"CROSS SECTIONAL STUDY OF TINEA INCOGNITO WITH CLINICAL AND MICROBIOLOGICAL CORRELATION."

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LIST OF ABBREVIATIONS USED

Sl. No.	Abbreviation	Expansion
1	T	Trichophyton
2	E	Epidermophyton
3	M	Microsporum
4	DTH	Delayed type of hypersensitivity
5	IFN	Interferon
6	KOH	Potassium hydroxide
7	SDA	Sabouraud Dextrose Agar
8	RPMI	Roswell Park Memorial Institute
9	MFC	Minimum fungicidal concentration
10	RCT	Randomised Control Trial.
11	PDT	Photodynamic therapy

ABSTRACT

INTRODUCTION

Ive and Marks coined the term tinea incognito in 1968 to characterise the abnormal clinical presentation of dermatophytic infections caused by earlier use of topical or systemic drugs.. This phrase also refers to dermatophytosis that has been transformed as a result of use of various immunomodulators, such as topical calcineurin inhibitors.

Dermatophytes break down dead keratin, causing an eczematous reaction. This response causes the fungus to have a limited supplying keratin, limiting infection. Steroids and other immunosuppressants suppress protective eczematous response, allowing fungal growth to flourish.

These lesions are less scaly, lower elevated edge than classic dermatophytosis. They're usually large, pruritic, erythematous, pustular, they can look like other skin conditions.Because pharmacists frequently dispense drugs without prescription, many individuals seek care from them first.

We tried to evaluate clinical manifestations, most prevalent sites , clinical patterns, causative agents, the usefulness of laboratory procedures in validating clinical diagnosis , species identification in the study of tinea incognito .

Objective: Determine the incidence of tinea incognito and the causative agent using KOH and culture and to investigate relationship of topical steroids and severity of clinical lesions.

Materials and method: A cross sectional study was done on one hundred clinically suspected cases of tinea incognito, with a history of topical or systemic steroid use for a period of at least six weeks. They were subjected to direct microscopy and fungal culture.

The information gathered with mean and standard deviation expressed the continuous data (SD). The Chi-square test, test of proportion, and Fisher's exact test were used to compare categorical data expressed in rates, ratios, and percentages.

Results- In the present study the commonest age group was 31 to 40 years comprised of 30% of the patients. 61 %of the patients were males and 39% were females. The male to female ratio was 2:1. majority of the subjects with SES class III. Duration of symptoms is 7-12 months for 67% of the subjects and minimum, maximum duration observed was 5 months and 18 months respectively. 82% of the subjects did not have any associated symptoms. 61% subjects gave history of application of triple combination,followed by 34% steroid use and sapat lotion 5%. The commonenst presentation T. corporis was seen in 30% . Frequent site observed is Trunk /Body i.e 52%, followed by other sites 34%. KOH positivity was seen in 75 % and cultural positivity seen in 85 %, both positive seen in 85% cases. Most common organism seen in our study T. mentagrophytes i.e 67.1%, followed by T.rubrum 24.7%, T.tonsurens 8.2%.

Conclusion Tinea incognito is a commonly encountered, yet poorly reported entity in the study population. An increased level of awareness and vigilance on the sale of steroid containing compounds will help control this dermatological condition.

In their regular practise, dermatologists are bound to encounter patient's with tinea incognito. Patients prefer pharmacist-dispensed medications as they are well-known and

provide immediate comfort. Steroid antifungal combination medications are widely available, inexpensive, and comes with a lengthy number of false indications written in regional languages.

Dermatophytosis can mimic wide spectrum of dermatoses, making clinical diagnosis difficult. Simple diagnostic tests have high success rate, to easily detect and treat. It is necessary to reach out to areas where misinformation exists and educate people about the hazards of steroid usage, which can turn mild and treatable infections into chronic and persistent dermatological disorders.

Dermatological associations need to take action at state and national levels. It is high time for strict legislative restrictions to be established on the free distribution of such pharmaceuticals, which is double-edged blade, becoming a bane rather than a benefit.

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INTRODUCTION

Ive and Marks coined the term tinea incognito in 1968 to characterise the abnormal clinical presentation of dermatophytic infections caused by earlier use of topical or systemic drugs.[1]

Clinical atypia, according to Atzori et al., is not merely a side effect of corticosteroid therapy, but is present from the start of the illness. It could be caused by dermatophyte invasive capacity, invasion site, individual physiology, and acquired variables like excessive washing or sun exposure. They propose the term "tinea atypica" instead of "tinea incognito" to describe all kinds of dermatophytosis that didn't have traditional signs of primary or secondary disease. [2]

Dermatophytes break down dead keratin, causing an eczematous reaction. This response causes the fungus to have a limited supplying keratin, limiting infection. Steroids and other immunosuppressants suppress protective eczematous response, allowing fungal growth to flourish.[2]

These lesions are less scaly, lower elevated edge than classic dermatophytosis. They are usually large, pruritic, erythematous, pustular, they can look like other skin conditions. [2] Because pharmacists frequently dispense drugs without prescription, many individuals seek care from them first, rather than dermatologists.

We tried to evaluate clinical manifestations, most prevalent places , clinical patterns, causative agents, the usefulness of laboratory procedures in validating clinical diagnosis , species identification in tinea incognito study.

AIMS AND OBJECTIVES

PRIMARY OBJECTIVE:

- To determine incidence of tinea incognito and the causative agent .

SECONDARY OBJECTIVE.

• Using KOH and culture for determining causal agent.

• To Investigate relationship of topical steroids and severity of clinical lesions.

REVIEW OF LITERATURE

History

Terminology and clinical description

Superficial fungal infections, known as dermatophytosis, have been documented since the dawn of time. Disease first named "Herpes" by Greeks, Herpes circinatus or Herpes desquamans was added. Because it resembled circular holes in textiles eaten by moths, Romans assumed that condition was caused by insects, and named "Tinea," which means bug larva. The earliest documented record of the word "Tinea" is credited to John de Trevisa [3]

Ive and Marks named tinea incognito in 1968, with unusual clinical appearance caused by past use of Steroids (topically or systemically). Tinea incognito refers as tinea versicolor that have been changed dermatophytosis as a result of various immunomodulators, like topical calcineurin inhibitors.[1]

According to Atzori et al., clinical atypia is evident from beginning of illness and caused by dermatophyte invasive capacity, invasion site, individual physiology, and acquired variables such as excessive washing or sun exposure. "Tinea atypica" refers to any dermatophytosis that develops atypical features as a result of primary or secondary disease.[2]

Mycological history

Hook is credited with founding the field of mycology in 1677, followed by Agostino Bassi's discoverd fungal origins with silk worm sickness in 1835. [4]

Medical mycology, as it relates to human disease, originated in mid-nineteenth century with the discovery of the fungal aetiology of favus, and was centred on three European physicians: Remak, Robert, David Gruby, Johann L. Schonlein. Remak originally identified microscopic structures appearing as rods and buds in crusts from favic lesions in 1835, according to Seeliger. In 1839, Schonlein was first to describe mycotic nature. Remak, on other hand, verified that the etiologic agent favus was infectious, cultivated with apple slices, correctly named it Achorion schoenleinii, after his instructor and first discovery. [5]

The original inventor of dermatomycology was David Gruby. He defined causal agent of favus, both clinically and in microscopic details of the crusts, based on his discoveries from 1841 to 1844, and established the disease's infectious character. He also identified ectothrix invasion of the beard and scalp, designating the etiologic agent Microsporum audou (which refers to the minute spores that surround the hair shaft). He studied clinical, microscopic signs with symptoms of thrush in children. [5]

Raimond Sabouraud, well-known and influential of early medical mycologists, began his scientific studies of the dermatophytes around 1890, and his contributions included studies on taxonomy, morphology, and methods of culturing the dermatophytes, with dermatophytosis therapy. [6]

Sabouraud divided the dermatophytes into four genera: Achorion, Epidermophyton, Microsporum, and Trichophyton, based with clinical characteristics of condition, cultural and microscopic investigations. As a result, he is rightfully referred to as the "Father" of Medical Mycology. Sabouraud glucose (dextrose) agar, which he invented, is still used for cultivating fungi today (albeit the components have changed) and is named after him. [6]

Chester Emmons revised Sabouraud and colleagues' taxonomy approach in 1934, establishing the present classification of dermatophytes based on spore morphology and accessory organs. Basis of mycological principles, he deleted the genus Achorion and identified only 3 genera Microsporum, Trichophyton, and Epidermophyton. [7]

Libero Ajello, Lucille K. Georg, and colleagues made dermatophyte identification easier and reduced the number of species and variations. Using Vanbreuseghem's hair bait technique, Dawson and Gentles discovered the teleomorphs of numerous dermatophytes and allied keratinophilic fungus quickly in 1959. Griffin and Stockdale obtained Microsporum gypseum complex teleomorphs in 1961. [8]

Mycology

Botanical classification

Emmons divides fungi pathogenic to humans into four kinds based manner of reproduction (1977).[9]

Classification of fungi[9]

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

Allegedly imperfect growth cycles, the dermatophytes are classified as "Fungi Imperfecti." Many dermatophytes found in perfect condition, and they are being classed.

Classification of dermatophytes[4][7]

Epidermophyton, Microsporum, and Trichophyton are three dermatophytosis-causing anamorphic (asexual or imperfect) taxa of the Deuteromycota's Hyphomycetes class (Fungi Imperfecti). Descriptions of genera largely follow Emmons' categorization method, which based on conidial morphology , conidial development, and are updated as new species are discovered.[10]

Dermatophytes are classified into 3 genera:[5]

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

Each genus is further divided number of species. There are 42 recognised species, according to recent available data: 24 in genus Trichophyton, 16 in genus Microsporum, and 2 in genus Epidermophyton. Only the keratinized tissues of humans and animals are parasitized by these dangerous dermatophytes. Fungal cultures must be performed to look for microconidia, macroconidia, and hyphae for correct identification.[5]

Skin, hair, and nails are commonly infected by Trichophyton species. Skin and hair are infected by Microsporum species. Skin and nail infections with Epidermophyton species. [5]

Trichophyton species

Trichophyton is characterised with formation of smooth-walled micro and macroconidia. Macroconidia have thin or thick walls and are borne directly on hyphae or pedicels, measuring 4-8 x 20-50 um in size. There may be only a few or none at all in many species. Microconidia range in size from 2-3 × 2-4 um and are pyriform, spherical, clavate, or irregular in shape. [5]

Microsporum species

On short conidiophores, Microsporum species produce both macro and microconidia. Macroconidia are hyaline, multiseptate, fusiform, diverse in form, spindle-shaped to obovate, with a thin or thick echinulate to verrucose cell wall, ranging in size from 20 to 60 um. Their shape, size, cell wall properties are all significant for species identification. Microconidia are hyaline, single celled, pyriform to clavate, smooth walled, and 3.5 x 4-7 um in size. They are not diagnostic for any species.[11]

Epidermophyton

Epidermophyton species develop clusters exhubit smooth, thin-walled macroconidia show sprout directly from the hyphae. A large number of chlamydoconidia develop. Microconidia do not develop.[5]

- Ecological Classification

  Anthropophilic species

These parasites have evolved to infect humans instead of dirt or animals. In nature, they're frequently epidemic. They usually create a non-inflammatory infection , mostly

seen on the body's exposed parts (feet, groin). Direct touch or fomites cause spread of illnesses from one person to another. [12]

Geophilic species

Geophilic species typically produce occasional infections that are inflammatory in nature. Although M. gypseum strains recovered from soil are low in virulence, isolated within humans are more virulent and are responsible for epidemic transmission of diseases under more suitable environments.[10,11] Many species of soil-dwelling keratinophiles have been identified, the vast majority of them are non-pathogenic to humans. [13]

Zoophilic species

In humans, they produce infections that are both inflammatory and suppurative. Infection is spreading rapidly among domestic animals and pets. Infection likely occurs in exposed areas of body.[14]

Clinical classification

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

1. Based on site of involvement [5]

- Clinical types Site of involvement
- Tinea corporis (Glabrous skin)
- Tinea cruris (Groin)
- Tinea capitis (Hair and scalp)
- Tinea faciei (Face)

- Tinea manuum (Palm)
- Tinea unguim (Nail)
- Tinea pedis (Feet)
- Tinea barbae (Beard area)

2. Based on morphology[15]

a) Tinea corporis

Non-inflammatory

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

Inflammatory

- Herpetiform
- Kerion of glabrous skin
- Majocchi's granuloma
- Nodular granulomatous perifolliculitis of leg
- Agminate folliculitis
- Subcutaneous abscess/Tinea profunda
- Mycetoma

b) Tinea cruris

- Papulosquamous
- Vesiculopustular

c) Tinea faciei

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

d) Tinea barbae

- Inflammatory
- Superficial/ Sycosiform
- Circinate/ Spreading

e) Tinea manuum

- Non-Inflammatory hyperkeratotic
- Inflammatory Vesicular

f) Tinea pedis

- Chronic Intertriginous
- Chronic Hyperkeratotic
- Vesicular
- Acute Ulcerative

g) Onychomycosis

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

h) Tinea incognito

3. Anatomo clinical classification of dermatophytes[15]

There are four groups:

a. Dermatophytoses limited to keratinized structure

- Tinea corporis, Tinea cruris, Tinea faciei, by non-perforating folliculitis
- Tinea of palms and soles
- Tinea capitis (excluding kerion , favus)
- Tinea unguim

b. Perforating Dermatophytosis:

- Chronic perforating folliculitis
- Kerion and sycosis
- Favus

c. Dermatophytosis actively invading dermal tissues (dermatophytic granuloma)

- Dermatophytic granuloma following invasion with hair follicles
- Majocchis granuloma

d. Generalized Dermatophytosis

- Dermatophytic disease

Epidemiology

India is ,world's most populous Superficial dermatophytosis is a prevalent infective dermatosis that affects 20 to 25% of global population. It was originally was simple-to-treat tropical , subtropical infection that appeared only during the summer and rainy seasons, but it has now become a perennial and difficult-to-treat entity in India.

Recent studies, incidence of dermatophytosis has increased across the country last decade, particularly in last 5–6 years. [16]

In India, the prevalence is currently estimated to be between 6 and 61 percent. In studies from south India, prevalence of 6.09 % to 27.6 % found, while north India has a high rate of 61.5 %. Interestingly, despite expectations that dermatophytosis would be more widespread in south India's hot and humid climes and less so in north India, no such correlation exists. Dermatophytosis has been on the rise in our country for the past 5–7 years. [17][18]

Incidence of chronic, relapsing, and recurrent dermatophytosis are prevalent, with symptoms lasting months to years. These tendencies are peculiar to India's current epidemic-like dermatophytosis scenario. Tinea incognito affects 3.8 percent of tinea cases each year, with more than half of them affecting the face, trunk, and groin [19]

Age and sex

Exception of tinea capitis, dermatophytosis common in postpubertal hosts, and men are more likely to be affected than women because men have a significantly higher incidence of tinea cruris, tinea pedis, and tinea unguium, and outdoor work exposes men to hot, humid, and sweaty conditions conducive to dermatophyte growth. However, incidence of dermatophytosis in women and children has recently raised. [20]

Because bulk of Indian studies with superficial dermatophytosis included adult patients, it's impossible to know how common disease is in children. In babies, toddlers, children of all ages, morphological types of dermatophytosis, such as widespread tinea cruris, tinea corporis, and tinea faciei, are frequent. [21]

Chronic dermatophytosis (6 months or more ,with or without recurrence despite treatment) is more common fifth decade, due to waning immunity, comorbidities such as diabetes, family history, topical steroid-antifungal cream use, immunosuppressive drug use, and other factors. [22]

Familal Cases

Greater prevalence of dermatophytosis history among close contacts is documented.

Rising incidence of familial infection is most likely due to chronicity and recurring infection. Dermatophytosis of connubium is very prevalent. Dermatophytosis affects entire family rather frequently. People who in overcrowded homes, slums, hostel rooms, dorms have a particularly more prevalence of the disease.[23]

Urban versus rural areas and literacy

Dermatophytic infections are common in urban and rural settings. The rural preponderance because of high frequency outdoor work, such as agriculture, which causes higher perspiration. However, research conducted in last five years have revealed that patients from metropolitan regions account for a rising proportion of patients (approximately 80%).[24]

Initially shift attributed metropolitan population's increasing awareness, literacy, beauty concerns, which prompted seeking medical help. A contributing cause could be the comparatively simple creams containing illogical combinations of strong topical steroids, antifungal, antibacterial chemicals in metropolitan regions and rural locations. According to a recent survey, majority of patients had a medium level of education .[25]

Socioeconomic status

Patients with dermatophyte infections from lower socioeconomic sectors, with studies indicating a predominance of 61–67%, followed by those from middle socioeconomic strata. Lower socioeconomic groups, poor living conditions, unhygiene, overcrowding, poor nutrition have aided establishment of dermatophytes, increasing risk of infection, chronicity, recurrence.[21]

Occupation

People who engage in outdoor activities are having higher risk of infection because dermatophytes thrive in these conditions. Recent studies, manual labourers are the most typically afflicted. Farmers are particularly vulnerable to fungal diseases because of increased exposure to the environment ,contact with soil , animals.[26]

Homemakers having active infection because the heated cooking environment, combined with increased sweating, promotes growth of dermatophytes, making them vulnerable. Students observed ,a higher rate of dermatophytosis, which is attributed to increased perspiration during school , sports activities, wearing school uniforms and footwear for lengthy periods of time. Furthermore, wearing tight-fitting synthetic clothing, which is popular among younger people, causes damp and heated skin conditions, which may contribute to rise occurrence, recurrence, chronicity of these infections in them. [26]

Topical steroid abuse

Common cause of persistent , recalcitrant dermatophytosis is use of topical steroids. Because of increased availability and inappropriate use of combo creams in last 4-5 years, there's dramatic surge in chronic, recurrent, refractory cases (antifungal

steroid ,antifungal antibiotic). Steroid-containing potent creams are widely accessible without a prescription and are frequently recommended by pharmacists, friends, prescribed by general practitioners, patients use them irregularly for months or years.[16]

Patients with dermatophytosis frequently self-administer oral/topical azoles, topical steroids, either alone or combination. Last 1-2 years, research have found that patients use topical steroid-containing combo creams more frequently (42 %–81%)[19].

Causative organism

Before 1935, Trichophyton mentagrophytes (mainly Trichophyton interdigitale) was most common organism causing superficial dermatophytosis all over world. Following that, Trichophyton rubrum began to replace Trichophyton mentagrophytes dominating organism in several countries, continents including India, where it was claimed having incidence 80%.[28]

This shift attributed environmental elements such as humidity, temperature, shock, internal factors such as host-parasite interaction, host vulnerability, and immunological factors, thus organism's shifting virulence. The causal agent until the recent emergence of dermatophytosis, of whom Trichophyton mentagrophytes is the most common organism.[29]

Prior epidemiological shift, are responsible. Moreover, rising probability of zoophilic Trichophyton mentagrophytes acclimatised itself ,undergone anthropization leading to easy transmission among humans as suggested. Trichophyton mentagrophytes not only increased in prevalence over the last decade, but also supplanted Trichophyton rubrum within few studies.[16]

Trichophyton mentagrophytes have been discovered > 90% of dermatophyte isolates in recent research. Trichophyton mentagrophytes/ Trichophyton inter-digitale complex found (93.2 %) in recent large multicentric examination, while Trichophyton rubrum was found 6.8%. Another study found that Trichophyton mentagrophytes complex (97.2%) of isolated dermatophytes, with Trichophyton interdigitale being widely distributed species. Trichophyton mentagrophytes grows exponentially in 5–7 days, which could explain the widespread involvement, inflammatory lesions, fomite transmission that are now common.[30]

Pathogenesis

Dermatophytes are not pathogens which are produced from body. Dermatophytes are passed from person to person by three different routes, each with its own set of symptoms.[31]

Category

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

Injured skin, scars, burns, well as increased hydration and maceration, are all possible entrance points for dermatophytes in host body. Artrospores or conidia are sources of infection.[31]

There are three primary steps in a dermatophyte infection:[31]

a) Keratinocyte binding
b) Penetration through and between cells
c) Induction of a host response

a. Keratinocyte binding

In order for the infectious ingredient, arthroconidia, to adhere to keratinized tissue, surface fungus must overcome various hurdles. Dermatophytes must withstand competition from regular flora, ultraviolet light, temperature and moisture variations, and keratinocyte-produced sphingosines.[31]

• The capacity of Trichophyton rubrum to adhere to epithelial cells has been attributed with carbohydrate-specific adhesins expressed on microconidia.[31]

• During the adhesion phase, T. mentagrophytes has morphological fibrillar projections; fibrils (long , sparse) attach fungal arthroconidia to keratinocytes with each other. Inner skin layers of newly created arthroconidia have thin, short appendages covering their whole surface, which gradually fade as a substantial contact area within conidia , skin tissue is produced.[31]

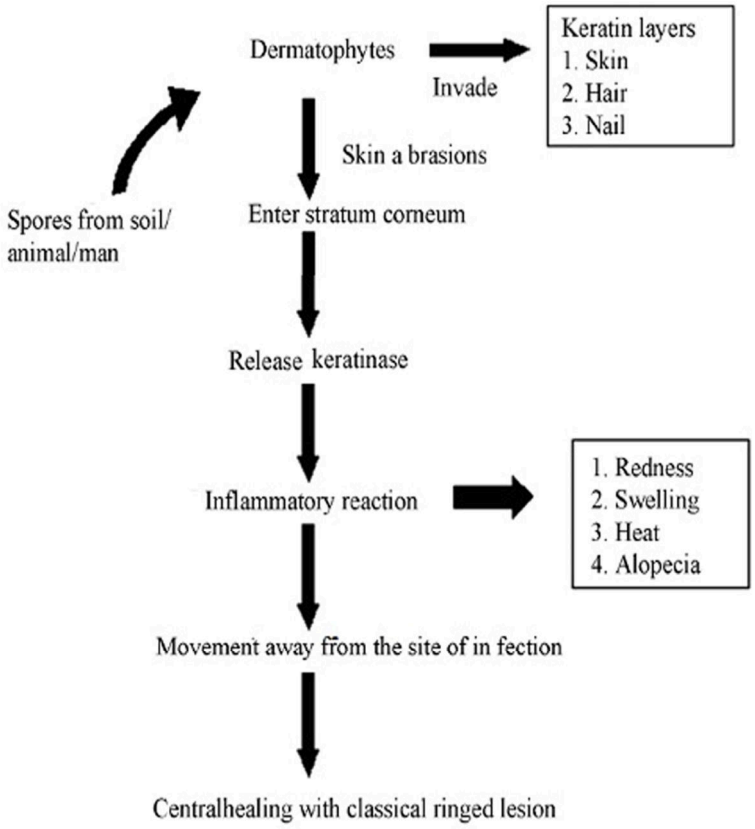
b) Penetration through and between cells

- Spores germinate and enter stratum corneum faster than desquamation after adhesion. Proteinases, lipases, and mucinolytic enzymes, which also offer nutrition to fungi, are secreted to allow penetration.[31]
- Fungal mannans found in cell walls of dermatophytes slows proliferation of keratinocytes. Once deeper layers of the epidermis are reached, new defences emerge, such as unsaturated transferrin's competition for iron and perhaps progesterone's prevents fungal growth. [31]

c) Induction of a host response

• Metabolic fungal products infiltrate within malphigian layer, causing pruritus, vesicles, erythema, and possibly pustule development. Within hair shaft, activity is restricted to develop keratin , Adamson's fringe.[31]

• Acute dermatophytosis associated with a delayed type hypersensitivity skin response to them, but persistent disease is associated with prompt hypersensitivity responses to mononuclear leukocyte production of Th2 cytokines, well as high levels of IgE and IgG4 antibodies.[31]



- **Figure 1. The schematic route of entry of dermatophytes into the host system and onset of immune response in the host in response to the pathogen entry**[32]

PATHOGENESIS OF TINEA IN-COGNITO

• Dermatophytes metabolise dead keratin , elicit an eczematous response, resulting lack of keratin supply for the fungus and hence restricting infection. Steroids and other immunosuppressants suppress this protective eczematous response, allowing fungal growth to flourish.[2]

Immunology

a) Acquired resistance

DTH's effective , protective response to dermatophytosis is often cell-mediated response characterised by macrophages as effector cells, interferon production from type 1 T-helper lymphocytes, important cytokines like as IFN's. Chemotactic factors ,alternative complement pathway activation are involved in immune recognition and chemotaxis. The immunological response , inflammation process, on the other hand, differ depending on dermatophyte species, host species, host's pathophysiological condition.[31]

• In general, zoophilic organisms generate more inflammatory infections that heal on their own, resulting relative resistance to reinfection. Anthropophilic organisms more use in induce persistent, less confined infections, resulting in lower resistance to reinfection.[31]

b) Hypersensitivity ("Trichophytin" Reaction)

- • The term "trichophytin reaction" refers dermatophyte antigens injected intradermally in humans causing cutaneous hypersensitivity. There are both rapid and delayed effects, with the latter being more commonly connected with infection.[31]

- Liquid medium containing beef extract, peptone, and maltose, Trichophyton species isolated by patients with deep-seated trichophytosis. The growth is ground and filtered after 2 -3 months at room temperature.[31]
- Individuals having deep-seated trichophytosis, parenteral injection of "trichophytin" triggered toxic effects similar in tuberculosis patients following tuberculin injection: high fever, sweat, lack of appetite, headache, and joint pain. At the injection site, there was inflammation, pustule development, and burning.[31]
- Dermatophytid reactions are generalised ,inflammatory eczematous, allergic skin reactions that occur at places other than the original fungal infection (4–5% of patients). Although it is KOH , culture negative, linked to DTH response to trichophytin test , can occasionally reveal local DTH response to a systemically absorbed antigen.[31]

c) Antibodies

- Production of antibodies doesn't appear protective. T lymphocytes multiply, travel to infected location, and cause inflammation after dermatophyte antigen processed by epidermal Langerhans cells , delivered to them in nearby lymph nodes.
- Epidermal barrier becomes permeable to transferring ,migrating cells, allowing lesions to resolve spontaneously. The trichophytin test is positive, and the second infection clears up faster.[31]

• Rivalier claimed dermatophytic infection causes 'le phenomene 'by inflammatory forms (kerion) generated from zoophilic species , doent always follow more chronic anthropophilic infections. When fungi develop horny layer

of the smooth skin, they do not have the same immunity as when they invades hair follicle.[31]

d) Non-Specific Resistance

- Immunological and non immunological systems are involved in natural defences against dermatophytes. Many nonspecific factors contributes natural infection resistance. It is mostly related to "serum factor," a fungistatic molecule found in serum of healthy people and animals that prevents dermatophytes from invading live tissues by limiting their growth to the keratinized layers.[31]
- Host factors help limits infection with keratinized tissue includes preference for cooler skin temperatures than normal body temperature, serum inhibitory factors (beta-globulins, ferritin, metal chelators) binding iron essential for dermatophyte growth, and serum inhibitory factors (beta-globulins, ferritin, and other metal chelators) binding to iron essential for dermatophyte growth. By attaching hyphae, unsaturated transferrin limits the growth and development of dermatophytes. Serum includes , growth modifying keratin inhibitor. Adults' inherent resistance to T. capitis due to long chain saturated fatty acids, which are fungistatic . fungicidal after pubertal development.[31]

• Pityrosporum yeast, a commensal yeast, enhances lipolysis , increases fatty acids available to inhibit fungus.[31]

• Humoral immunity plays a modest impact in dermatophytosis acquired resistance.[31]

CLINICAL FEATURES

The incubation period for Dermatophytes is 1–3 weeks. Itching commonest symptoms in superficial dermatophytosis, and it may be extremely uncomfortable for both adults ,children. Patients normally have pruritus paroxysms frequently ; some have it for extended lengths of time. Another symptom is "burning . itching," which is increases with sweating, heat, hot water, disrobing (atmokinesis), which leads to sleep disturbances .[33]

Patients report nighttime pruritus aggravation. Many patients who have been totally treated complain persistent itching, which is caused due to xerosis or a loss of barrier function due to scratching ,topical corticosteroid misuse. Many dermatologists have anecdotally noticed luliconazole, a topical antifungal, causing xerosis, which can lead to itching.[34]

Tinea corporis et cruris ,most prevalent presentation, followed tinea corporis and tinea cruris. Female patients frequently develop lesions in inframammary area and at occlusion locations like waist. In tinea unguium, tinea pedis, and tinea capitis. Unusual areas, such as the genital area, are involved. Tinea pubogenitalis is the combination of tinea cruris and tinea pubogenitalis.[35]

Tinea corporis usually appears as a well-defined, clearly delimited oval or circular scaly patch or plaque with a raised leading edge that is mildly erythematous. The ringworm lesion begins as a flat scaly region that spreads centrifugally and clears centrally to form a distinct annular lesion. The centre area becomes hypopigmented or brown and less scaly as the active border goes outward. Most cases, border is circular and irregular,some situations, border can be papular, vesicular, or pustular.[36]

Circinate and arcuate lesions common shapes for lesions. Pruritus, particularly mild pruritus, is extremely common. It's not uncommon for lesions to be distributed asymmetrically. When numerous lesions are present, they may coalesce into polycyclic patterns. Tinea corporis is most commonly observed on adults' exposed skin. In toddlers and teenagers, trunk is ideal location.[36]

Tinea incognito cutaneous fungal infection that lost its traditional morphological characteristics results of the usage of calcineurin inhibitors , corticosteroids. Tinea incognito has a wide range of clinical symptoms. Tinea incognito has less erythema , scaling, has a less clear boundary, and is usually more widespread. Pruritus is insignificant or non-existent. Face, the rash may resemble eczema, rosacea, discoid lupus erythematosus, trunk and limbs may resemble eczema, impetigo, or rosacea.[37]

Tinea corporis incognito was most common subtype, with many individuals having a widespread and comprehensive appearance. Tinea incognito its morphological appearances and can mimic a different skin illnesses, including psoriasis, acute ,chronic eczema, and SLE. As a result, we can call tinea incognito "the new imitator," making it the third well-known imitator.[38]

Tinea incognito has been dubbed "the new imitator," making 3rd most well-known of imitators (others being, drug eruptions and syphilis). Tinea incognito lesions, on the other hand, were often characterised with tendency for centrifugal extension with hyperactivity border. Hands lesions, particular, may march toward the nails, destroying them. Eczema-like lesions on the hands with nail involvement should be properly explored .Tinea incognita, a skin condition that affects the hands and nails, can be confused with acute or chronic eczema.[39]

In tinea, erroneous administration with fixed-dose combinations , steroid creams causes inflammation to turn on , off, resulting in "double-edged" tinea. In such circumstances, level of inflammation is frequently severe, and even after effective treatment, permanent postinflammatory hyperpigmented "double edges" might occur. Double edges are occasionally well-defined, running parallel with central crusting ,erosions, providing pseudoporokeratotic look.[40]

Tinea pseudoimbricata is a concentric ring of tinea that develops over time as a result of recurrent cycles of inflammation. Smaller scaly tinea lesions in a centrifugally spreading bigger annular lesion, plenty borders, is not uncommon. Double- edged tinea, tinea pseudo imbricata, and tinea "rings within rings" are all signs of steroid addiction, particularly erratic usage of steroid-containing combinations. Tinea recidivans uncommon illness that causes lesions to develop around the borders of previously healed lesions.[41]

Large Dermatophytosis lesions with diverse morphology are discovered, result of centrifugal spread or the coalescence several lesions. Circular , annular lesions, acuate and dumbbell-shaped lesions, annular lesions containning pustular borders, strange geographic lesions, are also common. In both immunocompromised and immunocompetent persons, dermatophytosis can manifest as erythrodermic dermatophytosis.[42]

Pustular lesions commonly seen near borders of tinea inflammatory erythematosa lesions that have been treated with strong topical steroids. Each ring of a tinea pseudoimbricata lesion studded with pustules, forming a cockade . Bullous tinea characterised with elevated, vesicular margins on annular lesions.[42]

Majocchi's granuloma is a dermatophyte infection were dermis is invaded as a result of impaired local immunity caused by topical steroid misuse. Histological examination of such instances reveals pruritic papules, pustules, nodules with underlying dermatophytic infection , granulomatous perifollicular inflammation.[16]

Superficial dermatophytosis mimicks lupus erythematosus, psoriasis, lichenoid lesions, atopic eczema, nummular eczema, erythema multiforme, granuloma annulare, granuloma faciale, lymphocytic infiltration of the skin, pityriasis rosea, seborrheic dermatitis, leprosy, molluscum contagiosum,rosacea, annular secondary syphilis,pustular psoriasis, Sweet's syndrome and impetiginized herpes have been described. [42]

Prevalent side effect of topical steroids is striae, is observed on flexures remaining areas of the body where skin is thin, such as inner thighs. They are usually erythematous at first and thereafter turn white . Striae can become ulcerated, leads to bacterial infection. Pseudoedematous striae have been observed.[43]

Lesional , perilesional hypopigmentation, which can appears in 3–4 weeks is often associated by atrophy , telangiectasia, is another typical side effect of fixed-dose steroid combinations. Local immunosuppression generated with topical steroids, concurrent bacterial infections and recurrent furunculosis , abscesses might worsen the dermatophyte-infected locations.[44]

> Cushingoid syndrome with tinea patient is reported within variety of patients, including children, who have long-standing tinea lesions in many areas that wax and wane with history of using fixed-dose combo creams. Many are truncal obese, with prominent striae, hypopigmentation, telangiectasias, acneiform eruptions, local hypertrichosis, occasionally hirsutism.[44]

LAB DIAGNOISIS

• Although the condition can be diagnosed on conventional clinical presentation, those with unusual symptoms may require test confirmation.[45]

For the best outcomes, the quantity ,quality of material reviewed are critical. Scrapings from active margin should be collected and transported on presterilized black chart paper specimen kept to dry , bacteria contamination from developing too quickly. The following is a list of the several laboratory tests can be used for confirming diagnosis of dermatophytosis.[45]

Direct microscopic examination

A quick and inexpensive bedside method to detect dermatophytic infection with skin sample with 10–20 percent potassium hydroxide . Presence of refractile, lengthy, smooth, undulating, branching, septate hyphal filaments with , without arthroconidiospores characterises positive scrapings. In 15% of cases, false negative outcomes are observed. The most sensitive method for microscopically detecting fungi in skin scales, nails, and hair is fluorescent staining with optical brighteners (diaminostilbene). These chemicals binds chitin, the major component of fungi's cell walls.[46]

Antifungal sensitivity and culture , most often used isolation media for dermatophytosis is Sabouraud dextrose agar i.e 4 percent peptone, 1 percent glucose, agar, water. Its medium were most morphologic descriptions are based. Colony develops within 7–14 days .Because chroramphenicol inhibits saprophytic fungal development, modified SDA with gentamicin, chloramphenicol, cycloheximide more selective for dermatophytes. Dermatophyte test is a non-phenol red-containing . Incubated for 5–14 days at room

temperature. Dermatophytes consume protein, resulting alkaline environment and an increase in ammonium ion, turning medium from yellow to brilliant red.[46]

Direct microscopy positive in 85 cases in study on tinea in cognito. Trichophyton was most prevalent species isolated, accounting for sixty of the 63 instances with culture positive. In 35 cases, Trichophyton rubrum was found most common.[47]

Antifungal susceptibility testing

The microdilution method is broth microdilution assay for antifungal susceptibility testing The standard approach is the Standards Institute's M38 A2. Fluconazole final concentrations range from 0.13 to 64.0 g/ml, while terbinafine , itraconazole final concentrations range from 0.06 to 32.0 g/ml. [23] To make a homogenous inoculum, the microconidia are counted microscopically. Cultures in SDA grown within 7 days at 35°C to yield conidia.[48]

To remove conidia from hyphal mat, cultures are gently swabbed with cotton-tipped applicator, sterile normal saline (85 %) is poured to agar slant. The suspension transferred into sterile centrifuge tube, which is filled to a capacity of 5 ml with sterile normal saline. Resulting suspension is counted on a hemacytometer, diluted in RPMI 1640 medium for desired concentration. When setting up microdilution plates, reference method is used.

After 4 days incubation at 35°C, microdilution plates are visually inspected. The lowest inhibitory concentration is defined as concentration at which organism's growth is reduced by 80% compared to control well.[48]

Determination of minimum fungicidal concentration ,100-l aliquots are withdrawn from the test wells that show no apparent growth on conclusion of incubation , streaked onto

SDA plates to determine the MFC. Plates are incubated for 7 days at 30°C. MFC is the lowest medication concentration that resulted in no apparent fungal growth .[48]

Colony features, microscopic morphology, physiologic testing used to identify dermatophytes. Shape of macroconidia distinguishes dermatophytes from other parasitic fungi. Few physiological tests used to validate the identity of a species. Trichohyton species can also be distinguished from others specific amino acid , vitamin needs. To hydrolyse urea differentiates T. mentagrophytes (urease positive) from T. rubrum (urease negative).[49]

Histopathology

Cases when KOH examination of scale on surface is negative, histology used to diagnose Majocchi's granuloma. When hyphae are present, can be detected in stratum corneum using hematoxylin , eosin staining. Special stains Periodic acid-Schiff , Gomori methanamine silver are frequently used to highlight hyphae.[49]

Dermoscopy

Comma hairs, slightly bent, broken hair shafts, corkscrew hairs have been reported as dermoscopic markers demonstrating tinea capitis. Broken or dystrophic hairs seen , presence of vellus hair on dermoscopy in tinea corporis implies that systemic therapy is required.[50]

Amplification based polymerase chain reaction and nucleic acid sequence .These tests aid not only in rapid , early detection of infection, also in the determination of drug resistance.[51]

New molecular approaches, such as matrix aided laser desorption ionisation time ,flight mass spectrometry, are based on detection of biochemical features, such as proteolytic breakdown products, result of mycological infection or noninfectious disease activity. To depict them, proteolytic breakdown products.[52]

Reflectance confocal microscopy provides in vivo imaging of epidermis , superficial dermis at cellular level resolution, which is used to detect cutaneous fungal and parasite infestations. Branching hyphae detectes across an erythematous annular scaly zone. [53]

Treatment

Adolescent counselling Improvements in skin, nail, and hair hygiene, avoidance of humidity and occlusive clothing, discontinuation of corticosteroid-containing topical antifungal , comprehensive assessment of likely carriers, such as family members and pets, important treatment measures.[54]

Dermatophytosis is treated with oral , topical antifungal drugs, or combination depending on extent and severity of illness, site of infection, causative organism. Topical antifungals are generally considered first-line therapy for uncomplicated, superficial dermatomycoses due to their high efficacy , low risk of systemic side effects.[54]

Depending on region of involvement, drugs incorporated variety of carriers, such as creams, lotions, gels, or sprays, to promote penetration and efficacy. They easily penetrate stratum corneum when applied to skin's surface, killing or suppressing fungal growth , achieving clinical and mycologic eradication.[54]

Despite the limited data, naftifine (1%) found more effective. Azole-corticosteroid combinations were more effective than azoles alone. Due to a rapid improvement in inflammatory component ,symptoms, leads to improved patient compliance. Unintentional use of this combination drug has been associated to treatment failure and negative effects.[54]

Luliconazole, azole antifungal, its fungicidal effect on Trichophyton species that is comparable to or greater than terbinafine. It's effective once-daily application for 1–2 weeks for dematophytic infection , is available in a 1% cream formulation. It's positive safety profile was approved by US Food and Drug Administration for treatment of interdigital tinea pedis, tinea cruris, tinea corporis.[38] For tinea pedis, Econazole nitrate foam preparation demonstrated more effective than foam vehicle.[55]

Systemic antifungals are used in extensive fungal infection or when topical therapy fails. Terbinafine ,Itraconazole are two of the most often prescribed systemic antifungals. Griseofulvin , fluconazole are also effective, but used for a long time. Systemic antifungals shown to be effective in RCTs [56],two weeks of therapy, itraconazole 100 mg/day has significantly superior clinical and mycological outcome than ultra micronized griseofulvin 500 mg/day for tinea corporis , tinea cruris.[56]

Photodynamic therapy (PDT) includes the systemic or topically delivery of a photosensitizing medication, followed by selective lighting of the target lesion with light of appropriate wavelength, resulting in generation of free radicals and cell death. Aminolevulinic acid and methylene blue are most commonly utilised photosensitizing compounds in PDT.[57]

METHODOLOGY

Various participants in this study were clinically diagnosed as Tinea incognito from all ages , genders, who visited Dermatology, Venereology, and Leprosy OPD's at KLE'S Dr Prabhakar Kore Hospital and Medical Research Centre in Belagavi.

Inclusion criteria

Age 18 to 65, all new cases of Tinea incognito presented to Department of Dermatology.

Exclusion Criteria

Patients on antifungal treatment.

Sample size

sample size formula based on prevalence is

$$n = \frac{z_\alpha{}^2 P(1-P)}{d^2}$$

where P is the percentage of prevalence and d is the percentage likely difference in the prevalence.

z_α is linked with the level of significance. For 5% level of the significance $z_\alpha = 1.96$.

With P = 63% and d = 15% of P = 9.45%,

the sample size is 100.

Ethical clearance

Prior commencement, ethical clearance was obtained through Institutional Ethics Committee, JNMC, Belgaum.

Informed consent

Patients who met selection criteria , were informed about the study purpose and gave their written consent (Annexure III).

History

Detailed history regarding duration of topical , systemic medications usage, source , reason for using , response prescribed medications,relapses, remissions were taken and recorded in proforma. (Annexure II).

Examination

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

Specimen Collection

To eliminate surface pollutants, affected area was first carefully cleansed with 70% alcohol. After the alcohol had dried, skin scraping were taken from border of active lesion's with a blunt sterile scalpel held perpendicular to skin surface. A sterile paper envelope was used to collect the specimen.

Direct Examination

Potassium Hydroxide Preparation

A clean, sterile glass slide , with few drops of 10% KOH is used to fix the sample. The slide was gradually warmed by passing , over a flame. The specimen was viewed under a low power microscope for 15-20 minutes before being inspected under a high power microscope with a low condenser for presence of hyphae or arthrospores.

Culture

Regardless of presence of fungal elements on KOH mount, the specimens were inoculated on to Sabourauds Dextrose Agar containing Chloramphenicol (50mg/l) and cycloheximide (500mg/l). Each sample was placed in a pair of tubes for inoculation. One tube was treated with antibiotics, while other was not. Both tubes were incubated at 270°C. For four weeks, the cultures were checked everyday. Slopes that had not grown

for four weeks were eliminated. Identification based on colony morphology and microscopic appearance growth was established with Sabourauds dextrose agar.

Macroscopic Examination Of Culture

The colony shape, colour of the surface, texture of the surface, topography, and rate of growth on Sabourauds dextrose agar were all studied.

Microscopic Examination

Tease Mount: A part of a fungal fragment was excised and teased on a glass slide with a drop of Lactophenol Cotton Blue stain to prepare a mount. Under microscope, cover slip was placed and examined.

Statistical analysis

Information gathered was entered into a Microsoft Excel spreadsheet (Annexure II). The mean and standard deviation were used to express the continuous data (SD). Chi-square test, test of proportion, and Fisher's exact test were used to compare categorical data expressed in rates, ratios, and percentages.

Statistical significance defined as a probability value ('p' value) of less than or equal to 0.050.

RESULTS

The present one year cross sectional study was conducted in the Department of Dermatology, Venereology and Leprosy, KLES Dr. Prabhakar Kore Hospital and Medical Research Centre attached to Jawaharlal Nehru Medical College, Belgaum during the period of January 2020 to December 2020.

A total of 100 patients with dermatophytosis were included in the study. The information gathered was coded and analysed using a Microsoft Excel spreadsheet. The following are the findings and observations.

Table 1. Age distribution

AGE (Years)	Distribution (n=100)	
	Number	**Percentage**
<20	3	3
21-30	28	28
31-40	30	30
41-50	26	26
51-60	12	12
61-70	1	1
Total	**100**	37.14±10.87

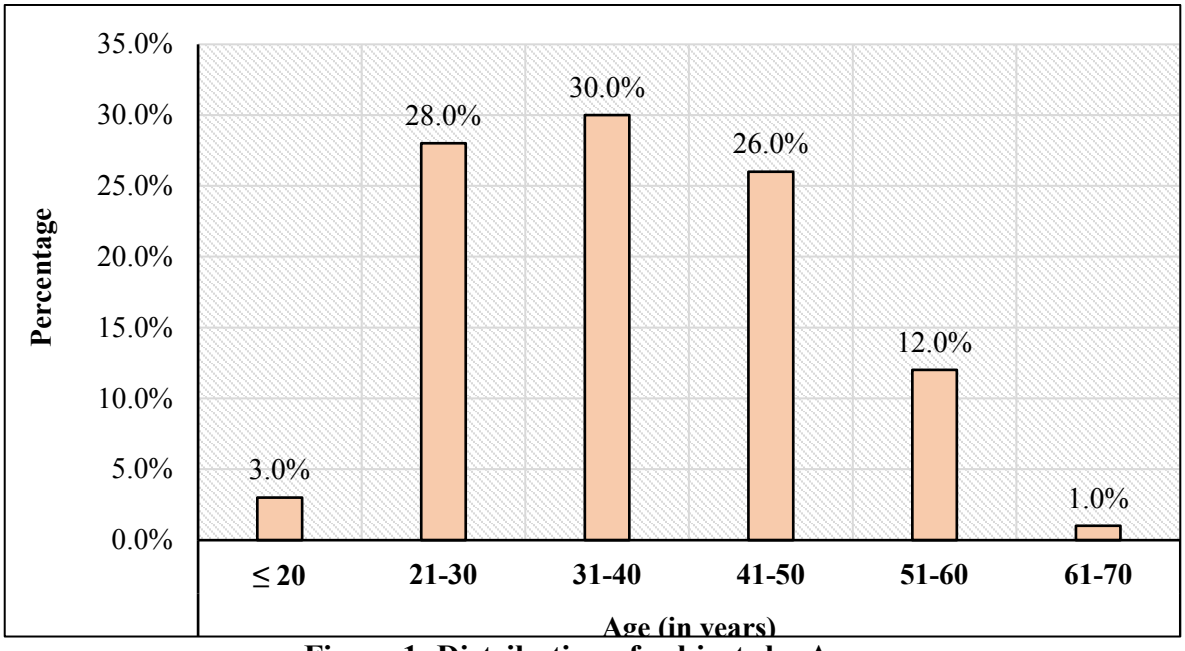


Figure 1: Distribution of subjects by Age.

In the present study the commonest age group was 31 to 40 years comprised of 30% of the patients. The distribution of other age groups is as shown in Table 1 and Figure 1.

Table 2. Sex distribution

SEX	Distribution (n=100)	
	Number	Percentage
Male	61	61
Female	39	39
Total	**100**	

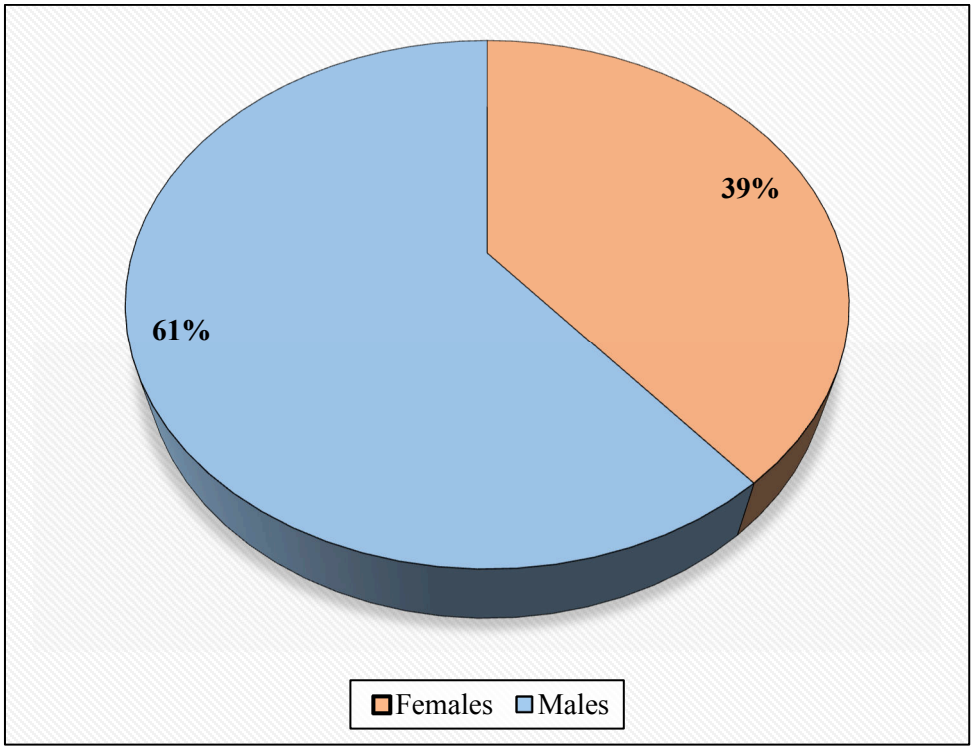


Figure 2: Distribution of subjects by Sex.

In the present study 61 %of the patients were males and 39% were females. The male to female ratio was 2:1.

Table 3. Socio economic status

SOCIO ECONOMIC	Distribution (n=100)	
	Number	Percentage
I	1	1
II	29	29
III	61	61
IV	8	8
V	1	1
Total	100	

TABLE 4:

Revision of Prasad's socioeconomic status classification revised for the year 2021 (base year 2016=100

Social class	Original classification of per capita income (Rs./month)	Revised classification for 2021 (Rs./month)
I (upper class)	100 and above	7770 and above
II (upper middle class)	50-99	3808-7769
III (middle class)	30-49	2253-3808
IV (lower middle class)	15-29	1166-2253
V (lower class)	<15	<1166

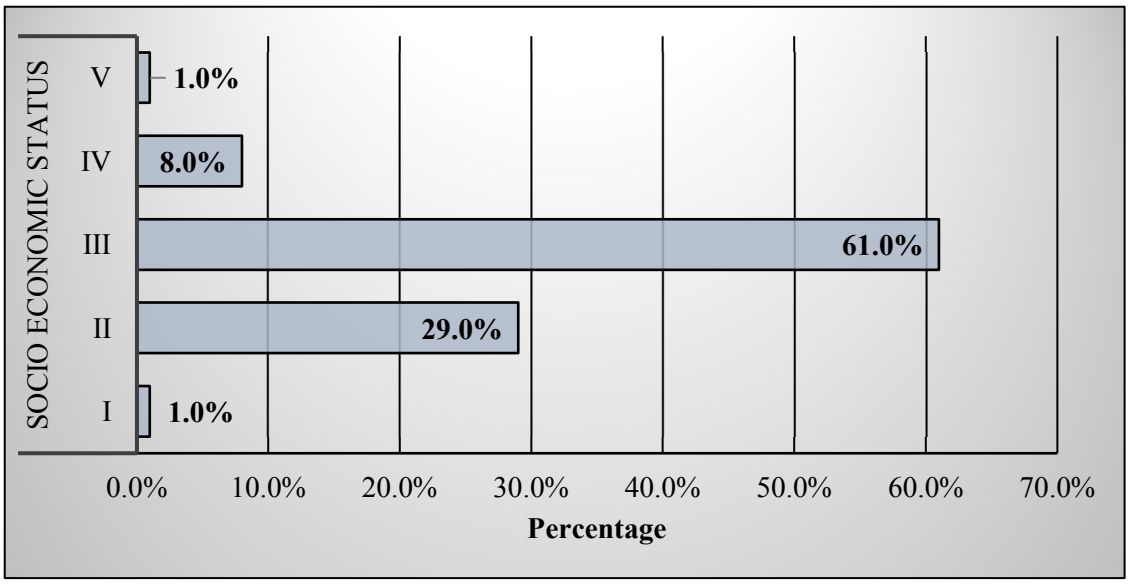


Figure 3: Distribution of subjects by SES.

In the study, majority of the subjects belong to SES class III according to B.J Prasad revised classifaction.(Table 4)

Table 5. Duration

DURATION	Distribution (n=100)	
	Number	**Percentage**
<6 Months	14	14
7-12 Months	67	67
13-18 Months	19	19
AVERAGE	**8.9+2.56**	**5,8**

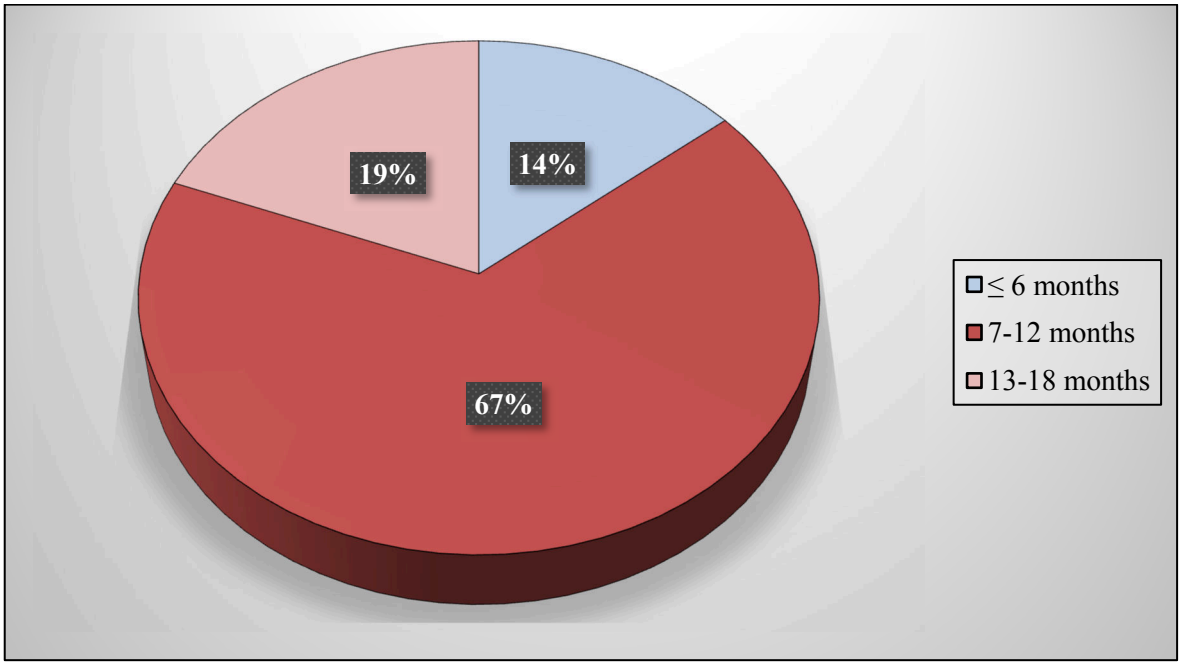


Figure 4: Distribution of subjects by duration of symptoms.

Duration of symptoms is 7-12 months for 67% of the subjects and minimum, maximum duration observed was 5 months and 18 months respectively

TABLE 6: Associated disease

ASSOCIATED DISEASE	Distribution (n=100)	
	Number	Percentage
HTN	8	8
Diabetes mellitus	10	10
Absent	82	82
Total	**100**	**100.00**

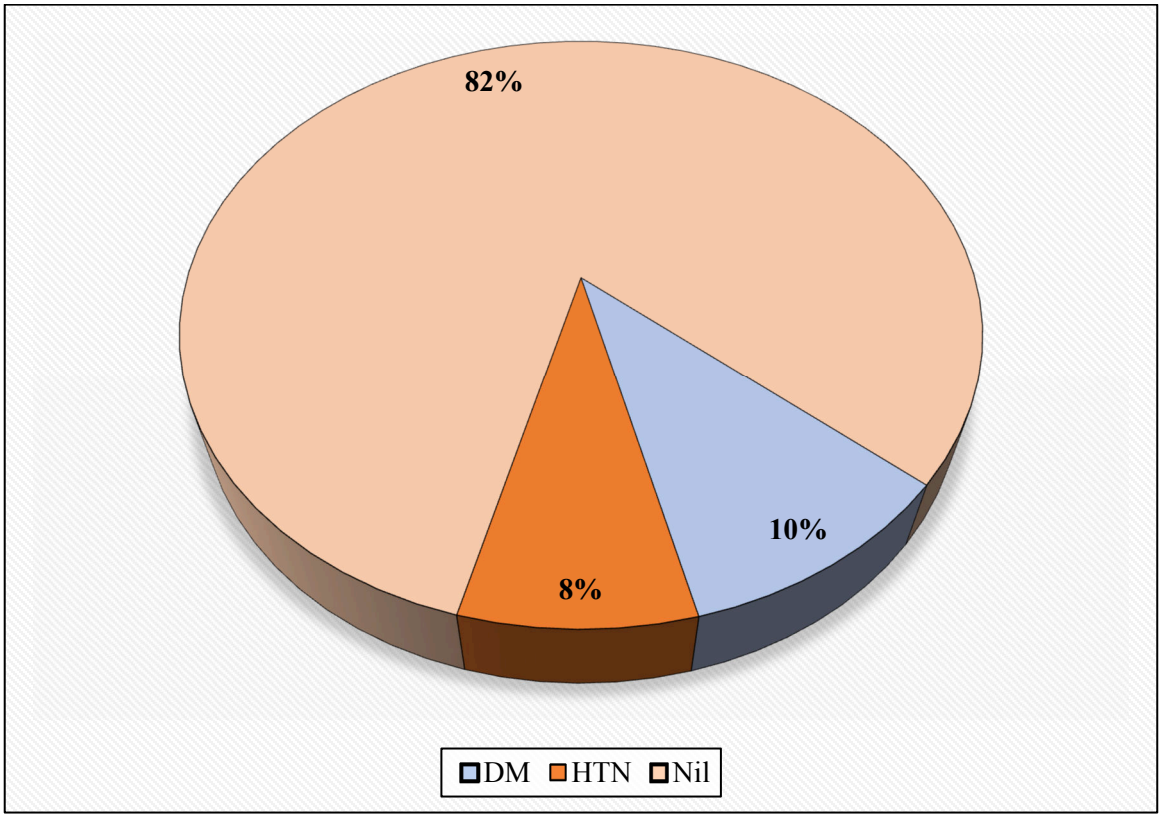


Figure 5: Distribution of subjects by associated disease.

In this study ,82% of the subjects didn't have any associated symptoms.

TABLE 7:STERIOD HISTORY

STERIOD USE	Distribution (n=100)	
	Number	Percentage
TRIPLE COMBINATION	61	61
STERIOD	34	34
SAPAT LOTION	5	5
Total	**100**	

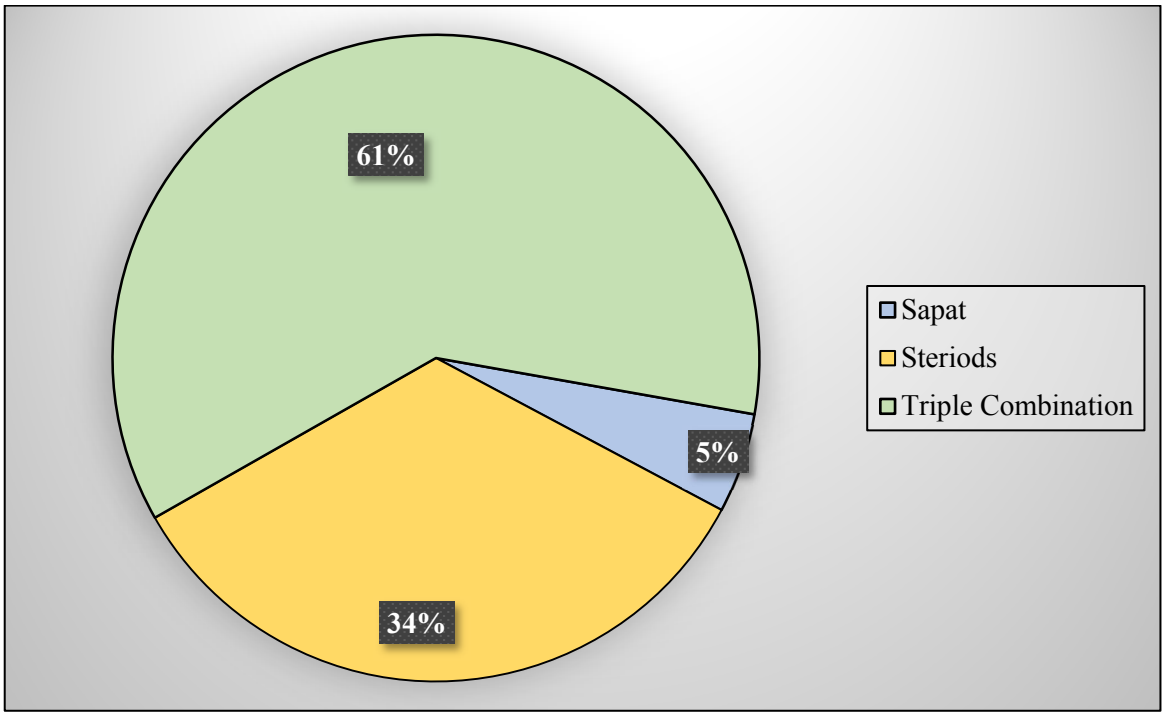


Figure 6:Distribution according to steroid history.

61% Subjects gave history of application of triple combination , followed by 34% steroid use and sapat lotion of 5%(FIG 6)

Table 8. Clinical diagnosis

Type	Distribution (n=100)	
	Number	**Percentage**
ECZEMA	27	27
PSORIASIS	25	25
TINEA INCOGNITO	48	48
Total	**100**	

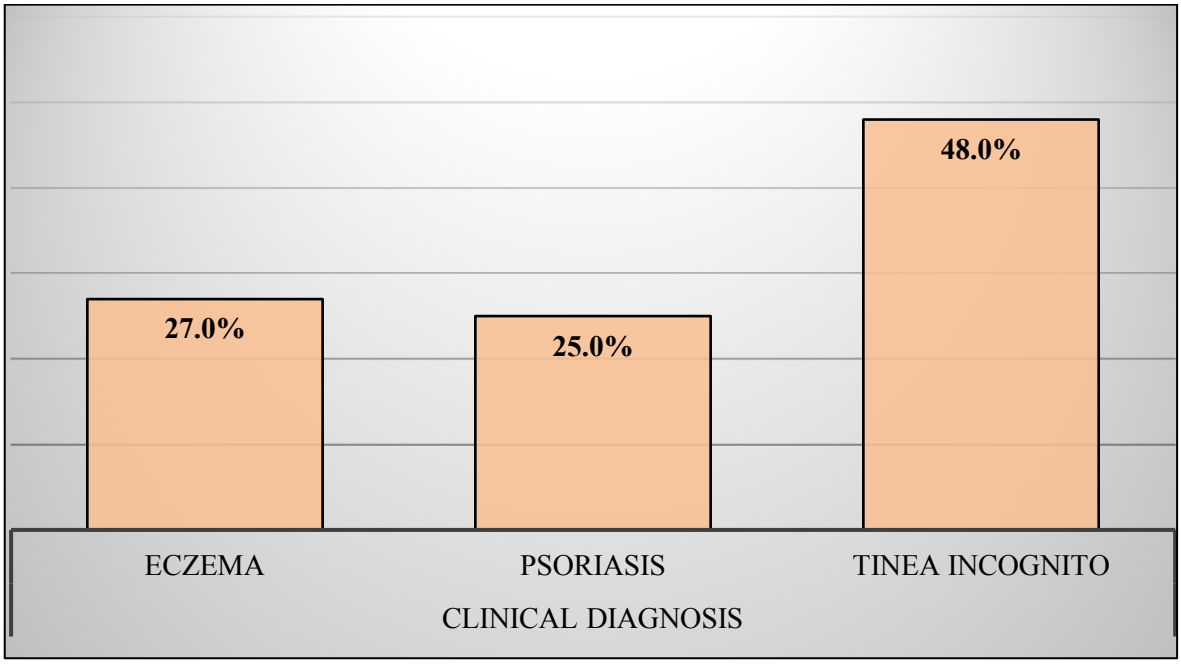


Figure 7: Distribution of subjects by diagnosis.

Typical annular lesions seen in 48% subjects, mimicking psoriasis was observed in 25% and eczematous changes seen in 27%.(Fig 7)

TABLE 9:SITE OF PRESENTATION

Type	Distribution (n=100)	
	Number	Percentage
FACE	14	14
TRUNK/BODY	52	52
OTHER SITES	34	34
Total	**100**	

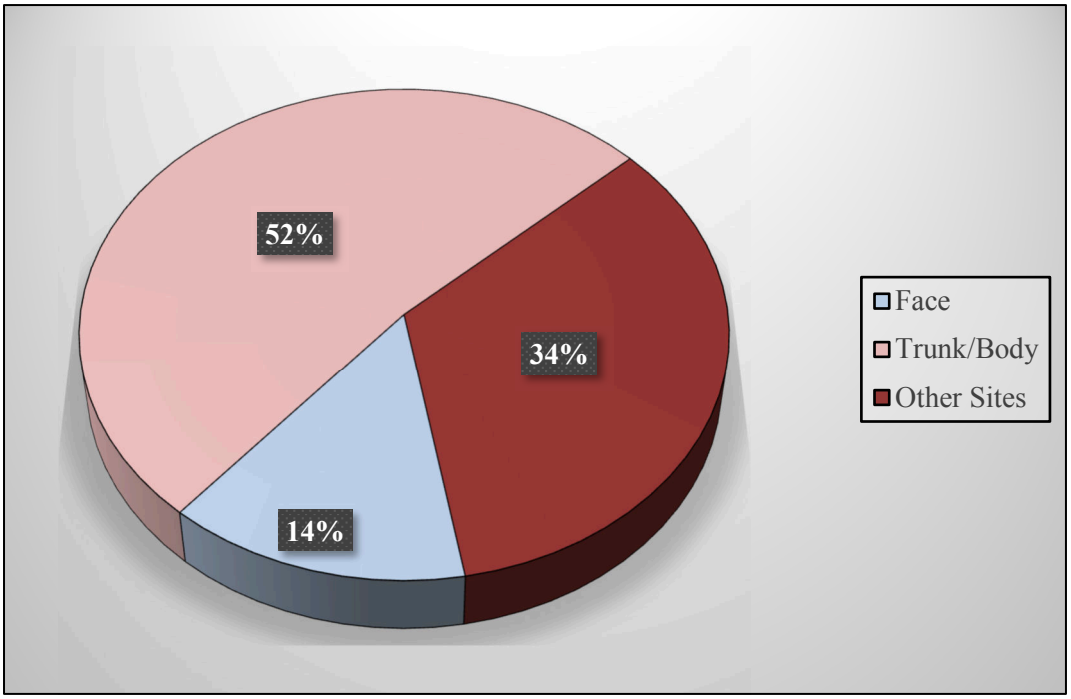


Figure 8: Distribution of subjects by site

Most common site observed is on Trunk /Body i.e 52%, followed by other sites 34%.

(Fig 8)

TABLE 10:SPECIES ISOLATION

Type	Distribution (n=100)	
	Number	Percentage
T.Mentagrophytes	67	67.1
T.Rubrum	25	24.7
T.tonsurens	8	8.2
Total	**100**	

Most common organism isolated from the study is T. mentagrophytes i.e 67.1%, followed by T.rubrum 24.7%, T.tonsurens 8.2%.(fig 9)

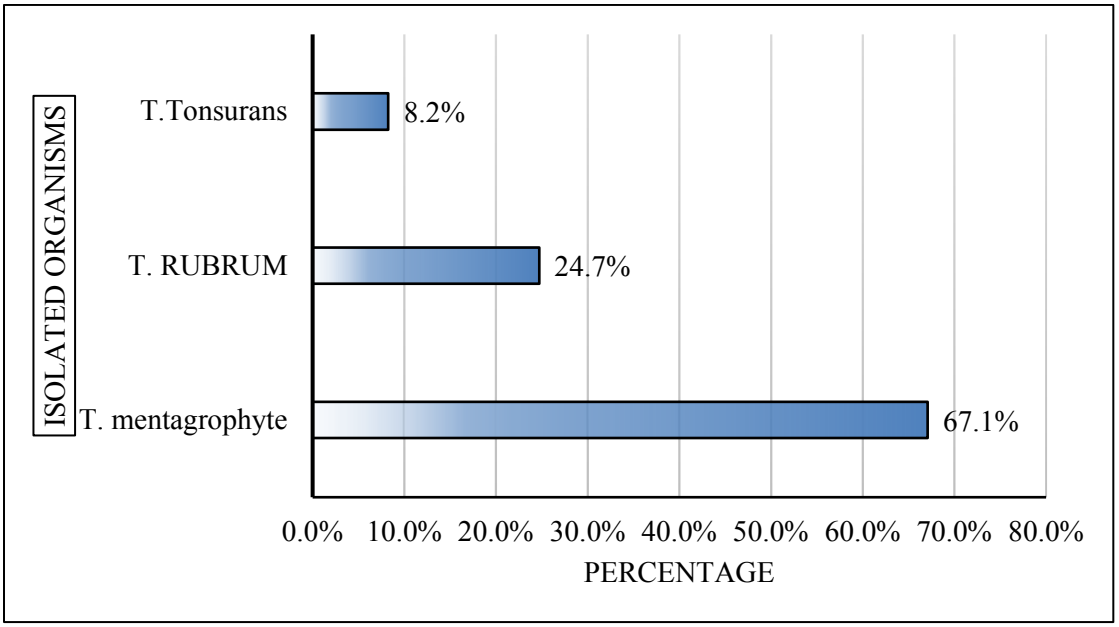


Figure 9: Distribution of subjects by isolated organism.

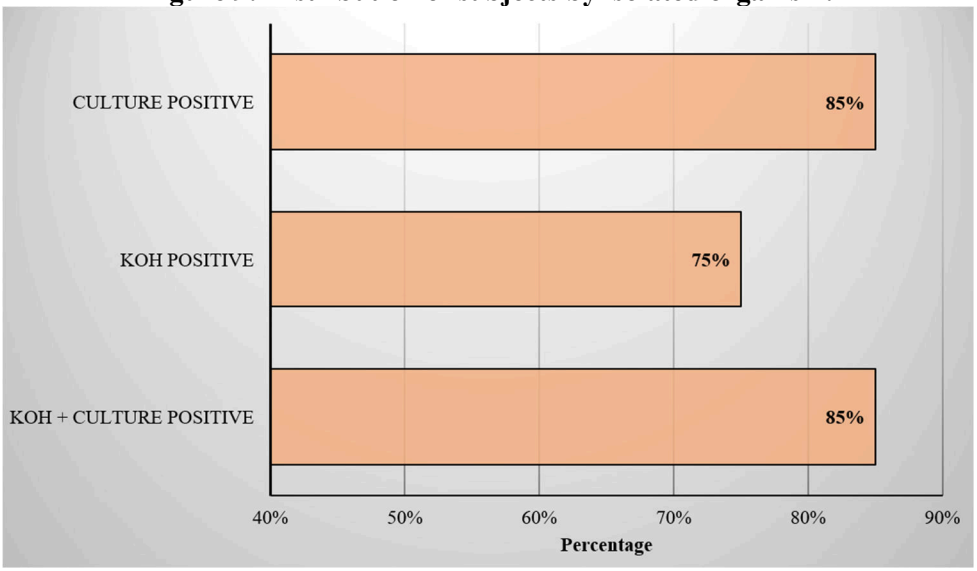


Figure 10: Comparison of result between KOH and culture medium.

Below table gives the agreement between KOH and culture medium.

Table 11: Comparison of KOH and Culture.

		Culture		Kappa value (p-value)
		Positive	**Negative**	
KOH	**Positive**	75 (75%)	0 (0%)	0.6923 (<0.00001*)
	Negative	10 (10%)	15 (15%)	

By Cohen's Kappa we observe that, there is significant moderate agreement present between KOH medium results and culture results and agreement between these two observed as 90%. Below table gives the sensitivity, specificity and other parameters of KOH compared to culture.

Table 12: Diagnostic parameters of KOH over culture.

Parameters	Percentage (Confidence interval)
Sensitivity	88.2 (79, 94)
Specificity	100 (78, 100)
Positive predictive value	100 (95, 100)
Negative predictive value	60 (39, 79)
Area under curve	0.8 (0.702, 0.898)

Below plot is ROC for KOH over culture.

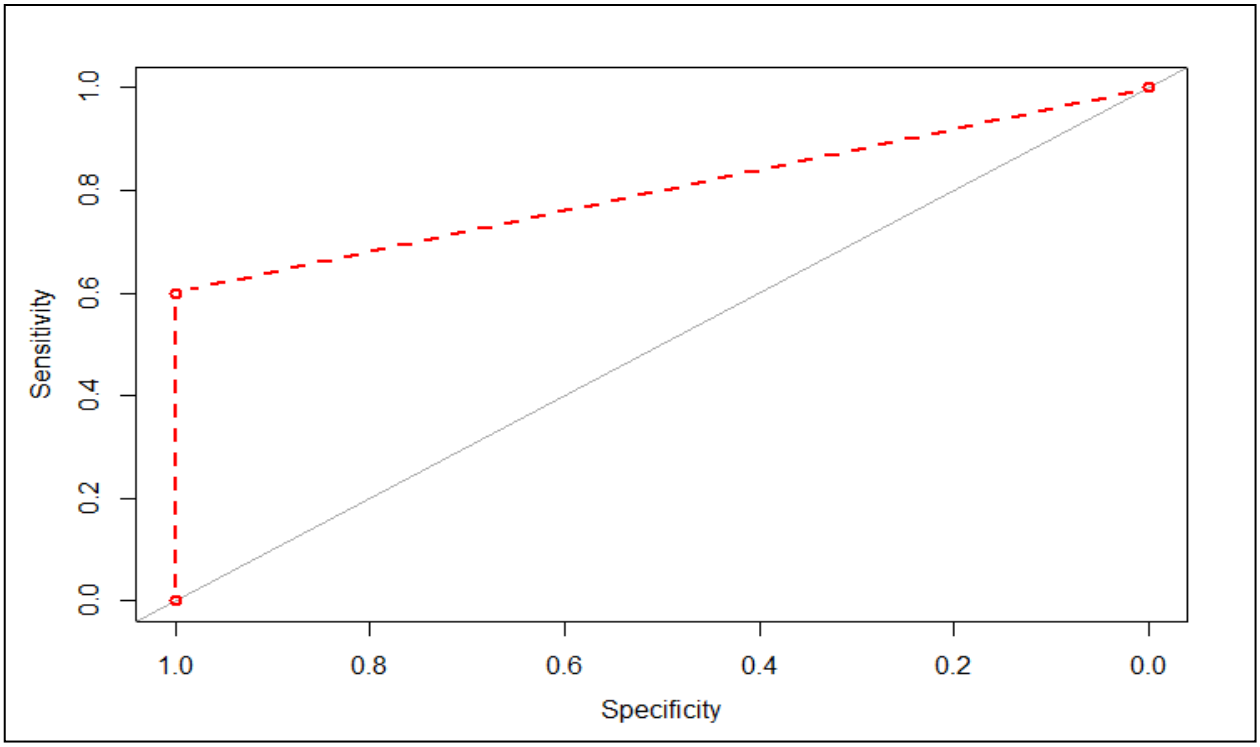


Figure 11: ROC for KOH in comparison with culture results.

DISCUSSION

Ive and Marks first described tinea incognito in 14 individuals with dermatophytic infection who had an abnormal clinical presentation due to previous steroid treatment in 1968. This happened in 1960s, after these medications were introduced for the topical treatment with variety of dermatologic illnesses. Since then, more cases involving the topical administration of pimecrolimus and tacrolimus were reported, though the topical or systemic usage of Corticosteroids remains most common. [58]

As some nations have over-the-counter access to steroids and other immunosuppressants, as well as an increase in drugs containing steroids, tinea in-cognito is more common, and the diagnosis is sometimes missed . These medications impair the usual cutaneous immune response to dermatophytes, allowing fungal superficial infections to spread faster. Some doctors, particularly non dermatologists, give steroid-antifungal mixtures like betamethasone and clotrimazole, which had betamethasone with greater affect than the antifungal, exacerbating superficial dermatophytosis.[59]

Male patients (61%) were found to be infected more than female(39%) patients in our study . Our study also discovered that the age group most typically affected was 31-40 years, followed by 20-30 years. The age range of the group was 37.14 yrs. Italian research of tinea incognito,[60] the gender distribution and mean age was identical (42 years). Kim. J.W. et al. [61] found an equal gender distribution with a younger mean age (32.6 yr). According to the findings of all of these research, tinea incognito primarily affects people between the ages of 30 and 40.

61% of the participants in our study were from the lower-middle socioeconomic strata. Hanumanthappa et al. showed that the majority of dermatophyte infections occur in the lowest socioeconomic strata, followed by the lower-middle and medium socioeconomic strata. Poor living circumstances, lack of hygiene, overcrowding, and poor nutrition stimulate the growth of dermatophytes in lower socioeconomic groups, increasing the risk of infection, chronicity, and recurrence.[62]

In our analysis, pharmacists treated the vast majority of patients (61 %). As per Ansar et al,

64.3 %of their patients were treated at home by themselves, friends, or family, 21.4% by health personnel, and 14.3 % by dermatologists. Another Korean study found that 40.6 % tinea in-cognito patients had previously had therapy from a dermatologist, 43.8 %had received treatment from non-dermatologists, and 15.5 %had self-medicated.[63]

Self-medicated patients used only topical steroids (34%), whereas dermatologists and non-dermatologists used diversity of treatment modalities, including topical/systemic steroids, topical/systemic antibiotics, topical calcineurin inhibitors, intralesional steroid injections, or a combination of those drugs. We found no examples of tinea incognito producing a topical calcineurin inhibitor, implying a higher cost and later onset.

In the present investigation, well to ill defined plaque with less scaling and more erythema seen in 48 % of the 100 clinically identified instances with Tinea in-cognito, 27 % of the cases matched the clinical findings of eczema, and 23 % psoriasis. According to several research, tinea incognita clinical symptoms are frequently mistreated as eczema and impetigo.[64]

Most common anatomical site of distribution with tinea incognito lesions was glabrous skin, notably trunk and groin (30 %), followed by the face (14 %). Similar studies in

Italy and Korea demonstrated that glabrous skin and the face are the most typically affected areas.[61]

KOH positivity was 75 % in our survey, and cultural positivity was 85 %. The percentage of KOH positive was 52.2 % in a study conducted by Banarjee U in Delhi.[65] Gerceker Turk et al. reported 80.95 % KOH positive and 90 % culture positivity in another investigation. In this study, Trichophyton mentagrophyte (T. mentagrophyte) was shown to be the most usually recognised dermatophyte. Tigga et al.[30] unearthed that Trichophyton mentagrophytes account for 97.2 %of isolated dermatophytes.

Trichophyton mentagrophytes not only has increased prevalence over the last decade, but it has also supplanted Trichophyton rubrum in a few studies.[66]

Trichophyton mentagrophytes grows rapidly in 5–7 days, which could explain the widespread involvement, inflammatory lesions, and fomite transmission seen today.[16]

CONCLUSION

Despite the fact that this entity was first reported about 50 yrs ago, continues to be a source of diagnostic conundrum and therapeutic concern. In their regular practise, dermatologists are bound to encounter patient with tinea incognito. Patients prefer pharmacist-dispensed medications as they are well-known and provide immediate comfort. Steroid antifungal combo medications are widely available, inexpensive, and come with a lengthy number of false indications written in regional languages.

Dermatophytosis can mimic wide spectrum of dermatoses, making clinical diagnosis difficult. Simple diagnostic tests have high success rate, to easily detect and treat. It is necessary to reach out to areas where misinformation exists and educate people about the hazards of steroid usage, which can turn mild and treatable infections into chronic and persistent dermatological disorders.

Dermatological associations need to take action at state and national levels. It is high time for strict legislative restrictions to be established on free distribution of such pharmaceuticals, which is double-edged blade, becoming a bane rather than a benefit.

SUMMARY

Dermatophyte distribution varies by geographical area and environmental circumstances. There is also a lot of variance spectrum of dermatophytic isolates. The focus of this research was to learn more about the clinical , mycological characteristics of dermatophytosis.

This one year cross sectional study was carried out in the Department of Dermatology, Venereology and Leprosy, KLES Hospital and Medical Research Centre, Belgaum dated January 2020 to December 2020. Total of 100 patients presenting with dermatophyte skin infection were studied. The patients subjected to clinical evaluation, KOH examination for fungi and culture.

Most patients were males (61%) with male to female (39%) ratio of 2:1 .The commonest age group 31 to 40 years (30%). Most patients (67%) reported duration of 7-12 months(67%) application of steroids. On clinical examination ,the commonest morphological variant was noted as annular (48%%), with most common presentation seen on trunk i.e Tinea corporis(30%). The KOH examination for fungus was positive in 75% of the cases and culture positive in 85% of the cases. Among patients with positive culture, *T. mentagrophyte* was the commonest isolate noted in 67% of patients.

Dermatophytosis has a wide range of clinical and mycological traits, according to this study. Monitoring the abundance of these fungi allows for the early detection of new organisms and may aid in treatment and efficacy of current pharmaceutical regimens.

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ANNEXURE I – ETHICAL CLEARANCE LETTER

K.L.E. ACADEMY OF HIGHER EDUCATION AND RESEARCH
(Deemed – to- be- University)
Accredited 'A' Grade by NAAC (2nd Cycle) Placed in Category 'A' by MHRD (GoI)
JAWAHARLAL NEHRU MEDICAL COLLEGE,
NEHRU NAGAR, BELAGAVI-590010 (KARNATAKA-INDIA)
Website: http//www.jnmc.edu
E-Mail : dome@jnmc.edu
Phone: (+ 91-(0)831 Office : 2472550
Principal: 2471701
Fax No. +91 (0)831 – 2470759

Ref: MDC/DOME/ 280. **Date: 24/12/2019**

To,

BT0119005
PG student in Dermatology, Venereology & Leprosy,
J.N.Medical College,
BELAGAVI.

Sub: Institutional Ethical Clearance for the study.

With reference to the above, we wish to inform you that your proposed research project titled **"CROSS SECTIONAL STUDY OF TINEA INCOGNITO WITH CLINICAL AND MICROBIOLOGICAL CORRELATION",** is ethical and justifiable. The proposed research project has been cleared by the JNMC Institutional Ethics Committee on Human Subjects Research.

(Dr. Anita Dalal)
Member Secretary
JNMC Institutional Ethics Committee
on Human Subjects Research,
J.N.Medical College, Belagavi.

(Dr. Roopa M Bellad)
Chairman,
JNMC Institutional Ethics Committee
on Human Subjects Research,
J.N.Medical College, Belagavi.

ANNEXURE II – PROFORMA

CROSS SECTIONAL STUDY OF TINEA INCOGNITO WITH MICROBIOLOGICAL CORRELATION

Case No. **OP/IP No.**

Name:

Age:

Sex:

Occupation:

Income:

Address with phone number:

Presenting complaints and duration:

History of present illnes:

:

Past History:

Family History:

Treatment History:

Personal History:

Diet: veg/mixed
Sleep: normal/disturbed
Appetite: normal/disturbed
Bowel and Bladder: normal/disturbed
Habits:

General Physical Examination:

Built: Poor/moderate/Good

Vitals: Pulse

BP:

Temperature:

Weight:

Pallor / Icterus/cyanosis /

Clubbing/Lymphadenopathy

Inspection:

Clinical examination of lesions

- PATTERN:
- DISTRIBUTION:
- SITE:
- SYMMETRY:
- SECONDARY CHANGES:
- STERIOD INDUCED CHANGES:

- **Palpation:**

  - Surface texture of surrounding skin
  - Tenderness
  - Temperature

HAIR EXAMINATION:

NAIL EXAMINATION:

Systemic Examination:

Cardiovascular system:

Respiratory system:

Per abdomen:

Central nervous system:

Investigations

KOH-

CULTURE-

Diagnosis:-

Signature:

Guide's Signature

INFORMED CONSENT FORM

I.D.NO: | | | |

Title of the study. : "CROSS SECTIONAL STUDY OF TINEA INCOGNITO WITH CLINICAL AND MICROBIOLOGICAL CORRELATION "

Respected Sir/Madam,

We invite you to participate in our study as you are eligible for the same. During the study you will be asked some questions in detail regarding your present complaints.

- **Purpose of the study:** To evaluate various clinical manifestations, most common sites, predisposing factors and causative agents of Tinea incognito.

Procedure:

You will be asked to give a detailed history of your disease, undergo a physical examination and sample will be taken for KOH microscopy and culture.

Risks and Benefits:

The result of you taking part in this research would help health care providers towards a better understanding adverse effects of the disease, and thus we will be able to provide improved patient care.

Alternatives:

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

Privacy and confidentiality:

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

Relations with the Institutional policy:

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

Financial incentives:

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

Authorization to publish results:

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

STATEMENT OF CONSENT

I.D.NO: | | | |

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

Participant's name:

Signature or left thumb print of participant:

Witness name:

Signature of witness:

Signature of the investigator:

Date:

STATEMENT OF ASSENT

I.D.NO: | | | |

I Mr/Ms/Mrs ---------- parent/guardian/ward of---------- consent to enrol my daughter/son to participate in this study. I have read the consent document or it has been read to me in my vernacular language. I give my acceptance on behalf of my daughter/son for her/his participation in the study. All the information regarding this study is provided to me and I have understood the same. I have been given the opportunity to ask questions and obtain appropriate answers.

Participant's name:

Participant's parent/guardian name:

Signature or left thumb print of parent/guardian of participant:

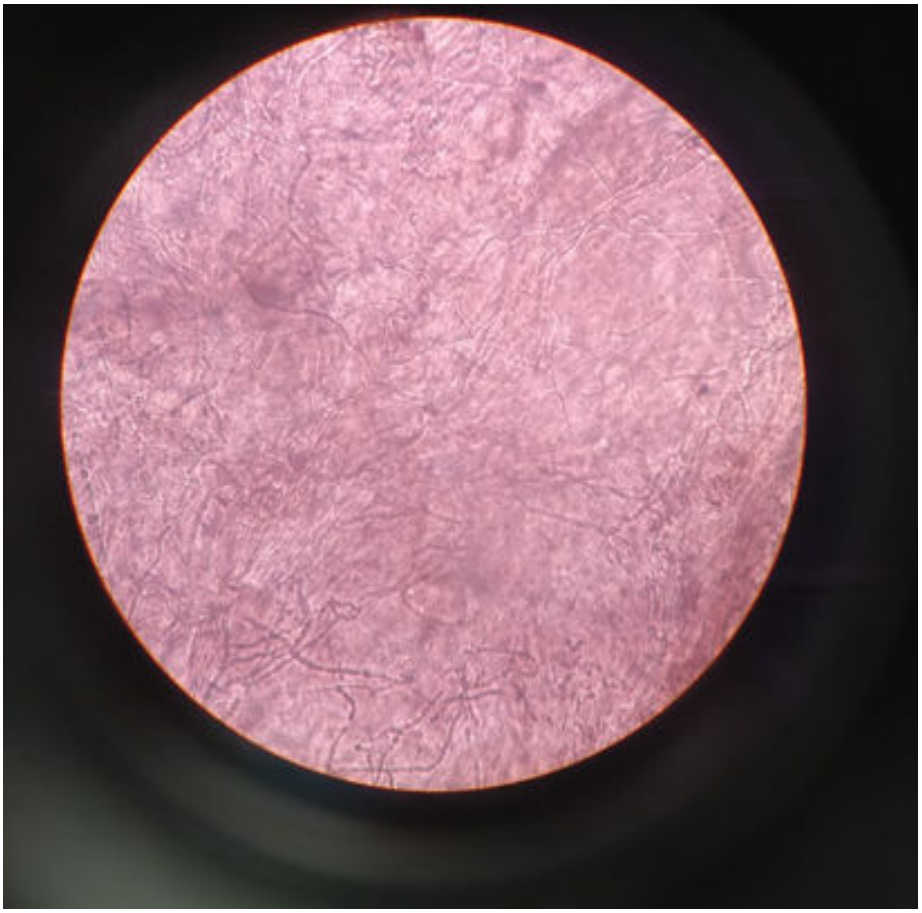
Witness name:

Signature of witness:

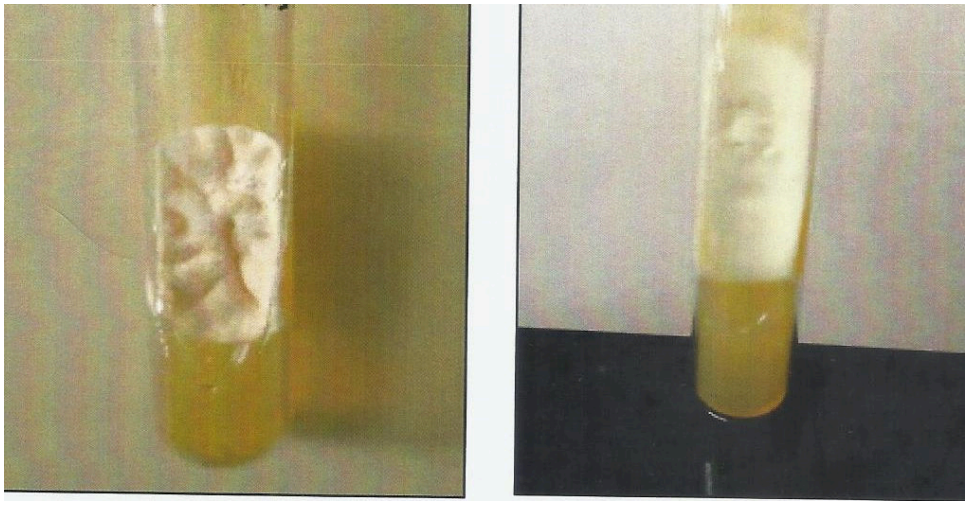
Signature of the investigator:

Date:

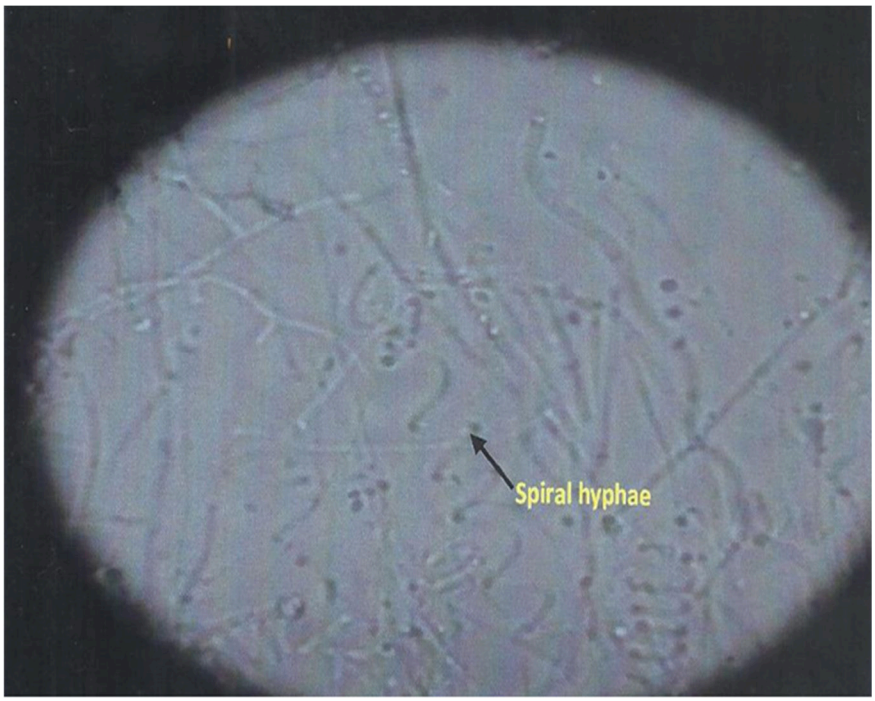
ANNEXURE IV – PHOTOGRAPHS



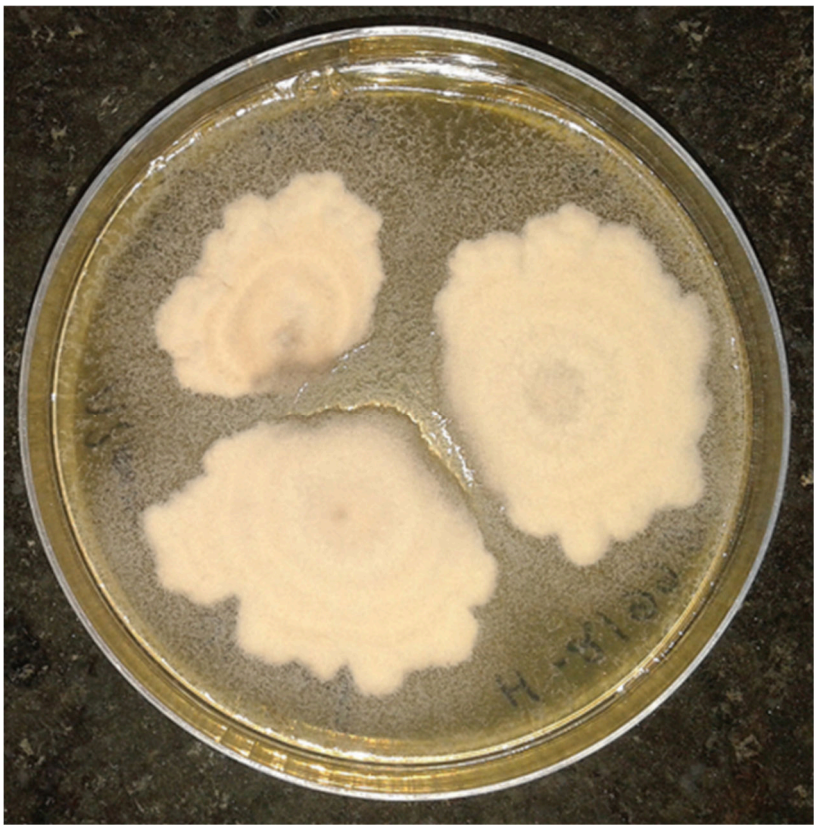
PHOTOGRAPH 1:KOH preparation



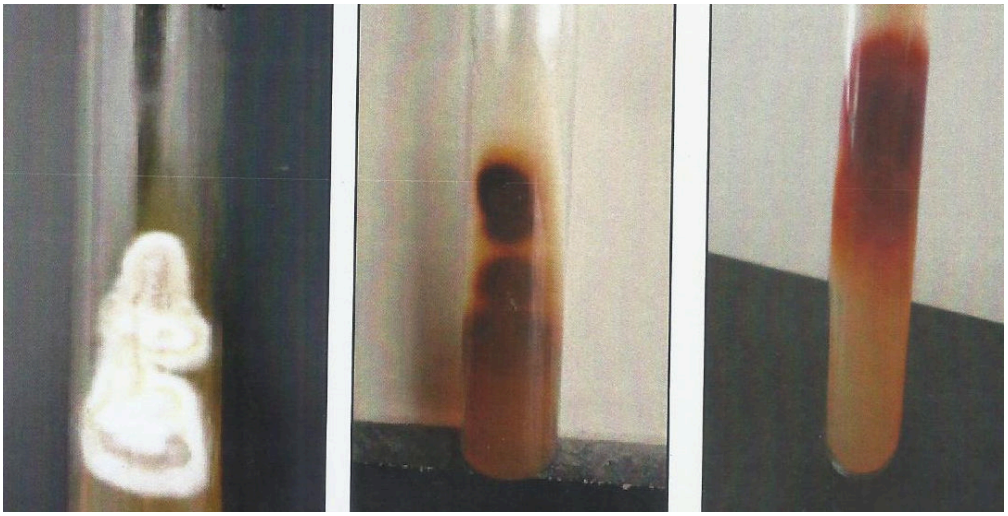
PHOTOGRAPH 2: T. Mentagrophytes in SDA



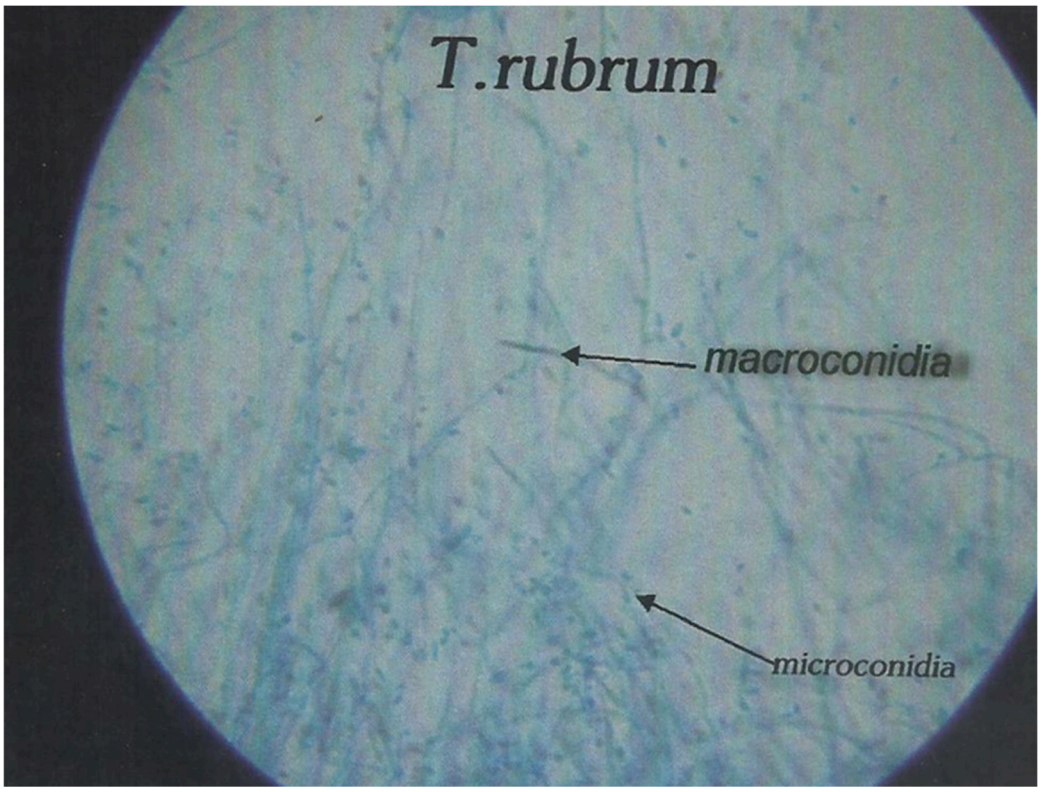
PHOTOGRAPH 3 : LPCB preparation of T. Mentagrophytes.



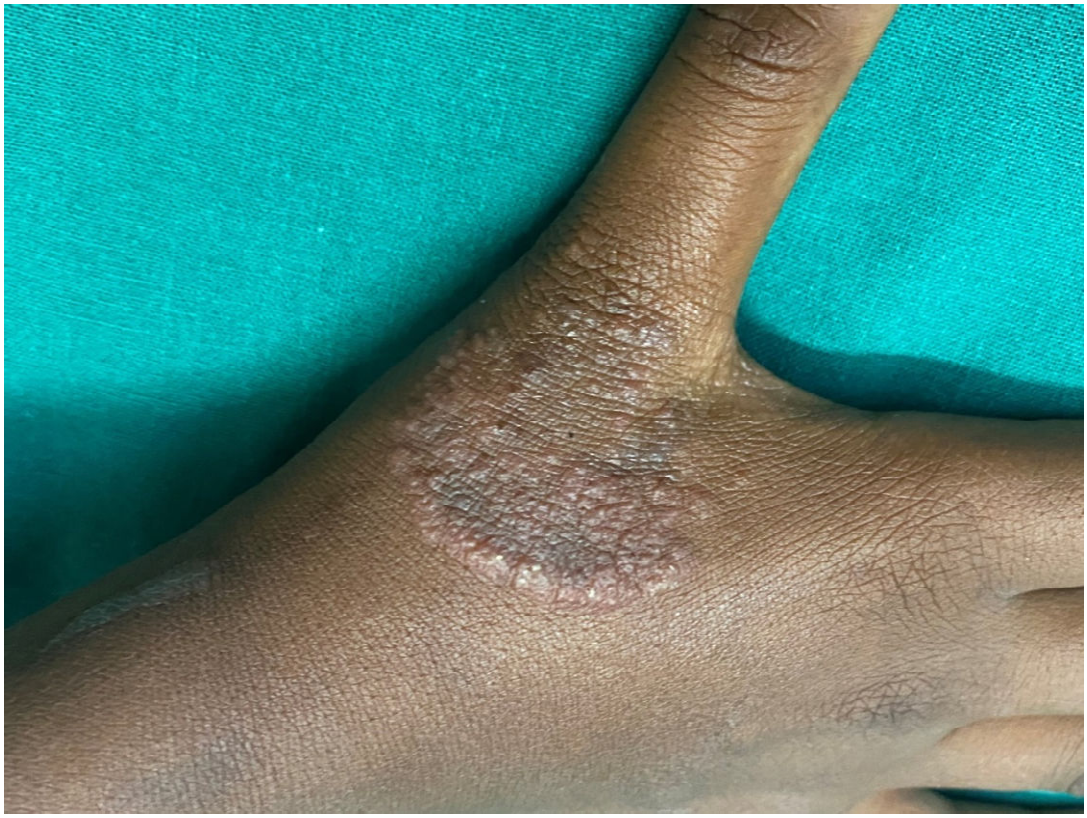
PHOTOGRAPH 4: White to tan, cottony colony of Trichophyton mentagrophytes.



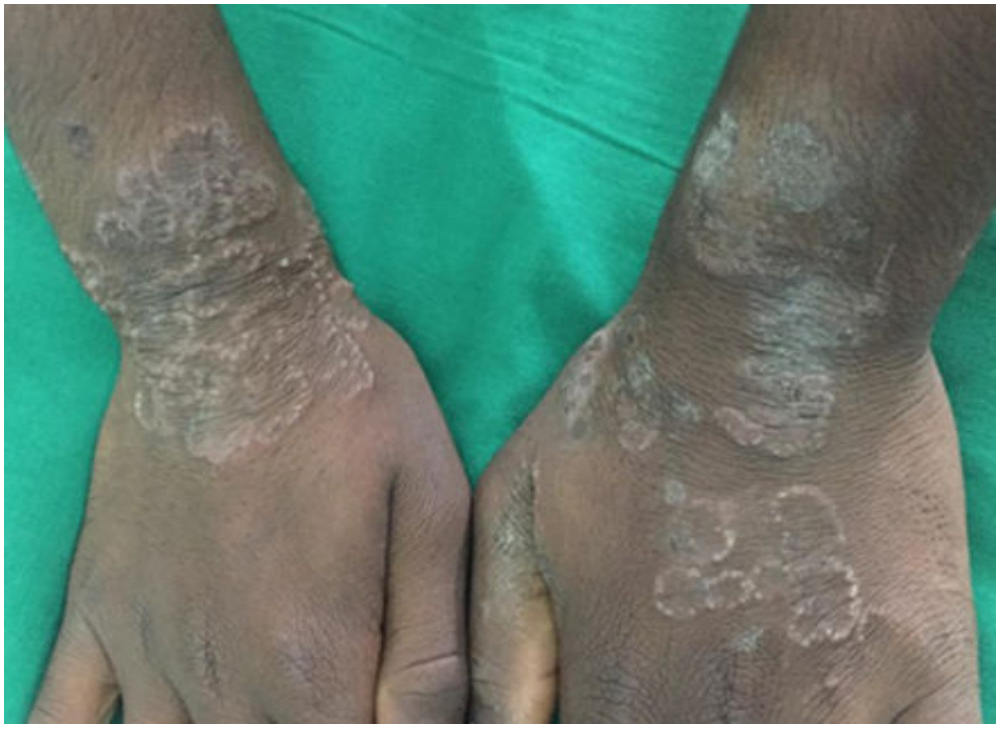
PHOTOGRAPH 5: **T. Rubrum in SDA.**



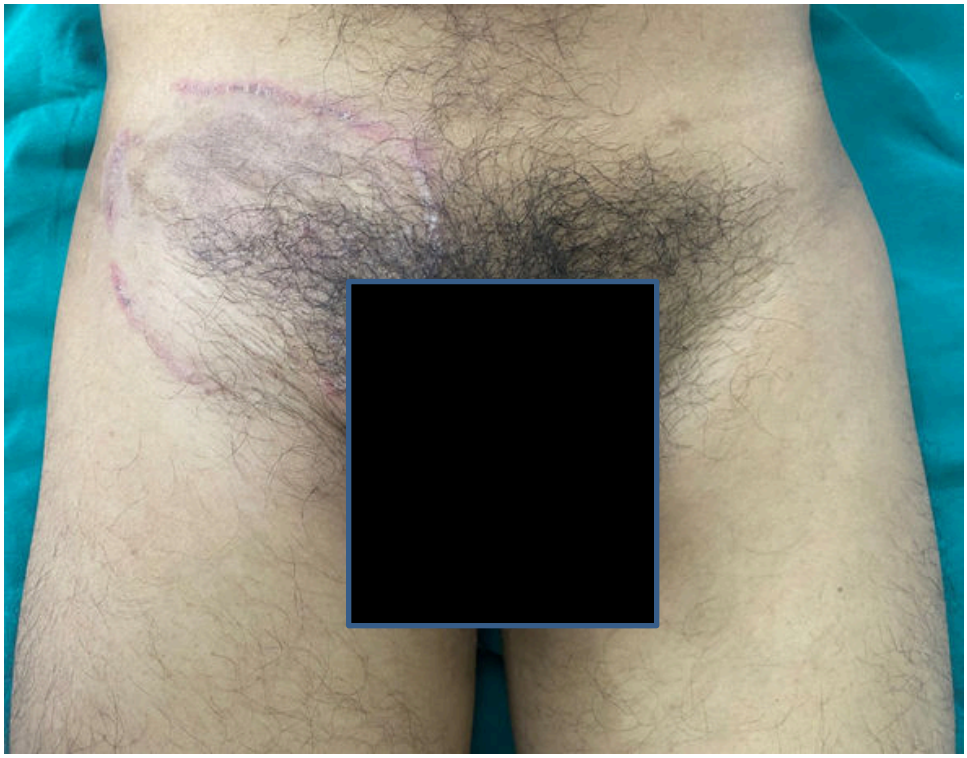
PHOTOGRAPH 6. LPCB preparation of T. Rubrum.



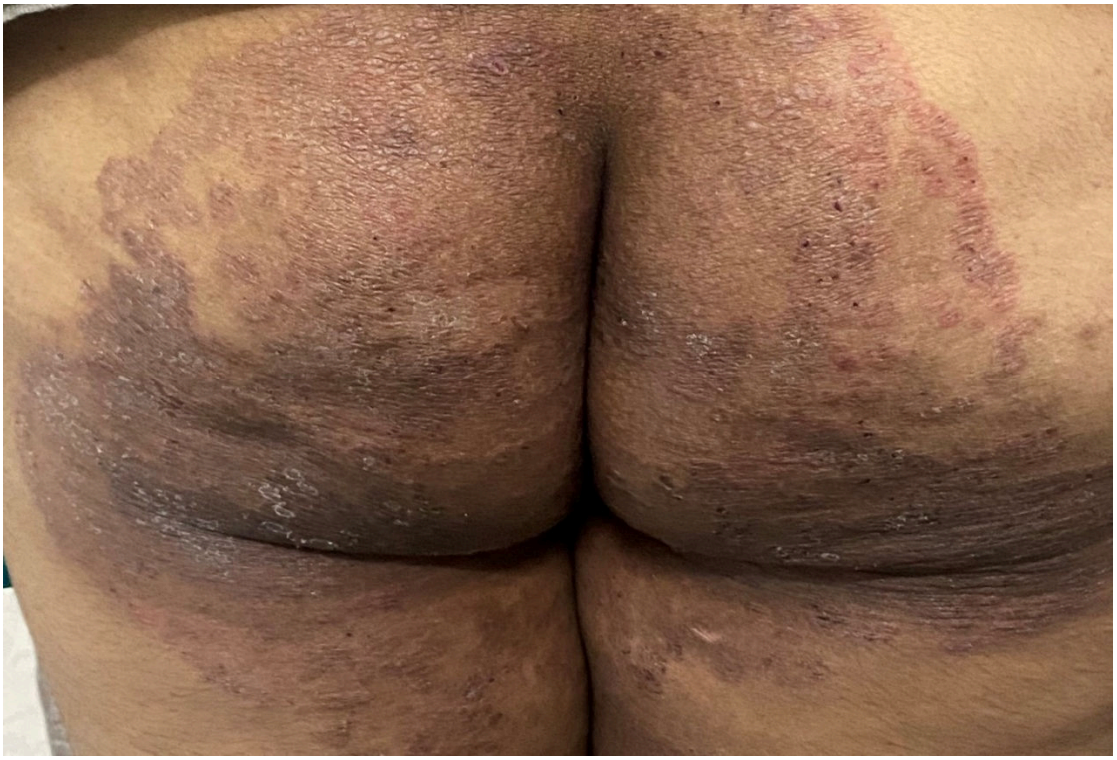
PHOTOGRAPH 7: Tinea incognito masquerading as eczema.



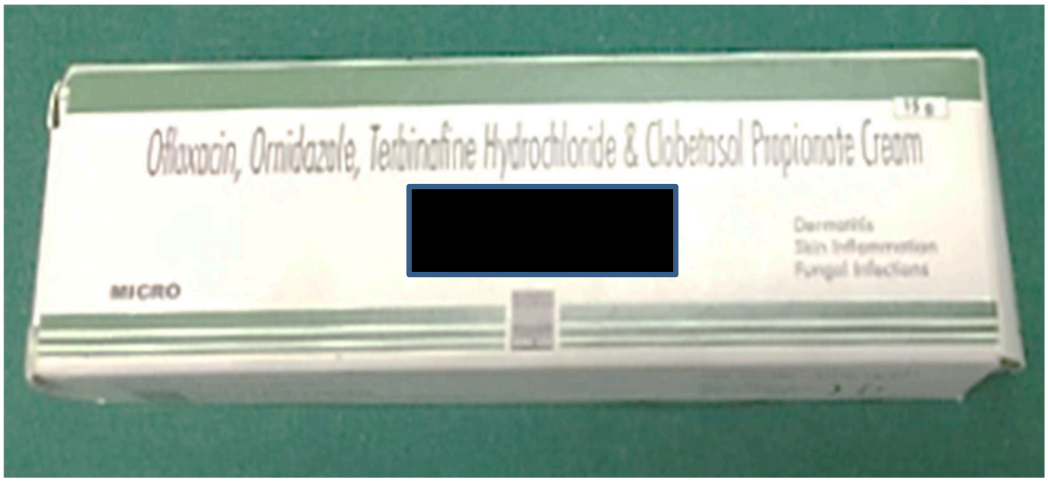
PHOTOGRAPH 8:Tinea mannum.



PHOTOGRAPH 9: Tinea cruris and tinea corporis.



PHOTOGRAPH 10:Tinea corporis.



PHOTOGRAPH 11: Example of a triple combination cream.

Serial Number	Out / In Patient number	Age (Years)	Sex	Occupation	Socio economic status					Clinical diagnosis	Clinical type		Investigations	
						Duration (Months)	Pruritis	STERIODS	Associated disease		Clinical type	Distribution	KOH	Culture
1	5602773	20	M	FA	IV	8	+	TC	-	T INCO/ECZEMA	ECZEMATOUS	GN	-	-
2	5745321	28	F	HW	IV	9	+	TC	-	T.INCOGNITO	T.CORPORIS	GN	+	*T. mentagrophyte*
3	5754602	48	M	FR	III	10	+	STERIODS	DM	T.INCOGNITO	T.CORPORIS	GN	+	*T. RUBRUM*
4	5626629	25	M	ST	III	9	+	_	_	T INCO/ECZEMA	ECZEMATOUS	GN	-	-
5	5570781	34	F	CO	IV	7	+	TC	_	T.INCOGNITO/SLE	T. FACEI	LC	+	*T. mentagrophyte*
6	5751878	44	F	HW	III	12	+	TC	HTN	T.INCOGNITO	T.CORPORIS	GN	+	*T. mentagrophyte*
7	5731185	39	M	W	V	9	+	TC	_	T.INCOGNITO/PSO	T CORPORIS	GN	+	*T.Tonsurans.*
8	5731183	47	M	DR	III	7	+	STERIODS	HTN	T.INCOGNITO/ABCD	T.CORPORIS	GN	+	*T. mentagrophyte*
9	5418821	20	F	ST	IV	8	+	TC	-	T.INCOGNITO/PSO	T.CORPORIS	GN	-	*T.Tonsurans.*
10	3644972	54	M	W	III	6	+	TC	-	T.INCOGNITO	T.CORPORIS	GN	-	*T. mentagrophyte*
11	5433290	37	M	W	III	8	+	TC	=	T INCO/ECZEMA	T.CORPORIS	GN	-	-
12	5454271	43	F	W	III	9	+	-	DM	T INCO/PSO	PSQ	GN	-	-
13	5427133	27	F	HW	II	7	+	STERIODS	-	T INCO/ECZEMA	T.CORPORIS	GN	-	-
14	5073763	26	M	ST	III	12	+	TC	-	T.INCOGNITO	T.CORPORIS	GN	+	*T. RUBRUM*
15	3881939	57	M	FA	III	18	+	TC	-	T.INCOGNITO	T.CORPORIS	GN	+	*T. mentagrophyte*
16	4259689	47	F	HW	III	12	+	-	DM	T INCO/ECZEMA	PSQ	GN	-	-
17	5786821	26	M	W	III	7	+	STERIODS	-	T.INCIGNITO	T.CORPORIS	GN	+	*T. mentagrophyte*
18	5832221	24	M	ST	II	9	+	-	-	T INCO/PSO	PSQ	GN	-	-
19	5496247	53	M	FA	II	12	+	TC	-	T.INCIGNITO	T.CORPORIS	GN	+	*T. RUBRUM*
20	5832194	39	M	FA	III	6	+	TC	-	T INCO/ECZEMA	PSQ	LC	-	-
21	3940477	45	F	HW	IV	6	+	-	HTN	T.INCO/SLE	T.FACEI	FA	+	*T. mentagrophyte*
22	5750543	30	M	DR	II	8	+	-	-	T.INCO/P VER	PSQ	GN	+	*T. mentagrophyte*

23	5597409	27	F	ST	III	6	+	STERIODS	_	T.INCO/P VER	PSQ	LC	+	*T. mentagrophyte*
24	5597527	44	F	ST	III	8	+	TC	-	T.INCIGNITO	PSQ	GN	-	*T. mentagrophyte*
25	5626598	22	M	FA	III	10	+	TC	-	T.INCIGNITO	PSQ	GN	-	*T. RUBRUM*
26	5429144	48	M	ST	III	9	+	TC	DM	T.INCIGNITO/PSO	Plaque	GN	-	*T. mentagrophyte*
27	5715459	40	F	BA	II	13	+	_	_	T.INCIGNITO	PSQ	GN	+	*T. RUBRUM*
28	1695499	34	M	FA	III	8	+	-	_	T.INCIGNITO/PSO	PSQ	LC	+	*T. mentagrophyte*
29	1006810	35	F	HW	III	18	+	STERIODS	-	T.INCIGNITO	PSQ	GN	+	*T. mentagrophyte*
30	5865377	39	M	ENG	II	6	+	-	HTN	T INCO/ECZEMA	PSQ	LC	-	-
31	5602457	24	F	ST	III	5	+	TC	-	T INCO/PSO	PSQ	LC	-	-
32	5834819	37	M	W	II	7	+	_	-	T INCO/EZCEMA	PSQ	LC	-	-
33	5362158	30	M	FA	III	9	+	TC	-	T.INCOGNITO	T.CORPORIS	GN	+	*T. mentagrophyte*
34	5425987	25	M	ST	II	8	+	TC	-	T.INCOGNITO/PSO	T.CORPORIS	LC	+	*T. mentagrophyte*
35	5025955	28	M	FA	III	6	+	TC	-	T.INCOGNITO/PSO	T.PEDIS	LC	+	*T. mentagrophyte*
36	5731183	37	M	ST	III	8	+	TC	-	T.INCOGNITO/ECZ	T.CORPORIS	GN	+	*T. mentagrophyte*
37	5916604	57	F	HW	II	7	+	STERIODS	DM	T.INCOGNITO	T.CORPORIS	GN	+	*T. mentagrophyte*
38	5916602	21	M	ST	III	8	+	STERIODS		T.INCOGNITO	T.CRURIS	LC	+	*T.Tonsurans*
39	5716851	37	M	BA	III	10	+	STERIODS		T.INCOGNITO	PSQ	GN	+	*T. mentagrophyte*
40	5850927	27	M	ST	III	6	+	-	-	T INCO/ECZEMA	PSQ	LC	-	-
41	5715450	40	F	W	II	8	+	TC	-	T.INCOGNITO	T.CORPORIS	GN	+	*T. RUBRUM*
42	4933609	43	M	DR	III	9	+	TC	-	T.INCOGNITO	T.CORPORIS	GN	+	*T. mentagrophyte*
43	2458174	43	F	HW	II	8	+	TC	-	T.INCOGNITO/PSO	T.MANUM	LC	+	*T.Tonsurans*
44	5754553	50	M	BA	III	8	+	STERIODS	-	T.INCOGNITO/ABCD	PSQ	GN	+	*T. RUBRUM*
45	5699866	24	F	ST	II	9	+	TC	-	T.INCOGNITO/LE	PSQ	GN	+	*T. mentagrophyte*
46	2960881	41	F	HW	III	10	+	STERIODS	-	T.INCOGNITO	Annular,Plaque	GN	+	*T. mentagrophyte*
47	1005022	59	M	BA	II	8	+	SAPAT	-	T.INCOGNITO/RO	T.FACEI	LC	+	*T.Tonsurans*
48	5751878	43	F	ST	III	9	+	TC	-	T.INCOGNITO	Annular	GN	+	*T. mentagrophyte*
49	5597628	40	M	FA	III	8	+	TC	-	T.INCOGNITO/PSO	PSQ	GN	+	*T. mentagrophyte*

50	5742780	19	M	ST	II	12	+	-	-	T.INCOGNITO	PSQ	GN	+	*T. rubrum*
51	5714166	36	M	ST	III	6	+	STERIODS	-	T.INCO/ECZEMA	Annular	LC	+	*T. mentagrophyte*
52	5896505	40	F	HW	II	8	+	TC	-	T.INCOGNITO	Annular	GN	+	*T. mentagrophyte*
53	5551258	69	M	RT	III	9	+	TC	-	T.INCOGNITO	PSQ	GN	+	*T.Tonsurans*
54	3008069	53	F	BA	II	8	+	-	-	T.INCO/RO	T.FACEI	LC	+	*T. mentagrophyte*
55	4721972	56	M	FA	III	9	+	-	-	T.INCO/PSO	T.CORPORIS	GN	+	*T. rubrum*
56	5834565	23	M	ST	III	12	+	-	-	T INCO/EZCEMA	PSQ	LC	-	-
57	5925956	34	F	FA	III	8	+	TC	-	T.INCOGNITO	Plaque	GN	+	*T. mentagrophyte*
58	5438769	44	F	FA	III	9	+	TC	DM	T.INCOGNITO	Annular	LC	+	*T. mentagrophyte*
59	5892521	25	M	ST	II	7	+	STERIODS	-	T.INCOGNITO	Annular	GN	+	*T. mentagrophyte*
60	5923462	39	M	W	III	9	+	TC	-	T.INCOGNITO/ECZ	PSQ	LC	+	*T. mentagrophyte*
61	5393428	45	F	HW	II	8	+	SAPAT	-	T.INCOGNITO	Vesicular	LC	+	*T. rubrum*
62	3919223	55	M	FR	III	7	+	TC	DM	T.INCOGNITO	PSQ	LC	+	*T. mentagrophyte*
63	5903367	31	F	HW	III	8	+	TC	-	T.INCOGNITO/ECZ	PSQ	LC	-	*T. mentagrophyte*
64	5849153	34	M	FR	II	9	+	TC	-	T.INCOGNITO	Annular	GN	+	*T. mentagrophyte*
65	5898964	29	M	DR	III	12	+	TC	-	T.INCOGNITO/PSO	T.MANUM	LC	+	*T. mentagrophyte*
66	5898986	22	F	ST	II	6	+	-	-	T INCO/EZCEMA	Eczema	GN	-	-
67	5949103	56	F	HW	III	9	+	TC	HTN	T.INCOGNITO	Annular	LC	+	*T. mentagrophyte*
68	5948500	32	M	DR	III	12	+	TC	-	T.INCOGNITO	PSQ,Annular	GN	+	*T. mentagrophyte*
69	5948574	44	M	FA	III	7	+	-	-	T.INCO/ECZEMA	T.CRURIS	LC	+	*T. rubrum*
70	5950689	53	M	FA	II	6	+	STERIODS	HTN	T.INCOGNITO	T.CORPORIS	GN	+	*T. mentagrophyte*
71	5871836	33	M	FA	IV	7	+	STERIODS	-	T.INCOGNITO	PSQ	LC	+	*T. mentagrophyte*
72	598975	25	F	ST	III	9	+	SAPAT	-	T.INCOGNITO	T.CORPORIS	GN	+	*T. rubrum*
73	5895728	44	F	HW	III	7	+	TC	-	T.INCOGNITO/SLE	T.FACEI	LC	+	*T. mentagrophyte*
74	4483758	45	M	W	II	6	+	TC	-	T.INCOGNITO	T.CORPORIS	LC	+	*T. mentagrophyte*
75	5971355	44	F	HW	III	12	+	STERIODS	-	T.INCOGNITO	Plaque,PSQ	GN	+	*T. mentagrophyte*
76	5973777	29	F	HW	II	8	+	TC	-	T.INCOGNITO	Annular	LC	+	*T. rubrum*

77	2050979	34	M	DR	II	9	+	TC	-	T.INCOGNITO/PSO	T.PEDIS	LC	+	*T. mentagrophyte*
78	5976486	32	F	BA	II	12	+	STERIODS	-	T.INCOGNITO	PSQ	LC	+	*T. mentagrophyte*
79	5979278	29	M	W	I	11	+	TC	-	T.INCOGNITO/ECZEMA	PSQ,Eczema	GN	+	*T. mentagrophyte*
80	5974116	43	M	PO	III	8	+	STERIODS	DM	T.INCOGNITO	Plaque	LC	+	*T. rubrum*
81	5990400	23	F	ST	II	8	+	STERIODS	-	T.INCOGNITO/ECZ	Plaque	LC	+	*T. rubrum*
82	6002494	43	M	FA	III	5	+	TC	-	T.INCOGNITO	Plaque,Annular	GN	+	*T. rubrum*
83	3680463	21	M	ST	II	12	+	SAPAT	-	T.INCOGNITO/SLE	T.FACEI	LC	+	*T. mentagrophyte*
84	5968420	33	M	W	II	9	+	TC	DM	T.INCOGNITO/PSO	Annular	LC	+	*T. mentagrophyte*
85	690074	22	M	DR	III	18	+	TC	-	T.INCOGNITO	PSQ	LC	+	*T. mentagrophyte*
86	2977145	42	F	HW	III	15	+	TC	-	T.INCOGNITO	PSQ	GN	+	*T. rubrum*
87	6008751	32	F	T	II	7	+	TC	-	T.INCOGNITO	PSQ	LC	+	*T. rubrum*
88	5472742	23	M	ST	III	9	+	STERIODS	-	T.INCOGNITO/PSO	T.MANUM	LC	-	*T. rubrum*
89	5932148	53	M	FA	III	7	+	TC	HTN	T.INCOGNITO	Annular	LC	+	*T. rubrum*
90	5569171	42	F	HW	III	8	+	SAPAT	-	T.INCOGNITO	PSQ	LC	+	*T. mentagrophyte*
91	5928928	32	M	FA	III	6	+	STERIODS	-	T.INCOGNITO	T.CORPORIS	LC	-	*T. mentagrophyte*
92	6000898	43	M	FA	IV	12	+	TC	DM	T.INCOGNITO	PSQ	LC	+	*T. mentagrophyte*
93	5916604	53	F	DR	III	9	+	TC	-	T.INCOGNITO	Plaque	LC	+	*T. mentagrophyte*
94	5471654	38	M	FA	III	12	+	STERIODS	-	T.INCOGNITO	PSQ	LC	-	*T. rubrum*
95	6025747	33	F	W	IV	8	+	TC	-	T.INCOGNITO/ECZEMA	PSQ	LC	-	-
96	6025599	32	M	FA	III	7	+	TC	-	T.INCOGNITO	Annular	LC	+	*T.Tonsurans*
97	5997330	44	M	FA	III	12	+	STERIODS	-	T.INCOGNITO	Annular	LC	-	*T. mentagrophyte*
98	6037670	24	F	FA	III	9	+	TC	-	T.INCOGNITO/PSO	PSQ	LC	+	*T. mentagrophyte*
99	5565703	32	M	W	III	10	+	TC	-	T.INCOGNITO	PSQ	GN	+	*T. mentagrophyte*
100	6096943	44	M	PO	III	9	+	STERIODS	HTN	T.INCOGNITO/PSO	T.CORPORIS	GN	+	*T. mentagrophyte*

KEY TO MASTER CHART

FA -FARMER

W -WORKERS

PO -POLICE

HW -HOUSEWIFE

DR -DRIVER

ST -STUDENT

BA -BANKER

ENG- ENGINEER

TC -TRIPLE COMBINATION

PSO -PSORIASIS

ECZ -ECZEMA

PSQ -PAPULOSQAMOUS

GN -TRUNK/BODY

LC -LOCALISED

T -TRICHOPHYTON

RO -ROSACEA

LE -LEPROSY

T .INCOGNITO-TINEA INCOGNITO

HTN -HYPERTENSION

DM -DIABETES