“DERMATOPHYTE INFECTIONS IN CHILDREN: A PROSPECTIVE STUDY IN A TERTIARY CARE HOSPITAL”

Submitted by

Dr. ANASWARASREE

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Under the guidance of

DR. ARUN C. INAMADAR, M.D, D.V.D, F.R.C.P

PROFESSOR

DEPARTMENT OF DERMATOLOGY, VENEREOLOGY AND LEPROSY

DR. ANNAPURNA G SAJJAN, M.D, MHA

PROFESSOR AND HEAD,

DEPARTMENT OF MICROBIOLOGY

B. L. D. E. (DEEMED TO BE UNIVERSITY)

SHRI B. M. PATIL MEDICAL COLLEGE HOSPITAL &

RESEARCH CENTRE, VIJAYAPURA,586103

B.L.D.E. (DEEMED TO BE UNIVERSITY)

SHRI B. M. PATIL MEDICAL COLLEGE, HOSPITAL AND RESEARCH CENTRE, VIJAYAPURA.

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DATE:

PLACE: VIJAYAPURA **DR. ANASWARASREE**

B.L.D.E. (DEEMED TO BE UNIVERSITY)

SHRI B. M. PATIL MEDICAL COLLEGE, HOSPITAL AND RESEARCH CENTRE, VIJAYAPURA.

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Signature of the Guide

DR. ARUN C. INAMADAR
PROFESSOR,

DEPARTMENT OF DERMATOLOGY,

VENEREOLOGY AND LEPROSY,

B. L. D. E. (Deemed to be University)

SHRI. B. M. PATIL

MEDICAL COLLEGE, HOSPITAL AND

RESEARCH CENTRE,

VIJAYAPURA

Date:

Place: Vijayapura

Signature of the Co-Guide

DR ANNAPURNA G SAJJAN

PROFESSOR & HEAD,

DEPARTMENT OF

MICROBIOLOGY,

B. L. D. E. (Deemed to be

University)

SHRI. B. M. PATIL

MEDICAL COLLEGE, HOSPITAL

AND RESEARCH CENTRE,

VIJAYAPURA

Date:

Place: Vijayapura

B.L.D.E. (DEEMED TO BE UNIVERSITY)

SHRI B. M. PATIL MEDICAL COLLEGE, HOSPITAL AND RESEARCH CENTRE, VIJAYAPURA.

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Seal & Signature of the HOD

Dr.Keshavmurthy Adya,

Professor and Head,

Department of Dermatology,

Venereology & Leprosy

B. L. D. E. (Deemed to be University)

Shri B. M. Patil Medical College Hospital & Research Centre, Vijayapura.

Date:

Place: Vijayapura

Seal and Signature of the Principal

Dr. Aravind V. Patil M.S.

Principal,

B.L.D.E. (Deemed to be University) Shri B. M. Patil Medical College Hospital & Research Centre, Vijayapura.

Date:

Place: Vijayapura

B.L.D.E. (DEEMED TO BE UNIVERSITY)

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Date:
DR. ANASWARASREE

Place: VIJAYAPURA

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Date:

Dr. ANASWARASREE

Place: Vijayapura

LIST OF ABBREVIATIONS

M. audouinii	*Microsporum audouinii*
M. ferrugineum	*Microsporum ferrugineum*
M. canis	*Microsporum canis*
M. gallinae	*Microsporum gallinae*
M. equinum	*Microsporum equinum*
M. nanum	*Microsporum nanum*
M. persicolor	*Microsporum persicolor*
M. gypseum	*Microsporum gypseum*
T. concentricum	*Trichophyton concentricum*
T. tonsurans	*Trichophyton tonsurans*
T. violaceum	*Trichophyton violaceum*
T. interdigitale	*Trichophyton interdigitale*
T. rubrum	*Trichophyton rubrum*
T. schoenleinii	*Trichophyton schoenleinii*
T. equinum	*Trichophyton equinum*
T. simii	*Trichophyton simii*
T. mentagrophytes	*Trichophyton mentagrophytes*
T. verrucosum	*Trichophyton verrucosum*
T. ajeloii	*Trichophyton ajeloii*
T. soudanense	*Trichophyton soudanenses*
E. floccosum	*Epidermophyton floccosum*
TLR	Toll like Receptor
DTH	Delayed type hypersensitivity

Th cell	T helper cell
CMI	Cell mediated immunity
IgE	Immunoglobulin E
IgG4	Immunoglobulin G4
DLSO	Distal and Lateral subungual onychomycosis
PSO	Proximal subungual onychomycosis
WSO	White superficial onychomycosis
EO	Endonyx onychomycosis
TDO	Total dystrophic onychomycosis
IL	Interleukin
KOH	Potassium Hydroxide
PCR	Polymerase Chain Reaction
MALDI-TOF	Matrix assisted laser desorption ionization time of flight
SDA	Sabouraud's Dextrose Agar
DTM	Dermatophyte test media
PCR-ELISA	Polymerase Chain Reaction- Enzyme Linked Immunosorbent Assay
AFA	Antifungal Agents

OD	Once Daily
BD	Twice Daily
MIC	Minimum Inhibitory Concentration
SD	Standard Deviation
BSA	Body Surface Area

ABSTRACT

BACKGROUND:

The prevalence of dermatophytic infections has recently surged worldwide, particularly in tropical nations like India. This is associated with change in the clinical pattern and mycological profile with poor response to treatment. This clinico-epidemiologic study helps to clarify the determinants of the trends associated with these dermatophytosis and the health burden of illness in the pediatric population.

AIMS AND OBJECTIVES:

1. To determine the epidemiological profile of dermatophytosis among children (less than 15 years of age) attending dermatology OPD at tertiary care hospital.
2. To assess clinico-mycological characteristics of dermatophytosis in pediatric population.

MATERIALS AND METHODS:

It is a hospital-based cross-sectional study. Children below 15 years of age clinically diagnosed to have dermatophytosis were be enrolled for the study. Detailed history about duration of disease, socioeconomic status, past history of medication and consultation, personal and family history were recorded. The Patients were examined to determine the clinical type of dermatophytosis. Specimen (skin scraping, hair, or nail clippings) for microbiological investigations was collected from the lesion. It was utilized for preparing a 10% KOH mount for direct microscopy for visualization of fungal hyphae. Irrespective of KOH mount result, the specimen was also inoculated in 3 media, Sabouraud dextrose agar without chloramphenicol and cycloheximide (SDA), Sabouraud dextrose agar with Chloramphenicol, and cycloheximide

(SDA with antibiotics) and Dermatophyte test media (DTM). It was further sent to the microbiology laboratory for the purpose of incubation and isolation of species.

RESULTS

The most commonly affected age group in this study was 13-15 years with male predominance. Maximum patients (39.9%) presented with 1-2 months duration of the lesion with 27.5% of the patients having history of self-medication. Past history of similar complaints was observed in 21.6 % of the patients and 41.8 % of the patients had similar complaints among family members or close contacts. Tinea Corporis was the most common clinical diagnosis followed by tinea Capitis. Tinea Corporis with tinea Capitis was the commonest presentation among the mixed type of dermatophytosis. Direct microscopy of a 10% KOH mount demonstrated fungal hyphae in 81 % of patient's samples and culture positivity for dermatophytes seen was in 51 %. *Trichophyton mentagrophyte* (32.7 %) was the most common specie isolated followed by *Trichophyton rubrum* (12.4%).

CONCLUSION:

- Topical corticosteroids in combination with antifungal agents available over counter are grossly abused leading to chronic treatment resistant dermatophytosis.
- Clinical failure from antifungal medication due to the persistence of risk factors, poor personal hygiene and inadequate knowledge of the infection and its features of treatment in the general population contribute to treatment resistant dermatophytosis.
- This clinic-epidemiologic study helps to determine the epidemiological trends, the nature of the disease, the predisposing factors and the causative species of dermatophytosis in the pediatric population.

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INTRODUCTION

The most common skin infections in children are superficial mycotic infections, and their prevalence is rising. [1,2,3]

Superficial dermatophytosis of glabrous skin is a significant public health issue due to the rise in chronic, recurring, resistant, and steroid-modified, challenging-to-treat tinea in recent years. [4] Hot and humid climate, poverty, poor hygiene, migration, and socioeconomic factors like overcrowding may be the contributing factors. [5]. Dermatophytic infections spread through direct skin-to-skin contact with affected individuals, sharing objects with infected individuals or by contact with contaminated surfaces. [6, 7]

Even though the problem of recalcitrant dermatophytosis is growing leaps and bounds in India, there is a large void in the published epidemiological, clinical, and laboratory research in this field. [8] This study was done to improve diagnostic approach of dermatophytosis in children by evaluating clinico-mycological features and correlating the results of fungal cultures and KOH mount with the clinical profile of these patients.

AIMS AND OBJECTIVES

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REVIEW OF LITERATURE

"Dermatophytes are infections caused by a specialized group of fungi, the dermatophytes, which are able to invade and colonize stratum corneum of the skin and keratinized tissues such as hair and nails." [9] The fungal infection is termed tinea, whereas fungal organisms that cause tinea are referred to as dermatophytes. [10]

History

The Roman encyclopaedist Aulus Cornelius Celsus is credited with the first documented description of a dermatophytic illness in the book De Re Medicina, which was written around 30 A.D. Celsus explained suppurative infection of scalp known as kerion of Celsus. [5,11]

The mycotic etiology was discovered for these skin diseases in 19 th century. [11]

- Remak discovered favus, a chronic infection of scalp, in 1837, it was the first time that a fungus was thought to be the source of an infection. [11,12]
- The filaments were first identified by Schoenlein in 1839 as belonging to molds. Remak isolated fungus in 1845, named it Achorion schoenleinii (now Trichophyton schoenleinii), placing it in the genus Achorion. [11]
- Gruby also wrote regarding the fungus that cause favus in 1841 and identified different forms of hair invasion in tinea capitis, naming the fungus responsible for ectothrix-type infections Microsporum audouinii. [11]
- In 1910 Les Teignes, a classic book written by Sabouraud that collected his research on the taxonomy, morphology, diagnosis, and treatment of dermatophytoses, was published. [11]
- On the basis of clinical manifestation of the disease and morphological characteristics of fungi, dermatophytes were classified as 4 genera-Achorion, Epidermophyton, Microsporum and Trichophyton. [11]

- New species and genera were described in subsequent years with distinctions being made solely on the basis of minute variations in morphology of fungus and type of lesions produced. [11]
- More than 300 species from 40 genera have been proposed by 1934. However, Emmons in 1934 classified the dermatophytes into three genera—Microsorum, Trichophyton and Epidermophyton—consisting 19 species, based on the morphology of the spores. [11]

Etiological agent:s

Dermatophytes

Evolution has made the mycelial and keratinophilic fungi of mold group, dermatophytes to adapt themselves to animal and human parasitism which was originally saprobic. [5]

They can reproduce via both sexual and asexual means. The dermatophyte species involved and the host's immune response determine the severity of illness. [5]

Table 1**: Scientific classification of Dermatophytes.** [13]

Kingdom:	Fungi
Phylum:	Ascomycota
Order:	Onygenales
Family	Arthrodermataceae
Genus	Trichophyton, Microsporum, Epidermophyton, Arthroderma, Nannizzia

Based on life cycle stages, fungi are classified as anamorphic and telomorphic state. [14]

Anamorphic state	distinct morphology, asexual or somatic reproduction occur
Teleomorph state	Morphologically differentiated from anamorph, undergo sexual reproduction.

Table 2 **: Classification of fungi based on life cycle stages**

Fungi are divided into four major divisions according to Botanical taxonomy. [5]

- Zygomycota
- Ascomycota
- Basidiomycota
- Deuteromycota

- Majority of pathogenic fungi belong to Deuteromycota. They have septate hyphae and reproduce asexually by producing conidia. Few are capable of acting as keratinolytic agents and penetrate natural keratin.[5]
- The first person to group dermatophytes into three anamorphic (asexual, imperfect) genera was Emmons (1934). [5]
  - Epidermophyton
  - Trichophyton
  - Microsporum

Epidermophyton

Firstly, discovered by Sabouraud in 1910, who also invented specie Epidermophyton rubrum "(Epidermophyton interdigitale—Kanfmam Wolf)." [5]. They invade the epidermis, but they also have a remote possibility of destroying hair. [5] This species is positioned within Trichophyton genera known as Trichophyton rubrum according to new classification. [15] Another species is Epidermophyton floccosum, which is entirely an Anthropophilic fungus. [11] The causing ability of these genera is termed as epidermophytosis. [5] Epidermophyton have massive conidia and branches with thin wall and clusters [16.]. Based on species, variations occur in cell wall thickness and form.. Microconidia measure between 2 and 3 μm and have a pyriform shape. [17]

Trichophyton

In 1845, Malmsten discovered the species T. tonsurans, which led to the identification of the genus Trichophyton.[15]. Both macroconidia and microconidia with smooth walls are produced by this genus. [18] Macroconidia is cigar-shaped and has thin walls. [18]. Microconidia are irregularly shaped or pyriform, measuring between 2 and 3 μm. [18]. The common agents of dermatophytosis are members of the genus Trichophyton.[5] They are especially relevant in cases of onychomycosis, but they can also invade skin and hair and cause infections that have a high morbidity rate.. [11]

Microsporum

Gruby discovered M. audouinii in 1843, which led to the identification of Microsporum. [15] It

was confirmed microscopically in 1956 at USA. [15] The genus reproduces both micro- and macroconidia. [18]

Macroconidia are multiseptated, may be numerous or few, having a spindle-shaped echinulate cell wall that is either thin or thick. [18,19] Microsporosis, caused by Microsporum, typically results in a single inflammatory skin or scalp lesion. [20]

Table 3**: Patterns Of Dermatophytes infections**-[21]

GENERA	SKIN	HAIR	NAIL
Microsporum	+	-	+
Trichophyton	+	+	+
Epidermophyton	+	+	-

Classification based on Natural habitat: -

- **Anthropophilic**
- **Zoophilic**
- **Geophilic**

Table 4: **Species of Dermatophytes Based on Ecological classification** [4]

Anthropophilic	Zoophilic	Geophilic
Microsporum *M.audouinii,* *M.ferrugineum*	**Microsporum** *M.canis,* *M.gallinae,* *M.equinum,* *M.persicolor*	**Microsporum** *M. nanum,* *M.amazonicum,* *M.gypseum,* *M.cookei*

Trichophyton *T. concentricum* *T. violaceum* *T. schoenleinii* *T. rubrum* *T. interdigitale* *T. tonsurans*	**Trichophyton** *T.mentagrophytes,* *T. simii,* *T.equinum,* *T. verrucosum*	**Trichophyton** *T. ajeloii,* *T.phaseoliforme,* *T.flavescens,* *T.gloriae*
Epidermophyton *E.floccosum*		**Epidermophyton** *E.stockdaleae*

Table 5**: Features of Dermatophytes Based on Ecology**. [13]

	Anthropophilic	**Zoophilic**	**Geophilic**
Natural habitat	Human	Fur of reservoir animals	Soil around burrows of specific terrestrial mammals.
Sexual phase	No	Mostly mating	Vigorously mating
Transmission	Human to human	Animals to human	Via environment
Clinical features	Chronic,mild, noninflammatory	Moderately inflammatory infection inhuman; rare outbreaks.Symptomatic or asymptomatic in animals	Acute,self-resolving,inflammatory mycoses in humans

PATHOGENESIS: -

The intricate interactions between the agent (dermatophytes), host (innate host defense and host immunological response), and environment factor are a component of the pathogenesis. [8]

Host factor

Various host factors influence the occurrence of dermatophytosis. Predisposing factors include the patient's age, the site of invasion, obesity, physiological changes in the host skin barrier, immunosuppression, acquired conditions such as excessive washing or sun exposure. [8]

Evidence show that the appearance of infection in familial or genetically predisposed individuals is mediated through specific defects in innate and adaptive immunity. Jaradat *et al*, attributes low defensin beta 4 in patients, as a possible predisposing factor for all dermatophytes. [22,23] The site of involvement determines the susceptibility to develop dermatophytic infection. Growth of the fungus in intertriginous areas (web spaces and groins) occurs as a consequence of excess sweating, maceration, and alkaline pH. [22]

Proteases, serine-subtilisins, and fungolysin facilitate the infection's progression through adherence and penetration after inoculation into the host skin. This process breaks down the keratin network into oligopeptides or amino acids and serves as a strong immunogenic stimulus. [24]

Environmental Factors:

There are three primary sources of infection for dermatophytes, which are prevalent in various ecological niches. Humans are the most prevalent source among them, followed by soil and animals. [8] Dermatophytoses from these three sources can infect humans directly or indirectly, resulting in the development of dermatophytoses in various clinical entities. [8]

Immunology of dermatophytosis: -

When dermatophytes infect a host, the immune system can react in a variety of ways, including humoral and cell-mediated immunological reactions. [22]. According to the currently accepted

theory, cell-mediated immunity regulates dermatophytosis. [22]

Innate immune response

Innate immune systems, including Dectin-1 and Dectin-2, identify the β-glucan molecules found in dermatophytes' cell walls and activate TLR-2 and TLR-4. Dectin-1 increases the synthesis of IL-17, IL-6, IL-10, and tumor necrosis factor-α and promote adaptive immunity. [22]. The keratinocytes release IL-8, a strong neutrophilic chemo-attractant in the presence of dermatophyte antigens known as trichophytin.[22]

Adaptive immune response: -

Humoral immunity:

There is no protection from humoral immunity to dermatophytes. Patients with chronic dermatophytosis have high levels of specific IgE and IgG4, which causes positive (IgE-mediated) IH tests for Trichophyton. Conversely, patients with a positive delayed type hypersensitivity (DTH) skin test have low Ig levels. The IH skin test for Trichophyton is associated with the presence of serum IgE and IgG (mainly IgG4) against Trichophyton antigens, which are markers of a Th2 response.[22] Here, CD4 T-cells (Th2 cells) produce IL-4, which causes antibody isotype switching to IgG4 and IgE. [22]

Cell-mediated immunity:

Various studies demonstrated that DTH mediates dermatophytosis resolution. Th1 or Th2 subsets may control immunity to infections, which will eventually determine the outcome of infection. While a persistent infection is linked to low DTH and high IH. A positive DTH skin test result for trichophyton is associated with an acute inflammatory reaction, and recovery from illness. [25]

Nonspecific response:

It has been discovered that unsaturated transferrin inhibits dermatophytes by attaching itself to their hyphae. Commensal pityrosporum promotes lipolysis and enhances the supply of fatty

acids that can be utilized for inhibiting fungal growth. [22]

Clinical features: -

The type of fungus that causes the infection largely determines the clinical presentation of dermatophytosis. This is due to differences in the keratinase production by dermatophytes, cytokine secretion by keratinocytes and other inflammatory cells in response to infection. Site and size of the infection and immune status of the host and therapy can further modify the clinical presentation.

Table 6 :Dermatophyes are classified based on anatomical site of involvement. [13]

Clinical type	**Synonym**	**Description**	**Etiology**	**Subtypes/special Types**
Tinea capitis	-	Dematophyic Infection of the scalp hair and intervening skin.	*T.violaceum, T.tonsurans M.canis*	Gray patch, Black dot, Kerion,Favus
Tinea barbae	Barber's itch	Dermatophyte infection of the beard and moustache hair and intervening skin	*T.violaceum, T.tonsurans, M.canis*	Sycosiform, Kerion type, Non inflammatory
Tinea Faciei	-	Dermatophyte infection of glabrous skin of face.	*T.rubrum, T.mentagrophytes, E. floccosum*	-

Tinea Corporis	Ringworm	Dermatophye infection of glabrous skin of body.	*T. rubrum, T.mentagrophytes, T.violaceum*	T.incognito, T.rubrum syndrome
Tinea cruris	Jock itch, Dhobi itch,eczema marginatum	Dermatophyte infection of the glabrous skin of the groin.	*T.rubrum,T.mentagrophytes*	-
Tinea mannum	-	Dermatophyte infection of palms.	*T.interdigitale ,T.rubrum*	Non inflammatory squamous, Inflammatory vesicular
Tinea Pedis	Athlete's foot	Dermatophyte infection of the feet.	*T.interdigitale ,T.rubrum*	Interdigital,chronic hyperkeratotic,vesicular,ulcerative
Tinea unguim	Onychomycosis	Dermatophyte infection of nails.	*T.interdigitale ,T.rubrum*	Distal lateral subungual, White superficial , Proximal subungual , Total dystrophic onychomycosis.

TINEA CAPITIS

Tinea capitis, dermatophyte infection of the scalp is mostly prevalent in pediatric population. It is caused mainly by 2 species of fungi:*Trichophyton* and *Microsproum.* Alopecia with scaly patches, alopecia with black dots, or mild hair loss combined with scalp

scaling are clinical manifestations of tinea capitis.[13,26]

Classification of Tinea capitis based on the size and location of spores. [13]

- **Endothrix**
- **Ectothrix**
- **Nonfluorescent large spore ectothrix type**.

Enodothrix(spores inside the hair shaft) **variant** is caused by *T. soudanense*, *T. tonsurans, T. violaceum, or T. rubrum*. [13] It is identified by a black dot ringworm pattern, larger hair stubs, and hair shaft breaks at the scalp level. [13]

Ectothrix (spores outside the hair shaft) **variant** is caused by fluorescent small pore infections *M.audouinii, M.canis, M.audouinii, M.canis, M.gypseum, M.distortum, M.ferrugineum*.[13] The hair shaft at the mid follicle level is affected. This results in scaly, inflammation of the skin along with hair shaft breaking 2-3 mm above the scalp.s [13]

Nonfluorescent large spore ectothrix type: It is caused by *T.mentagrophytes,T.equinum,Trichophyton megninii* and rarely *T.rubrrum.* [13]

Table 7 **:Classification of Tinea capitis based on inflammatory component. [13]**

Inflammatory Tinea capitis	**Non inflammatory Tinea capitis**
Kerion Favus	Gray patch Black dot

Clinical type	**Clinical features**	**Most common agent**
Gray patch	Circular patches of partial hair loss with dull gray,lustreless,broken hairs.	*M.audonii*

Black dot	Black dot appearance due to broken hairs with diffuse scaling of the scalp and areas of polygonal hair loss.	*T.tonsurans,T.violaceum*
Kerion	Boggy, tender, induarted swelling consist of broken or unbroken hairs.	*T.mentagrophytes,* *T.verrucosum,* *M.canis*
Favus	Characterized by yellow cup shaped crust with central depression surrounding the hair that merges to form prominent dense masses with mossy odor.	*T.schoenleinii*

Inflammatory Tinea capitis

Kerion (Kerion celsi):

The clinical presentation includes pustular folliculitis (agminate folliculitis) to kerion formation. “It is an inflammed boggy,tender swelling that is studdded with broken or unbroken hairs, vesicles,pustules resulting in matting of adjacent hair and formation of sinus.” Lymphadenopathy and secondary bacterial infections can occur frequently. It heals mostly with scarring. Dermatophytid reactions may occur in patients. Eczematous scaly patches or plaques, follicular popular lesions, pustules are the various forms of id eruptions which occur before or after initiation of systemic antifungal therapy. [13,27,28]

Favus:

It begins early in life and extends to adulthood. Clinically present as yellow cup shaped crusts with central depression consist of mycelia and epithelial debris. Due to its shield-like shape it is known as scutulum. A hair pierces the cup with concavity facing upward, which surrounds the opening where the cup has formed. The scutulae enlarges and merges,resulting in formation of yellowish crusts with a central healing with scar (cicatricial alopecia). Other presentations include pityroid favus (desquamation in large plaques); papyroid favus (adherent parchment like crusts); impetignoid favus (suppurative crusty lesions). [13,29]

Non inflammatory Tinea capitis

Gray patch Tinea capitis:

Patients with these lesions are asymptomatic or present with mild itching sensation. Initially present as small erythematous papules surrounding the hair shaft and spread centrifugally. Clinically present as circular patches with numerous broken hairs and overcoating with arthrospores showing dull gray and lusterless appearance, fine scaling with minimal inflammation. [30]

Black dot Tinea capitis:

Clinically present with minimal hair loss, inflammation and diffuse scaling. Hair shaft breaks at the level of scalp and it is extremely brittle.On clinical examination, remaining hair in the infected follicle appears as a black dot. [13]

TINEA FACIEI:

Tinea Faciei is dermatophytoses of the glabrous skin of face which exclude mustache and beard areas in an adult male. Patients present with itching, burning sensation and photosensitivity. Lesions initially appear as erythematous scaly macule that extends peripherally and progress

to annular plaque with raised borders. [13] Scaling is evident in less than two-thirds of cases. Atypical features are more commonly seen in tinea faciei due to corticosteroid usage, invading agent, host immunity, site of invasion, excessive washing and sun exposure. [31] Erythematous plaques with vesiculopustules, plaques with broad edges and instinct central clearing are the atypical presentation seen. [31]

TINEA CORPORIS:

Synonyms: Tinea glabrosa, Tinea circinata

The occurrence of dermatophytosis on the trunk, neck, arms, or legs is defined as tinea corporis. [31] Typical lesion of tinea corporis is characterized by an annular or polycyclic pattern with a clear center surrounded by an erythematous and vesicular or scaly border. The clinical pattern is modified by the defective cellular immune responses. It shows variable degree of inflammation based on the species of fungus, immune status of the host and extend of follicular invasion. The lower trunk area including lower abdomen, waistline, gluteal region are the common site involved. The occluded areas or areas of trauma are affected if the tinea corporis is caused by anthropophilic organism but in case of infection caused by zoophilic organism exposed skin (head, neck, face arms) are affected. [13]

Tinea profunda, Tinea imbricata, Bullous tinea corporis, Psoriasiform, Lichenified plaques, Eczematous type, Majocchi's granuloma, Vasculitis like lesions and Erythema induratum like plaques are the clinical variants of Tinea Corporis. [13]

Tinea Profunda:

Similar to a kerion on the scalp, tinea profunda is the outcome of an overly inflammatory reaction to dermatophytes. It could appear verrucous or granulomatous. frequently confused with a dimorphic fungal infection, or cutaneous tuberculosis, squamous cell carcinoma. [32]

Tinea Imbricata (Tokelau):

The anthropophilic dermatophyte *T.concentricum* is the source of this persistent superficial dermatophytytic infection.[24] Clinically present with concentric annular rings resembling erythema gyratum repens. It is postulated that the development of this fungal infection occurs under the influence of genetic, environmental, and immunologic factors. Majority of the patients have autosomal recessive mode of inheritance with a minority having an autosomal dominant pattern. There is a decrease in the cellular immunity suggested by the presence of specific antibodies to *T.concentricum*. It also occurs associated with iron deficiency, dietary influences, and malnutrition. [32]

Majocchi's Granuloma:

It is defined as a deep dermatophytic folliculitis with disruption of the follicular wall. The long-standing superficial fungal infection disseminates into subcutaneous tissue.[33] Most frequently caused by *T. rubrum*. [33] Trauma induced mechanical injury of skin facilitates fungal penetration into the reticular dermis by causing cellular death and a drop in dermal pH, which creates an environment more conducive to fungal survival. Immunocompromised hosts are easily susceptible. It occurs due to Immunosuppression by topical steroid application. [32]

TINEA CRURIS:

Synonyms: 'Dhobi's itch', jock itch, gym itch, eczema marginatum

Tinea cruris is dermatophytoses of inguinal region, affecting the inner aspect of thighs (upper) and crural folds.

A warm, humid environment in the groin area promotes the growth of fungi. Most commonly seen in males.

TINEA MANUUM:

The dermatophytic infection of palm or interdigital folds of one or both hands is known as Tinea manuum. Mostly it occurs associated with tinea pedis, but rarely localized forms may solely affect the hands.

Table 8 **:Clinical types of Tinea manuum:-**

Clinical types of Tinea manuum	**Clinical presentation**
Non-inflammatory squamous form	Diffuse hyperkeratosis, with accentuation of the flexural creases and hyperhidrosis of palms and fingers.
Inflammatory vesicular/dyshydrotic/eczematous form:	Vesicles, usually in clusters and frequently multiloculated are present over the palms. The lesion is arranged in an annular or segmental pattern.

TINEA PEDIS:

Synonyms: Ringworm of the foot, Athelet's foot

Tinea pedis is dermatophytic infection of the feet or toes. [(22)] It is mostly seen in adults than in children.

Table 9 **:Types of Tinea pedis:-**

Types of Tinea pedis	**Clinical presentation**
Chronic intertriginous type: (Commonest type)	Fissuring, scaling, maceration in interdigital or sub digital areas.
Chronic papulosquamous type:	Patchy or diffuse moccasin-like scaling over the soles associated with inflammation.
Vesicular or vesiculobullous type:	Vesiculopustular or studded by small vesicles on the mid-anterior plantar surface of foot.
Acute ulcerative variant:	maceration, weeping, denudation and ulceration of sizable areas in the soles.

TINEA UNGIUM AND ONYCHOMYCOSIS:

Dermatophytic infection of the nail is clinically defined as Tinea ungium. The Greek words onyx (nail) and mykes (fungus) are the origins of the word onychomycosis. Infections of the nail caused by dermatophytes, non-dermatophyte molds and yeasts is referred as Onychomycosis. [13,34]

Clinical types of Onychomycosis- [13,34]

- Distal and lateral subungual onychomycosis (DLSO)
- Proximal subungual onychomycosis (PSO)
- White superficial onychomycosis (WSO)
- Endonyx onychomycosis (EO)
- Total dystrophic onychomycosis (TDO)

Table 10 : **Clinical types of Onychomycosis**

	Distal lateral subungual	**White superficial**	**Proximal subungual**	**Endonyx**	**Total dystrophic**

Nails affected	Toenail and fingernail	Toenail	Toenail	Fingernail and toenail	
Entry	Hyponychium	Direct nail plate invasion	Proximal nail fold	Distal free edge of nail	total destruction of the entire nail apparatus
Clinical features	Subungual hyperkeratosis of distal free edge or lateral nail fold area and onycholysis	Well-circumscribed powdery white patches.	Whitish to brownish discoloration of proximal nail plate.	Milky white patchy lesions on nail plate without subungual hyperkertosis or onycholysis	Diffusely thickened, friable, dystrophic, crumbled nail plate
Special features	Melanonychia if caused by melanin-producing agents	Friable surface	Associated with HIV disease.	Rare type	end-stage of onychomycosis that follow other subtypes.

TINEA INCOGNITO:

Tinea incognito is dermatophytic infection which lost its typical appearance due to topical usage and systemic immunosuppressants. Ive and Marks initially described it in 1968.

Topical steroids application causes immunosuppression and decreases inflammation, modified clinical appearance, transforming the typical lesions and mimicking other skin disease. Clinically pruritus is reduced, raised margins, scaling is lost and erythema is reduced. Topical steroid application results in a significant change in the nature of the lesion whereas, systemic steroids cause only slight changes in appearance. Both of which make it challenging to diagnose and provide the appropriate treatment.

Chronic Dermatophytosis

Synonyms: Tinea corporis generalisata, tinea rubrum syndrome, dry-type tinea rubrum infection and generalized chronically persistent rubrophytia. [22]

Persistent dermatophytosis with remission and exacerbation episodes over an extended period of time is referred to as chronic dermatophytosis. Despite the lack of a universally accepted definition, chronicity can be defined as duration and frequency of infection recurrences. Numerous pathogenic agent, host, and pharmacologic factors may have contributed to the emergence of such cases. [13,22]

TRICHOPHYTON RUBRUM SYNDROME

It involves at least 4 body sites, hands (palmar),feet (plantar),nails and an additional site, excluding the inguinal area. All four sites have microscopic fungal detection, and at least three of the four sites have positive cultures. [26] *T. rubrum* is ubiquitous and resilient fungus. It has arsenal of factors which helps to evade host immune system. It survives as a spore on the body until it finds a warm, wet area of the skin, where it quickly colonizes. Adhesion between arthroconidia and keratinocytes initiates dermatophyte invasion of the epidermis, which is then followed by penetration and proliferation. [8]

“Two Feet One Hand Syndrome”: -

Skin of one hand and both feet are infected in “Two feet one hand syndrome”, which is a form of tinea pedis.Toenails and/or fingernails are also frequently involved. [26] Causative organisms are Trichophyton rubrum Trichophyton mentagrophytes. Clinically it presents as chronic bilateral, papulosquamous lesion with minimal inflammation, patchy or diffuse mocassin-like scaling. In cases of "two feet, one hand syndrome," tinea pedis typically appears before tinea manuum.

Tinea Indecisiva/ Tinea Pseudo Imbricata:

The dermatophytes other than *Trichophyton concentricum.* causes “Tinea Pseudo Imbricata”.[35] Lengthy duration of alternating topical steroids and antifungals may result in a clinical picture resembling tinea imbricata. Widespread circular concentric erythematous rings that are caused due to cyclical immunosuppression from topical corticosteroids and reinfection from early topical antifungal medication withdrawal are its defining feature.

Laboratory investigations

Dermatophytosis is primarily diagnosed clinically, but laboratory tests are used to determine the etiology when morphology is altered.

- Wood’s lamp
- Potassium Hydroxide (KOH) wet mount for direct microscopy
- Dermoscopy
- Histopathological examination
- Fungal culture

Newer methods include:

- Polymerase chain reaction.

- Matrix-assisted laser desorption ionization-time of flight mass spectrometry.
- Reflectance confocal microscopy.

Wood's Lamp Examination

Wood's lamp identify dermatophytes by emitting long-wave ultraviolet light. due to pteridine induced fluorescence.

Uses of Wood's lamp in dermatophytosis

It helps in the diagnosis of inconspicuous scalp lesions.

Apart from the diagnostic utility, Wood's lamp helps in the selection of infected skin and hairs for laboratory investigation.

Dermatophytoses can be differentiated from other clinically similar-looking non-fungal skin conditions. e.g., erythrasma.

Fluorescence causing dermatophytes are generally members of the Microsporum genus.

Tinea capitis: Blue-green (most Microsporum species).Occasionally dull yellow (Microsporum gypseum) and dull blue *(T. Schoenleinii)*.

Potassium Hydroxide (KOH) wet mount for direct microscopy

10–20% potassium hydroxide (KOH) to a skin specimen is a simple and low-cost bedside method to demonstrate dermatophytic infection. Positive scrapings have hyphal filaments that are refractile, long, smooth, undulating, branched with or without arthroconidiospores.15% of instances have false negative outcomes. [36]

Fluorescence staining using optical brighteners (diaminostilbene) is the sensitive method for microscopically identification of fungus. The main component of fungal cell walls, chitin, is bound by these chemicals. [37]

Dermoscopy

Dermatophytosis is limited to stratum corneum of skin, hair, and nail. [38]

Table 11: **Dermoscopic findings of dermatophytosis**

Types	Dermoscopic findings
Tinea Corporis	Diffuse erythema, with whitish scales, follicular micropustules. Brown spots surrounded by a white-yellowsish halo. Wavy and broken hairs, Morse code hairs of vellus hairs.
Tinea Cruris	Similar to Tinea corporis, Morse code hairs
Tinea Capitis	Comma hairs, corkscrew hairs, zigzag hairs, black dots. Short vellus, bar code (morse code hairs), inter follicular scales. Follicular pustules or Abscesses present as Kerion, Cigarette ash hairs.
Tinea Pedis/Manuum	Brownish scales displaying dry vesicles, whitish scales in the palmar and plantar creases. Areas of intense erythema.
Tinea Unguium	Spikes, longitudinal striae of various colors (aurora borealis pattern),pseudoleuconychia. Proximal jagged edge of onycholysis.
Tinea Incognito	Morse code hairs of vellus hairs with follicular micropustules. Concentric areas of erythema with scaling. Easily deformable hairs which is weak and translucent with unusual bends.

Histopathology

Tinea capitis and Tinea barbae:-

Colonization of stratum corneum of perifollicular epidermis initiates follicle infection. Initially

enter the cuticle in subcuticular region of cortex, hyphae spread outward from the follicle and infiltrate the hair, reaching the upper limit of the zone of keratinization. In endothrix infections (*T. tonsurans or violaceum*), rounded, box-like arthrospores can be detected inside the hair shaft. They penetrate the hair shaft's surface. It create a sheath around it in ectothrix infections. The dermis has a perifollicular mononuclear cell infiltration of variable intensity, although fungi are rarely seen there. Dermis may contain neutrophils and multinucleated giant cells in case of follicular disruption. [39]

Kerion celsi-The zoophilic fungi cause a marked inflammatory tissue reaction. This is indicated by interfollicular neutrophilic infiltration,severe chronic inflammatory infiltration and pustule production around hair follicles. [39]

Tinea of the glabrous skin- fungi occur only in the horny layers of the epidermis without invading hair follicles. Except *T. rubrum and T. verrucosum*, both of which can invade hair follicles, resulting in perifolliculitis. [39]

Only hyphae are seen in infections caused by Microsporum or Trichophyton, whereas chains of spores are observed in infections caused by E. floccosum. If fungi are found in the horny layer, it is typically "sandwiched" between the cornified cells, with the lower zone having some parakeratotic cells and the top zone being orthokeratotic. [39]

The“sandwich sign” indicate for the application of a fungal stain for confirmation. The presence of neutrophils in the stratum corneum is also an indicator for diagnosis. The histologic findings are simiar to acute, subacute, or chronic spongiotic dermatitis. [39]

Majocchi granuloma or nodular perifolliculitis. It is caused by *T.rubrum.* It shows numerous hyphae and spores within hair follicles on staining with PAS or GMS. The spores located in dermis, within multinucleated giant cells, have a diameter up to 6 μm and spores within hair follicles have a diameter of around 2 μm. Hyphae and spores can be seen within hair follicles in PAS-stained sections of agminate folliculitis caused by T. verrucosum (faviforme).

However, the dermis around hair follicles does not contains fungi. In well-established lesions, the inflammatory infiltrate contains numerous plasma cells, microabscesses, and small aggregates of foreign body giant cells.

Tinea unguium, Nail plate biopsies are the preferred diagnostic technique. Inadequate sampling causes false-negative results.The fungus on PAS-D-stained sections is frequently seen in nail clippings or nail biopsies obtained using the punch technique or scalpel under local anesthesia. Slender, uniform mycelial components are visible in the parakeratotic nail plate (PAS-D stain).

Fungal culture

The gold standard method of diagnosis of dermatophytosis is isolation and identification of dermatophytes from the clinical samples. Culture is usually done for academic purposes. Dermatophytes can be cultivated in an artificial media containing an organic source of nitrogen. (40)

Most Commonly used media are as follows: (40)

1.Sabouraud's dextrose Agar (SDA)

2.Emmon's modified SDA

3.Potato flake agar:

4.Dermatophyte test media (DTM)

Sabouraud's dextrose Agar (SDA): (40)

The most commonly used medium. SDA medium includes: -

● Peptone 1%

● Dextrose 4%

● Agar 2%

● PH 5.6

● Chloramphenicol 0.05 g/L

● Cycloheximide 0.5 g/L

Emmon's modified SDA:

It consists of: - [40]

● 2% Dextrose instead of 4%

● Neopeptone instead of peptone

● PH: 6.8-7

Chloramphenicol prevents contamination by bacteria. Cycloheximide inhibits the growth of non dermotophytic molds. Cycloheximide free medium should be used for isolation of non-dermatophytic mold infections in palms, soles and nails.

Potato flake agar:

It promotes rapid conidiation and colony pigment development. [32]

1.**Microscopic examination dependent techniques**

- Bright-field light microscope
- Phase-contrast light microscope
- Fluorescence microscope

2.Modified rapid culturing techniques

- Dermatophytes test medium (DTM)
- Dermatophytes identification medium (DIM)

- Screening culture-slide method

3.Matrix-assisted laser desorption/ionization-time of flight mass spectrometry-based techniques (MALDI-TOF)

4.Molecular diagnostic techniques

Conventional PCR

Quantitative/real-time PCR (qPCR)

Nested PCR

Multiplex PCR

PCR-ELISA

PCR-Restriction fragment length polymorphism (PCR-RFLP) [38]

5.Lateral flow-based techniques [38]

- Immunochromatography lateral flow assay (ILFA).
- Nucleic acid lateral flow immunochromatographic assay (NALFIA).
- Nucleic acid lateral flow assay (NALFA).

Modified rapid culturing techniques: -

Dermatophyte test media (DTM)

The earliest media developed for rapid presumptive dermatophyte identification. [41]

Dermatophytes identification medium (DIM)

A DTM modification that circumvents the non-specific and false-positive NDM is a comparatively quick medium for dermatophyte presumptive identification. In this medium

incubation is done at 37°C and concentration of cycloheximide was increased and has higher sensitivity and specificity.[41]

MALDI-TOF

This molecular approach is extremely reliable, precise, simple to use, and straightforward to include into the standard laboratory workflow. Drawbacks are high cost, long-time consumption and low specificity.

Molecular diagnostic techniques

1. Conventional PCR

It is highly specific, sensitive, accurate diagnosis and low cost compared to other molecular techniques. Species-specific primers are used to detect dermatophytes at the diagnostic level. The mostly used pan dermatophyte primers are Internal transcribed spacer (ITS) and 28s ribosomal DNA. This is followed by further outcome amplicon size determination by gel electrophoresis. Disadvantage of conventional PCR is it requires post-amplification steps and quantitative analysis of fungus is not possible.

2. Quantitative/real-time PCR (qPCR)

This molecular technique is a very sensitive, specific, and precise method for quantifying the fungal load of an infected sample. It is less susceptible to contamination and relies on a pair of primers and labeled probes that target species-specific genes. It has higher sensitivity and specificity.[42]

3.Nested PCR

Several targets nested PCR has been built up with the goal of increasing PCR specificity in the diagnosis of dermatophytosis, particularly in the use of pan primers. [42]

Following the initial primer pair amplification cycle, a primary amplicon will undergo a second cycle using a different primer pair. The resulting primary amplicon serve as a template for

second set of primers in second cycle. [42]

4.Multiplex PCR

It is used in sample limitation to detect multiple causative agents in the same sample. PCR reaction with two or more sets of primers are used. [42]

5.PCR-ELISA

A hybrid method that combines PCR with ELISA, wherein a labelled nucleic acid amplicon is utilized on microculture ELISA plates in place of the target analyte protein. Despite its extensive use and time commitment, it was less expensive per sample than qPCR. It requires more time and effor. It is not considereds for routine diagnosis.

6. **PCR-Restriction fragment length polymorphism (PCR-RFLP)**

The primary element of this assay is the amplification of target sequences, which vary depending on the species. The amplicon was subsequently treated with particular restriction enzymes, and the molecular weight of the bands was compared to a standard ladder using either amplicon sequencing or gel electrophoresis. This approach is not included in the standard dermatophyte diagnosis scheme because to its time-consuming, exhaustive, and arduous nature, as well as its demand for restriction enzymes.

Lateral flow-based techniques.

It is used for the rapid detection of various infectious and agents, fulfilling World Health Organization's (WHO) ASSURED criteria: “affordable, sensitive, specific, user-friendly, rapid and robust, equipment-free, and deliverable to end users”.

Treatment of dermatophytosis

Dermatophytic infection has a straightforward treatment as it is a common occurrence of the skin and its appendages but lately, it has become notably challenging due to chronicity and

recurrence. Treatment options available are monotherapy, combination therapy, or sequential therapy. [43]

General measures [44]

Steroid-containing antifungal preparations must be strictly avoided. Strong counseling emphasizing the clear dangers of these FDCs is required.

- Wearing tight clothing, such as jeans, leggings, and jeggings, is discouraged.
- Clothing should be loose fitting, cotton or synthetic garments.
- Refrain from sharing clothes, towels, or bed linens. Towels and bed linens should be regularly washed.
- Taking regular showers. Putting on clothing only when the body has completely dried
- Bed linens and clothing should be cleaned in hot water and dried in sun.
- Dermatophytes are known to be destroyed by sunlight. In the absence of sunlight, ironing clothes might also be beneficial.
- Patients with tinea cruris are advised to wear "boxer shorts" rather than form-fitting ones.
- Clothes should be dried inside out. Wearing well dried inner garments.
- Washing infected clothes separately.
- Use of absorbent powders and deodorants (decrease perspiration) can be encouraged.
- Remove waistband, wristband, etc.
- Advice footwear which are nonocclusive.
- The spore load in the environment can be reduced by vacuuming, wet mopping, cleaning the house before using detergents.

Topical therapy [44]

Topical antifungals and systemic antifungals are used generally as a combination therapy.

Indications for topical antifungal agents.

- Localized lesions.
- Hepatic failure/severe systemic disease in which systemic antifungals are contraindicated.

Topical therapy includes azoles, triazoles, allylamines, imidazoles, tolnaftate, benzylamines, ciclopiroxolamine.

"The topical antifungals should be applied 2cm beyond the margin of the lesion for atleast 2 weeks beyond clinical resolution- **Rule of Two".**

Systemic therapy [44]

Indications of systemic therapy: -

- Multiple site involvement.
- Tinea corporis is extensive.
- Localized infection unresponsive to topical antifungal agents.
- Recurrent or chronic dermatophytosis
- Tinea capitis, Tinea unguim, Tinea pedis
- Immunocompromised

Systemic treatment options available against dermatophytes are itraconazole, terbinafine, griseofulvin, ketoconazole and fluconazole.

Table 12 : **Treatment of Cutaneous Tinea-**[43]

Topical therapy	Azoles OD or BD for 2-4 weeks. Terbinafine 1% twice daily for 2 weeks.

Systemic therapy First line	Terbinafine for 2-4 weeks 62.5mg/day for weight<20 kg, 125mg/day for weight 20-40kg 250 mg/day for weight >40 kg or3-6mg/kg/day OR Itraconazole 5mg/kg/day for 1to 2weeks.
2nd line	Griseofulvin >1 month of age: 10-20mg/kg/day for 2 to 4 weeks.

Treatment of Tinea Capitis [43]

Systemic therapy First line	Griseofulvin-higher efficacy against *Microsporum* species. Dosage:weight <50 kg :15-20 mg/kg/day for 6 to 8 weeks. Weight >50 kg :1g/day for 6 to 8 weeks. Terbinafine-higher efficacy against *Trichophyton* species.

	Dosage:Weight <20 kg:62.5 mg/day for 2 to 4 weeks. Weight 20-40 kg:125mg/day for 2 to 4 weeks. Weight >40 kg:250 mg/day for 2 to 4 weeks.
Alternative systemic therapy	Itraconazole -effective against both *Trichophyton* and *Microsporum* species. Dose:50-100mg/day for 4 weeks or 5mg/kg/day for 2 to 4 weeks.
Topical therapy (only to prevent transmission)	2% ketoconazole or 1-2.5% selenium sulfide or 1-2% zinc pyrithione or 2.5% povidone iodine shampoo.

Treatment of Onychomycosis- [43]

Systemic therapy (First line)	First choice:Terbinafine (daily continuous) 62.5 mg/day for weight <20 kg 125mg/day for weight 20-40 kg 250 mg/day for weight >>40 kg or3-6 mg/kg/day 6 weeks for fingernails and 12 weeks for toe nail onychomycosis. Second choice: Itraconazole Pulse therapy (5mg/kg/day for one week every month) 2 pulses for fingernail and 3

	pulses for toenail onychomycosis or 5 mg/kg/day for 2 to 3 months.
Alternative systemic therapy	Fluconazole 3-6 mg/kg once weekly for 12 -16 weeks for fingernail infection and 18-26 weeks for toe nail onychomycosis. Griseofulvin Above 1 month of age:10 mg/kg/day for 6-9 months in fingernail and 12-18 months in toe nail onychomycosis.
Topical therapy	Ciclopirox 8%OD, Amorolfine 5% once/week, Effinaconazole 10% OD, Tavabarole 5% OD for 48 weeks.
Adjunctive therapy	1.Surgical/Chemical nail avulsion (Partial/Total) 2.Laser therapy (Nd:Yad/CO2) 3.PDT

Newer therapy

Properties of newer antifungal

- Drugs with pharmacological similarities to older medications that share target

molecules, a lower minimum inhibitory concentration, with specific indications (e.g., Isavuconazole, Micafungin, Luliconazole). [45]

- • The current medications, such as calcineurin inhibitors, target of rapamycin inhibitors, Hsp90 inhibitors and synergy with azoles, which use an ancient chemical with a well-established pharmacology either by itself or in combination with another medicine for recent uses. [46]

Table 13: **Newer antifungal drugs**

Newer antifungal	**Mechanism of action**
NB-002	Highly refined oil, ethanol, polysorbate 20, CPC, and water were combined to create the nanoemulsion. Broad spectrum, uniform, rapid fungicidal action against dermatophytes, candida albicans. Active against terbinafine-resistant *Trichophyton rubrum* [47]
PH 151 and PH 153	8-Hydroxyquinolone derivatives. Lipophilic. Acts on the cell walls of dermatophytes by metal chelation. [48]
Efinaconazole	10% solution approved by FDA for onychomycosis. [49]

Fosravuconazole	Oral triazole, prodrug of ravuconazole. Potent broad-spectrum and antifungal activity. Blocks ergosterol biosynthesis. lower inhibitory effect on cytochrome 3A4 than itraconazole. [48]
ME1111	(2-(3, 5-Dimethyl-1H-pyrazol-1-yl)-5-methylphenol) is a novel antifungal. It has fungicidal effect by specifically inhibiting fungal succinate dehydrogenase (complex II), an enzyme involved in mitochondrial respiratory electron transport that blocks generation of ATP.[50]
AR-12	Celecoxib derivative that inhibits acetyl coenzyme A synthetase enzyme. [51]
Tavaborole	fungal protein synthesis inhibitor that forms a boron-based bond at the enzyme-editing site to prevent catalytic turnover of leucyl-tRNA synthetase.[52]

MATERIALS AND METHODS

SOURCE OF DATA:

Patients with clinically diagnosed dermatophytic infection, attending outpatient department of Dermatology, Venerology and leprosy of B.L.D.E(deemed to be university) Shri B.M. Patil Medical College Hospital and Research Centre, Vijayapura were enrolled for the study.

Period of study:

The study was conducted during the period of May 2023 to January 2025

Study design:

A hospital based cross sectional study.

Sample size:

With anticipated Proportion of superficial dermatophyte infection among children under 18 years 19 %, the study would require a sample size of 95 subjects with 95% level of confidence and 8% absolute precision.

Formula used

$$n = \frac{z^2\, p*q}{d^2}$$

Where Z= Z statistic at α level of significance

d 2 = Absolute error

P= Proportion rate

q= 100-p

STATISTICAL ANALYSIS:

The data obtained was entered in a Microsoft Excel sheet, and statistical analysis was performed using JMP® Pro 16 software Version 16, SAS Institute., Cary, NC,1989-2021. Results were presented as Mean (Median) ±SD, counts and percentages and diagrams. Pearson Chi-square ($\chi 2$) test was used for association between two categorical variables.

METHOD OF COLLECTION OF DATA:

Inclusion criteria:

Children below 15 years of age clinically diagnosed to have dermatophytosis were enrolled for the study.

Methods:

Detailed history with respect to the onset and duration of dermatophytic infection and any treatment for infection was be recorded.

Initial clinical examination of the patient was done to determine the clinical type of dermatophytosis based on site of involvement. These findings were recorded in the proforma. Informed consent for the study was undertaken from the parents.

Methodology:

Specimen collection:

The affected area is swabbed with 70% alcohol, skin scales, crusts and pieces of nail were collected according to the involved site.

Skin specimen collected by scraping across the erythematous, peripheral, actively growing margins of the lesions. The skin scales are flaked onto a glass slide using a blunt edge of a sterile surgical blade.

Nail specimen collected after washing of hands with soap and water with emphasis on nails followed by drying and decontamination of nail with 70% alcohol taking clipping of the infected nail from nail bed, proximal nail plate and subungual region of the nail.

In case of hair involvement, the dull lusterless hair and stubs of hairs are chosen and plucked by sterile forceps'

Direct microscopic examination:

KOH mount:

Sample collected over a glass slide is taken and 10% KOH is added over collected material and covered with a cover slip and gently preheated before examining the fungi. In case of nail involvement 20%KOH is used. The samples after keratolysis are examined for the presence of filamentous, septate, branched hyphae with or without arthrospores.

Fungal culture:

The samples irrespective of demonstration of fungal elements by direct microscopic examination, are inoculated on test tubes containing.

1.Sabouraud dextrose agar without Chloramphenicol And cycloheximide.

2.Sabouraud dextrose agar with Chloramphenicol and cycloheximide

3.Dermatophyte Test Media.

They are incubated at 32◦c for a period of 4 weeks. The fungal cultures are identified by colony morphology, pigment production, rate of growth. Lactophenol cotton blue preparations are made to detect the presence of macroconidia, microconidia, chlamydospore and special hyphal structures.

RESULTS

A hospital based prospective study was conducted from May 2023 to January 2025 at a tertiary care centre in Vijayapura. 153 pediatric patients with clinical diagnosis of dermatophytosis were included in this study.

Age distribution

Children below 15 years of age clinically diagnosed to have dermatophytosis were enrolled for the study. The most commonly affected age group was 13-15 years. (30.7 %). Mean age is 9yrs with a standard deviation of 4.5

Age	No. of patients	Percentage
<1 year	8	5.2
1-2 year	6	3.9
3-4 year	22	14.4
5-6 year	14	9.2
7-8 year	14	9.2
9-10 year	18	11.8
11-12 year	24	15.7
13-15 year	47	30.7
Total	153	100.0

Table 14: **Age wise distribution of patients**

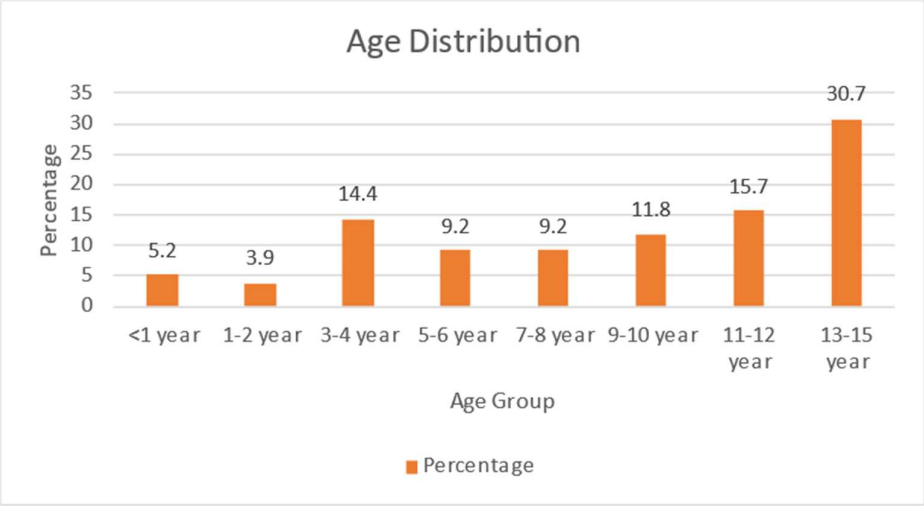

Figure 1: **Age wise distribution of patients**

Gender distribution

Out of 153patients enrolled in the study, 111 (72.5%) were males and 42 (27.5%) were females.

Male to female ratio was 2.65:1

Gender	No. of patients	Percentage
Female	42	27.5
Male	111	72.5
Total	153	100.0

Table 15: **Gender wise distribution**

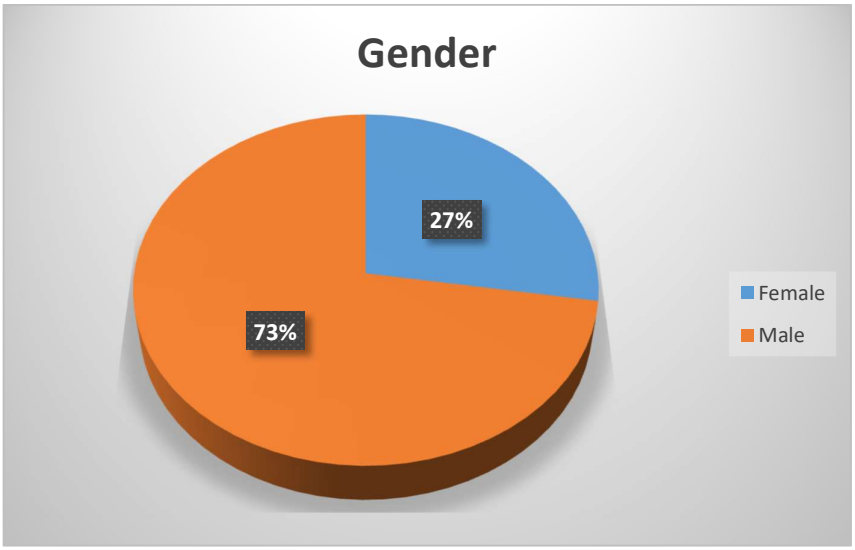

Figure 2: **Gender wise distribution**

Correlation Between Age and Sex Distribution

Maximum number of affected males belonged to the age group of 13-15 years with 39 males (35.1%) followed by 11-12 years with 17 males (15.3%). Affected females mostly belonged to 13-15 years age group with a total of 8 females (19.0 %).

Age (Years)	Gender		Total
	Female	Male	
<1 year	3 (7.1%)	5 (4.5%)	8
1-2 year	3 (7.1%)	3 (2.7%)	6
3-4 year	6 (14.3%)	16 (14.4%)	22
5-6 year	5 (11.9%)	9 (8.1%)	14
7-8 year	7 (16.7%)	7 (6.3%)	14
9-10 year	3 (7.1%)	15 (13.5%)	18
11-12 year	7 (16.7%	17 (15.3%)	24
13-15 year	8 (19.0%)	39 (35.1%)	47
Total	42 (100. %)	111(100.0%)	100.0

Table 16 :**Sex distribution in relation to age**

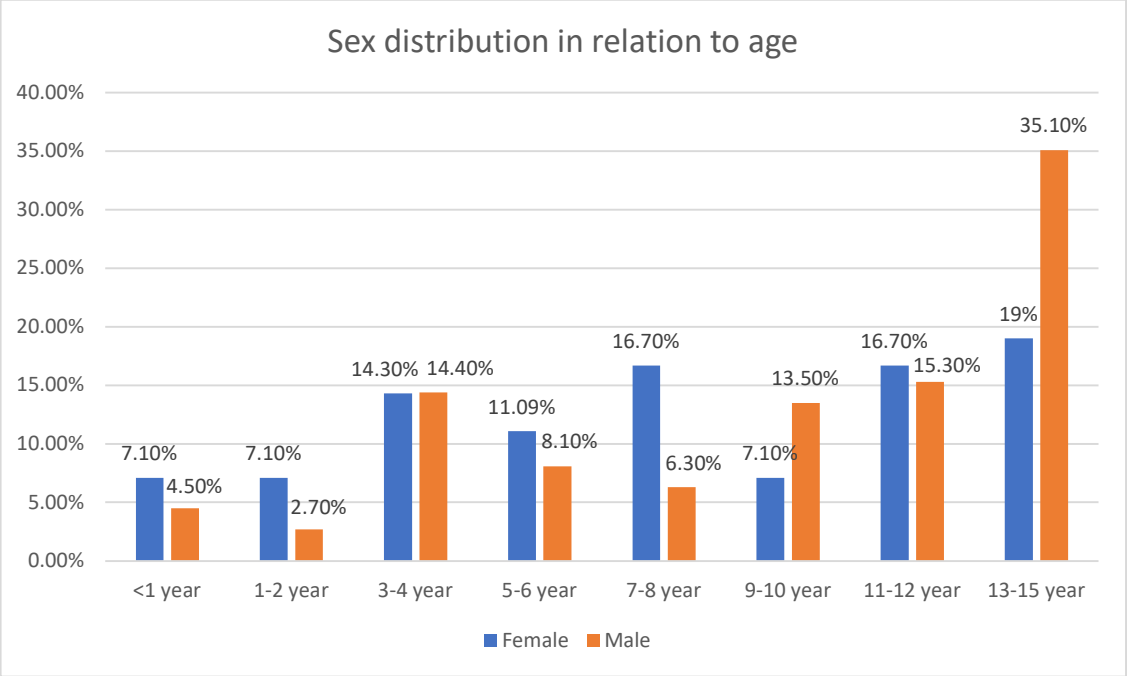

Figure 3: **Sex distribution in relation to age**

Socioeconomic status

Majority of patients 84 belongs to lower middle socioeconomic status (54.9%)

Socio economic status	No. of patients	Percentage
Lower	0	0
Upper lower	24	15.7
Lower middle	84	54.9
Upper middle	42	27.5
Upper	3	2.0
Total	153	100.0

Table 17**: Distribution of patients on the basis of socio-economic status.**

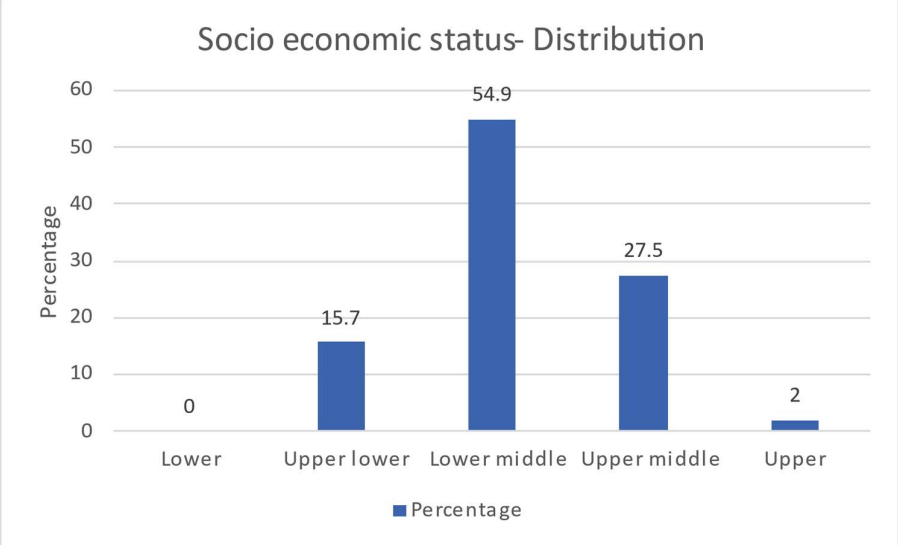

Figure 4: **Distribution of patients based on socio-economic status.**

Duration of Lesions

The duration of the infection varied from 2 weeks to 1 year. Majority had, 61patients (39.9%) infection for 1-2months.

Duration of lesion in months	No. of patients	Percentage
< 1.00	43	28.1
1 – 2	61	39.9
2- 3	21	13.7
3- 4	7	4.6
4– 5	3	2.0
5—6	0	0
6– 7	6	3.9
7—8	0	0
9. +	12	7.8
Total	153	100.0

Table 18: **Distribution of patients based on duration of lesions.**

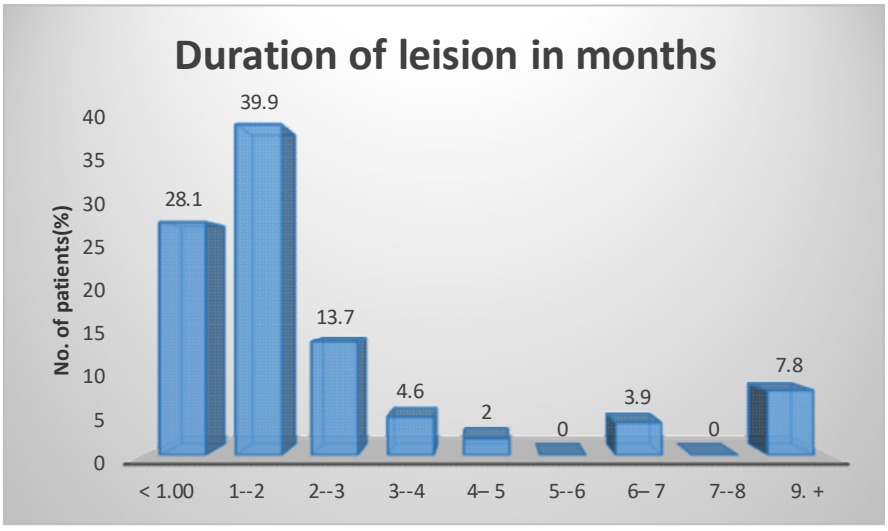

Figure 5: **Distribution of patients based on duration of lesions.**

History of previous medication

History of taking treatment of any form, prior to consultation at the tertiary care centre was observed in 83 patients (54.2%). Among them 7 patients (4.57%) had history of taking oral medications along with application of topical medication while 2 patients (1.30%) had used only oral medication and 31 patients (20.26 %) had history of application of topical steroid combination medication alone. History of either using medications, whose details were not known to the patient or usage native medication was seen in 43 patients (28.10%).

Type of medication	No. of patients	Percentage
Oral + Topical	7	4.57
Oral Medication	2	1.30
Topical steroid Combination	31	20.26
Unknown medication	43	28.10
Nil	70	45.7
Total	153	100.0

Table 19: **Distribution based on history of previous medication**

History of prior consultation

History of self-treatment by using medication taken from the pharmacy was elicited in 42 patients (27.5%). Treatment following consultation of a doctor for the skin ailment was seen in 34 patients, among them 26(17%) patients had consulted a general practitioner while 8 patients (5.2%) had consulted a dermatologist.

Consultation	No. of patients	Percentage
Nil	77	50.3
DERM	8	5.2
GP	26	17.0
OTC	42	27.5
Total	153	100.0

Table 20 : **Distribution based on prior consultation**

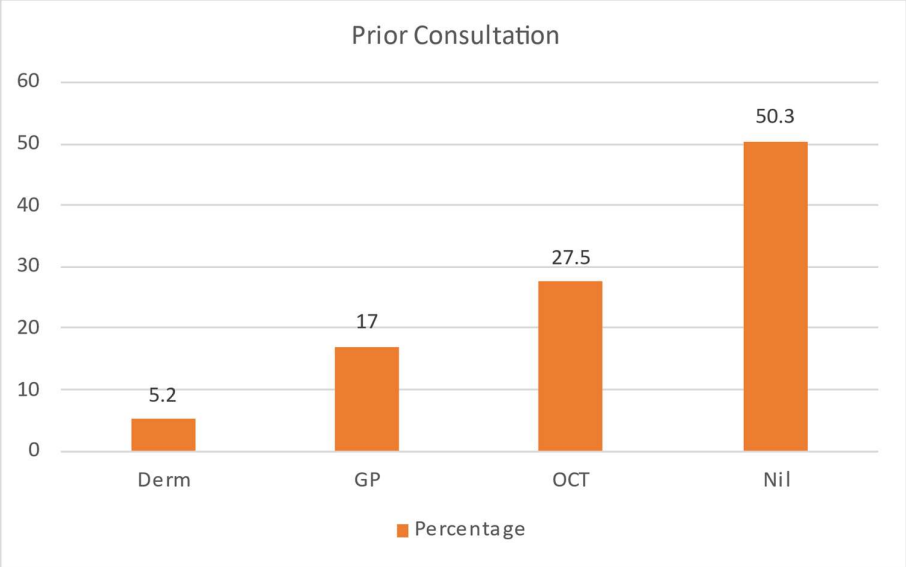

Figure 6: **Distribution based on prior consultation**

Past history of similar complaints

History of similar complaints in the past was present among 33 patients (21.6%). Among them 16 patients (10.5%) had similar complaints 1 year back, while it was present 6 months back in 11 patients (7.2%) and 3 months back in 6 patients (3.9%)

H/o similar complaints	No of patients	Percentage
Present	33	21.6
Absent	120	78.4
Total	153	100.0

Table 21a : **Distribution based on past history of similar complaints**

Duration of similar Complaint	No of patients	Percentage
3m back	6	3.9
6m back	11	7.2
1year ago	16	10.5

Table 21b : **Distribution based on duration of past history of similar complaints**

Family History

Similar complains among family members or close contacts was seen in 64 (41.8 %) patients.

Family history	No. of patients	Percentage
Present	64	41.8
Absent	89	58.2
Total	153	100.0

Table 22: **Distribution of patients according to Family history**

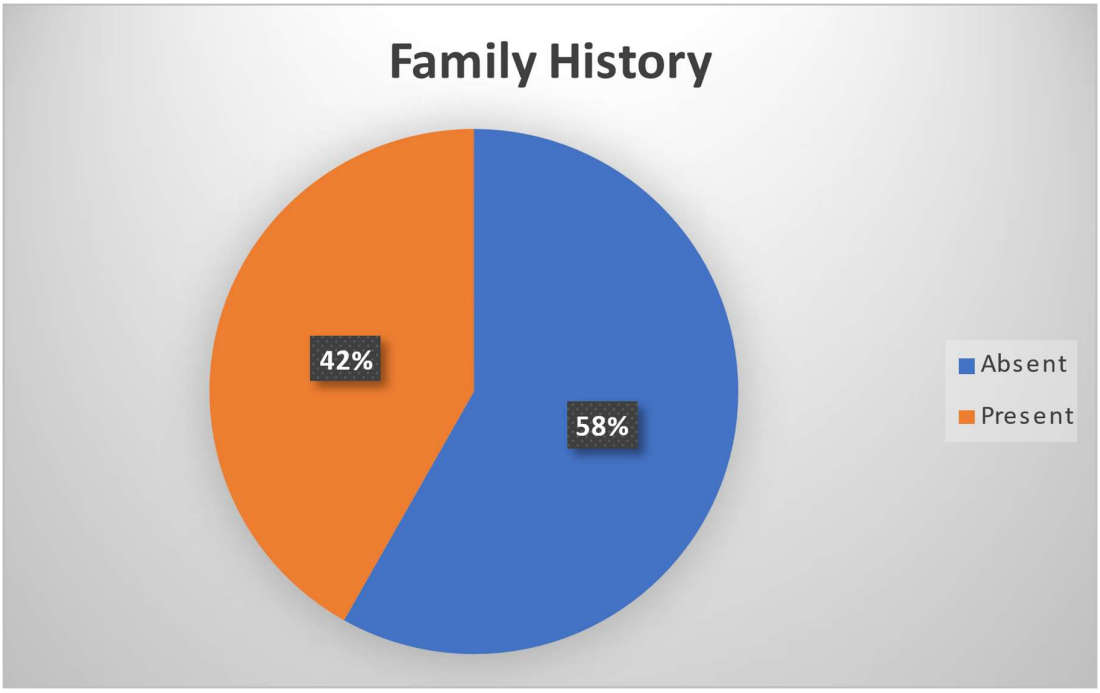

Figure 7: **Distribution of patients according to Family history**

Personal History

1.Personal hygiene

Out of 153 patients, 100 (65.4 %) patients gave history of taking bath everyday while 32 (20.9 %) patients gave history of taking bath on alternate days and 21 (13.7 %) patients took bath once in 2 or more days.

Frequency of Bathing (Days)	**No. of patients**	**Percentage**
Every day	100	65.4
Alternate days	32	20.9
>=2 days	21	13.7

Table 23: **Distribution of patients based on bathing history**

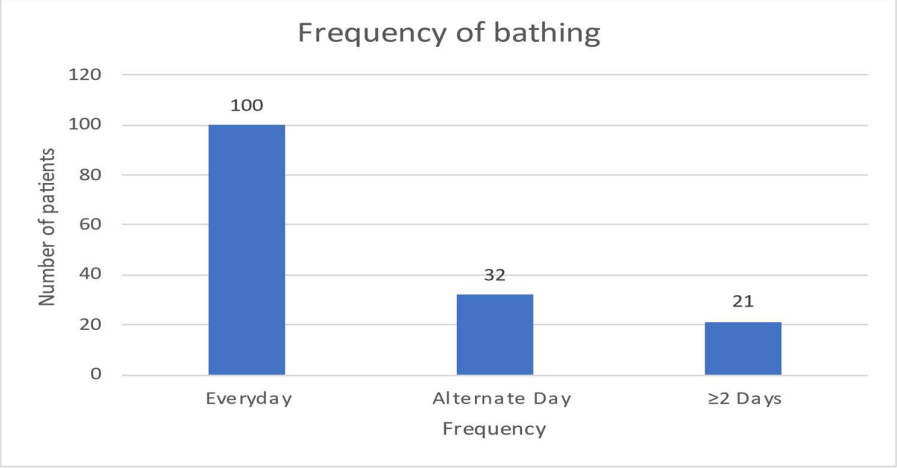

Figure 8: **Distribution of patients based on frequency of bathing**

2. **History of tight clothing**

Frequently wearing tight garments like leggings, jeans was seen in 68 patients (44.4 %). Tinea lesions were commonly seen in areas prone for occlusion due to tight fitting clothing.

H/o tight clothing	No of patients	Percentage
Present	68	44.4%
Absent	85	55.6%

Table 24 : **Distribution of patients based on history of tight clothing**

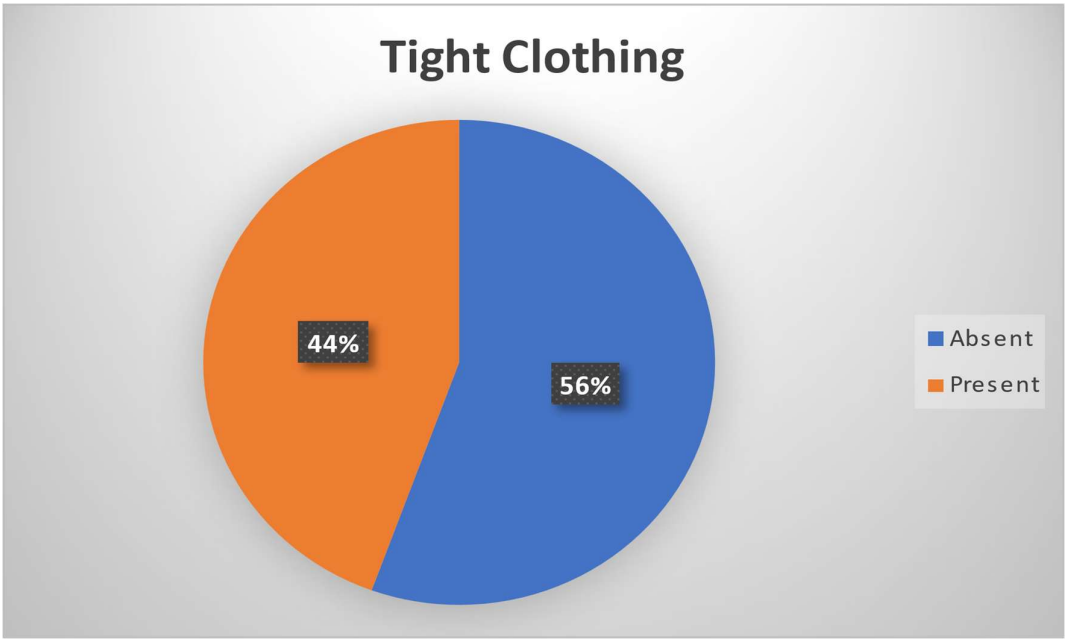

Figure 9: **Distribution of patients based on history of tight clothing**

3. **History of sharing fomites**

Sharing of clothes, combs, pillows and beds among close contacts or family members was habitual among 57(37.3%) patients.

H/o sharing fomites	No. of patients	Percentage
Present	57	37.3%
Absent	96	62.7%

Table 25: **Distribution of patients based on sharing of fomites**

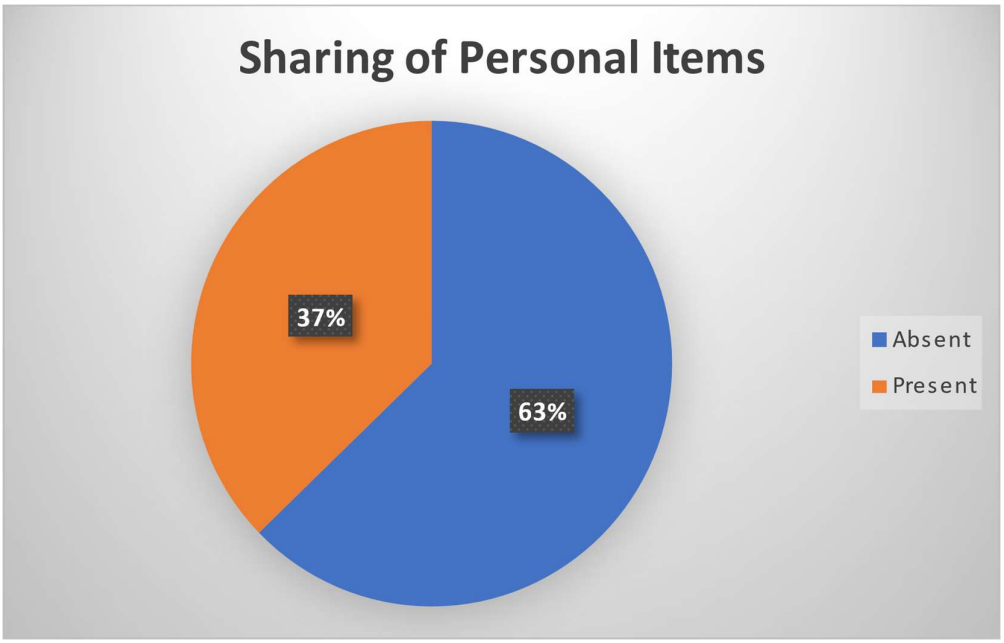

Figure 10: **Distribution of patients based on sharing of fomites**

4. **Pet history**

History of contact with animals (dog/cats) present in 47 patients (30.7%).

H/O contact with Animals	No. of patients	Percentage
Present	47	30.7
Absent	106	69.3

Table 26: **Distribution based on contact with pets**

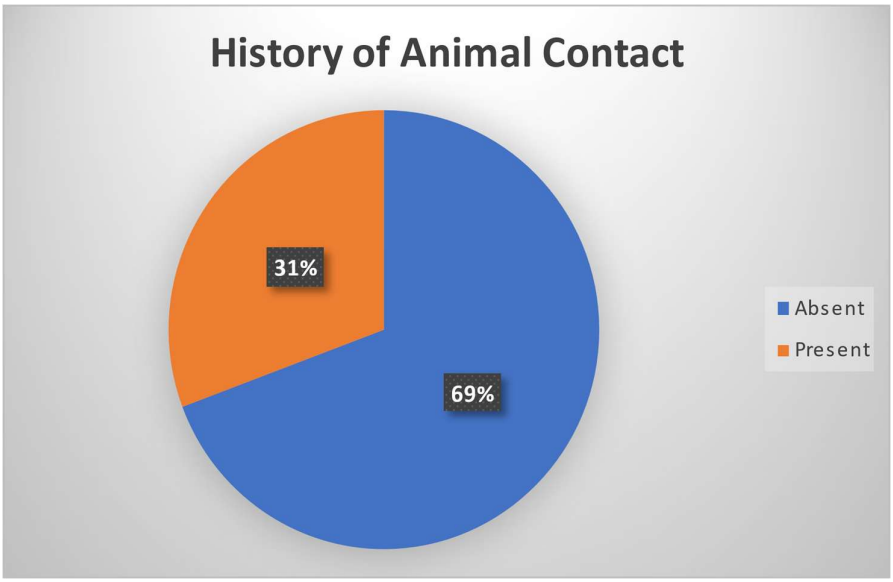

Figure 11: **Distribution based on history of animal contact**

Examination

Body Surface Area Involvement

24 patients (15.7 %) had body surface area (BSA) involvement of 5-10%, followed by 11(7.2 %) patients with less than 5% body surface area involvement while 7 patients (4.6 %) had more than 10% body surface area involvement.

BSA	No. of patients	Percentage
<5%	52	34
5-10%	73	47.7
>10%	28	18.3
Total	153	100.0

Table 27 : **Distribution of patients on the basis of BSA involvement**.

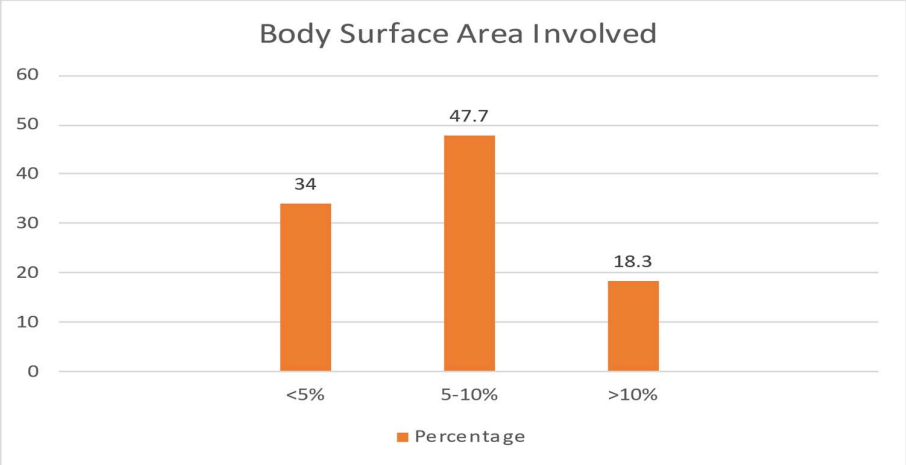

Figure 12: **Distribution of patients on the basis of BSA involvement.**

Cutaneous Examination

Most common cutaneous site of involvement was found to be Scalp (24.2 %) followed by Groin area (23.5%) while least common site over all was found to be Soles and Nails.

Site of presence of lesion	No. of patients	Percentage
Scalp	37	24.2
Face	23	15
Neck	9	5.9
Chest	5	3.3
Back	13	8.5
Abdomen	22	14.4
Upper limb(except hands	26	17.0
Lower limb(except feet)	24	15.7
Gluteal region	30	19.6
Groin	36	23.5
Dorsum of feet	5	3.2
Dorsum of hands	6	3.9
Palms	2	1.4
Soles	0	0
Nails	0	0

Table 28 : **Distribution of patients based on site of tinea lesion**

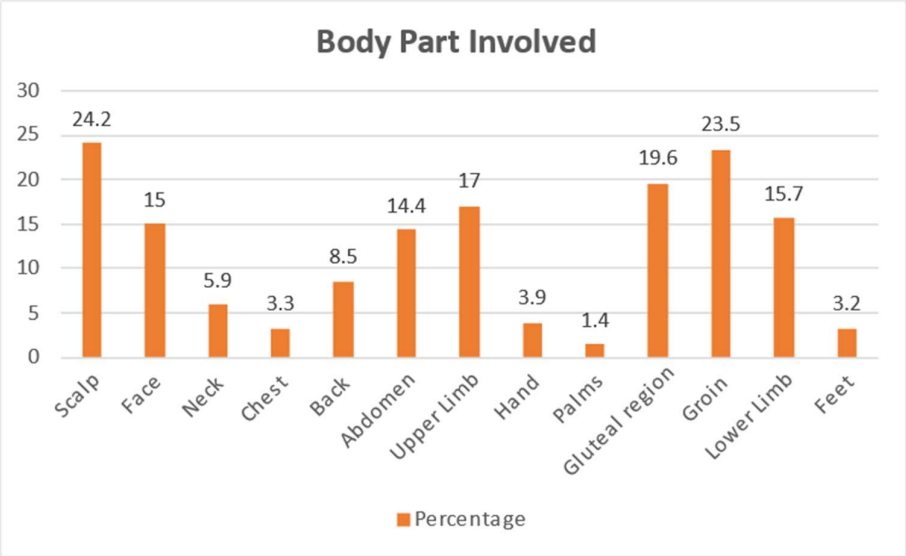

Figure 13 : **Distribution of patients on the basis of site of lesion.**

Scaling

Scaling over the lesion was present in 145 (94.8%) patients.

Scaling	No. of patients	Percentage
Present	145	94.8
Absent	7	4.6
Total	153	100.0

Table 29**: Distribution of patients based on presence of scaling**

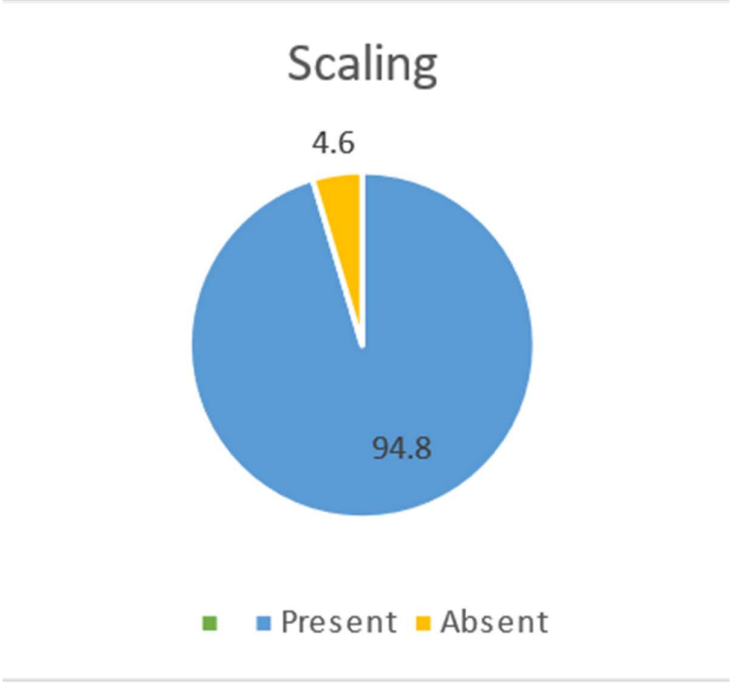

Figure 14 : **Distribution based on presence of scaling**

Atypical Presentation

Unusual presentation of tinea was seen in 19 patients (12.4 %) with 11 patients having eczema like ,4 patient with psoriasiform like tinea and 4 patients with Tinea pseudoimbricata.

Examination	No. of patients	Percentage
Eczema like	11	7.2
Psoriasiform like	4	2.6
T.pseudoimbricata	4	2.6

Table 30: **Distribution of patients based on atypical presentation**

Clinical Diagnosis

Most common clinical diagnosis was Tinea Corporis (82 %) followed by Tinea Capitis (22.2 %) while least common was Tinea pedis (3.3%) and Tinea unguim (0 %).

Examination	**No. of patients**	**Percentage**
Tinea capitis	37	22.2
Tinea faciei	23	15.0
Tinea corporis	126	82
Tinea cruris	36	21.6
Tinea manuum	6	3.9
Tinea pedis	5	3.3
Tinea unguim	0	0
Total	153	100.0

Table 31: **Distribution of patients according to Clinical diagnosis**

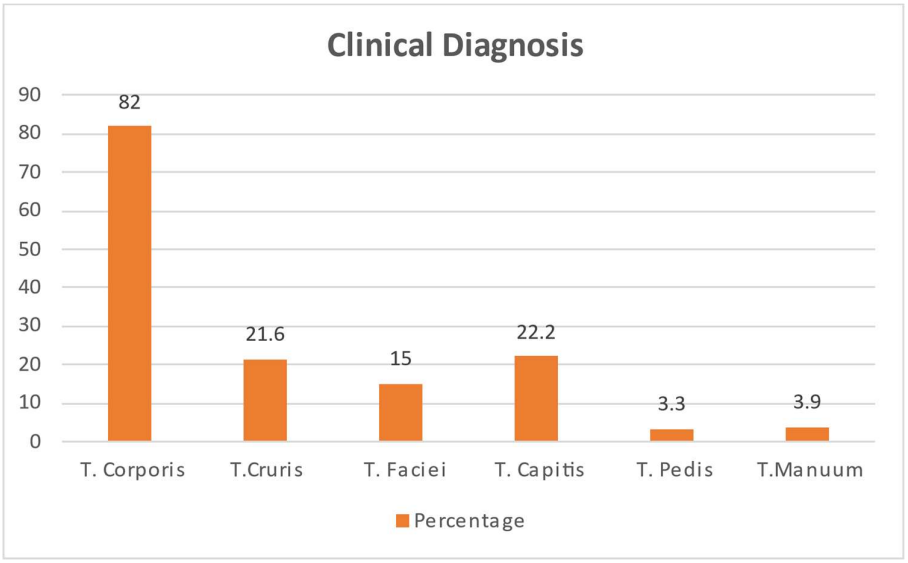

Figure 15 :**Distribution of patients according to Clinical diagnosis**

Mixed Clinical type of dermatophytosis

Most commonly seen mixed type of clinical diagnosis was Tinea Corporis along with Tinea Cruris, present in 23 patients (15.2 %) followed by Tinea Capitis along with Tinea Corporis seen in 4 patients (2.6%).

Clinical diagnosis	No.of Patients	Percentage
T. Corporis +T.Cruris	23	15.2
T.Capitis +T.Corporis	4	2.6
T.Capitis+T.Corporis+T.Cruris	1	0.7
T.Capitis+T.Cruris+T.Corporis+T.Faciei	1	0.7
T.Corporis+Cruris+Pedis	1	0.7
T.Corporis +Cruris +Pedis +Manuum	2	1.4
T.Corporis+T.Faciei	3	2.0
T.Corporis+.Manuum	1	0.7
T.Corporis+T.Manuum+T.Pedis	1	0.7
T.Cruris +T.Corporis+T.Faciei	1	0.7
Total	38	25.4

Table 32**: Distribution of patients based on mixed type of dermatophytosis**.

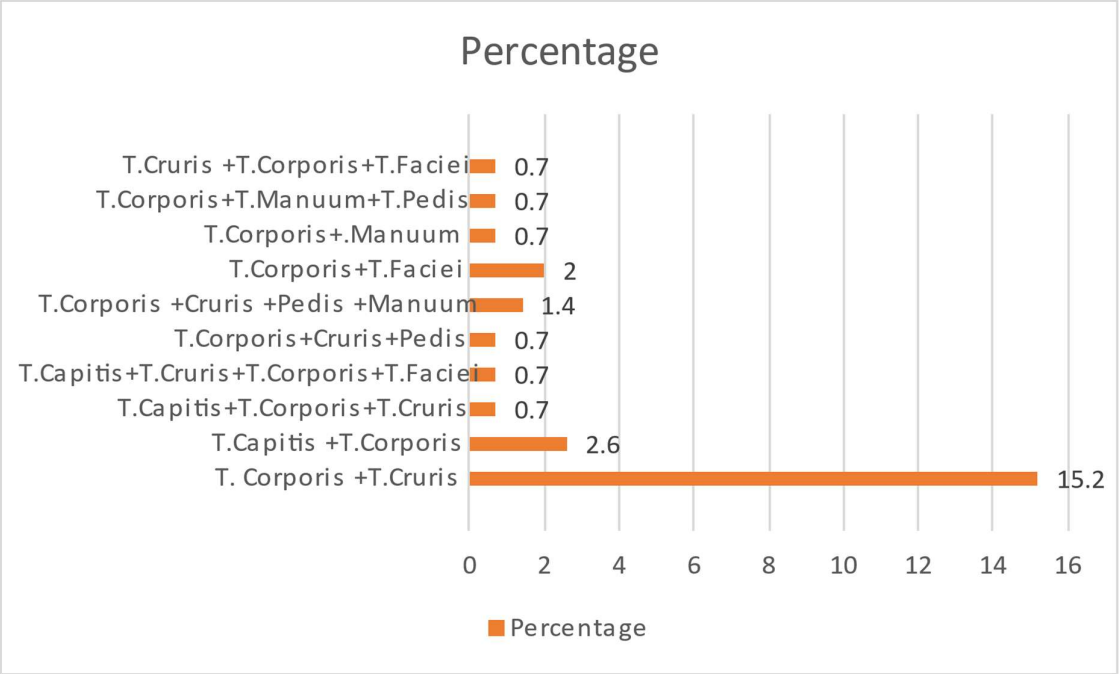

Figure 16: **Distribution of patients based on mixed type of dermatophytosis**.

10% KOH Mount for Direct Microscopy

Examination of skin scrapings with 10% KOH solution showed presence of fungal hypahe in 124 patients (81%).

Fungal hyphae on KOH mount	No. of patients Percentage	Percentage
Present	124	81.0
Absent	29	19.0

Table 33: **Direct microscopy of KOH mount**

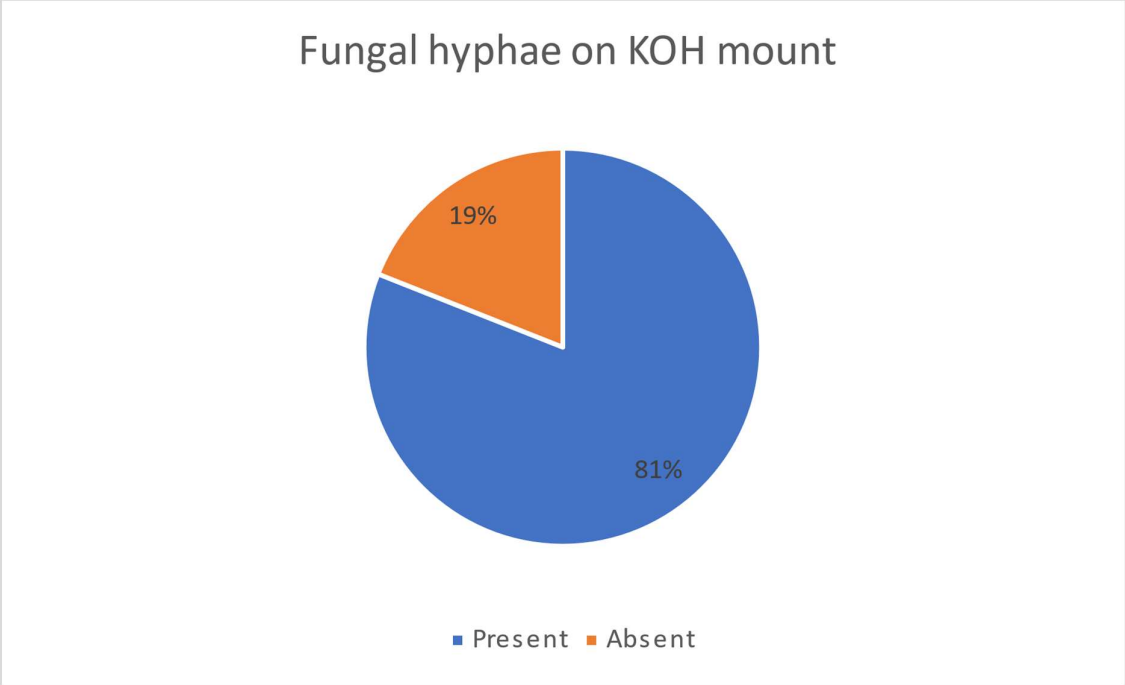

Figure 17 : **Direct microscopy of KOH mount.**

Correlation between KOH mount and fungal culture

Both microscopy and culture were positive in 73 samples (58.9 %) while 51 (41.1%) samples were positive by microscopy but negative by culture. Negative by microscopy but culture positivity was seen in 5 (17.2%) samples and both negative was evident in 24 cases (82.8%). Based on Pearson Chi-square test value 16.299 with a 'p' value of 0.001 which is statistically significant, there is a correlation between KOH positivity and culture positivity.

Culture positive for dermatophytes	10% KOH mount	
	Absent	Present
Positive	5 (17.2%)	73 (58.9%)
Negative	24 (82.8%)	51 (41.1%)

Table 34: **Comparison of 10% KOH mount and Dermatophytic growth on culture media.**

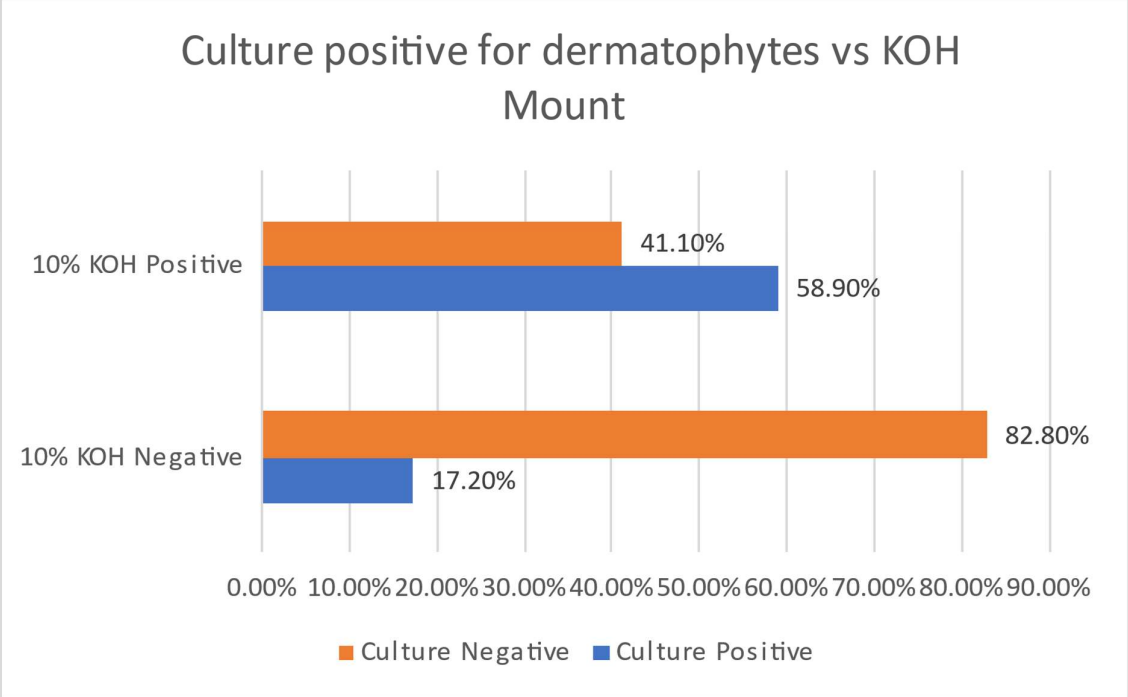

Figure 18: **Comparison of 10% KOH mount and dermatophytic growth on culture media.**

Fungal culture

Culture positive for growth was seen in 95 samples (62.1%). Culture positive growth for dermatophytes was seen in 78 samples (51%).

Culture positive for Growth	**Number of samples**	**Percentage**
Present	95	62.1
Absent	58	37.9
Total	153	100
Culture positive for Dermatophytes	**Number of samples**	**Percentage**
Present	78	51.0
Absent	75	49.0
Total	153	100

Table 35 : **Fungal growth on media**

Dermatophytic growth on culture media

Among the medias with dermatophytic growth (78), 77 patient samples had growth on dermatophyte test media (98.7 %) ,63 patient samples had growth on SDA with antibiotics media (80.7 %) while 31 patient samples showed growth on plain SDA media (39.7%).

Dermatophytic growth on media	**Number of medias**	**Percentage (%)**
SDA without antibiotic		
Present	31	39.7
Absent	47	60.25
SDA with Cycloheximide and Chloramphenicol		
Present	63	80.8
Absent	15	19.23
Dermatophyte test media		
Present	77	98.7
Absent	1	1.28
Total	78	100

Table 36: **Dermatophytic growth on culture media**

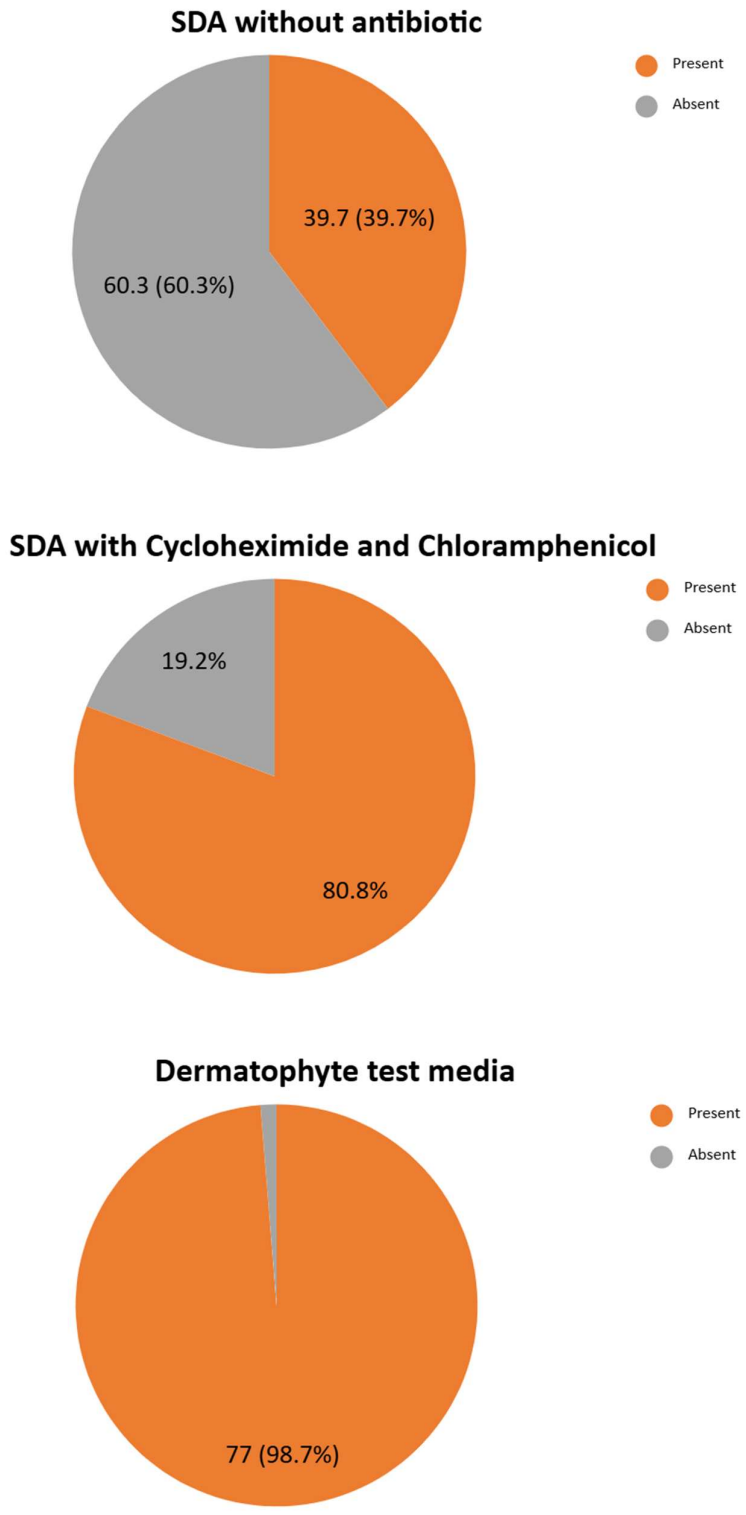

Figure 19 : **Dermatophytic growth on culture media.**

Isolated Species of Dermatophytes

In this study a specie isolation was possible from 81 samples (52.9%) out of 153 samples collected. *T. mentagrophytes* was isolated from a maximum of 50 patient samples (32.7%). *T. rubrum* from 19 (12.4%), *T tonsurans* from 7(4.6 %), and *M. canis* from 5 (3.3%) patient samples were isolated. *T. mentagrophyte* (32.7%) was noted to be the most commonly isolated specie followed by *T. rubrum* (12.4 %).

Organism	No. of species Isolated	Percentage (%)
T.mentagrophyte	50	32.7
T.rubrum	19	12.4
T.tonurans	7	4.6
M. canis	5	3.3
Total	81	100.0

Table 37 : **Distribution of isolated species of dermatophytes**

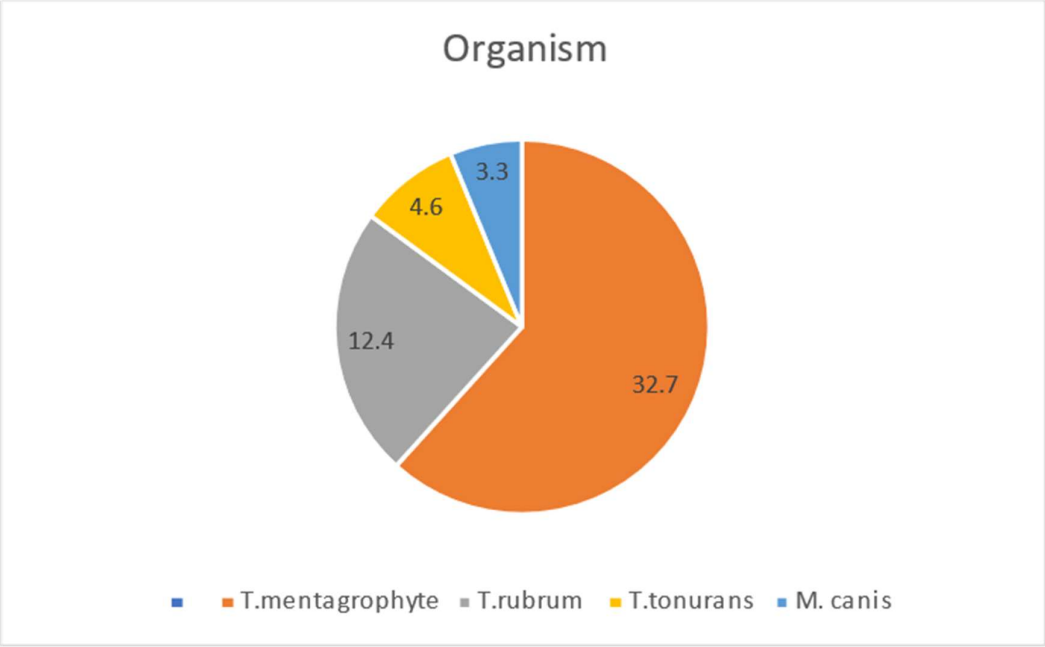

Figure 20 : **Distribution of isolated species of dermatophytes**

IMAGES

Fig 21a: Tinea capitis (kerion)-solitary inflamed boggy swelling with crusting

Fig 21b: Tinea capitis-multiple patchy loss of hair with scaling

Fig 22: Tinea corporis-extensive erythematous patches with well-defined border and scaling

Fig 23: Tinea pseudoimbricata, -ring within ring appearance

Fig 24a: Tinea Faciei with raised active margin and central clearance

Fig 24b: Tinea faciei with erythema and scaling

Fig 25: Irritant contact dermatitis secondary to topical steroid + salicylic acid application in Tinea faciei patient

Fig 26: Tinea manuum with fingers and palmar skin involvement

Fig 27: Interdigital Tinea pedis presenting with erythema and scaling

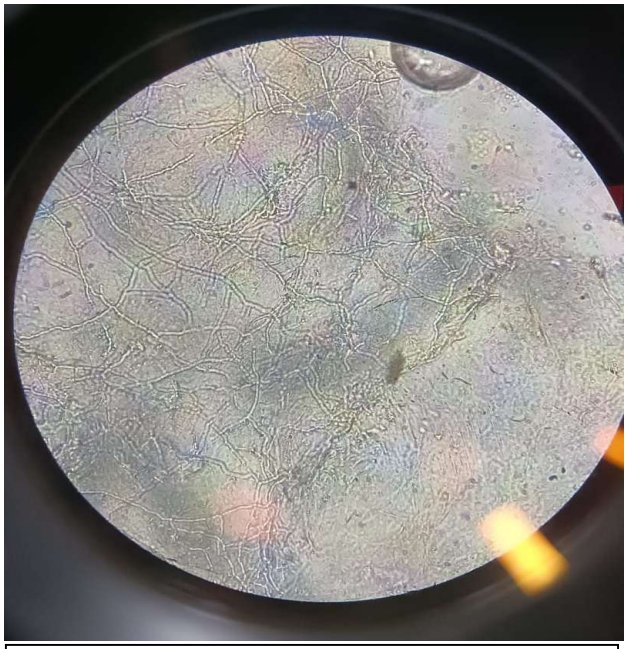

Fig 28: Direct microscopy of 10% KOH mount-hyaline long branching septate hyphae

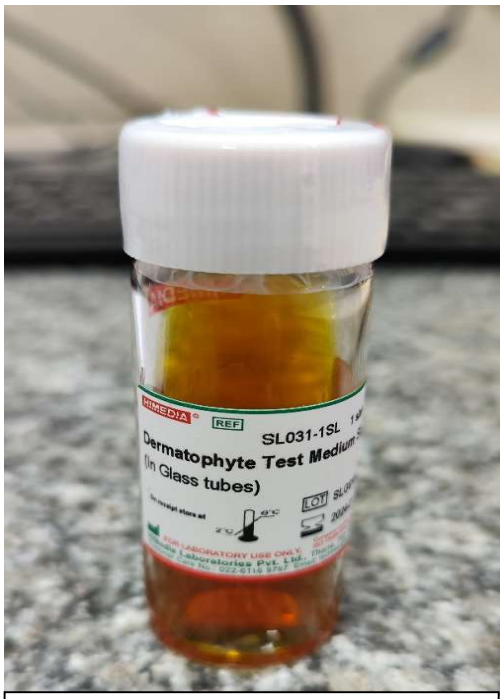

Fig 29: Dermatophyte test media prior to inoculation

Fig 30: Change in colour of DTM from yellow to pink following dermatophytic growth.

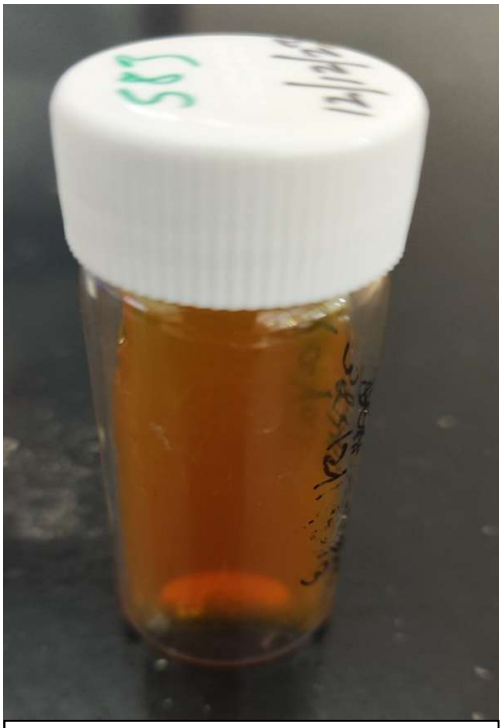

Fig 31: SDA media

Fig 32: White cottony growth on SDA media

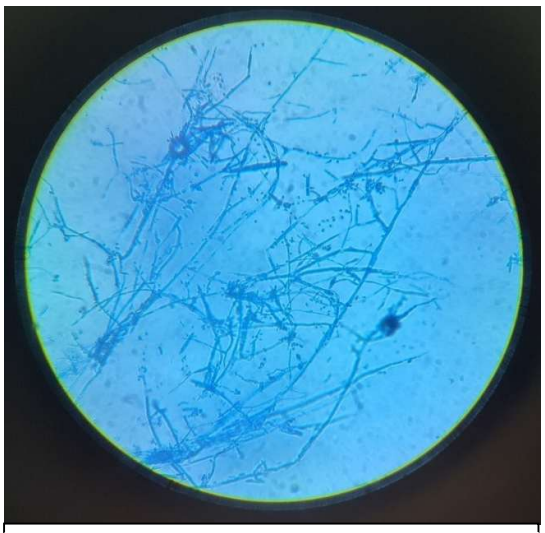

Fig 33: Microscopic morphology of *T.mentagrophytes* on Lactophenol cotton blue mount-Spiral hyphae with spherical microconidia in cluster.

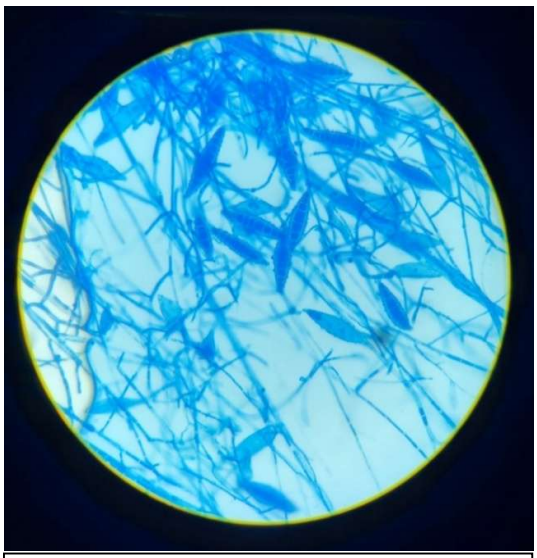

Fig 34: Microscopic morphology of M.*Canis* on lactophenol cotton blue mount-thick walled roughened ,beaked macroconidia.

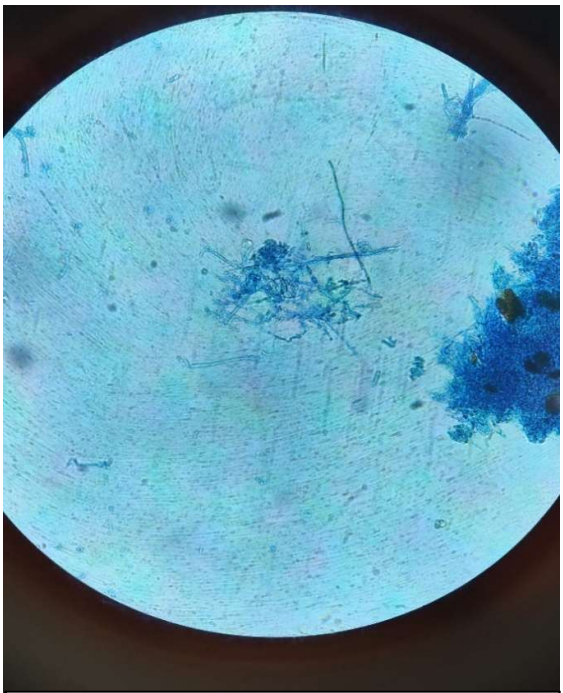

Fig 35: Microscopic morphology of *T. rubrum* on Lactophenol cotton blue mount -slender clavate microconidia

DISCUSSSION

Age

The study highlighted that dermatophytosis commonly affected 13-15 years (30.7%) age group of patients followed by 11-12 years (15.7%) age group. This is in accordance with studies done by Ray A *et al* [53] and Dash M *et al.* [54]

Studies on dermatophytosis by George IO *et al* [55] reported age group of less than 10 years as commonly affected. Predisposing factors attributed to rising dermatophytic infection in children are poverty, overcrowding, more participation in outdoor activities with increased sweating and negligence of personal hygiene.

Gender

Dermatophyte infection was more commonly seen in males than females in our study with a sex ratio of 2.65:1.

Almost all the studies have reported male predominance [56] and [54, 57] except few studies showed female predominance. [58]. It has been postulated that low prevalence in female could be associated with the fact that majority tend to practice better personal and hair hygiene when compared to males. [57]

Socioeconomic status

The highest incidence of dermatophytosis occurred in lower middle SES group followed by upper middle SES group. This was similar to the findings of Dash *et al*. [54] and George and Altraide [55]. This finding has been explained due to lack of personal hygiene and poor health care facilities in this area. [54]

Duration of lesion

Maximum number of patients had history of duration of lesion for 1-2 months. Similar finding was noted by Ray A *et al* [53] and Mishra *et al* [59]. Patient's careless behaviour, self-medication habit, benign nature of the condition and limited access to higher health care centres can be attributed for prolonged duration of lesion at consultation.

Long duration negatively impacts the child's quality of life with considerable psychological distress, especially in recurrent cases. [60]

Past history

History of previous medication

History of taking treatment of any form, prior to consultation at the tertiary care centre was observed in 83 patients (54.2%).

History of application of topical steroid combination medication was seen in 20.26%. Most of over-the-counter topical drug combinations contain clobetasol propionate in combination with topical antifungals and/or antibacterials. Due to their widespread availability and low-price community misuses, such topical preparations. [61] Persistent or recurrence of infection resulted in clinical failure of antifungal therapy.

History of prior consultation

Majority of patients had history of self-medication and only few patients (5.2%) had history of consulting a dermatologist.

This signifies that those who failed to visit a dermatologist were not diagnosed and treated appropriately. This is explained by the widespread availability of over-the-counter antifungal-steroid combination treatments and need for rapid relief from dermatophytosis symptoms. However, this relief is only temporary, and abuse of the steroid medication increases the risk

of developing recalcitrant dermatophytosis [62]. Dash *et al.* [54] have reported that a majority of their participants (61.11%) too were treated by non-dermatologists and with steroid creams.

History of similar complaints

In this study 21.6% of the children had history of similar complaints in the past. This is similar to the study done by Kashyap *et al.* [63] This fact points to prevalence of recurrent dermatophytosis and steroid-modified tinea in the rural population where this study was conducted owing to illiteracy, overcrowding and lack of personal hygiene.

Family history

History of similar complaint among family members or close contacts was seen in 64 (41.8 %) patients. Studies done by Ray *et al* [53] and Kashyap *et al* [63] showed 70% and 60% cases having a history of contact with an infected family member. This can be accredited to the practice of sharing of towels, pillows and other fomites among family members.

Thus, to prevent recurrences and chronicity of infection it is imperative to ensure adequate treatment for all affected family members.

Personal history

Personal hygiene

A great proportion of the patients had good personal hygienic practices while 34.6% had average to poor personal hygiene. This result is similar to study done by Ray A *et.al* [53]. Illiteracy, overcrowding, low socioeconomic conditions can be the reasons for poor personal hygiene.

Clothing

Sharing of clothes, combs, pillows and beds among close contacts or family members was habitual among 57(37.3%) patients. This was explained in a study done by Gupta A *et al* [(64)]. In the background of hot and humid climatic conditions, tightly fitted clothes along the groin, waist line, infra-mammary area provide moist and occlusive environment, favors the growth of dermatophytes. [(62)]

Pets

History of contact with animals such as dog/cats was present in 47 patients (30.7%). Personal history of pets helps to determine the source of infection as they are possible risk factor for recurrent dermatophytosis. [(65)]

Examination

The study showed 47.7% of patients with body surface area involvement of 5-10%, followed by 34% patients with less than 5% body surface area involvement, followed by 18.3% of patients with body surface area involvement of more than 10%. There is no study mentioning BSA in children but in adults BSA is reported. [(61)]. The reason for changing trend of larger size and increased number of lesions in patients is due to history of steroid abuse, prolonged duration of lesion, delayed consultation and recalcitrant nature of the disease. [(44)]

Cutaneous examination

On examination, most common cutaneous site of involvement was found to be Scalp (24.2 %) followed by Groin area (23.5%) while least common site over all was found to be Soles and Nails. These findings are similar to previous studies in which scalp was predominant reported site. [(66, 67, 68)].

Atypical presentation

Tinea pseudoimbricata was seen in 2.6% of patients and eczema like tinea lesion was seen in 7.2% of patients.

Tinea pseudoimbricata was seen in (4.4%) of patients in a similar study by Satheesh *et al.*

Eczematization results from both, inflammatory reaction caused by viable dermatophytes and due to inadequate clearing of the dermatophytes caused by topical steroid application.

Clinical diagnosis

Most common clinical diagnosis was Tinea Corporis (82 %) followed by Tinea Capitis (22.2 %). Studies done by Ray A *et al*, [53] Dash M *et al* [54] and Mishra N *et al* [59] also showed Tinea Corporis as the most common clinical diagnosis. Study done by George IO et al showed Tinea capitis as the most common clinical diagnosis.

Laboratory investigation

In our study, overall KOH positivity and culture positivity was 81% and 51% respectively. 58.9% of the cases were both KOH and culture positive. Satheesh *et al* reported (86.7%) positivity by KOH and (77.9 %) positivity by culture.

Overall culture positivity was 62.1% and culture positive for dermatophytosis was 51%. Non-dermatophytic growth included Aspergillus species and candida species.

The culture negativity may be due to bacterial contamination during collection of samples, nonviable fungus as a consequence of prior usage of topical anti-fungal agents or insufficient sample size due to reduced scaling as a result of topical steroid application.

Maximum dermatophytic growth was seen on dermatophyte test media 98.7% followed

by SDA with Cycloheximide and Chloramphenicol (80.8%). T. mentagrophyte (32.7 %) was noted to be the most commonly isolated specie followed by T. rubrum (12.4 %). Other species isolated included T. tonsurans (4.6%) and M. canis (3.3%). These findings align with the findings of previous study done by Satheesh *et al* [56] and Mishra *et al* [59]. Contrary to the observation the predominant species isolated was T. rubrum, followed by T. mentagrophytes in studies done by Kashyap *et al* [63].

CONCLUSION

A hospital based cross sectional clinico-mycological study, enrolling 153 patients was done to determine the epidemiological trends associated with dermatophytosis at a tertiary care centre. A detailed history was elicited regarding duration of the lesion, occupation, significant past, personal and family history. The patients were further evaluated to determine the clinical type of dermatophytosis. Specimens collected from the lesion were subjected to microbiological investigation such as 10% KOH mount to visualize fungal hyphae and culture on SDA, SDA with antibiotics media and DTM for species isolation.

It was observed from the study that the most commonly affected patients were 13-15 years and essentially males. A majority of the patients belonged to lower middle socioeconomic strata. Frequently patients had lesions for a duration of 1-2 months at consultation and a striking 54.2% of patients had history of using various forms of medication prior to consultation. Subsequently, many patients had super-added irritant contact dermatitis. History of similar complaints in the past was observed in 21.6% of the patients and similar complaints among family members or close contacts was found in 42% of patients. A multitude of patients had a history of wearing tightly fitted garments 44%, sharing of fomites 37% and close contact with animals 31%.

According to the study, Tinea corporis was the commonest clinical presentation of dermatophytosis followed by Tinea capitis. Tinea Corporis along with Tinea cruris was the common pattern among the combination types. Direct microscopy of 10% KOH mount demonstrated fungal hyphae in 81% of patient's samples. Culture positivity for growth was seen in 95 samples (62.1%). Culture positive growth for dermatophytes was seen in 78 samples (51%). Predominant causative fungal species isolated were *Trichophyton mentagrophytes* followed by *Trichophyton rubrum.*

In recent years, a significant shift in the clinical pattern of dermatophytosis is observed, with

larger lesions and increased involvement of body surface area. Topical corticosteroids used in combination with antifungal agents available over the counter are widely abused, resulting in treatment resistant chronic dermatophytosis.

Treatment-resistant dermatophytosis is a result of clinical failure from antifungal medication brought on by the persistence of risk factors, poor personal cleanliness, and a lack of general community information about the infection and its treatment characteristics. Species isolation from the study is following the trend in epidemiological transformation of most common causative species of dermatophytosis from *T. rubrum* to *T mentagrophytes.*

SUMMARY

- A hospital based cross sectional clinico-mycological study of dermatophytosis at a tertiary care hospital, to determine its epidemiological trends was conducted from May 2023 to December 2024.
- A total of 153 patients, clinically diagnosed cases of dermatophytosis in children up to 15 years of age group irrespective of gender were included in the study.
- The age group most commonly affected was 13-15 years.
- Males were predominantly affected than females.
- Patients in the study, predominantly belonged to lower middle socioeconomic status.
- Maximum patients 39.9%presented with 1-2 months duration of lesion.
- Only 50.3% of the patients had no history of previous treatment.
- 27.5% of the patients in the study had history of self-medication.
- Similar complaints in the past were seen in 21.6% of the patients.
- 41.8% of the patients had similar complaints among family members.
- Wearing tightly fitted garments (44.4%), sharing of fomites (37.3%) and close contact with animals (30.7%) was seen associated in the development of dermatophytosis.
- Multiple body site involvement and presence of larger lesions were far more common in this study than single site infection.
- Tinea Corporis (82%) was the commonest clinical presentation followed by Tinea Capitis (22.2%).
- Among the combination types, Tinea Corporis with Tinea Cruris was the most common pattern.
- Direct microscopy of 10% KOH mount demonstrated fungal hyphae in 81% of patients.
- Culture positive growth for dermatophytes was seen in 78 samples (51%).

- *Trichophyton mentagrophytes* was the most common organism isolated in this study, followed by *Trichophyton rubrum, Trichophyton tonsurans*, and *M. canis*.

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ANNEXURE I

ETHICAL CLEARANCE CERTIFICATE

Azadi Ka Amrit Mahotsav

BLDE
(DEEMED TO BE UNIVERSITY)
Declared as Deemed to be University u/s 3 of UGC Act, 1956
Accredited with 'A' Grade by NAAC (Cycle-2)
The Constituent College
SHRI B. M. PATIL MEDICAL COLLEGE, HOSPITAL & RESEARCH CENTRE, VIJAYAPURA

BLDE (DU)/IEC/ 884/2022-23 10/4/2023

INSTITUTIONAL ETHICAL CLEARANCE CERTIFICATE

The Ethical Committee of this University met on **Saturday, 18th March, 2023 at 11.30 a.m. in the CAL Laboratory, Dept. of Pharmacology,** scrutinizes the Synopsis/ Research Projects of Post Graduate Student / Under Graduate Student /Faculty members of this University /Ph.D. Student College from ethical clearance point of view. After scrutiny, the following original/ corrected and revised version synopsis of the thesis/ research projects has been accorded ethical clearance.

TITLE: "DERMATOPHYTE INFECTIONS IN CHILDREN: A CROSS SECTIONAL STUDY IN A TERTIARY CARE HOSPITAL".

NAME OF THE STUDENT/PRINCIPAL INVESTIGATOR: DR.ANASWARASREE

NAME OF THE GUIDE: DR.ARUN C.INAMADAR, PROFESSOR,
DEPT. OF DERMATOLOGY, VENEROLOGY AND LEPROSY.

Dr. Santoshkumar Jeevangi
Chairperson
IEC, BLDE (DU),
VIJAYAPURA

Chairman,
Institutional Ethical Committee,
BLDE (Deemed to be University)
Vijayapura

Dr.Akram A. Naikwadi
Member Secretary
IEC, BLDE (DU),
VIJAYAPURA

MEMBER SECRETARY
Institutional Ethics Committee
BLDE (Deemed to be University)
Vijayapura-586103. Karnataka

Following documents were placed before Ethical Committee for Scrutinization.

- Copy of Synopsis/Research Projects
- Copy of inform consent form
- Any other relevant document

Smt. Bangaramma Sajjan Campus, B. M. Patil Road (Sholapur Road), Vijayapura - 586103, Karnataka, India.
BLDE (DU): Phone: +918352-262770, Fax: +918352-263303, Website: www.bldedu.ac.in, E-mail:office@bldedu.ac.in
College: Phone: +918352-262770, Fax: +918352-263019, E-mail: bmpmc.principal@bldedu.ac.in

ANNEXURE-II

PROFORMA

B.L.D.E.U'S SHRI B. M. PATIL MEDICAL COLLEGE HOSPITAL AND RESEARCH CENTRE, VIJAYAPURA.
Department of Dermatology, Venereology and Leprosy.

DERMATOPHYTE INFECTIONS IN CHILDREN: A CROSS-SECTIONAL STUDY IN A TERTIARY CARE HOSPITAL.

S.No: Date:

Name: Hospital Number:

Age / Sex:

Address and Contact Details:

Socioeconomic status:

HISTORY:

Present history:

Duration of illness:

Past history

Treatment history:

Similar history in family members:

H/o sharing soap/towels/combs/pillows with infected person:

H/o contact with animals:

EXAMINATION:

Height:

Body weight:

Pallor:

Nutritional status: well-nourished/poorly nourished:

Cutaneous examination:

Site of lesion and number:

Scalp:

Face:

Trunk

Upper limbs:

Lower limbs:

Groin:

Palms:

Soles:

Nails:

Others:

Number:

Clinical pattern

Scaling: Present/Absent

CLINICAL DIAGNOSIS:

MICROBIOLOGICAL INVESTIGATION:

Direct microscopy-KOH mount:

Culture-SDA without antibiotic:

SDA with Cycloheximide an Chloramphenicol:

Dermatophyte test media

ANNEXURE– III

CONSENT FORM

B.L.D.E. (Deemed to be University) SHRI B.M PATIL MEDICAL COLLEGE HOSPITAL AND RESEARCH CENTRE, VIJAYAPURA-586 103

TITLE OF THE PROJECT: - "DERMATOPHYTE INFECTIONS IN CHILDREN: A PROSPECTIVE STUDY IN A TERTIARY CARE HOSPITAL"

PG GUIDE **: -** DR. ARUN C. INAMADAR

PG STUDENT **: -** DR. ANASWARASREE

PURPOSE OF RESEARCH: -

I have been informed that this project will assess epidemiological trends of tinea cases among the patients attending skin OPD at SBMP medical college and hospital

BENEFITS: -

I understand that my participation in this study will help the investigator to know the Trends and Epidemiology of tinea cases among patients attending skin OPD at SBMP medical college and hospital

PROCEDURE: -

I understand that relevant history will be taken and I will undergo detailed clinical examination after which treatment will be given.

RISK AND DISCOMFORTS: -

I understand the possible complications that may occur during and after the procedure, i.e., post procedure pain, swelling and erythema at the site of collection of skin/nail scrapings or hair specimen.

CONFIDENTIALITY: -

I understand that medical information produced by this study will become a part of my hospital records and will be subjected to the confidentiality and privacy regulation of the said hospital. Information of a sensitive personal nature will not be a part of the medical records, but will be stored in the investigator's research file. If the data are used for publication in the medical literature or for teaching purposes no names will be used and other identifiers such as photographs and audio or videotapes will be used only with my special written permission. I understand I may see the photographs, videotapes and hear the audiotapes before giving this permission.

REQUEST FOR MORE INFORMATION: -

I understand that I may ask more questions about the study at any time concerned. Dr. Anaswarasree is available to answer my questions or concerns. I understand that I will be informed of any significant new findings discovered during the course of this study, which may influence my continued participation.

REFUSAL OR WITHDRAWAL OF PARTICIPATION: -

I understand that my participation is voluntary and I may refuse to participate or may withdraw consent and discontinue participation in this study at any time without prejudice. I also understand that Dr. Anaswarasree may terminate my participation in this study at any time after she has explained the reasons for doing so and has helped arrange for my continued care by my own physician, if this is appropriate.

INJURY STATEMENT: -

I understand that in the unlikely event of injury to me resulting directly from my participation in this study and if such injury were reported promptly, then medical treatment will be available to me, but no further compensation will be provided. I understand that by my agreement for my participation in this study, I am not waiving any of my legal rights.

I have explained to (patient's / relevant guardian's name) the purpose of the research, the procedures required, and the possible risks and benefits to the best of my ability in patient's own language.

_____ _____

Investigator / P. G. Guide Date

I confirm that …….(Name of the PG guide / chief researcher) has explained to me the research, the study procedures that I undergo and the possible risks and discomforts as well as benefits that I may experience. I have read and I understand this consent form. Therefore, I agree to give my consent for my participation as a subject in this research project.

_____ _____

Participant / guardian Date

_____ _____

Witness to signature Date

ANNEXURE IV

KEY TO MASTERCHART

M	Male
F	Female
G	Good
Av	Average
Po	Poor
U	Upper
UM	Upper middle
LM	Lower middle
UL	Upper lower
T	Tinea
Cor	Corporis
Cru	Cruris
Cap	Capitis
Ungi	Ungium
Ped	Pedis
Fac	Faciei
Man	Manuum
BMI	Body Mass Index
10% KOH	10% Potassium Hydroxide
BSA- Body Surface Area	
A	<5%
B	5-10%
C	>10%

SDA without Antibiotic	Sabouraud's Dextrose Agar without Cycloheximide and Chloramphenicol
SDA with Cycloheximide and Chloramphenicol	Sabouraud's Dextrose Agar with Cycloheximide and Chloramphenicol
DTM	Dermatophyte Test Media
Medication History	
T/S	Topical Steroids
O+T	Oral+ Topical
UM	Unknown Medication
OM	Oral Medication
Nil	No Medication
Mode of taking Medication	
GP	General Practitioner
OTC	Over the Counter
Derm	Dermatologist
Bathing	
1	Once Daily
2	Alternate day
3	Once in two days or more
P	Present
A	Absent
T. ment	*Trichophyton mentagrophyte*
T. rub	*Trichophyton rubrum*
T. ton	*Trichophyton tonsurans*
M. can	*Microsporum canis*

Sl No.	Hospital No	Age	Sex	Personal Hygiene	Socioeconomic	Duration of leision in months	Past history			Family	Personal history						Clinical Examination																										Clinical Diagnosis								Microbiological Investigations						
							Medication		Reccurance		Bathing	Sharing clothes/shoes/comb/	Tight clothing	H/o animal contact	H/o systemic steroid	Immunocomprom	Height (cms)	Weight (kgs)	BMI	Cutaneous Examination																										T. Cor	T.Cru	T. Fac	T. Cap	T. Ped	T.Man	T.Ung		10% KOH mount	Cult.pos for growth	Cult. pos for dermatoph ytes	Growth			Organism
							Type of medication	Consultation												Scalp	Face	Neck	Chest	Back	Abdo men	Upper Limb	Hand	Palm	Gluteal region	Groin	Lower Limb	Feet	Soles	Nail	Other	Scaling	Unusual Presentation	BSA Involved													SDA without Antibioti	SDA with cyclohexi mi de & chloramph	Dermatoph yte test media	
1	118706	12	M	Po	LM	3	UM	GP	A	P	2	A	P	A			143	38	18.583		P												P		A			P					T. Fac	P	P					
2	118707	14	F	Av	UM	1	Nil		A	P	1	P	P	P			143	53	25.918					P				P	P				P		B	P	P							T.Cor	P	P	P		P	P	T.men
3	114748	6	M	G	UM	0.5	Nil		A	A	1	A	A	A			100	15	15	P													A		A			P					T.Cap	A						
4	119345	7	F	Po	LM	0.4	TS	OCT	A	A	2	A	A	A			112	18	14.349						P								P	ez	B	P							T.Cor	P	P	P		P	P	T.rub
5	120781	11	M	Av	LM	1	Nil		A	A	3	A	A	A			110	26	21.488	P													A		A			P					T.Cap	A						
6	123413	8	F	Av	LM	6	UM	OCT	P,3m	P	1	P	A	P			114	24	18.467							P	P						P		A						P		T.Man	A	P	P	P	P	P	T.men
7	134278	14	M	Po	LM	6	UM	GP	P,3m	P	1	A	A	A			136	32	17.301		P												P	ez	A			P					T.Fac	P	P	P		P	P	T.men
8	102460	11	F	Av	LM	0.25	Nil		A	P	2	A	A	A	Thalassemia		124	30	19.511	P													P		B			P					T.Cap	A						
9	135135	1	M	Av	UM	1.5	Nil		A	P	2	A	P	A			50	6.4	25.6					P					P				P		B	P	P							T.Cor+T.Cru	P	P	P			P	T.men
10	143547	11	F	Av	LM	1	Nil		A	P	1	P	P	A			122	30	20.156					P				P	P				P		C	P	P							T.Cor+T.Cru	P	P	P		P	P	T.rub
11	184829	3	M	Po	LM	3	TAF	DERM	A	P	1	P	P	A			68	12	25.952					P				P	P				P	ez	C	P	P							T.Cor+T.Cru	P	P	P	P	P	P	T.men
12	185282	9	M	Av	LM	3	Nil		A	P	3	A	A	P			114	21	16.159						P								P	T.ps	B	P							T.Cor	P	P					
13	185279	13	F	Po	UM	4	UM	OCT	P,1y	A	1	A	A	P			134	32	17.821					P				P	P				P		B	P								T.Cor	P	P	P		P	P	T.men
14	186216	11	M	Av	LM	1	Nil		A	A	1	A	A	P			130	26	15.385	P													P		A			P					T.Cap	P						
15	149581	8	M	Po	LM	1.5	UM	GP	A	P	2	P	A	A			104	20	18.491	P													P		B			P					T.Cap	A						
16	63356	13	M	Av	UM	1.5	Nil		P,1y	A	1	A	A	A			132	27	15.496					P						P			P		B	P								T.Cor	P	P	P	P	P	P	T.rub
17	191109	9	F	Av	UM	0.25	Nil		A	P	1	A	P	P			116	20	14.863		P	P	P	P		P					P		P	pso	B	P		P					T.Fac+T.Cor	P	P	P		P	P	T.men
18	193904	6	F	G	LM	0.25	Nil		A	A	2	A	A	A			100	22	22	P													P		B				P				T.Cap	A						
19	201928	14	M	Av	LM	0.1	Nil		A	A	1	P	P	P			136	30	16.22				P										P		C	P								T.Cor	P	P					
20	207227	5	F	Av	UM	1	Nil		A	A	3	A	A	A			88	15	19.37	P													P		A				P				T.Cap	A						
21	209115	9	M	Av	LM	1	Nil		A	A	1	P	P	A			118	21	15.082											P			P		B	P								T.Cor	P	P	P			P	T.men
22	123603	1	M	Av	LM	0.25	Nil		A	A	1	P	A	A	Atopic		52	11	40.68											p			P		B	P								T.Cor	P	P					
23	212501	13	M	Av	LM	2	Nil		A	A	2	A	P	P			140	40	20.408						P								P		B	P								T.Cor	P	P	P		P	P	T.men
24	221519	6	M	Po	LM	0.25	TS	OCT	A	A	2	A	P	P			110	21	17.355					P									P		B	P								T.Cor	P						
25	224183	14	F	Av	UM	2	Nil		A	A	1	P	P	A			150	38	16.889						P				P				P		C	P	P							T.Cor+T.Cru	P	P	P		P	P	T.men
26	228858	3	M	Po	LM	0.25	UM	OCT	A	A	1	A	P	A			90	11	13.58						P								P	pso	B	P								T.Cor	P	P					
27	229243	5m	M	Av	UM	0.25	Nil		A	P	3	A	A	A			58	6.7	19.917	P													P		A			P					T.Cap	P	P	P	P		P	T.ton
28	234108	11	M	Av	LM	12	UM		P,6m	A	1	P	P	P			144	31	14.95											P			P		B	P								T.Cor	P						
29	235192	15	M	Av	LM	0.25	Nil		A	P	1	P	A	A			142	60	29.756									P					P		B	P								T.Cor	P	P	P		P	P	T.men
30	236279	1	F	Po	LM	2	UM	GP	A	P	2	A	P	A			66	10	22.957					P									P	ez	B	P								T.Cor	P	P	P	P	P	P	T.men
31	237937	11	F	Av	LM	1	Nil		A	A	1	A	A	A	Acanthosis		126	36	22.676						P								P		C	P								T.Cor	P	P	P		P	P	T.men
32	242422	12	M	Av	LM	0.1	Nil		A	A	1	A	A	A			148	28	12.783	P													P		A				P				T.Cap	A	P	P			P	T.ton
33	250380	10	M	Av	UM	1	Nil		A	A	2	A	P	A			142	26	12.894									P					P		B	P								T.Cor	P						
34	189175	3m	M	Po	UM	0.25	UM	GP	P,1y	P	3	A	P	A			59	6	17.236					P									P	ez	B	P								T.Cor	P	P					
35	254126	6	F	Po	UL	0.25	TS+TAb,O	OCT	A	A	1	P	A	A			120	14	9.7222			P											P	ez	B	P								T.Cor	P	P	P		P	P	T.men
36	257295	12	M	Av	LM	1	Nil		A	A	1	P	A	A			146	35	16.42					P									P	pso	B	P								T.Cor	P						
37	237291	8	M	Po	UM	2	OAb	GP	A	A	1	A	A	A	P		120	19	13.194	P													P		A			P					T.Cap	A						
38	275978	6	M	Po	LM	1	UM	OCT	A	A	1	A	A	A			100	20	20						P			P		P			P		A	P								T.Cap	A	P	P	P	P		T.ton
39	276076	13	M	Av	U	1	Nil		A	A	1	P	P	A			150	46	20.444					P									P		B	P								T.Cor	P	P					
40	277071	10	M	Po	LM	6	UM	OCT	P,1 yr	A	1	A	P	P			120	43	29.861					P									P		C	P								T.Cor	P						
41	277823	3	M	Av	LM	0.25	Nil		A	A	2	A	A	P			88	10	12.913	P													P		A				P				T.Cap	A						
42	277827	8m	F	Av	LM	0.25	Nil		A	A	3	A	A	A			68	6.5	14.057	P													P		A				P				T.Cap	P						
43	277829	11	M	Po	UM	1.25	UM	OCT	P,6m	A	1	P	A	A			140	146	74.49		P												P		A					P			T.Fac	P	P	P		P	P	T.rub
44	238845	8	F	Av	LM	1	Nil		A	A	1	A	A	P			122	24	16.125	P													A		A				P				T.Cap	A						
45	281974	14	M	Po	UL	1	TS	OCT	P,1y	A	1	P	P	A			136	30	16.22			P	P			P							P	pso	B	P								T.Cor	P	P					
46	282034	3	M	Av	UL	1	Nil		A	A	3	A	A	A	Papular		66	15	34.435		P												P		A					P			T.Fac	P						
47	282036	5	M	Po	LM	1	UM	GP	A	A	2	A	P	A			88	14	18.079	P				P									P	ez	C	P				P				T.Cap+T.Co	P	P	P		P	P	T.men
48	288859	9	M	Av	UM	1	Nil		A	A	1	A	P	A			112	19	15.147						P								P	ez	B	P								T.Cor	P						
49	297346	13	M	G	UM	24	TS		P,1y	P	1	A	P	P			140	44	22.449		P			P									P		B	P		P						T.Fac+T.Cor	P	P					
50	299974	4	M	Av	LM	24	Nil		A	P	2	A	P	P			80	10	15.625									P	P				P		C	P	P							T.Cor+T.Cru	P						T.men
51	434360	7	F	Av	UL	3	Nil		A	A	1	P	A	A			102	11	10.573	P													P		A				P				T.Cap	P						
52	299901	14	M	Po	UM	0.75	UM	GP	A	P	1	P	P	A			144	40	19.29			P											P		B	P								T.Cor	P	P					
53	274680	10	M	Po	UL	2	UM	GP	A	A	1	P	A	P			128	30	18.311	P													P		A				P				T.Cap	A						
54	305789	7	F	Po	LM	2	UM	OCT	A	A	2	A	A	P			116	19	14.12									P	P				P		C	P	P							T.Cor+T.Cru	P	P	P	P		P	T.rub
55	306938	4	M	Av	UM	1	Nil		A	A	3	A	A	A			90	15	18.519	P													P		A				P				T.Cap	A						
56	306948	6	F	Av	LM	1	Nil		A	A	1	A	P	A			112	5	3.986	P				P		P					P		P		C	P			P					T.Cap+T.Co	P						

57	240445	3	M	Po	UL	0.1	UM	OCT	A	A	1	A	P	A	Verruca		108	12	10.288							P											P		B	P							T.Cor	P	P				
58	317629	11	F	Av	UL	2	Nil		A	A	1	A	A	A			118	42	30.164							P											P		B	P							T.Cor	P					
59	223911	12	M	Av	LM	1	TAF		A	P	1	P	P	P			130	44	26.036	P						P											P		C	P			P				T.Cap+T.Co	P	P	P		P	P	T.rub
60	321703	7	M	Po	UM	1.5	UM	OCT	A	A	2	P	A	P			126	18	11.338	P									P	P						P	ez	B	P	P		P				T.Cap+T.Cor+T.	P					
61	321701	7	M	Av	LM	1	Nil		A	A	1	P	A	A			120	26	18.056		P					P				P						P		B	P	P	P					T.Fac+T.Cor+T.	P	P	P	P		P	T.men
62	321702	4	F	Av	U	1.5	Nil		A	A	1	A	A	A			96	13	14.106	P	P	P		P	P	P			P		P					P		C	P	P	P	P				T.Cap+T.Fac+T.Co	A	P				
63	1979	10	M	Po	LM	1	TAF	Derm	A	A	1	A	A	P			124	21	13.658	P															P		A				P				T.Cap	P	P	P		P	P	T.rub
64	336175	13	M	Av	LM	1	Nil		A	P	1	A	P	A			140	42	21.429											P						P	T.ps	A		P						T.Cru	P					
65	336174	14	M	Av	UM	1	Nil		A	P	1	P	P	P			138	40	21.004										P	P						P		B	P	P						T.Cor+T.Cru	P	P	P	P		P	T.men
66	339073	14	M	Po	UL	24	OAF	Derm	A	A	1	P	A	A			140	65	33.163							P			P	P						P		B	P	P						T.Cor+T.Cru	P					
67	340507	14	M	Po	LM	0.25	UM	GP	A	P	1	A	A	A			144	45	21.701							P				P						P	T.ps	C	P	P						T.Cor+T.Cru	P	P	P	P	P	P	T.men
68	350007	14	M	Po	LM	2	UM	OCT	A	P	1	A	A	P			136	45	24.33						P												P		A	P							T.Cor	P					
69	348271	6	F	Po	UM	1	O+T	GP	A	A	2	P	A	A			114	14	10.773	P															P		A				P				T.Cap	P	P				P	T.rub
70	360958	13	F	Po	LM	1	TAF +OAI	Derm	P,1y	A	1	A	P	A			136	40	21.626						P												P		B	P							T.Cor	P	P				
71	151098	4	F	Av	UM	12	Nil		A	A	3	P	A	A			86	13	17.577							P				P						P		B	P	P						T.Cor+T.Cru	P	P	P		P	P	T.men
72	364735	14	M	Av	UL	4	Nil		A	A	2	A	P	A	N+Striae+P.Alb		158	64	25.637						P					P						P		C	P	P						T.Cor+T.Cru	P					
73	366683	14	M	Av	UL	2	Nil		A	P	1	P	A	A			140	39	19.898						P												P	T.ps	B	P							T.Cor	P	P	P	P	P	P	T.men
74	373686	11m	F	Po	LM	2	S+TAb+T	OCT	A	P	3	P	A	A			144	6.3	3.0382										P							P		C	P							T.Cor	P					
75	372532	8	F	Av	UL	0.1	Nil		A	A	2	A	A	P			110	17	14.05		P															P		A			P					T.Fac	P	P	P			P	T.men
76	383951	1	M	Av	LM	1	Nil		A	A	3	A	A	A			68	10	21.626	P															P		B				P				T.Cap	P	P	P	P	P	P	M.can
77	201901	11	F	Av	UL	0.25	Nil		P,3m	A	2	A	A	A			122	28	18.812		P															P		A			P					T.Fac	P					
78	42435	14	M	Po	LM	12	UM	GP	P,6m	P	1	P	A	P			148	42	19.175						P					P						P		B	P	P						T.Cor+T.Cru	P	P	P		P	P	M.can
79	387414	15	F	Av	UM	1	UM	OCT	A	P	1	A	P	A			140	42	21.429				P				P		P	P		P	P			P		C	P	P			P	P		T.Cor+T.Cru+T.Ma	P	P	P	P	P	P	T.men
80	387413	9	M	Po	LM	1	TS	OCT	A	P	1	A	A	A			120	25	17.361							P											P		A	P							T.Cor	P	P				
81	387411	12	F	Av	UM	2	Nil		P,1y	P	1	P	P	P			138	45	23.629												P					P		B	P							T.Cor	P	P	P		P	P	T.men
82	406620	3	M	Av	LM	0.25	Nil		A	A	3	P	A	A			76	12	20.776					P													P		B	P							T.Cor	P	P	P				T.men
83	358920	11	M	Po	LM	2	TS	OCT	P,1y	P	1	A	A	A			136	40	21.626		P															P		A			P					T.Fac	P					
84	72814	13	M	Po	UL	2	UM	GP	P,6m	P	1	A	P	P			138	31	16.278										P	P	P					P		B	P	P						T.Cor+T.Cru	P	P	P	P		P	M.can
85	408927	14	M	Av	UL	0.75	Nil		A	P	1	A	A	A			136	40	21.626	P				P		P											P		C	P			P				T.Cap+T.Co	P	P	P			P	T.men
86	43217	4	F	Av	LM	1	Nil		A	A	2	P	A	A			90	15	18.519	P															P		A				P				T.Cap	A					
87	18858	12	M	Po	UM	6	UM	OCT	A	P	1	A	A	A			126	30	18.896		P															P		A			P					T.Fac	P	P	P	P	P	P	T.rub
88	19446	13	M	Po	LM	1	UM	OCT	A	P	1	A	P	A			140	38	19.388										P							P		B	P							T.Cor	P	P	P	P		P	T.men
89	21146	15	M	Po	LM	12	S+TAF+O	GP	P,1y	A	1	A	A	P		Scabie	142	44	21.821	P															P		A				P				T.Cap	A					
90	199561	12	M	Av	LM	0.25	Nil		A	A	1	P	P	P	N+Striae+P.Alb		122	36	24.187						P					P						P		C	P	P						T.Cor+T.Cru	P	P	P		P	P	T.rub
91	25580	14	M	Po	UL	3	UM	GP	A	A	2	A	P	A			138	40	21.004										P	P						P		B	P	P						T.Cor+T.Cru	P	P	P	P	P	P	T.men
92	32424	13	F	G	UM	2	TS	OCT	A	A	1	A	A	A			134	50	27.846						P					P						P		C	P	P						T.Cor+T.Cru	P	P	P		P	P	T.men
93	38737	12	M	Po	LM	9	UM	OCT	P,6m	P	1	P	P	P			130	26	15.385		P															P		A			P					T.Fac	P					
94	39536	4	M	Av	UM	0.1	Nil		A	A	3	P	P	A			80	15	23.438	P															A		A				P				T.Cap	A					
95	42720	15	M	Po	LM	6	O+T	Derm	P,1y	P	1	A	P	P			142	60	29.756											P						P		B		P						T.Cru	P	P	P		P	P	T.men
96	44316	3	F	Po	UM	1	TAF	Derm	P,1y	A	2	P	A	A			70	13	26.531		P															P		A			P					T.Fac	A					
97	44310	10	M	Av	UM	0.1	Nil		A	A	1	P	P	A			120	30	20.833	P															P		A				P				T.Cap	P					
98	50527	2	F	Po	LM	12	TS+TAF	OCT	A	A	3	A	A	A			70	10	20.408		P															P		A			P					T.Fac	A					
99	59050	10	F	Po	UM	24	UM	GP	P,6m	A	1	A	A	A			130	22	13.018	P															P		A				P				T.Cap	A	P	P	P	P	P	T.rub
100	99324	13	M	Av	LM	1	Nil		A	A	1	P	P	A			144	42	20.255			P		P						P						P		B	P	P						T.Cor+T.Cru	P	P	P	P	P	P	T.men
101	103819	9	M	Po	UM	0.75	OAb+TAI	Derm	A	A	1	P	A	A	Neurofibro		124	34	22.112	P															P		A				P				T.Cap	A					
102	101569	3	M	Av	LM	1	Nil		A	P	2	A	A	P			76	12.5	21.641	P															P		B				P				T.Cap	A					
103	120512	3	M	Av	UM	12	Nil		A	P	3	A	A	A			80	11	17.188												P					P		B	P							T.Cor	P	P	P			P	T.men
104	121353	3	M	Av	LM	0.25	Nil		A	P	2	A	A	A	Periporitis+		88	16	20.661	P															A		B				P				T.Cap	A					
105	125601	8	M	Po	UM	1	TS	GP	A	A	1	A	P	A			114	20	15.389							P											P		C	P							T.Cor	P	P		P			T.ton
106	123205	6	M	Po	LM	2	UM	OCT	A	P	1	P	P	A			122	16	10.75										P	P						P		C	P	P						T.Cor+T.Cru	P	P	P		P	P	T.men
107	130207	14	M	Av	UM	0.25	Nil		A	A	1	A	A	P			142	35	17.358														P			P		A					P		T.Ped	P					
108	132065	11	M	Av	LM	2	Nil		A	A	1	A	A	A			145	35	16.647				P		P						P					P		A	P							T.Cor	P	P	P			P	T.men
109	132078	15	M	Po	LM	1	TS	OCT	A	P	1	P	A	P			144	40	19.29											P	P					P		B	P	P						T.Cor+T.Cru	P	P	P	P	P	P	T.men
110	136174	15	M	Po	UM	1	TS	OCT	P,6m	P	2	A	A	P			142	44	21.821		P															P	ez	A			P					T.Fac	P					
111	136152	12	M	G	LM	0.25	UM	OCT	A	A	2	A	A	A			138	45	23.629		P															P		A			P					T.Fac	P	P	P		P	P	T.rub
112	141705	7	M	Av	LM	1	Nil		A	A	1	P	A	P			116	20	14.863	P															P		B				P				T.Cap	P					
113	142241	13	M	Po	LM	2	TS	OCT	A	A	1	P	P	A			142	38	18.845												P					P		B	P							T.Cor	P	P	P	P	P	P	M.can
114	14930	6m	M	Po	LM	1	TS	OCT	A	P	3	A	A	A	tile		68	6.3	13.625										P							P		B	P							T.Cor	P					
115	405487	6m	M	Po	UL	1	TS	OCT	A	P	3	A	A	P			72	5.3	10.224					P													P		C	P							T.Cor	P	P	P	P	P	P	T.men
116	87387	1	F	Av	LM	0.75	Nil		A	A	3	A	A	P			76	10	17.313	P															P		A				P				T.Cap	A					
117	48750	2W	F	Av	LM	0.05	Nil		A	A	3	A	P	A			48	5	21.701																	P		B								T.Cor	P	P	P		P	P	T.men
118	198725	7	F	Po	UM	1	TS	GP	A	A	1	P	A	P			106	26	23.14	P															P		A				P				T.Cap	P	P	P		P	P	T.rub
119	185940	12	M	Po	LM	2	UM	OCT	A	P	1	P	P	A			144	33	15.914												P					P		B	P							T.Cor	A					
120	93047	6	M	Po	LM	1	TS	OCT	A	P	1	A	P	A			120	26	18.056											P						P		A		P						T.Cru	P	P	P		P	P	T.men
121	273878	8	M	Av	UL	3	UM		P,1y	P	1	A	P	A			114	23	17.698										P		P				P	ez	B	P							T.Cor	P	P	P	P	P	P	T.rub

122	134438	15	M	Av	UM	1	Nil		A	P	1	A	P	P			142	39	19.341						P											P		B	P							T.Cor	P						
123	107683	10	M	Av	LM	0.25	Nil		A	P	1	A	A	A			120	28	19.444		P															P		A			P					T.Fac	A	P	P	P	P	P	T.rub
124	104778	14	F	Po	LM	6	TS	OCT	P,1y	A	1	A	P	A			138	35	18.378										P							P		B	P							T.Cor	P						
125	380102	14	F	G	UM	0.25	Nil		A	A	1	A	A	A			148	42	19.175	P										P						P		B		P						T.Cru	P	P	P			P	T.ton
126	34780	3	F	Po	UL	1.5	UM	OCT	A	A	2	A	A	A			84	17	24.093		P															P		A			P					T.Fac	A						
127	222971	13	M	Po	LM	1	TS	GP	A	P	1	A	P	A			148	44	20.088												P					P		B	P							T.Cor	P	P	P	P	P	P	M.can
128	2222	11	M	Av	LM	1	Nil		A	P	1	A	P	A	Thalassemia		136	26	14.057						P											P		B	P							T.Cor	P						
129	50783	5	M	G	UM	0.05	Nil		A	A	1	A	A	A			96	22	23.872		P															P		A			P					T.Fac	A						
130	283442	12	F	Av	UL	0.1	Nil		A	P	1	P	P	P			138	28	14.703							P	P									P		B	P					P		T.Cor+T.Ma	P	P	P		P	P	T.men
131	286331	9	F	Av	UL	1	Nil		A	A	1	A	A	P			132	34	19.513			P				P										P		B	P							T.Cor	P	P	P		P	P	T.men
132	69261	15	M	Av	LM	1	Nil		A	A	1	P	P	A			148	44	20.088					P						P		P	P			P		C	P	P			P			T.Cor+T.Cru+T.	P	P	P	P	P	P	T.men
133	289698	4	M	Po	UM	1	UM	GP	A	P	1	A	A	A			80	15	23.438										P		P					P	Y	B	P							T.Cor	P	P					
134	289711	10	M	Po	LM	1	UM	OCT	P,6m	P	1	A	P	A			122	28	18.812										P							P		B	P							T.Cor	P	P	P		P	P	T.Men
135	289734	6	M	Po	LM	0.05	TS	GP	A	A	1	A	A	P			110	24	19.835			P									P					P		B	P							T.Cor	P	P	P		P	P	T.men
136	290987	4	M	Po	U	1	TS	OCT	P,3m	P	1	A	A	A			94	15	16.976										P		P					P		B	P							T.Cor	P	P	P	P	P	P	T.rub
137	200461	9	M	Po	UM	2	UM	GP	P,6m	A	1	P	P	A			135	36	19.753							P				P						P		C	P	P						T.Cor+T.Cru	P	P	P	P	P	P	T.men
138	280276	10m	M	G	UL	10	TS	OCT	P,3m	A	3	A	P	A			62	8	20.812												P							B	P							T.Cor	P	P	P		P	P	T.men
139		4	M	Po	LM	2	OAF+TA	Derm	A	P	2	P	P	A			96	18	19.531		P	P							P		P					P		C	P		P					T.Fac+T.Cor	P	P	P		P	P	T.men
140	213272	15	M	Po	LM	1	UM	GP	A	P	1	P	A	A	Undescede		145	34	16.171						P											P		C	P							T.Cor	P						
141	12312	14	M	Po	UL	1	UM	OCT	A	A	1	P	P	A			148	46	21.001											P						P		A		P						T.Cru	P	P	P		P	P	T.ton
142	300074	14	M	Po	UM	0.25	TAF	GP	P,1y	A	1	P	P	A			140	35	17.857												P					P		B	P							T.Cor	P						
143	30061	3	F	G	LM	0.25	Nil		P,6m	P	3	P	P	A			80	10	15.625		P															P		A			P					T.Fac	P	P	P		P	P	T.rub
144	26543	9	M	Po	LM	1	UM	GP	A	P	1	P	A	A			116	20	14.863							P										P		B	P							T.Cor	P						
145	232771	12	M	Av	LM	0.05	Nil		A	A	1	A	P	A			116	39	28.983							P				P						P		B	P	P						T.Cor+T.Cru	P	P	P		P	P	T.rub
146	21457	15	M	Av	LM	3	Nil		A	P	1	A	P	P			140	38	19.388										P							P		B	P							T.Cor	P	P	P	P	P	P	T.men
147	19177	10	M	Av	UL	0.25	TAF		P,1y	A	1	A	P	P			122	28	18.812								P	P	P		P					P		C	P					P		T.Cor+T.Ma	P	P	P		P	P	T.men
148	17343	14	M	Po	UM	0.25	UM	OCT	A	A	2	A	A	A			144	49	23.63											P						P		A		P						T.Cru	P						
149	52842	14	M	Po	UL	2	UM	OCT	P,6m	P	2	P	P	A			148	42	19.175										P	P						P		B	P	P						T.Cor+T.Cru	P	P	P		P	P	T.men
150	52812	14	M	Av	LM	4	TAF	GP	A	P	1	P	P	A			146	55	25.802		P						P		P	P		P				P		C	P	P	P		P	P		ac+T.Cor+T.Cru+T.	P	P	P		P	P	T.rub
151	61981	6	M	G	LM	1	UM	OCT	P,3m	P	2	A	A	P			112	24	19.133							P										A		B	P							T.Cor	P	P	P		P	P	T.ton
152	63741	15	M	Ao	LM	1	UM	OCT	A	P	1	P	P	P			154	59	24.878											P						A		A		P						T.Cru	P	P	P	P	P	P	T.men
153	70983	4	M	Av	LM	0.25	Nil		A	A	2	A	A	P			88	13	16.787								P									P		A						P		T.Man	P						