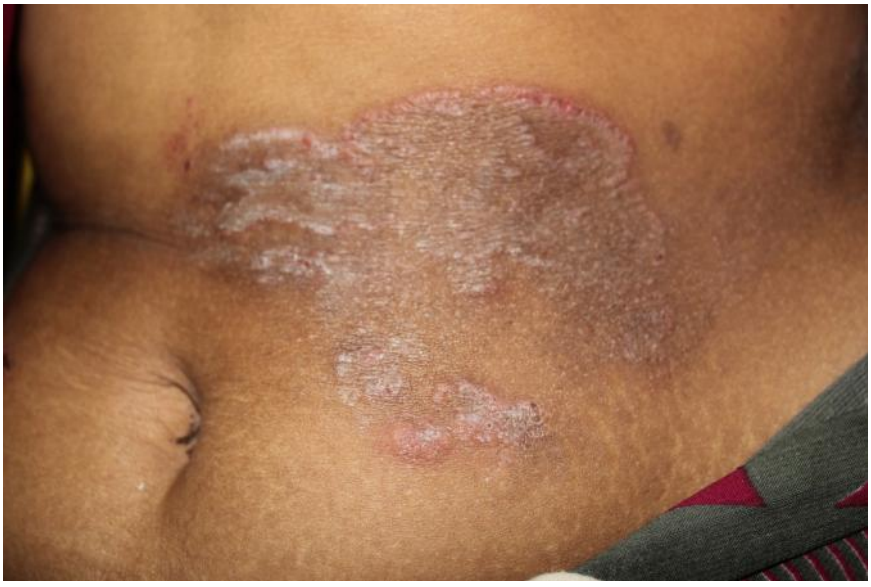
Table 25. Clinical diagnosis and KOH for fungus

Clinical diagnosis	KOH			
	Negative (n=27)		Positive (n=98)	
	Number	Percentage	Number	Percentage
T. corporis	14	51.85	51	52.04
T. cruris	12	44.44	42	42.86
T. pedis	2	7.41	10	10.20
T. unguim	3	11.11	6	6.12
T. capitis	0	0.00	3	3.06
T. mannum	0	0.00	2	2.04
T. faciei	0	0.00	2	2.04
T. barbae	0	0.00	1	1.02

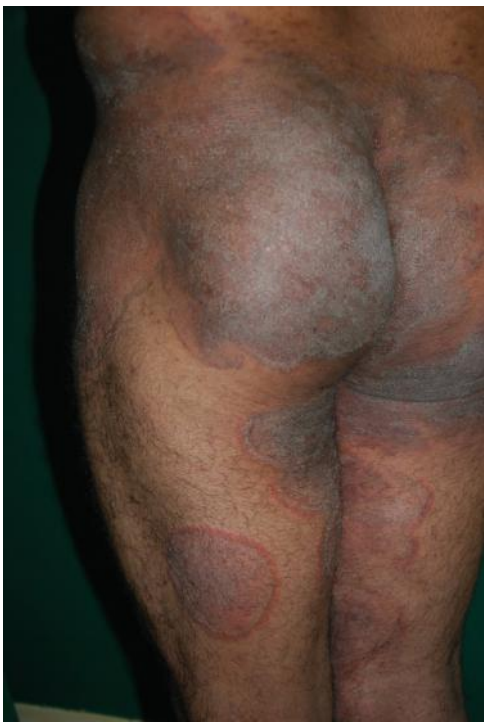
In this study Culture was positive in 98 cases. Of these, maximum (51 cases, 52.04%) were positive on T. corporis followed by T. cruris (42 cases, 42.86%) and T. pedis (10 cases, 10.20%).



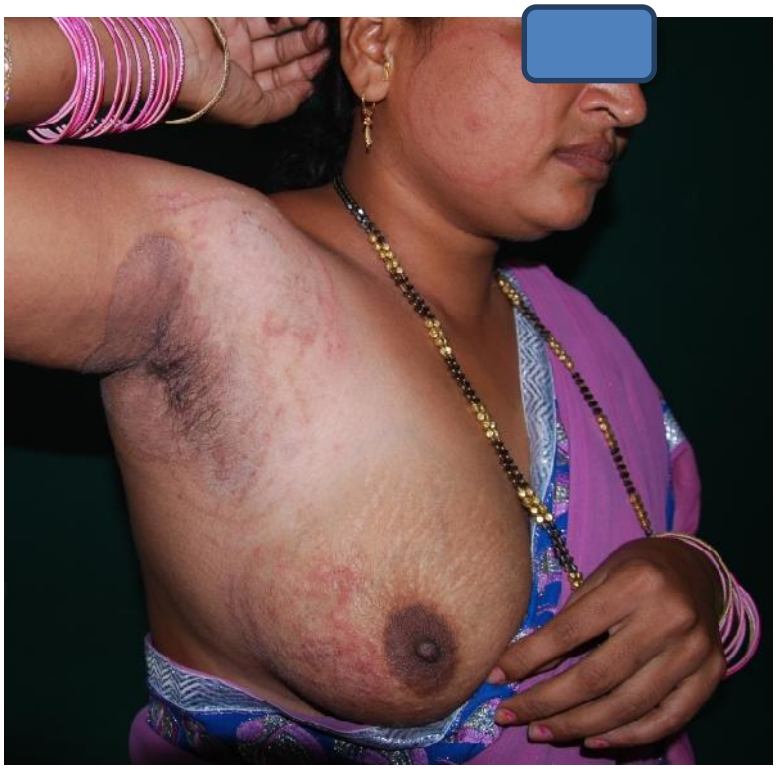
Photograph 11. Tinea faciei



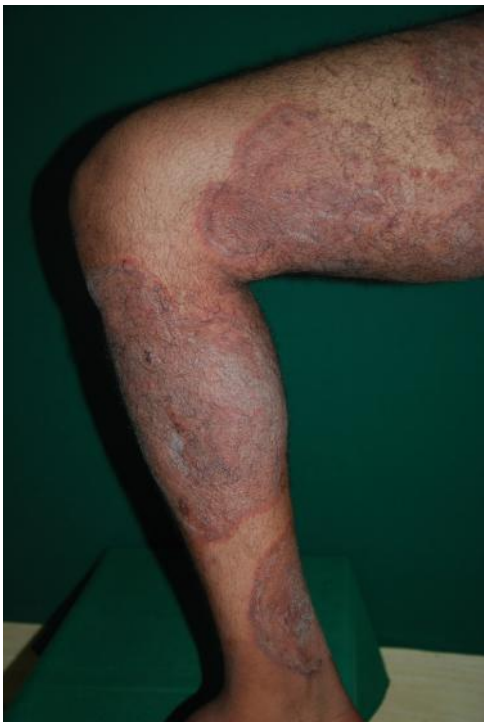
Photograph 12. Tinea corporis



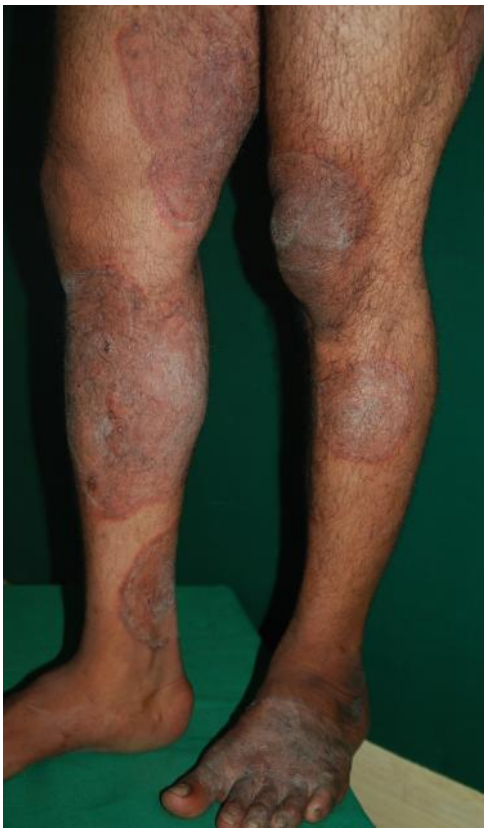
Photograph 13. Tinea corporis



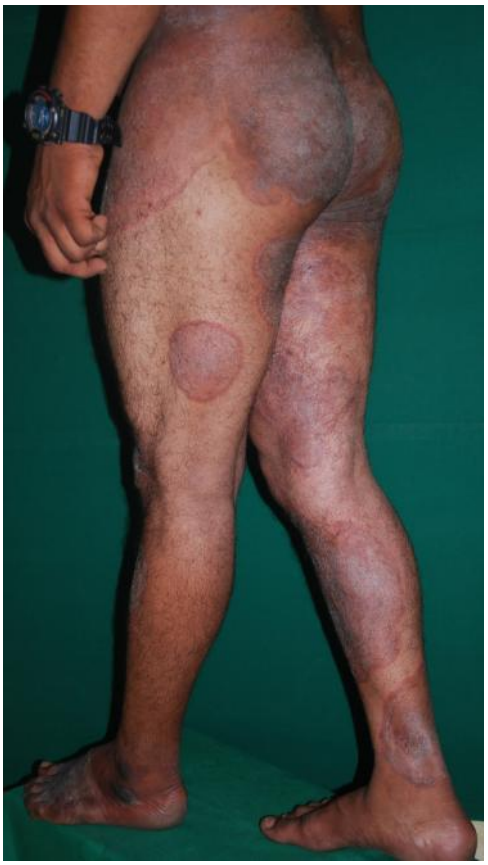
Photograph 14. Tinea corporis



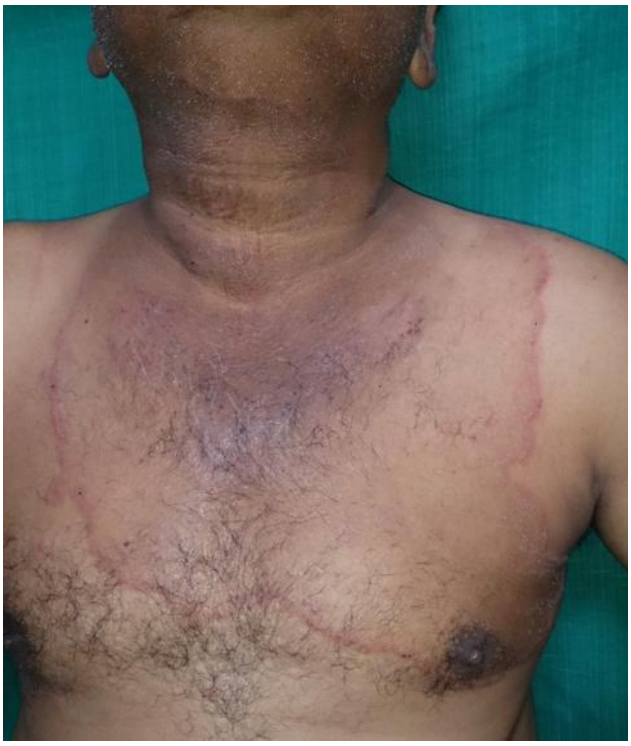
Photograph 15. Tinea corporis



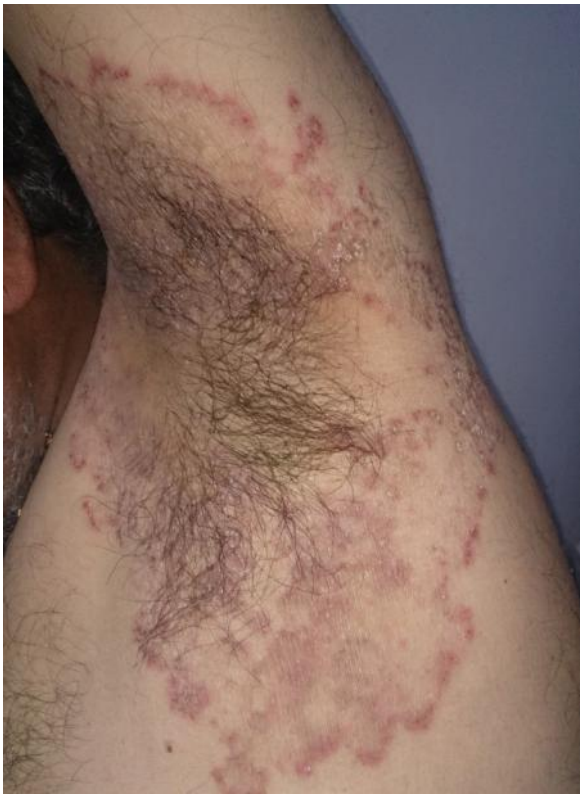
Photograph 16. Tinea corporis and tinea pedis



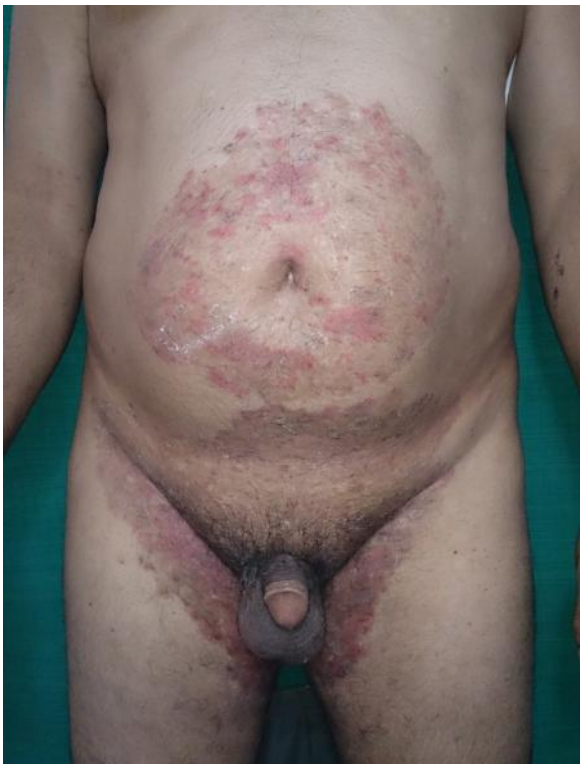
Photograph 17. Tinea corporis



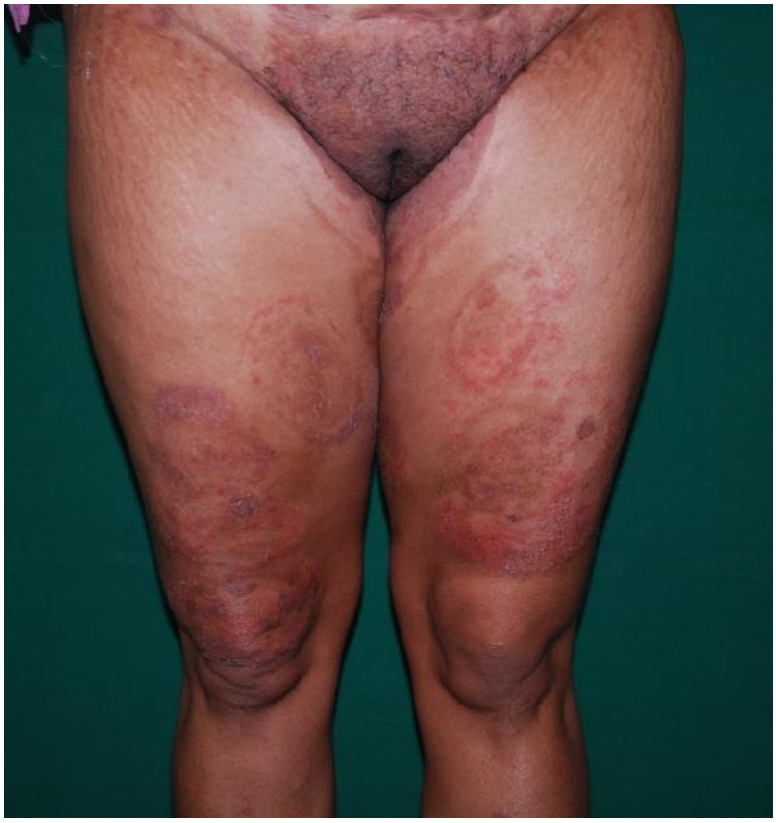
Photograph 18. Tinea corporis



Photograph 19. Tinea corporis

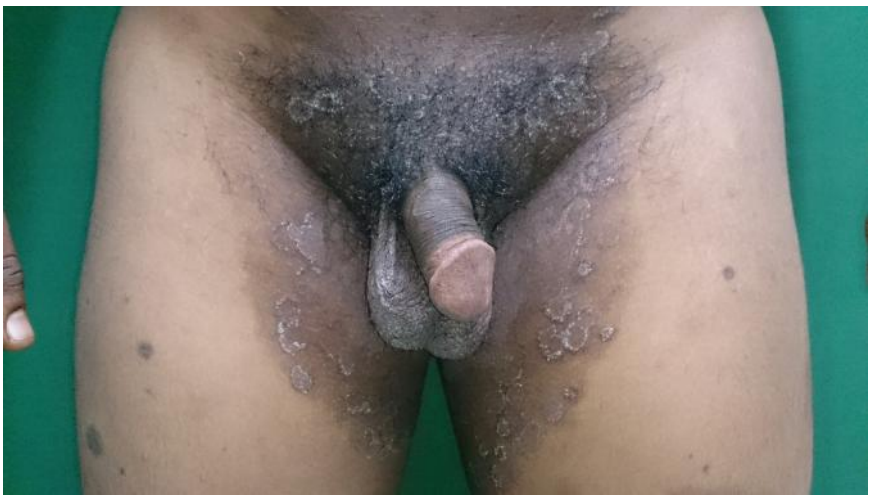


Photograph 20. Tinea corporis and cruris

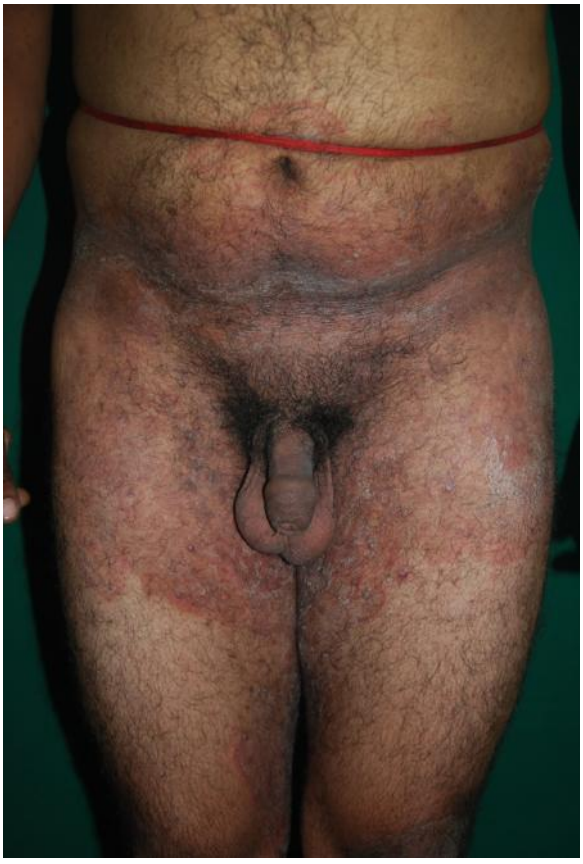


Photograph 21. Tinea corporis and cruris

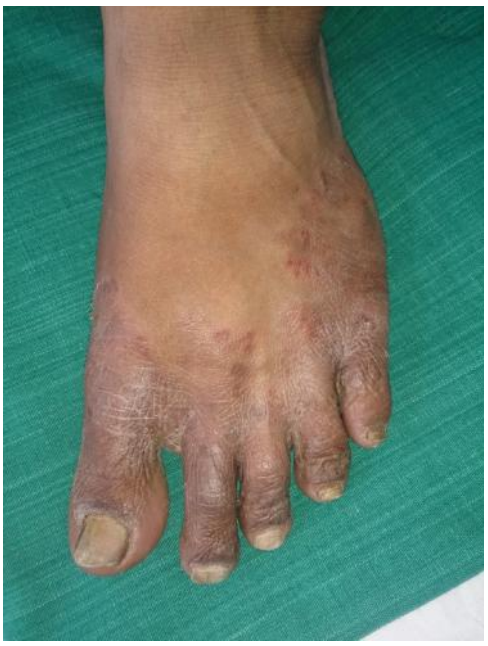
Photograph 22. Tinea cruris and tinea corporis



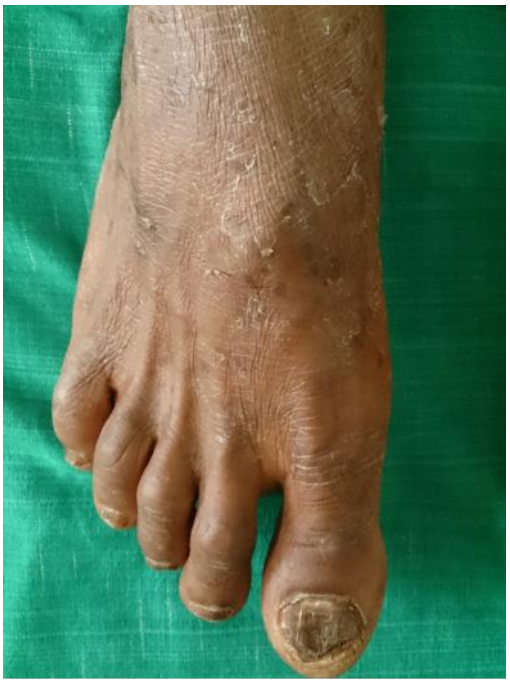
Photograph 23. Tinea cruris



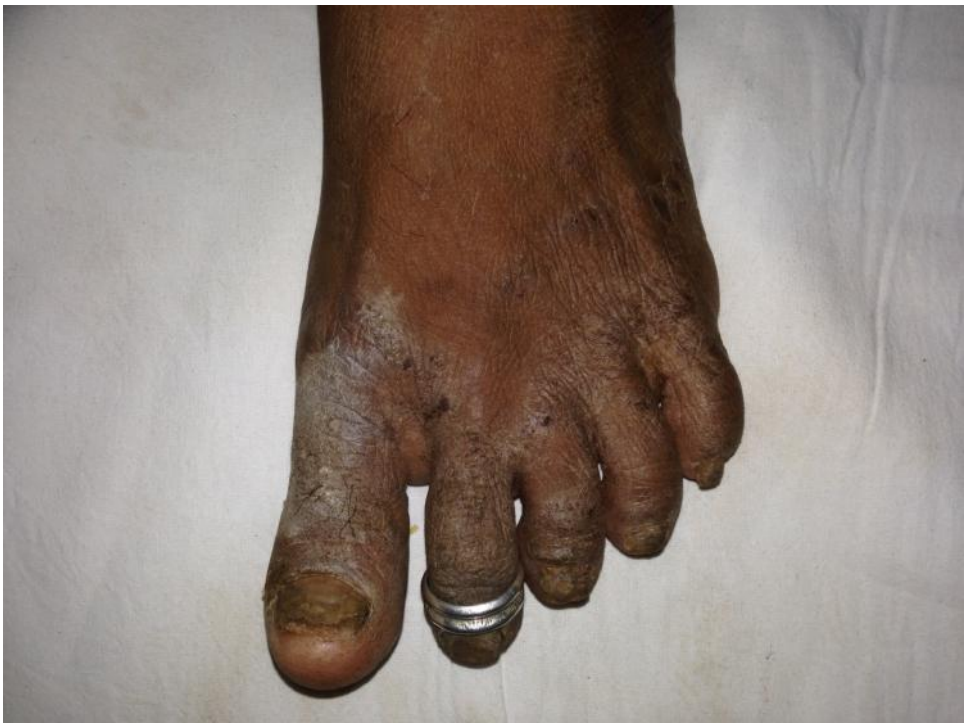
Photograph 24. Tinea cruris and corporis



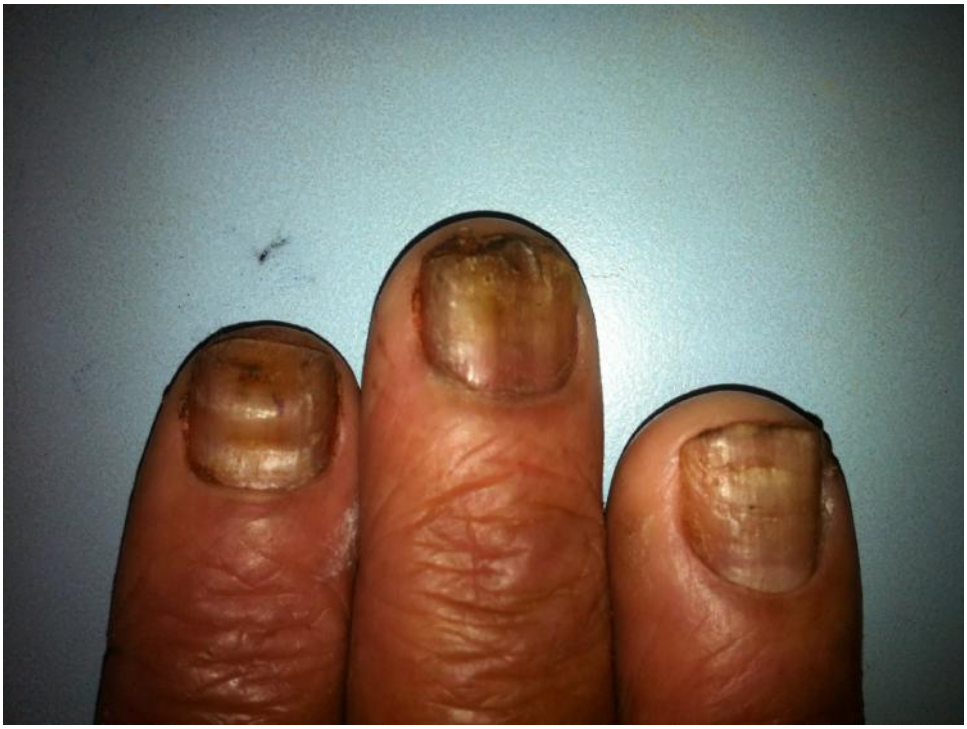
Photograph 25. Tinea pedis



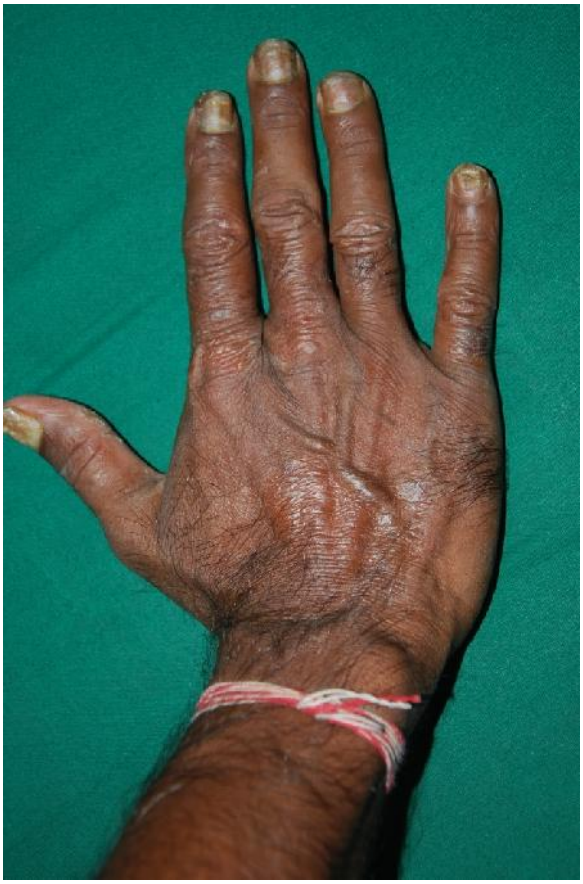
Photograph 26. Tinea pedis with Tinea unguim



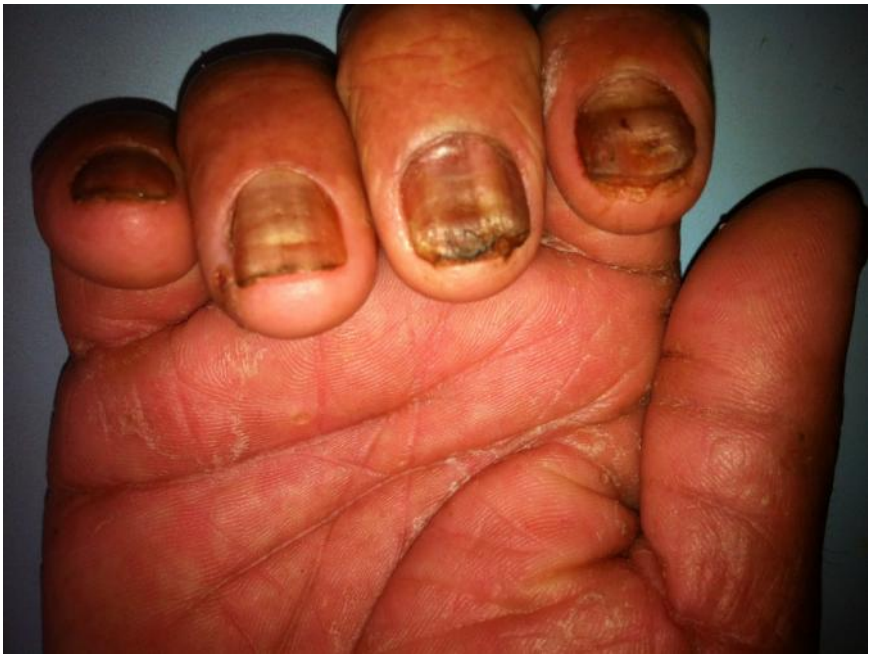
Photograph 27. Tinea pedis with Tinea unguim



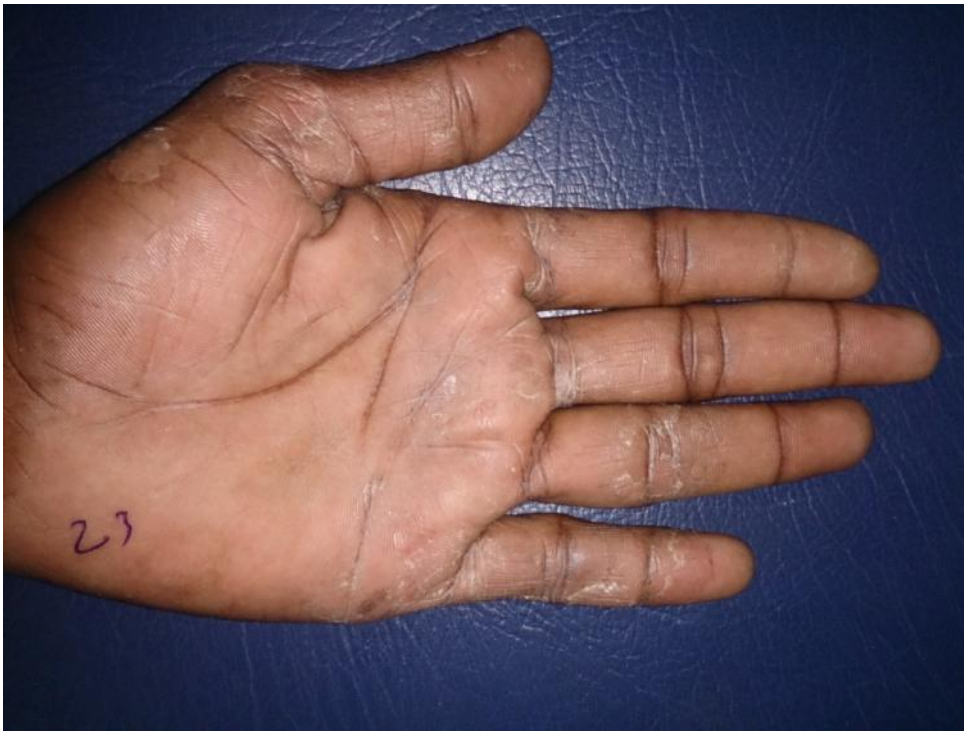
Photograph 28. Tinea unguim



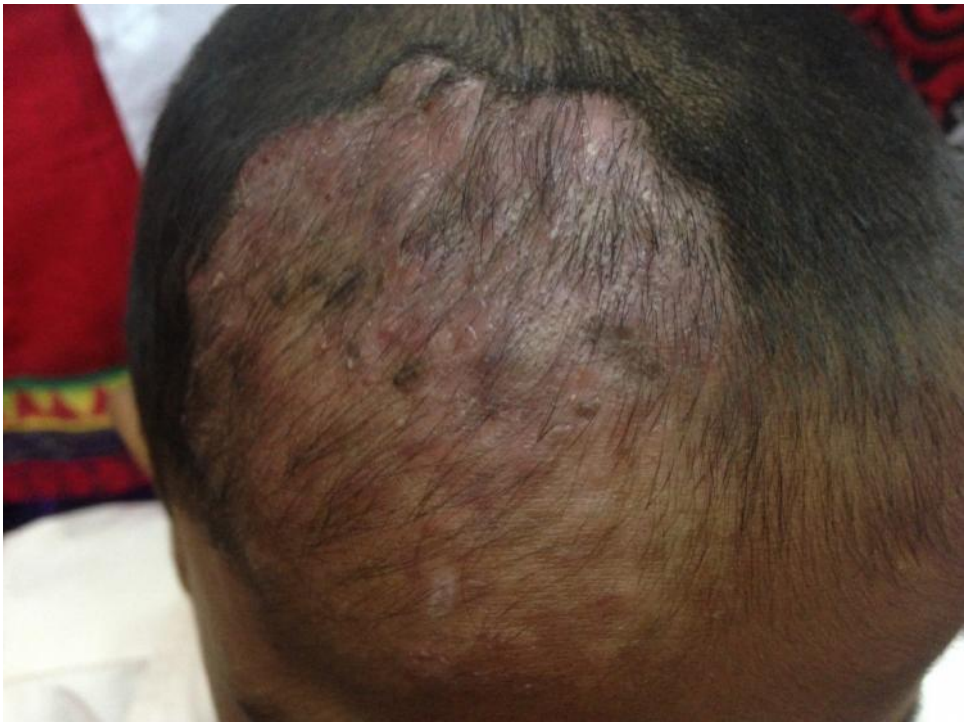
Photograph 29 Tinea unguim with tinea corporis



Photograph 30. Tinea mannum with Tinea unguim



Photograph 31. Tinea mannum



Photograph 32. Tinea capitis (Kerion)

DISCUSSION

Dermatophytosis is characterized by the infection of keratinized tissues such as the epidermis, hair and nails. This condition is caused by a group of closely related filamentous fungi commonly known as dermatophytes. *Epidermophyton*, *Microsporum* and *Trichophyton* are the genera of dermatophytes implicated in superficial mycoses. These organisms are assuming greater significance due to the excessive use of immunosuppressive drugs for controlling serious infectious as well as non infectious conditions. They produce keratinases which degrade the keratin and thus, invade the superficial skin tissue. The infections due to these pathogens are generally cutaneous and restricted to the non-living, cornified layers of the skin.[42]

Distribution of dermatophytes varies with geographical area. Further, there is wide variation in the spectrum of dermatophytic isolates. To understand the burden and trend of dermatophytosis, surveillance of the disease plays an important role. Considering the above facts the present study was designed to know the clinical and mycological profile of dermatophytosis so as to elaborate the epidemiological data in the region which will help in understanding the disease pattern and burden which may not only aid in taking adequate measures to prevent the transmission but also help in preventing spread of infection thereby reducing the disease burden.

This one year cross sectional study of 125 patients presenting with dermatophytes skin infection was conducted in the Department of Dermatology, Venereology and Leprosy, KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belgaum from January 2013 to December 2013.

In this study various clinical forms dermatophytic infections were noted. The commonest clinical form was Tinea corporis (52%) followed by Tinea cruris (43.2%), Tinea pedis (9.60%), Tinea unguim (7.2%), Tinea capitis (2.40%), Tinea faciei and Tinea mannum (1.60% each), and Tinea barbae (0.8%). A recent study from Mysore Karnataka by Surendran KAK et al[43] also observed Tinea corporis (44.3%) as the most common clinical pattern followed by Tinea cruris (38.2%), Tinea pedis (2.7%), Tinea manuum (3.3%), Tinea unguium (8.1%), Tinea barbae (2.1%), and Tinea faciei (1.3%). Further the finding of the present study were comparable with other studies from the different places of India by Bindu V et al[44] (54.6%), Singh S et al,[45] Sen SS et al[46] (48%) and Neetu J et al[47] (37%). Tinea cruris was the second most diagnosis noted in this study among 43.2% which was comparable with previous studies done by Siddappa K et al,[48] Mishra M et al[49] and Sen SS et al[46] and Peerapur BV et al.[50] In another clinicomycological study of superficial mycosis in north-east India,[51] Tinea pedis (29.2%) was the most common dermatophytosis followed by Tinea cruris (26.2%), which differs from the present study as well as other studies.[43-50]

In this study multiple sites were involved among 23 cases. Of these, Tinea cruris and Tinea corporis were present in 17 (73.91%) and Tinea corporis and Tinea pedis in three (13.04%) cases. Further one case (4.35%) each of Tinea unguim and Tinea corporis, Tinea cruris and Tinea pedis and Tinea cruris and Tinea barbae were noted. In a study from Mysore by Surendran KAK et al,[43] 46 mixed clinical types were noted and maximum patients had Tinea corporis with Tinea cruris (34 cases, 73.91%). Similar findings have been reported by Peerapur et al.[50]

In the present study males were commonly affected that is, almost two third of the patients (67.2%) were males with male to female ratio of 2:1. There is wide variation in the male to female ratio among the previous studies. An earlier study conducted at Davangere, Karnataka by Siddappa K. et al[48] reported male to female ratio as high as 4:1 during 1982. In contrast, Madhuri JT. et al[52] from Assam reported equal distribution of disease among males and females (M:F ratio 1:1.08) in 2002. During the past decade Bindu V. et al[44] (2002) from Vishakhapatnam, Sumana V et al[53] from Khammam (2004) and Sen SS et al[46] from Guwahati (2006) reported male to female ratio of 2.06:1, 3:1 and 2.85:1 respectively. Overall, all the studies including present study observed male predominance which may be attributed to the increased outdoor physical activities and increased sweating.

In the present study Tinea corporis was the commonest clinical type of dermatophytosis among males (48.81%) while in females it accounted among 58.54% of the patients. Sen SS et al[46] and Jain N et al[47] reported 48% and 37% of the male with Tinea corporis while Bindu V et al[44] reported 54.6% of males.

In this study, maximum patients belonged to age between 21 to 30 years (36%) and the next common age group was 31 to 40 year (19.2%). This was in accordance with a recent study from Mysore by Surendran KAK et al,[43] Karnataka where maximum number of cases encountered in the age group of 16-30 years (44%) followed by the age group of 31-45 years (26%). Other studies by Sen SS et al[46] from Guwahati in 2006 and Sahai S et al[54] from Lucknow in 2011 also reported commonest age group as 21 to 30 years (44% and 32.4% respectively). Although the majority of studies have observed higher incidence in the third decade, the study

done at Calicut by Bindu V et al[44] who observed higher incidence in the second decade.

In this study of the 45 patients with age between 21 to 30 years, 25 (56%) had Tinea corporis and 8 (18%) had Tinea cruris. Similar findings were noted by Bindu V et al,[44] Singh S et al,[46] Sen SS et al[46] and Jain N et al.[47]

In the present study, dermatophytosis was most commonly seen in housewives (23.2%) and students (20%) The above findings are comparable with the observations of Veer P et al[55] and Sumana V et al.[53] This higher incidence of dermatophytosis may be due to increased wet work involving in household activities.

In this study infection was most common in patients with above poverty line (85.6%). In contrast Ranganathan S et al[56] reported 69.2% of infected people were from low income group and 23.2% from middle income group. The authors attributed these findings to poor hygienic condition, overcrowding, sharing clothes without washing them properly and also due to poor nutrition. However the present study considered the socio economic status based on the Government Norms hence could not be commented upon.

In this study maximum cases were noted between June to September (37.6%) with peak in the month of August (16%) which is similar to the findings of Kalla G et al[57] and Sumana V et al.[53] The higher incidence during monsoon, post-monsoon months could be due to increased humidity and moisture. Lower incidence in extreme summer and winter could be attributed to the dry, arid climate during this period of the year.

In this study the commonest morphological variant was noted as Annular (37.6%) followed by plaque and papulosquamous (19.2% each).

In the present study KOH examination for fungus and culture was positive in 78.4% and 64.8% of the cases respectively highlighting the importance of both direct microscopy and culture in definitive diagnosis of fungal infection. Of the 98 cases with positive KOH examination for fungus, 81 (82.65%) cases had positive culture for fungi ($p<0.001$) suggesting either culture or KOH examination was positive in 82.65% of the patients. Among the 65 patients with clinical diagnosis of Tinea corporis, 44 (68%) had both KOH and culture positive. A recent study from Mysore by Surendran KAK et al[43] demonstrated fungi on direct microscopy with KOH in 96 cases but overall positivity by culture was 39% as noticed in other studies.[44,58] However, a study by Belukar et al.[59] showed culture positivity of 71%, which was much higher and close to the present study. Further a study done at Aurangabad showed low rate of culture positivity that is, 22.8%.[58]

Distribution of the Dermatophyte varies with the geographical area and during the course of time leading to a change in the spectrum of dermatophytic isolates.[54] In this study, *T. mentagrophyte* was the commonest isolate noted in 48.15% of the patients followed by *T. Rubrum* (43.21%). In a study recent study from Mysore by Surendran KAK et al,[43] *T. rubrum* was the chief organism isolated with a percentage of 67.5% while *T. mentagrophytes* (20%) isolates were found second in frequency. *T. mentagrophytes* are relatively more prevalent in south India.[43] However, in another study by Grover *et al*. in north-east India isolated *T. tonsurans* as the most common dermatophyte followed by *T. rubrum*, which differs from present study where *T. mentagrophyte* was the most common fungal

pathogen[51] suggesting wide variation in the spectrum of isolates in the different regions in India.

In the present study, the commonest dermatophyte isolated was noted as *Trichophyton* (88.64%). The *Trichophyton* genera dominated with 90% of the isolates followed by *Epidermophyton* (5%) and *Microsporum* (5%), in majority of the studies undertaken.[44,45,51]

In this study of the 39 cases with *T. mentagrophytes* clinical diagnosis of Tinea corporis was noted in 22 (56%) cases and Tinea cruris in 10 (26%). In a study by Surendran KAK et al from Mysore, correlation of clinical and mycological data found that in all clinical patterns, *T. rubrum* was the chief organism isolated (55.5%) followed by *T. mentagrophytes* (33.3%) which is in agreement with majority of other studies reported from India and other countries.[44,45,58]

CONCLUSION

The present study showed various clinical forms of dermatophytic infections. The commonest clinical form was Tinea corporis followed by Tinea cruris, Tinea pedis, Tinea unguim, Tinea capitis, Tinea faciei, Tinea mannum and Tinea barbae. Multiple clinical forms were noted among the one fifth of the study population and Tinea cruris with Tinea corporis was the commonest multiple clinical form. Male were affected more commonly and Tinea corporis was the commonest clinical type of dermatophytosis among males. Higher incidence was noted in the age group of 21 to 30 years Tinea corporis was the commonest clinical form among these patients. More than one third of the study population presented with duration of more than 35 weeks and females presented with higher duration. The commonest morphological variant was noted as annular followed by plaque. In patients with clinical diagnosis of Tinea corporis maximum patients had annular morphological variant

The KOH examination for fungus was positive in 78.4% of the cases and culture was positive for fungus in 64.8% and in patients with positive culture, *T. mentagrophyte* was the commonest isolate followed by *T. rubrum* (43.21%). Significantly higher number of patients with positive KOH examination for fungus had positive culture for fungi. Maximum patients had positive culture who were diagnosed to have Tinea corporis as clinical variant. The commonest dermatophyte isolated was *Trichophyton*.

Overall there is wide variation in the clinical and mycological profile of dermatophytosis. Further KOH examination for fungus and culture play an important role in the diagnosis of dermatophytosis.

SUMMARY

Distribution of dermatophytes varies with geographical area as well as climatic conditions. Also wide variation in the spectrum of dermatophytic isolates is reported. The present study was aimed to know the clinical and mycological profile of dermatophytosis.

This one year cross sectional study was carried out in the Department of Dermatology, Venereology and Leprosy, KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belgaum from January 2013 to December 2013. A total of 125 patients presenting with dermatophyte skin infection were studied. The patients were subjected to clinical examination, KOH examination for fungi and culture.

Based on the clinical examination findings the commonest clinical forms noted were Tinea corporis, Tinea cruris, Tinea pedis, Tinea unguim, Tinea capitis, Tinea faciei, Tinea mannum and Tinea barbae. Multiple clinical forms were present in almost one fifth of the study population and the commonest clinical form was noted as Tinea cruris with Tinea corporis. Most of the patients were males (67.2%) with male to female ratio of 2:1 and 48.81% of the males had clinical type of Tinea corporis. The commonest age group was 21 to 30 years (36%) and had Tinea corporis (56%) commonly. Most of the patients (36.8%) reported duration of > 35 weeks. Maximum cases were noted in the month of August (16%). The commonest morphological variant was noted as annular (37.6%). The KOH examination for fungus was positive in 78.4% of the cases and culture was positive for fungus in 64.8% of the cases. Among the patients with positive culture, *T. mentagrophyte* was

the commonest isolate noted in 48.15% of the patients. The commonest dermatophyte isolated was noted as *Trichophyton* (88.64%).

The present study shows wide variation in the clinical and mycological profile of dermatophytosis. Monitoring the incidence of these fungal species enables the detection of emerging organisms and may be helpful in the treatment and adequacy of current pharmacologic regimens.

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ANNEXURE I – CONSENT FORM

I.D.NO. | | | |

A one year cross-sectional clinical and mycological study of dermatophytosis in KLE Dr. Prabhakar Kore Hospital and MRC, Belgaum

The study is conducted by Dr. **** Post graduate student in M.D Dermatology under guidance of Dr. *** *****, Professor of Dermatology, J N Medical College, Belgaum.

Respected Sir/Madam, we invite you to participate in our study as, you are eligible for the same. During the study you will be asked some questions in detail regarding your present complaints.

Purpose of the study

The purpose of this study is to find out the prevalence and etiological agent of dermatophytosis. You are being asked to participate in this research because you have been diagnosed to have dermatophytosis. All patients attending the outpatient department, who are diagnosed to have this disease, will be requested to participate in this study during the period of one year.

Procedure and treatment

Should you choose to participate, you will be asked to give a detailed history of your disease, undergo a physical examination, and consent to a few routine investigations.

Risks and benefits

You may undergo some amount of discomfort during the process of investigations, which may include slight pain. However all necessary steps and precautions will be taken to ensure your safety. The result of you taking part in this research would help health care providers towards a better understanding of this disease, and thus we will be able to provide improved patient care

Alternatives

If you decide not to participate in this study, you will still be receiving the usual standard care for your disease.

Privacy and confidentiality

Your privacy will be respected and all information collected about you during the course of this study will be kept confidential. Your identity will remain undisclosed.

Relations with the Institutional policy

The J N Medical College will provide, within the limitations of the laws of the State of Karnataka, facilities and medical attention to patients who suffer injuries as a result of participating in this project.

Financial incentives

You shall not be receiving any payment or any financial incentives for participating in this study.

Authorization to publish results

The results of this study may be published for scientific purpose or presented to a scientific group. Your identity, however, will be maintained confidential at all times.

Voluntary participation

Your participation in this study is voluntary. Your decision whether or not to participate will neither affect the care of your current disease, nor your future relations with the doctor or the hospital. In the event if you suffer any physical injury as the result of your participation in this study, you may contact Dr. **** Telephone No. ***** ***** or Dr. ***** ******, Telephone No. ***** *****. In the event of an emergency, you should contact KLE'S Dr. Prabhakar Kore Hospital and MRC on Telephone No. ***** ******.

In case you need further information regarding your rights as a study participant, you may please contact The Chairman of the ethical committee, J N Medical College, Belgaum.

Statement of Consent

I.D.NO: | | | |

I Mr/Ms/Mrs _____ volunteer and consent to participate in this study. I have read the consent document or it has been read to me in my vernacular language. I accept to participate in the study. All the information regarding this study is provided to me and I have understood the same. I have been given the opportunity to ask questions and obtain appropriate answers.

Participant's name:

Signature or left thumb print of participant:

Witness name:

Signature of witness:

Signature of the investigator:

Date:

If the participants are Minors (under 18), the parents sign the form, rather than the participants.

ANNEXURE II – PROFORMA

ONE YEAR CROSS SECTIONAL CLINICAL AND MYCOLOGICAL STUDY OF DERMATOPHYTOSIS IN Dr.PRABHAKAR KORE,KLE HOSPITAL,BELGAUM

Case No. ☐☐ **OP/IP No.** ☐☐☐☐☐☐☐

Name: First name ☐☐☐☐☐☐ Middle name ☐☐☐☐☐☐☐

Last name ☐☐☐☐☐☐

Age: ☐☐

Sex:

1. Male
2. Female

☐

Occupation:

1. Business
2. Housewife
3. Professional
4. Farmer
5. Student
6. Any other

☐

Income: Monthly income (In Rs.):

1. Above poverty line 2. Below poverty line

☐

Address with phone number:

Presenting complaints and duration:

History of present illness:

Onset: Acute Insidious

Factors initiating the disease: Season Others

Associated skin diseases (if any):

Precipitating factor (if any):

1.Hyperhidrosis: ☐

2.Trauma: ☐

3.Personal Hygeine: ☐

4.Poor nutrition: ☐

5.Contact with animals: ☐

6.Warm, humid climate: ☐

7.Obese: ☐

8.Diabetes: ☐

9.Endocrine disorder: ☐

10.Topical steroid application: ☐

Site of lesion

Face ☐

1. Present
2. Absent

Upper and lower extremities ☐

1. Present
2. Absent

Neck ☐

1. Present

2. Absent

Back

1. Present ☐

2. Absent

Trunk ☐

1. Present

2. Absent

Palms /Dorsum of Hand ☐

1. Present

2. Absent

Soles/ Dorsum of foot

1 .Present ☐

2 . Absent

External genetalia

1 Present

3. Absent ☐

Nail

1 Present

2 Absent

Scalp

1 Present

2 Absent

Hair ☐

1.Pluckable

2.Dull

3.Lustreless

Clinical examination of lesions

Inspection**:**

- Type

  1.Plaque

  2.Papule

  3.Pustule

  4.Vesicle

- Number

  1.Single

  2.Discrete

- Size
- Shape
- Surface
- Border

  1. Raised

  2.Erythematous

  3.Scaly

  4.Vesicular

Centre

1.Clearing

2.Hyperpigmentation

3.Erythema

- Distribution
- Surrounding skin

- Presence of pruritus
- Secondary infection

  1.Present

  2.Absent
- Inflammation

  1.Present

  2.Absent
- Discharge

  1. Present

  2. Absent
- Scales

  1. Present

  2. Absent
- Erythema

  1.Present

  2.Absent
- Symmetry

  1.Present

  2.Absent
- Odor

  1.Present

  2.Absent
- Fissure

  1.Present

2.Absent

- Maceration

  1.Present

  2.Absent

- Ulceration

  1.Present

  2.Absent

- Pain

  1.Present

  2.Absent

- Lichenification

  1.Present

  2.Absent

Palpation

- Surface texture of surrounding skin
- Tenderness
- Temperature

Past History:

History suggestive of similar illness:

1. Present
2. Absent

History of any other medical disorders:_____

Family History:

Treatment History

Oral antifungal 1-On treatment ☐

2-Not on treatment

Topical antifungal 1-On treatment ☐

2-Not on treatment

Alternative Treatment if any ☐

Personal History:

Diet

1. Veg ☐
2. Mixed

Appetite ☐

1. Normal
2. Poor

Bowel/ Bladder ☐

1. Normal
2. Altered

Sleep

1. Normal ☐
2. Altered

Alcohol

1. Present ☐
2. Absent

Smoking

1. Present ☐
2. Absent

General Physical Examination:

Built

1. Poor
2. Moderate
3. Good

☐

Vitals

Pulse / min ☐☐☐

BP(mm/hg): Systolic

Diastolic ☐☐☐

Temperature ^{0}F

Weight ☐ (Kg) ☐☐☐

Pallor

1. Present
2. Absent

☐

Icterus

1. Present
2. Absent

☐

Cyanosis

1. Present
2. Absent

☐

Clubbing

1. Present
2. Absent

☐

Lymph nodes

1. Palpable

☐

2. Non palpable

Edema

1. Pitting ☐
2. Non Pitting
3. Absent

Systemic Examination:

Cardiovascular system: Heart sounds

1. Normal ☐
2. Abnormal;if abnormal specify the finding_____

Respiratory system: Breath sounds

1. Normal ☐
2. Abnormal;if abnormal specify the finding_____

Per abdomen:

1. Normal ☐
2. Abnormal;if abnormal specify the finding_____

Central nervous system: Neurological examination

1. Normal ☐
2. Abnormal; if abnormal specify the finding_____

Investigations

Diagnosis:-

Signature:

Guide's Signature

ANNEXURE III – KEY TO MASTER CHART

-	-	Negative
+	-	Positive
1	-	Above poverty line
2	-	Below poverty line
a	-	April
AT	-	Atopy
au	-	August
B	-	Business
ca	-	Contact with animals
Ch. Inter	-	Chronic intertriginous
Ch.Hyp	-	Chronic hyperkeratotic
d	-	December
DM	-	Diabetes mellitus
DSO	-	Distal subungual onychomycosis
E.	-	*Epidermophyton*
Eczema	-	Eczematous
f	-	February
F	-	Female
FR	-	Farmer
GN	-	Generalised
hd	-	Hyperhidrosis
HW	-	Housewife
Hyp.ker	-	Hyper keratotic

j	-	January
jl	-	July
ju	-	June
L	-	Labour
LC	-	Localised
M	-	Male
m	-	March
M.	-	*Microsporum*
ma	-	May
n	-	November
o	-	October
ot	-	Other
ob	-	Obesity
PR	-	Professional
PSQ	-	Papulosquamous
s	-	September
ST	-	Student
T.	-	Tinea
T.	-	*Trichophyton*
TDO	-	Total dystrophic onychomycosis
VPU	-	Vesiculopustular
VSV	-	Vesicular
wh	-	Warm humid climate