

**CLINICO-AETIOLOGICAL STUDY OF FOOT INTERTRIGO IN A
TERTIARY CARE CENTRE**



By
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**DOCTOR OF MEDICINE
IN
DERMATOLOGY, VENEREOLOGY AND LEPROSY**

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April 2015**

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LIST OF ABBREVIATIONS USED

CD	Contact Dermatitis
CICD	Cumulative Irritant Contact Dermatitis
CoNS	Coagulase Negative Staphylococcus
FI	Foot Intertrigo
GABHS	Group-A Beta- Hemolytic Streptococcus
GNB	Gram Negative Bacilli
GPC	Gram Positive Cocci
HKD	Hyper Keratotic Dermatoses
KOH	Potassium Hydroxide
LPCB	Lacto Phenol Cotton Blue Test
MRSA	Methicillin Resistant Staphylococcus aureus
MSSA	Methicillin Sensitive Staphylococcus aureus
NDM	Non Dermatophytic mould
SDA	Sabouraud's Dextrose Agar
TWI	Toe Web Infection

ABSTRACT

BACKGROUND: Intertrigo is an inflammatory skin eruption, infectious or non infectious, of two closely opposed skin surfaces caused by friction in conjunction with moisture trapped in deep skin folds. Foot intertrigo, occurring in the toe web spaces, is a relatively common and troubling disorder, especially in hot and humid climate, like in India. The principal etiologic factors in this disorder are mechanical, but chronic bacterial or fungal infection becomes operative, when suitable conditions are present. Clinicians often treat foot intertrigo as purely a fungal infection, overlooking role of predisposing factors and the possibility of added bacterial infection. Also, very few studies have been conducted internationally to evaluate the etiology of interdigital foot intertrigo and even fewer Indian studies addressing the same in Indian population.

AIMS: To evaluate the clinical features and pre-disposing factors of foot intertrigo and to determine the causative organisms of toe web space infections.

MATERIALS AND METHODS: The present study was a hospital based cross sectional study, carried out at Sri R.L.Jalappa Hospital and Research centre, Tamaka, Kolar, from January 2012 to June 2014 over a period of 18 months. All patients reporting to the Department of Dermatology at the above mentioned hospital were evaluated for inclusion into the study and a total of 115 patients having clinically diagnosed foot intertrigo were enrolled into the study. Detailed history including the presenting illness, predisposing factors, systemic diseases, past history and family history were taken. A

complete clinical examination including detailed dermatological examination and relevant investigations were performed. Specimens were collected from the lesions in the affected toe web spaces and subjected for bacteriological and fungal studies.

RESULTS: Majority of the patients with foot intertrigo belonged to the age group of 41 to 50 years (26.1%). Mean age was 44.7 ± 17 . Males were 50.4% and 49.6% were females. It was most commonly seen in housewives (33.9%) followed by agriculturists (19.1%). Onset of the disease was insidious in 77.4% and acute in 22.6%. Majority (61.7%) had the disease for at least more than a year and 27.8% had foot intertrigo for < 6 months. Most common predisposing factors were repeated exposure to wet conditions (49.6%) and occlusive foot wear (19.1%) followed by poor hygiene (11.3%), hyperhidrosis (8.7%), foot deformity (4.3%), and obesity (3.5%). Only 60% were symptomatic and most common symptoms were itching in 36.5%, pain in 20%, burning sensation in 13.9%. Most common morphologic presentation was maceration (72.2%) and scaling (38.2%). Bilateral involvement was seen in 53.9% and unilateral in 46.1%. Most commonly affected toe web space was the 4th toe web space (85.21%), followed by the 3rd toe web space. Diabetes mellitus was the most common associated disease noted in 24.3%, followed by concomitant fungal infection at other body areas seen in 21.7%. Cellulitis was seen in 4.3%. Fungal elements were seen in 25 (21.7%) cases, on KOH mount. Fungal culture showed growth in 16 (13.91%) cases of which *Candida albicans* was isolated in 8 cases, *Aspergillus niger* in 4 cases and *Candida tropicalis*, *Microsporum nanum*, *Aspergillus flavus* and *Fusarium* species were isolated in 1 sample each. Bacterial culture was positive in 100 cases. Skin commensals like Micrococci and CoNS

constituted 35 cases (30.43%). Gram positive bacterial pathogens were isolated in 26.9% and Gram negative in 50.4% cases. Most commonly isolated organism was MSSA in 24 cases (24.3%), *P.aeruginosa* in 14 cases (12.2%), *Acinetobacter* in 12 (10.4%).

In total of 34 mixed infections, Gram negative with Gram negative infection constituted 13 cases of mixed infections. Gram positive with Gram positive infection was seen only in 1 case. Incidence of fungal with Gram positive was seen in 8 cases and Fungal with gram negative bacteria seen in 9 cases.

Most common type of Intertrigo was mixed toe web intertrigo seen in 34 cases (29.6%), followed by single organism bacterial intertrigo in 32 cases (27.82%). Simple intertrigo was seen in 29 cases (25.2%), isolated fungal infection was seen in 8 cases (7%), contact dermatitis was seen in 6 cases (5.2%). Miscellaneous causes of intertrigo (5.2%) included 3 cases of soft corn, 2 cases of palmo-plantar psoriasis, 1 case of scabies.

CONCLUSION: Aetiology of foot intertrigo is variable and it occurs due to a complex interplay between various local predisposing factors, host factors, bacterial and fungal organisms. It can be simple intertrigo where only mechanical and physical factors play a role or it may be primarily or secondarily infected by various bacterial and fungal pathogens resulting in mixed toe web infections. Also clinical manifestations can be similar in majority of the cases with maceration being the most common presentation, hence, diagnosis cannot be made based only on clinical features, making appropriate microbiological studies imperative.

Keyword: Foot intertrigo, toe web infection, tinea pedis, Gram negative toe web infection

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**CLINICO-AETIOLOGICAL STUDY OF FOOT INTERTRIGO IN A
TERTIARY CARE CENTRE**

INTRODUCTION

INTRODUCTION

Intertrigo is an inflammatory skin condition, infectious or non infectious, of two closely opposed skin surfaces that presents as a moist erythematous lesion and scaling.^{1,2} This condition can affect people of all ages and either sex and can be symptomatic or asymptomatic.

The principal etiologic factors in this disorder are mechanical, but chronic bacterial or fungal infection becomes operative when suitable conditions are present. The condition is most commonly seen in groin, axillae, inframammary folds, interdigital areas, intergluteal areas.^{1,2} Foot intertrigo affecting the toe web spaces is a relatively a very common and troubling disorder encountered in tropical climate like in India.³

Obesity, hyperhidrosis, occlusion, poor hygiene, exposure to excessive heat, humidity and moisture and certain immunodeficient conditions like diabetes mellitus, chronic exposure to steroid therapy, malnutrition are some of the predisposing factors for intertrigo. Interdigital toe web intertrigo may be associated with close-toe or tight fitting footwear.^{1,4-6}

Toe web space provides a hospitable niche for subsequent colonization by several microorganisms. An occlusive environment of the web space can exacerbate a mild dermatophyte infection and favor the overgrowth of resident bacteria. Foot intertrigo is initially caused by dermatophytes and with time, a “complex” may develop in the setting of moisture and maceration that favours the growth of multiple fungal and bacterial organisms.^{3,7,8}

Although foot intertrigo may present as a chronic erythematous desquamative eruption, it is often characterized by malodorous maceration and mainly affects the interdigital regions of the feet. In addition to eczematous dermatitis and interdigital tinea pedis, the aetiologies of foot intertrigo are varied and include candidal intertrigo and bacterial intertrigo.^{3,7}

Clinicians generally treat foot intertrigo as fungal infection, overlooking the possibility of added bacterial infection. Therapeutic approaches should not concentrate solely on antifungal therapy, but should concentrate on addressing the predisposing factors, preventive measures along with appropriate topical or systemic antibacterial and antifungal agents.

Very few studies have been conducted internationally to evaluate the etiology of interdigital foot intertrigo and even fewer Indian studies addressing the same in Indian population.

The scarcity of studies in Indian population relating to microbial aetiology and predisposing factors of foot intertrigo, have led us to underestimate the relative frequency and importance of this condition.

Hence, clinical and microbiological studies are required to assist in the selection of appropriate treatment and prevention of important complications of foot intertrigo.

AIMS AND OBJECTIVES

AIMS AND OBJECTIVES

1. To evaluate the clinical features and pre-disposing factors of foot intertrigo.
2. To determine the causative organisms of toe web space infections in foot intertrigo.

REVIEW OF LITERATURE

REVIEW OF LITERATURE

DEFINITION:

Intertrigo, or intertriginous dermatitis, may be defined as superficial inflammatory dermatitis created by friction of opposing skin surfaces in conjunction with moisture trapped in deep skin folds.^{3,9}

HISTORICAL ASPECTS:

The term intertrigo is derived from the Latin language

Inter = between

Terere = to rub.¹⁰

Intertrigo is also known as “chafing”. Reports of superficial yeast infection involving the perineum and upper part of thighs in a negress was made by Engman in early 19th century. The yeasts causing similar infection in axillae, groins, gluteal folds and interdigital spaces of fingers and toes were given the name Intertrigo saccharomycetica. Similar cases were observed in Ceylon, by Castellani, in England by Whitfield, in the Philippine Islands by Phalen and Nichols, and in France by Hudelo where it attacked the intertriginous areas.¹¹

Many therapies have been used for treatment of Intertrigo ranging from herbal pastes to chemical agents. Corbleet in 1929 suggested the use of alcoholic solution of gentian violet for intertrigo. 3% sulphur, Chrysarobin in chloroform and castellani’s paint, zinc

oxide, emol keleet, boric acid ointment have been advocated for therapy of intertrigo earlier.^{11,12}

EPIDEMIOLOGY:

Incidence and prevalence: The true incidence and prevalence of intertrigo in general population is currently unknown. Prevalence of intertrigo in the large skin folds varies from 6% in hospital patients to 17% in nursing home clients and 20% in home care patients.^{9,13}

Internationally, intertrigo is common in tropical weather conditions, hot, humid environment.

Age: Intertrigo affects people of all age groups, more prevalent in the very young and elderly likely due to reduced immunity and immobilization.

Sex: Intertrigo has no sex predilection.

Race: Intertrigo has no racial predilection.

Occupation: Interdigital intertrigo is mainly seen in housewives, maids and others who have constant exposure to water, people working in hot and humid environment and in athletes who have increased sweating and wear occlusive footwear.

Mortality and Morbidity: Intertrigo can progress to a severe bacterial infection with pain, mobility problems, erysipelas, cellulitis, abscess formation, fasciitis, and osteomyelitis.¹⁴

ETIOLOGY AND RISK FACTORS:

Skin folds are prone for intertrigo due to following reasons:

1. These areas are commonly occluded. Occlusion, perspiration and maceration initiate and exacerbate the inflammatory process.¹⁵
2. Skin folds, in particular, groin, axilla and intragluteal regions are major sites of apocrine glands.¹⁶
3. Skin on skin rubbing promotes friction between the surfaces.¹

There are many risk factors associated with intertrigo. Some of them common, and a few other not so common risk factors. Having multiple risk factors, greatly increase the incidence of intertrigo.

Factors that promote friction, moisture and heat in the intertriginous areas are primarily responsible for the intertrigo.

Factors that increase skin friction:

- Obesity
- Clothing that chafes skin
- Activities that promote skin on skin rubbing
- occlusive footwear

Factors that increase moisture:

- Obesity
- Occlusive clothing

- Hyperhidrosis
- Occupational exposure to moisture

Factors that increase heat:

- hyperhidrosis
- occlusive footwear
- Occupational exposure to warm temperatures

Factors which influence immune response or promote fungal growth:

- Diabetes mellitus
- Corticosteroids- topical and systemic
- Immunodeficiency like HIV, Cancer chemotherapy, immunosuppressant medications
- Antibiotics
- Occupational exposure to sugar

1. Obesity: There is a linear increase in the severity of obesity and the presence of intertrigo.¹⁷ Patients who are obese sweat more profusely because of their thick layers of subcutaneous brown fat, generating more heat than persons with normal body mass.¹⁸ This increases thermal, frictional, and moisture components of the skin.¹⁷
2. Diabetes: Important risk factors for development of diabetic foot infections include neuropathy, peripheral vascular disease, and poor glycemic control. In the

setting of sensory neuropathy, there is diminished perception of pain and temperature thus, many patients are slow to recognize the presence of an injury to their feet. Motor neuropathy can lead to foot deformities, which lead to pressure-induced soft tissue damage. Peripheral artery disease can impair blood flow necessary for healing of ulcers and infections. Hyperglycemia impairs neutrophil function and reduces host defenses.¹⁹

3. Hot and humid environment: Sweating and its retention due to lack of ventilation results in creating a favourable environment for growth of fungi and bacteria
4. Hyperhidrosis: Heat, moisture and sweat retention combine to cause maceration and irritation, especially where the skin is thin.¹⁵
5. Strenuous exercises
6. Irritation from sweat, urine, faeces, vaginal discharge.
7. Persons who are bedridden or who wear medical devices that might trap moisture against the skin, such as in artificial limb, splint or brace.
8. Anatomic peculiarities such as deformed foot, spinal deformities.
9. Wearing occlusive footwear predisposes to foot intertrigo.
10. People involved in athletic activities, occupational and recreational exposure to moisture.⁴

PATHOPHYSIOLOGY:

Intertrigo occurs at sites of skin apposition where friction, moisture, perspiration, maceration, heat, and occlusion induce and exacerbate the inflammatory response. Thus intertrigo is an irritant dermatitis caused by frictional trauma.¹

Body folds (flexures) are most common areas of intertrigo due to:

- Relatively high skin temperature.
- Moisture from insensible water loss and sweat cannot evaporate.
- Friction from movement of adjacent skin results in chafing.²⁰

Heat, moisture, and sweat retention combine to cause maceration and irritation. Initially this moisture is derived from unevaporated eccrine sweat, but later the inflammation itself contributes to the moisture by “weeping”.¹⁵ As the stratum corneum becomes macerated because of hyperhydration, the friction intensifies and further damages the epidermis. This moist damaged skin associated with intertrigo is a fertile breeding ground for various microorganisms and forms an entry point for organisms causing secondary infections.^{4,14}

Infective organisms in intertrigo: Secondary skin infection can occur in the presence of intertrigo or may occur independently of any evidence of moisture associated skin damage.⁹

Table 1: INFECTIVE ORGANISMS PLAYING ROLE IN INTERTRIGO.¹

FUNGAL	- Candida (esp. in obesity or diabetes) - Dermatophytes: - Trichophyton mentagrophytes -Trichophyton rubrum -Epidermophyton floccosum
BACTERIAL	- Staphylococcus aureus - Beta hemolytic streptococci - Pseudomonas aeruginosa - E. coli - Proteus Sp. - Enterococcus faecalis - Erythrasma caused by Corynebacterium minutissimum
MIXED	Either 2 different bacteria or bacteria with fungal infection

CLINICAL FEATURES:

Intertrigo is often a chronic disorder that begins insidiously with the onset of pruritus, stinging, and a burning sensation in skin folds.¹⁴

Skin folds that may develop intertrigo are:

- Post-auricular fold
- Angles of lips
- Creases of neck
- Axillae
- Infra mammary (beneath pendulous breasts)
- Abdominal folds
- Umbilicus
- Finger and toe web spaces
- Crural folds
- Perianal/ natal cleft

Intertrigo usually begins as a well margined diffuse erythema confined to areas of skin contacts like axilla, crease of neck, intergluteal areas, genitocrural region, web spaces of the fingers and toes. When the rubbing between the skin continues, maceration and frank erosion of epidermis cause dermatitis. Left untreated, the dermatitis becomes superinfected subsequently. Superinfection and infectious eczematous dermatitis may result in eczematization outside the skin folds.¹

Simple intertrigo: Initially skin folds appear erythematous with peripheral scaling, later develop maceration followed by erosions and fissuring.^{14,15, 21}

Candidal intertrigo: is characterized by typical appearance of skin lesions with satellite vesiculo pustules or papules.^{5,22}

Bacterial intertrigo: Secondary infection with bacteria is associated with more severe inflammation and the lesions appear eczematous and progress to become pustular and crusted.²³

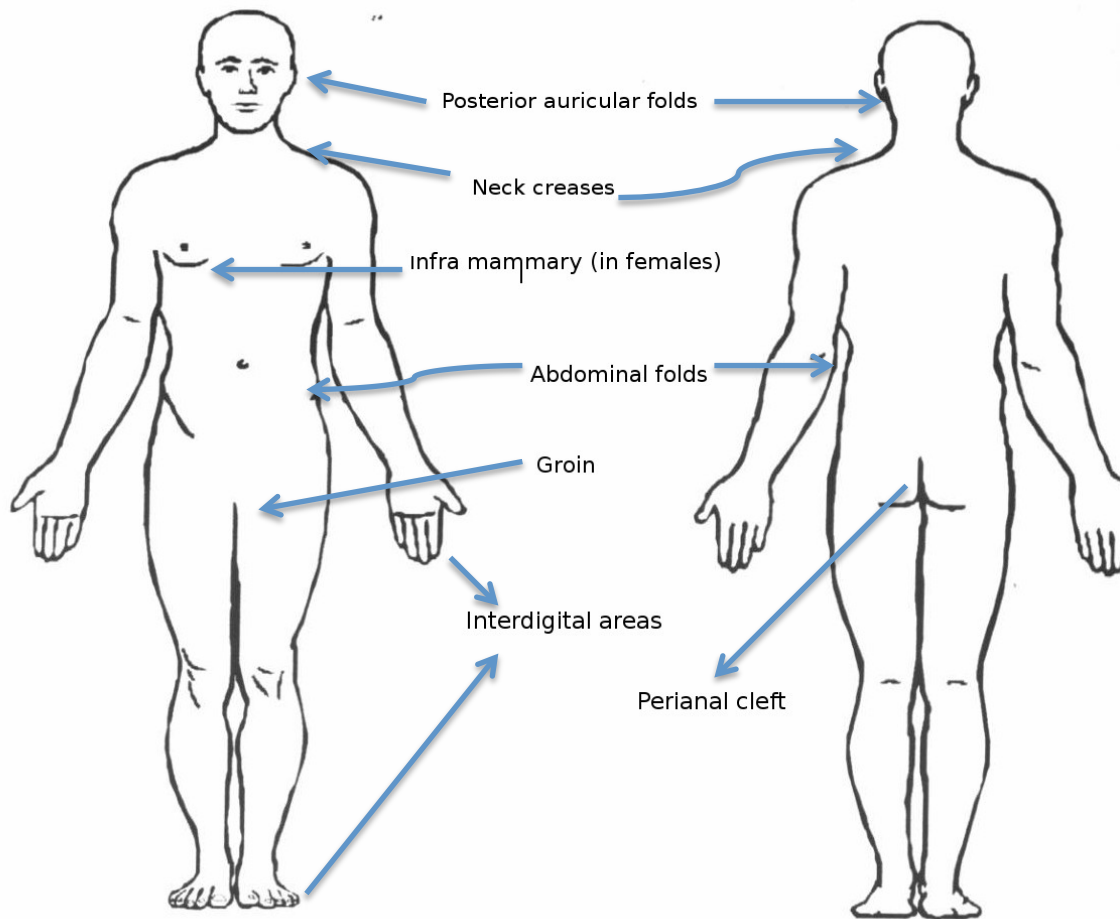


Figure 1: Intertriginous areas

DIFFERENTIAL DIAGNOSIS OF INTERTRIGO:

A) IN INFANTS AND CHILDREN¹

- Diaper dermatitis
- Seborrheic dermatitis
- Psoriasis
- Nutritional dermatitis – zinc deficiency, biotin deficiency, breast feeding in premature infants.
- Candida
- Congenital syphilis
- Granuloma gluteale infantum
- Letterer siwe disease.

B) IN ADULTS ¹⁴

- Infectious conditions:
 - Candidiasis (moniliasis): Superficial erythematous infection, commonly affecting moist, cutaneous areas of the skin; satellite pustules
 - Dermatophytosis: Pruritic infections of nonviable keratinized tissues, such as nails and hair; contains a leading scale
 - Erythrasma: Small, red-brown macules that may coalesce into

larger patches with sharp borders; may be asymptomatic or pruritic; fluoresces coral-red on Wood lamp examination

- Pyoderma: Aggressive infection with boggy, blue-red bullae that progress to deep ulcers with hemorrhagic bases
- Scabies: Infection with intense pruritus and minimal cutaneous manifestations, including intertriginous burrows and papules; the head is spared in all age groups except infants
- Seborrheic dermatitis: Yellow, greasy, scaly plaques with overlying erythema; most often affects the face, postauricular region, and chest

○ Non infectious inflammatory diseases

- Atopic dermatitis: Red or brownish patches with intense pruritus; personal and family history of allergies and asthma is common
- Pemphigus vulgaris: Serious, often fatal, autoimmune disease; flaccid bullae, nikolsky sign
- Psoriasis: Chronic scaling papules and plaques; often affects extensor surfaces of elbows and knees, associated with silvery scale

○ Non inflammatory diseases

- Acanthosis Nigricans: hyperpigmentation with velvety thickened skin, predominantly in neck and body folds
- Hidradenitis suppurativa: Chronic condition of apocrine gland bearing skin in the axillae or anogenital region

- Lichen sclerosus: Well demarcated, hypopigmented, atrophic plaques on genitalia, trunk and axillae
- Neoplasms:
 - Bowen disease: Solitary, enlarging, erythematous, well defined plaque
 - Paget disease: Erythematous plaques with scaling, crusting, exudation. Affects breast, axillae, or anogenital region
 - Superficial basal cell carcinoma: Pink or flesh coloured papule often containing telangiectatic vessel.

FUNGAL INFECTIONS :

(i) **Candidal Intertrigo**: Candida intertrigo is the most common cutaneous candidal infection of hair-bearing skin.²⁴

Etiology and Risk Factors: The warm, moist environment of the skin folds is ideal for the growth of candida. The risk of infection is increased by specific factors that increases skin friction, increase moisture within folds, or interfere with the immune response within folds and/or promote fungal overgrowth.^{1,6}

Candidiasis may affect the following sites:

- Inguinal folds
 - Axillae
 - Scrotum
 - Interluteal folds
 - Inframammary folds
 - Web spaces of the toes and fingers
 - Abdominal folds, particularly beneath a pannus.²⁵
-
1. Web spaces of the hands and feet: This is more common in warm climates and in those with frequently wet hands such as gardeners and housewives. The skin initially appears sodden, white and macerated followed by peeling. This condition is also called “Erosio interdigitalis blastomycetica”.
 2. Under breasts (submammary), axillae, in the groin or between the buttocks: This is characterized by abrupt onset of bright red irritable skin that may peel leaving behind erythematous moist and macerated plaques and erosions with a thin overhanging fringe of epidermis at the margins. There are usually tiny pinpoint “satellite” pustules outside the advancing border. Lesions are often pruritic and painful.
 3. Diaper candidiasis: Napkin dermatitis can get secondarily infected with candida and it is suspected from the involvement of the folds and occurrence of “satellite” pustules scattered along the margins of the larger lesions.²⁵⁻²⁸

4. Perleche: Elderly patients can present with soreness and cracks at lateral angles of the month (angular cheilitis) that is secondarily infected with candida.

It starts as a sore fissure in the depth of the skin fold followed by development of erythema, scaling and crust formation at the sides of the fold.

Intertrigo presents as erythematous, macerated plaques and erosions with delicate peripheral scaling and erythematous satellite papulopustules. The pustules are easily ruptured, leaving an erythematous base with a surrounding collarette of epidermis. The lesions are often pruritic and may be painful.^{25,29}

Laboratory Studies:

1. Direct examination under KOH reveals typical oval budding yeast cells with pseudohyphae.
2. Microscopic examination of skin scrapings prepared with calcofluor white stain is another simple way of detecting yeasts and pseudohyphae of *C.albicans*.
C.albicans binds non-specifically to polysaccharides found in fungal cell walls and produces a distinct bright color in a pattern characteristic for the organism when viewed under a fluorescence microscope.
3. Culture from an intact pustule, skin biopsy tissue or desquamated skin can help support a diagnosis. Growth on Sabouraud's dextrose agar treated with antibiotics yields whitish mucoid colonies after incubation.^{24,25}

Treatment:

1. Treatment is targeted at keeping the skin dry by using antifungal powders.
2. Topical antifungal agents are recommended for twice daily application until the symptoms resolve. Most commonly used agents are nystatin (polyene), miconazole, clotrimazole, ketoconazole (azoles), terbinafine (allylamine)
3. Oral antifungal agents: Patients with extensive or recalcitrant infection may require the addition of oral azoles.
 - Fluconazole (100 mg PO daily for 2-3weeks) or
 - Itraconazole (200 mg PO daily for 1-2 weeks).²⁵

Sundaram et al.³¹ noted that candidal intertrigo can be treated with filter paper soaked in Castellani paint.

(ii) Superficial dermatophyte infections in intertriginous sites.

Dermatophytes include a group of fungi that under most conditions have the ability to infect and survive only on dead keratin. Dermatophytes are responsible for the majority of skin, nail and hair fungal infections and they belong to three asexual genera:

Microsporum, Trichophyton and Epidermophyton.

Clinical Classification: Clinically dermatophyte infection are classified by body region affected.

Intertriginous areas infected by dermatophytes are:

- Groin and thigh region- Tinea Cruris
- Interdigital areas of fingers and toes- Tinea manum and tinea pedis
- Inframammary areas- Tinea corporis

Intertriginous infection with dermatophytes may be caused by *T. rubrum*, *T. mentagrophytes*, or *Epidermophyton floccosum*.

Tinea cruris, more frequently seen in adult males, is characterized by itchy, red, scaly, plaques on the upper medial thighs. Lesions tend to grow with a circular border, with central clearing. The macerated keratin compromises the cutaneous barrier and acts as a portal of entry for secondary bacterial infection leading to lymphangitis and cellulitis.

Tinea pedis: refers to dermatophytic infection of the feet or toes. Involvement often starts asymmetrically and then spreads to the other foot and, in susceptible individuals, to the hands. A secondary bacterial infection, often from the toe webs in a person with diabetes, can be life or limb threatening.^{29,31}

There are primarily 3 types of tinea pedis:

1. Interdigital type
2. Moccasin type
3. Vesiculobullous type

Interdigital tinea pedis is the most common form and usually manifests in the interspace of the fourth and fifth digits and may spread to the underside of the toes.^{32,33}

Patients often complain of itching and burning sensations on the feet accompanied by

malodor. Dermatophytosis complex, as it is called, may have fissuring of the interspace along with hyperkeratosis, leukokeratosis, or erosions.³⁴

The moccasin type is a more severe, prolonged form of tinea pedis that covers the plantar surface and lateral aspects of the foot. Its appearance is that of a slipper or moccasin covering the foot, hence the name. *T. rubrum* is most commonly associated with moccasin type tinea pedis. The skin of the inflamed area in this type of infection is often scaly and hyperkeratotic with erythema around the soles and sides of the foot.^{32,33} Papules may also be noted around the demarcation line of erythema that surrounds the foot.

Vesiculo bullous tinea pedis type comprises of pustules or vesicles on the instep and adjacent plantar surfaces of the feet and is less common.³³ Fluid filled vesicles are usually clear but pus usually indicates secondary bacterial infection, most often with *Staphylococcus aureus* or group A *Streptococcus*. This form of tinea pedis may be associated with dermatophytid or "ID" reaction.³⁵

Diagnosis:

KOH wet mount: Direct visualization under microscope shows branching hyphae in keratinized material.

Culture: Dermatophytes are aerobic and grow on surface of media. Three types of culture media used most often for isolation are dermatophyte test medium, mycosel agar, and Sabouraud's dextrose agar.

Treatment:

Topical: Topical terbinafine 1% cream or topical azoles can be applied twice daily for 2 weeks

Systemic:

Fluconazole 150mg per week for 4 to 6 weeks

Terbinafine 250mg per day for 2 weeks

Itraconazole 100mg per day for 2-4 weeks.^{29, 36}

The diagnosis can be confirmed by examining fungal scrapings of the skin surface keratin for the presence of septate hyphae in potassium hydroxide preparations. A positive culture on Sabouraud's agar can identify the specific organism, but culture can take up to a month. About 20% of fungal infections are negative on a potassium hydroxide test and on culture. Dermatophyte infection generally responds well to topical antifungal creams.³¹

2) BACTERIAL INFECTIONS AND INTERTRIGO

The moist, damaged skin associated with intertrigo is a fertile breeding ground for various microorganisms and secondary bacterial infections are commonly observed in these areas. Bacterial proliferation may be associated with keratinocyte necrosis.

Bacterial Infections can primarily be divided into

- Gram Positive infections- e.g., Staphylococcus, Streptococcus
- Gram Negative infections- e.g., Pseudomonas aeruginosa, Acinetobacter

- Mixed bacterial infections

Staphylococcus aureus may present alone or with group-A beta- hemolytic streptococcus (GABHS). Pseudomonas aeruginosa, Proteus mirabilis or Proteus vulgaris may also occur alone or simultaneously.⁴

Toe-web infections can be caused or worsened by gram positive and gram negative bacteria. Gram negative toe web infections are often caused by P.aeruginosa combined with other gram negative bacteria such as Moraxella, Alcaligenes, Acinetobacter, and Erwinia. Gram positive infections are usually caused by S.aureus and GABHS and occasionally by Staphylococcus saprophyticus or other coagulase negative Staphylococci.^{7,37}

Gram-negative mixed bacterial infection with organisms such as Moraxella, Acinetobacter, Pseudomonas, Proteus, and Erwinia species, may represent a mild secondary infection of tinea pedis. Over time, in the setting of moisture and maceration, multiple fungal and bacterial organisms may proliferate. The process may progress to advanced stages of gram-negative infection with sepsis.⁸

Streptococcal intertrigo presents as a well demarcated red, weeping intertrigo on neck folds, axilla or inguinal regions. It is characterized by foul odour and absence of satellite lesions.³⁸

Erythrasma: Erythrasma may complicate intertrigo of interweb areas, intergluteal and crural folds, axillae, or inframammary regions. Erythrasma is a bacterial infection caused by Corynebacterium minutissimum. It presents as small, red-brown macules that may

coalesce into larger patches with sharp borders. These lesions are often asymptomatic but may be pruritic in some instances.

Diagnosis:

1. Culture and sensitivity should be performed.
2. Wood's light examination identifies a *Pseudomonas* or *Erythrasma* infection more quickly than a culture. The Wood's light characteristically shows a green fluorescence with *Pseudomonas* infection and a coral-red fluorescence with *Erythrasma*.

Therapeutic modalities for intertrigo with secondary bacterial infection:

1. For patients with GABHS, an independent or concomitant regimen of topical therapies (e.g., mupirocin, erythromycin) is the best therapy. Oral antibiotics (e.g., penicillin, first generation cephalosporins) and low-potency topical steroids (e.g., hydrocortisone 1% cream) are useful. The latter may be particularly useful if the intertrigo is associated with seborrhoeic or atopic dermatitis.
2. Cutaneous erythrasma is best managed with oral erythromycin (250 mg four times daily for two weeks). Topical clindamycin, Whitfield's ointment, sodium fusidate ointment and antibiotic soaps also may be beneficial.
3. Toe-web infections can be serious, and severe cases may warrant hospitalization. Tissue removal may be needed to allow absorption of topical antibiotic agents, which promote healing and slow the spread of infection. Antibiotic sensitivity should determine what topical and systemic therapies are used. Third generation

cephalosporins and quinolones are active, together with aminoglycosides.

Ciprofloxacin 500 mg twice daily for 10 days is another option, but some patients need parenteral therapy instead (e.g., 1 to 3g daily of intramuscular ceftazidime/ 2g daily of cefotaxime for 10 days).

3) OTHER COMMON DIFFERENTIAL DIAGNOSIS:

Psoriasis:

Inverse psoriasis or the intertriginous form of psoriasis is symmetrically distributed and bright red in colour with a sharp margin. It is distinguished from other forms of psoriasis by the absence of a silvery scale even in untreated cases. Intertriginous psoriasis is most common in the groin, under the breasts, in the axillae and in the perianal area, but it can occur in other locations. There is usually an absence of satellite papules or pustules. Involvement of other areas may help to establish the diagnosis.

Seborrhoeic dermatitis of the flexural areas:

Seborrhoea of the flexural areas is common in otherwise healthy, young infants. It presents as yellow-pink erythema, sometimes with a peripheral greasy scale. As infants become older, it gradually improves. This condition is rare in older children or adults except in association with immunosuppression or immunodeficiency.

Contact dermatitis of the flexural regions:

80% of contact dermatitis is due to irritants and 20% is allergic in nature. Irritant contact dermatitis is often diffuse, whereas many contact allergies produce bright red

erythema with discrete margins. Irritant contact dermatitis is common, due to irritants in soaps, detergents, fabric softener residue in clothes, deodorants, antiperspirants and antimicrobial preparations.

Common contact allergens in the flexural areas include perfumes; preservatives such as formaldehyde and formaldehyde releasers; topical antimicrobials, such as neomycin, bacitracin, polymyxin and others; and occasionally topical steroids. The allergic reaction can be reproduced by the repeat open application test. Products can be screened by applying them twice a day for two to three days in a coin-shaped circle on normal forearm skin. Allergic reactions to irritants or sensitizing agents can be confirmed by patch testing.

Atopic dermatitis of the flexural areas:

Atopic individuals often have a decreased ability to sweat, altered immunity and susceptibility to eczema in the body folds. Atopic flexural eczema is most common in the antecubital and popliteal fossae, usually starts once individuals can walk with an upright posture, and is less common as they reach adulthood.²¹ Itch often leads to scratching and rubbing the involved areas, which can produce increased skin surface markings (lichen simplex chronicus).⁹

Pemphigus Vegetans:

A variant of pemphigus vulgaris seen usually in middle aged adults. The disease presents as flaccid bullae that rupture leaving either changes of erosions, vegetation or papillomatous proliferation characteristically seen in the body folds. Its clinical course

begins insidiously initially in the nose or mouth and it has been noted to affect the scalp as well later progressing to involve the body fold.¹ Lesions are primarily flexural although vegetations may occur at any site. There are 2 subtypes of pemphigus vegetans, the severe form also known as Neumann, and benign form Hallopeau type. Bullae and vesicles are predominant in the former and pustules are predominant in the latter.³⁹

Benign familial chronic pemphigus (hailey-hailey disease):

It is an autosomal dominant disorder that is characteristically distributed in the intertriginous areas as recurrent chronic bullous and vesicular dermatitis that usually presents during late teens to twenties. It occurs due to defect in the calcium pump protein ATP2C1. It presents as vesicle and erythematous plaques with overlying crusts in the genital as well as chest, neck and axillary areas. Heat, friction and infection exacerbate the symptoms. Biopsy usually distinguishes hailey-hailey from impetigo, pemphigus vulgaris or vegetans or Darrier's disease.^{1,40}

MICROBIAL ECOLOGY OF TOE WEB SPACES:

Normal skin is colonized by large number of harmless microorganisms which lives as commensals. Those organisms found regularly on skin constitute its normal flora. Normal flora has been classified into the following groups:

1. Resident flora – Resident flora are the bacteria that grow on skin and are relatively stable in number and composition at particular sites.
2. Transient flora- Transient flora are organisms that lie free on the skin surface without any attachments and are derived from exogenous sources. They are unable to multiply on the skin and vary widely in both type and number. They disappear from skin within a short time.

TABLE 2: NORMAL SKIN FLORA.⁴¹

BACTERIA			
<u>Aerobic</u>	Gram Positive	Cocci	-Staphylococci (e.g., <i>S.epidermidis</i> , <i>S.saprophyticus</i> , <i>S.hominis</i>) -Micrococci
		Rods	-Corynebacteria -Brevibacterium epidermidis -Propionibacterium (some species)
	Gram Negative		Acinetobacter (<i>Mima</i> , <i>Herellea</i>)
<u>Anaerobic bacteria:</u>			Propionibacteria (<i>P.acnes</i> , <i>P.granulosum</i> , <i>P.avidum</i>) <i>Peptococcus saccharolyticus</i> (anaerobic staphylococci) ⁴¹
FUNGII			
			<i>Candida</i> species <i>Pityriasporm</i>

GRAM POSITIVE BACTERIA:Staphylococci and Micrococci:

Coagulase positive staphylococci like *S.aureus* are pathogenic and are only transient residents in anterior nares and perineum. Humans have a high degree of resistance to their colonization. Coagulase negative staphylococci are most frequently found organisms of the normal flora.

Micrococci are much less frequently present than Staphylococci.^{41,42}

Coryneform bacteria:

These are gram positive rods, sometimes called diphtheroids. These are more common in intertriginous areas, especially toe web spaces with hyperhidrosis.⁴³

Brevibacterium:

They are penicillin resistant rapidly growing coryneforms. They are frequently isolated from toe web spaces, especially in patients with tinea pedis and have been implicated in foot odour.⁴²

Propionibacteria:

They are non spore forming, anaerobic gram positive bacteria and are normal inhabitants of hair follicles and sebaceous glands.⁴⁴

GRAM NEGATIVE BACILLI:

These are not part of the resident flora as they die quickly on dessication. They are only transient flora in the perineum and perianal region.⁴¹ E.Coli, Klebsiella, Enterobacter are the predominant Gram negative rods. These organisms are known to cause systemic infections. Extended spectrum Beta lactamase are the resistant strains known to cause gram negative septicemia.⁴⁵

FUNGI:

Candida:

Normally found in 40% of oral mucosa. *C.albicans* is the most common species found in blastospore form. Increased colonization of the skin is seen in diabetics, psoriasis, atopic dermatitis and in patients who are immunosuppressed.

Dermatophytes:

Dermatophytes are a group of taxonomically related fungi. Their ability to form molecular attachments to keratin and use it as a source of nutrients allows them to colonize keratinized tissues, including hair and nails.³⁶

Many external factors can alter the ecosystem of the skin, with resulting changes in microbial populations. These factors can alter or suppress the normal flora and instead promote colonization of other pathogenic organisms.

Environmental factors include temperature, humidity, salinity, and light exposure. Host factors include age, sex, immune status, hospitalization status, hygiene, use of medications (antibiotics, steroids), use of soaps and cosmetics, and presence of trauma.⁴⁶

Effect of climate on skin flora:

Increased temperature and humidity increase the density of bacterial colonizations and alter the flora.⁴⁷ Increase in heat and humidity also favours fungal growth. Cutaneous candidiasis, tinea versicolor and *Trichohyton rubrum* can be reproduced experimentally

when skin is occluded. The raised carbon dioxide produced by occlusion may favor conversion of yeasts and dermatophytes to more infectious stages.⁴²

Effect of body location:

Composition of the normal flora varies with body location. Face, neck and extremities are exposed areas and may have increased proportion of transient organisms and higher bacterial density. Head and upper trunk have more sebaceous glands and hence greater number of lipophilic organisms like propionibacterium. Axilla, perineum and toe web spaces have partial or complete occlusion with increased moisture and temperature. These areas are colonized with all organisms, particularly gram negative rods or coryneform organisms which need moisture for growth.⁴²

Effect of co-morbidities:

Systemic illness may have direct relation with the prevalence of infection and kind of organism that constitute the normal flora. Systemic illness may predispose to colonization or infection. In diabetes mellitus, skin glucose concentration is high and this factor may affect the colonization. Increased nasal carriage of *S.aureus* occurs in diabetic children compared to non-diabetics. Candidal infection is more prevalent in diabetics, more so in females.

Effect of Age:

Microflora can vary widely with age. In young children, micrococci, coryneform bacteria and gram-negative organisms are found more frequently compared to older children and

adults. *Pityrosporum* and propionic bacterium species are present at much lower levels before puberty. This may be attributed to the organisms requiring higher lipid levels for growth and multiplication, which is seen in puberty due to enhanced sebum production in puberty. Elderly patients have a decrease in sebum production and infection with these organisms is rare.⁴²

Effect of Sex:

Men carry higher absolute number of organism and more biotypes. This may be attributed to the higher rate of sweating in men as well as tendency to wear more occlusive clothing. Increased sebum production and hormonal differences may also contribute.

Effect of occupation:

Gram negative organisms, candida and coryneforms are microflora which have preference to hot and humid environment, hence occupations exposing individual to hot and humid environment may lead to infection with these organisms. Hospital workers are known to harbor more pathogenic organisms and more resistant strains.

Effect of soaps and disinfectants:

Repeated washing with soap makes skin more alkaline compared to washing with disinfectants. *Propionibacteria* are markedly increased when soap is used and reduced in number with use of medical disinfectants. Antibiotics suppress the normal flora and increase colonization by other organisms. They also impair bacterial adherence to epithelial cells and allow the natural selection of organisms like gram-negative rods,

candida or pityrosporum. Oral steroids and hormones are associated with change in the normal flora. Steroids suppress immune system and increase susceptibility to bacterial, fungal, viral and parasitic infections. Frequent use of topical antibiotics may induce resistant strains to appear.⁴²

FOOT INTERTRIGO (TOE WEB INTERTRIGO)

Foot intertrigo affecting the interdigital toe web spaces occurs due to a combination of factors such as increase in moisture content, temperature, friction and maceration in conjunction with overgrowth of yeasts, dermatophytes, and bacteria.

Some of the common predisposing factors of foot intertrigo are: ^{4,37,42,48}

- Occlusive footwear
- Hyperhidrosis
- Poor hygiene
- Excessive exposure to water and moisture.
- Athletic and sporting activities
- Repeated foot washing
- Use of communal baths
- Host factors such as diabetes mellitus, obesity and immunosuppression.

Intertrigo is very common in Toe web spaces because:

- Air circulation is limited
- Semi occlusive environment of the toe web spaces retains sweat and increases moisture content
- Friction between the toes is very common
- Hyperhidrosis is more predominant in the palms and feet

AETIOLOGY:

Any body site such as the toe web space, that is habitually colonized by polymicrobial flora creating a precariously balanced cutaneous ecosystem caused by mechanisms of biologic interference typically has a variable aetiology.^{49,50}

Interdigital foot intertrigo is commonly infected by dermatophytes, but it can also be infected with bacteria, yeasts or non-dermatophyte moulds.⁹

Dermatophytes that commonly complicate interdigital intertrigo include *Trichophyton rubrum*, *Trichophyton mentagrophytes*, *Epidermophyton floccosum*. Gram-positive and gram-negative bacteria can also complicate interdigital intertrigo. Gram-negative toe web infections are often caused by *Pseudomonas aeruginosa*. This may occur as an isolated pathogen, or as a part of polymicrobial infection with other gram negative bacteria such as *Escherichia coli*, *Proteus*, *Moraxella*, *Alcaligenes*, *Acinetobacter*, and *Erwinia* species. Gram-positive infections are usually caused by *Staphylococcus aureus* and Group A Beta Hemolytic *Streptococcus* and occasionally are caused by *Staphylococcus saprophyticus* or other coagulase-negative staphylococci.⁴ However, gram-negative and gram-positive infections occasionally occur simultaneously.^{4,7,37}

Yeasts such as *Candida albicans* also commonly cause interdigital toe web infections.⁵¹

PATHOPHYSIOLOGY:

Interdigital toe web infections have been traditionally regarded as strictly a fungal infection. However, this assumption has been revised, because often fungi cannot be isolated from the lesions throughout the disease course.⁵²

The inability to culture dermatophytes from macerated toe web spaces is now understood in terms of the dynamic interplay between fungi and bacteria.⁴²

Interdigital toe web infections often involve an ecologic interplay in which an initial dermatophyte infection provides a hospitable niche for subsequent colonization by bacteria. Dermatophytes which are usually the first invaders incite the damage to the stratum corneum, that allows bacterial overgrowth, which promotes maceration and hyperkeratosis. These dermatophytes produce natural substances with antibiotic activities that can affect the composition of the residing bacterial flora and determine antibiotic-resistant strains. The prevalence of particularly *Brevibacterium epidermidis*, *Micrococcus sedentarius*, *Corynebacterium minutissimum*, *Staphylococcus aureus* and various gram-negative species increases and the presence of these pathogenic and more virulent bacteria in presence of a damaged stratum corneum is responsible for clinically aggravating the lesions.⁵³⁻⁵⁶

A probable mechanism by which dermatophyte positive interspaces can be converted into macerated interspaces from which fungi can't be recovered involves the production of a variety of potent antifungal sulfur compounds such as methanethiol, ethanethiol, dimethyl sulfide and others produced by *M. sedentarius* and *B. epidermidis*.⁴²

In other cases, gram-negative intertrigo appears independently and is favored by pre-existing local conditions such as marked hyperhidrosis and cutaneous maceration, often caused by closed shoes or the practice of wet sports, especially in hot water.^{7,53,57}

Pseudomonas aeruginosa, mostly in association with other gram-negative bacteria, is the main causative agent of these clinical forms, which are characterized by an acute

worsening trend and create therapeutic problems related to the appearance of early antibiotic resistance.^{58,59}

CLINICAL MANIFESTATIONS OF FOOT INTERTRIGO:

The term foot intertrigo consists of different clinical manifestations; these may present with a chronic, more or less asymptomatic, erythematous desquamative type of lesions or they may include an acute form with an exudative, macerating, painful inflammatory process that may lead to discrete functional impairment. The etiology is variable and a differential diagnosis based on a clinical features alone is difficult because the initial phase of the infection is often common to interdigital tinea pedis, candidal intertrigo, and bacterial intertrigo.^{7,8} Interdigital toe web infection is usually associated with burning sensation but it may be mild and asymptomatic³ or it may be painful.¹⁴

During the initial stage of ‘dermatophytosis simplex’, there may be only an asymptomatic scaling. The occlusive environment of the toe web space can exacerbate this mild dermatophyte infection resulting in malodorous maceration and inflammation followed by a more severe, symptomatic ‘dermatophyte complex’ which occurs due to overgrowth of opportunistic bacteria at the cost of the dermatophytes.^{53, 54,60}

Clinical manifestations are similar in most of the patients and clinical features can include erythema, vesicopustules, erosions, purulent and profuse discharge, marked maceration and ulceration.³⁷ In severe intertrigo, patients may have a purulent discharge with edema and intense erythema of tissues surrounding the infected area.^{4,14} Green discoloration at the infection site is seen in patients with gram negative bacterial infections.

Interdigital intertrigo can progress to a severe bacterial infection with pain, mobility problems, erysipelas, cellulitis, abscess formation, fasciitis, and osteomyelitis causing pain that is so severe that the patient is unable to ambulate.

Patients with severe toe web intertrigo, who are overweight or who have diabetes are at a higher risk for cellulitis.⁴

DIFFERENTIAL DIAGNOSIS OF INTERDIGITAL FOOT INTERTRIGO:

1. INTERDIGITAL TINEA PEDIS: This is the most common type of tinea pedis which is predominantly caused by dermatophytes like *Trichophyton rubrum*, *Trichophyton mentagrophytes* and *Epidermophyton floccosum*. It usually begins as dry scaling of the interdigital and subdigital skin of the feet and later develops interdigital maceration, and fissures. It commonly affects the third and fourth toe web spaces but it may involve all the toe web spaces. Sometimes it spreads to involve the undersurface of the toes, sole and rarely the dorsum. It is most often associated with itching and malodour.^{26,36}
2. CANDIDAL INTERDIGITAL INTERTRIGO: Interdigital candidal infection of the hands and feet is called as *Erosio interdigitalis blastomycetica*.⁵ This form of candidiasis is typically seen as oval shaped area of white maceration in the third web space between the middle and ring fingers. Denudation of the maceration reveals a moist, pink erythematous area surrounded by a collar of overhanging white epidermis. Lesions in the toe web spaces are similar but the white

maceration is very thick, adherent and does not peel off freely and it most commonly involves the fourth toe web space.^{28,29}

3. BACTERIAL INTERTRIGO: This is usually much more macerated, malodorous, and exudative, and painful than T.pedis. It is caused by either gram positive or gram negative organisms or most often by mixed infections. Gram-negative toe web intertrigo, often caused by *Pseudomonas aeruginosa*, is more frequent and it is characterized by vesiculopustules, maceration, malodorous discharge and marked edema and erythema of the surrounding tissues. It may be associated with cellulitis and inability to walk.^{2,7,56}

Erythrasma is another bacterial infection that can affect the major skin folds and the interdigital spaces of the feet. It is caused by lipophilic, diphtheroid, filamentous, gram-positive bacillus *Corynebacterium minutissimum* and is characterized by erythematous, brown, scaly patches and maceration, and exhibits coral-red fluorescence under Woods lamp examination. These lesions are often asymptomatic but may be pruritic in some instances.^{4,61}

4. CUMULATIVE IRRITANT CONTACT DERMATITIS(CICD): This type of dermatitis develops as a result of a series of repeated and damaging insults to the skin. These insults may include both chemical irritants and a variety of harmful physical factors, such as friction, microtrauma, low humidity, the desiccant effects of powder, soil or water and temperature. Cumulative irritant dermatitis most

commonly affects the dorsa of the hands, fingertips and the webs of the fingers and toes.⁶²

5. SCABIES: It is an infestation of the epidermis with the mite, *Sarcoptes scabiei* var. *hominis* involving interdigital web spaces and other intertriginous sites. It is typically characterized by intense pruritis which is worse at night. The pathognomonic lesions of scabies are burrows, which appear as slightly raised, brownish, tortuous lesions characteristically seen over the flexor aspect of the wrist, finger web spaces, instep of the feet and genitalia. However, pruritic, inflammatory papules and papulo-vesicles constitute the more frequent and obvious findings. These lesions are predominantly seen over the interdigital spaces, axillae, flexor aspect of wrists, elbows and knees, periumbilical areas and also genital areas.^{63,64}

6. CORNS (clavi or helomata) OR CALLUSES (tylomata) are, respectively keratotic papules and plaques that occur in areas that are subject to sustained, excessive mechanical shear or friction forces. Crookedness of the lateral toes leads to prominence of the proximal and/or distal interphalangeal (IP) joints. This results in formation of corns or calluses either in between the toes or dorsal to the IP joints or on the lateral aspect of the fifth toe. The interdigital corn can be hard if they are adjacent to the IP joints or soft when present deep within the fourth interdigital space. Soft corns or calluses, sometimes with sinus formation, are common in the lateral toe web spaces, especially in women.^{26,65}

7. PSORIASIS: Psoriasis involving the intertriginous areas is most common in the groin, sub mammary areas, axillae and perianal area but it can occur in other areas.⁹ However psoriasis of the interdigital spaces has not been described frequently in the literature. The term “white psoriasis” or “psoriasis alba”, was first introduced by Waisman in 1961 to describe interdigital psoriasis. This is a distinct but atypical form of psoriasis that is often missed as it is commonly mistaken for interdigital fungal infection. If recalcitrant whitish, sodden patches are present, especially in patients with other psoriatic clinical features as well, if mycological cultures are consistently negative, and if there is no response to antimycotic treatment, interdigital psoriasis should always be considered.^{66, 67}

LABORATORY TESTS:

- Gram stain, bacterial culture and sensitivity testing, potassium hydroxide preparation, and fungal culture should be performed to determine the etiologic agents.
- Wood lamp Examination: It characteristically shows a green fluorescence with Pseudomonas infection and a coral-red fluorescence with erythrasma.
- Skin biopsy is usually not required because the histology of intertrigo shows no characteristic features.⁴
- In addition, complete blood cell counts with differential and fasting blood glucose levels may be used to screen the patient's immune status and to exclude diabetes mellitus.



FIGURE 2: Severe Gram-negative toe web infection showing denudation, discharge and greenish discolouration.⁶⁰



FIGURE 3: Erosio interdigitalis blastomycetica.⁵



FIGURE 4: Soft corn located deep within the 4th interdigital space.⁶⁵



FIGURE 5: Tinea pedis- interdigital type showing maceration, scales and erosion. ³⁶

TREATMENT:

There is relatively little evidence from controlled trials addressing the best therapy and strategies for prevention of intertrigo. However, the following basic treatment approach to intertrigo is generally successful.^{4,9,25}

PRINCIPLES FOR MANAGEMENT OF INTERTRIGO:

1. Address the predisposing and precipitating conditions
2. Anti infective therapy
3. Anti inflammatory therapy
4. Patient education
5. Preventive strategies for recurrences^{2,7,25}

1. CORRECTING THE PREDISPOSING AND AGGRAVATING FACTORS:

An important component to successful treatment is the correcting of predisposing factors and underlying conditions, such as diabetes mellitus, that may contribute to infection. When obesity is a contributing factor, weight loss should be encouraged. Occlusive footwear should be avoided and exposure to moist environment should be limited. If foot deformities are noted, then corrective surgeries for correction of foot deformities should be considered.

The main principle of therapy for simple or uncomplicated intertrigo is to eliminate the heat, friction and maceration by keeping the skin folds clean, dry, and cool.^{4,14,21}

BARRIER AGENTS: Topical barrier creams protect the skin from contact irritants and they include zinc oxide, petrolatum, liquid acrylates and silicone or dimethicone based creams.^{9,21}

DRYING AGENTS: Various topical drying agents have been employed to keep the skin folds dry, clean and cool. These include aluminum sulfate calcium acetate solution, antiseptic drying agents such as potassium permanganate solution, talcum powder, short chain fatty acid powders like undecyclic acid and antifungal powders.

Drying agents are typically used after an adequate course of antifungal therapy has been completed to help maintain a dry intertriginous environment and minimize the risk of maceration and reinfection. Patients at high risk for recurrence of intertrigo should use a drying powder indefinitely.

Drying agents cannot be applied simultaneously with antifungal creams or ointments as this will result in a sticky paste. If used together, the preparations should be applied several hours apart.^{14,25}

2. ANTI-INFECTIVE THERAPY - Intertrigo is often complicated by fungal or bacterial infections which should be promptly treated with appropriate antifungal or antibacterial medications based on the laboratory findings.

Topical antifungal agents used in treatment of intertrigo include:

- Polyenes like Nystatin- effective only against candida. ⁴
- Azoles like clotrimazole, ketoconazole, miconazole, econazole, and oxiconazole – are broad spectrum antifungals that are effective against both candida species and

dermatophytes.

- Allylamines- like terbinafine are effective against dermatophytes and a number of yeasts and moulds.⁴²

Oral antifungals: Systemic antifungal medications are rarely necessary for the treatment of intertrigo and treatment with an oral agent should generally only be used only in case of recalcitrant and severe infection.

Oral antifungals typically used include:

Fluconazole 50 to 100 mg daily for 2 to 3 weeks or 150 mg weekly for 3 to 4 weeks

Itraconazole 200 mg daily for 1 to 2 weeks

Ketoconazole 200 mg daily for 1 to 2 weeks

Terbinafine 250 mg twice daily for 2 weeks.^{4,25}

Antibacterial agents: Ideally antibiotic sensitivities should determine what topical and systemic therapies should be used. Tissue removal may be needed to allow absorption of topical antibiotic agents, which promote healing and slow the spread of infection.⁴

Topical antibiotic agents include:

Mupirocin - Mupirocin is highly effective against aerobic gram-positive cocci, especially *S.aureus*, for which it is bactericidal at the concentrations present in the commonly used 2% ointment. It is not effective against enterococci and generally has poor activity against gram-negative bacteria.⁶⁸

Topical aminoglycosides like clindamycin gel, amikacin gel, gentamicin cream are highly active against gram-negative organisms such as *Pseudomonas* and some gram-positive bacteria, including some staphylococcal strains.

Topical erythromycin – is useful in treatment of erythrasma.

Systemic antibiotics: Topical antibiotics are often inadequate in treating some cases of bacterial intertrigo. Systemic antibiotics should be considered in such patients if topical treatment fails or there is extensive disease.³ Some of the antibiotics found effective in treatment of bacterial intertrigo are:

Third generation cephalosporins

Quinolones- Ciprofloxacin

Aminoglycosides – gentamycin

Oral antibiotics combined with cleansing and debridement is often effective. But some patients may need parenteral therapy instead (e.g., 1 to 3 g daily of intramuscular ceftazidime , 2 g daily of cefotaxime for 10 days).^{3,4,37}

Miscellaneous agents:

Castellani paint: Castellani paint acquired its name from an Italian physician, Sir Aldo Castellani (1877-1971). It is also called carbol fuchsin paint. It is an antiseptic and a drying agent which has fungicidal and bactericidal properties along with an anesthetic effect on the skin.

The paint is a mixture of resorcinol (8 g), acetone (4 mL), magenta (0.4 g), phenol (4.0 g), boric acid (0.8 g), industrial methylated spirit 90% (8.5 mL), and water (100 mL).^{37,42}

Whitfield's ointment: It contains 12% benzoic acid and 6% salicylic acid and has non specific antifungal properties. It acts as a keratolytic agent causing desquamation of keratinized epidermis containing fungal organisms.⁴²

Surgical Care:

Occasionally, if the infection is advanced, superficial debridement may allow creams, ointments, or other antibiotic agents to reach infected areas faster, promoting healing and stopping the spread of the infection into surrounding areas.³⁷

3. ANTI INFLAMMATORY THERAPY

In case of simple uninfected intertrigo, mild topical corticosteroids may be added to reduce the inflammation.²

Topical corticosteroids – Although low potency topical glucocorticoids may be used in conjunction with antifungal therapy to treat associated pruritus, pain, and burning, these symptoms generally respond quickly to antifungal therapy alone. Thus the use of topical glucocorticoids is usually not necessary. Moderate and high potency glucocorticoid preparations should be avoided.

Topical calcineurin inhibitors: Topical tacrolimus or pimecrolimus may also be used in treatment of patients with intertrigo due to noninfectious inflammatory etiologies. It might also be expected to provide benefit in those patients with candida intertrigo in whom a topical antiinflammatory agent is indicated but who are unable to use topical glucocorticoids.^{2,25}

4. PATIENT EDUCATION:

Patients should be educated about the predisposing factors, and the chronic nature of disease.

Also, measures that are aimed at eliminating the moisture that provides the environment for infection and its recurrence should be explained to the patients. Patients should also be instructed about proper hygiene which is a primary preventive measure. Instructions about wearing open-toed shoes and avoiding skin maceration are essential. ^{37,42, 48}

5) PREVENTION OF RECCURENCES

- Keeping the area affected by intertrigo clean, dry and exposed to air can help prevent recurrences.
- Weight loss should be encouraged if obesity is a predisposing factor.
- A structured skin care routine based on gentle cleansing, moisturizing (preferably with an emollient) and application of a skin barrier protectant may help.
- Patients who engage in water-related sports should shower afterwards and ensure that interdigital spaces are thoroughly dried to prevent creating an environment for bacterial growth.
- Wearing open-toed shoes may help prevent toe web intertrigo
- The use of gauze pledgets between the toes helps prevent occlusion, and the use of astringent soaps reduces the number of gram-negative bacteria. ^{4,9,14,37}

TABLE 3: INTERTRIGO AND TREATMENT

CONDITION	TREATMENTS
Intertrigo	Topical: zinc oxide ointment, petrolatum, talcum powder, aluminum sulfate, calcium acetate solution
Intertrigo complicated by secondary bacterial infections	
Erythrasma	Topical: erythromycin, clindamycin, Whitfield ointment, chlorhexidine Oral: erythromycin
Group A beta- hemolytic streptococcus	Topical: mupirocin (Bactroban), erythromycin, low-potency steroids Oral: penicillin, cephalexin (Keflex), ceftriaxone (Rocephin), cefazolin, clindamycin
Intertrigo complicated by secondary fungal infections	
Candida	Topical: nystatin, clotrimazole, ketoconazole, oxiconazole (Oxistat), econazole Oral: fluconazole (Diflucan; used for resistant cases)
Dermatophytes	Topical: clotrimazole, ketoconazole, oxiconazole, econazole

MATERIALS AND METHODS

MATERIALS AND METHODS

The present study was a hospital based cross sectional study, carried out at Sri R.L.Jalappa Hospital and Research centre attached to Sri Devaraj Urs Medical College, Tamaka, Kolar, from January 2012 to June 2014 over a period of 18 months. All patients reporting to the Department of Dermatology, Venereology and Leprosy, at the above mentioned hospital were evaluated for inclusion into the study and patients having clinically diagnosed foot intertrigo were enrolled into the study after obtaining an informed consent.

CRITERIA FOR SELECTION:

Inclusion criteria

- ❖ All patients with clinically diagnosed foot intertrigo.

Exclusion criteria

- ❖ Patients who had taken systemic antibiotics or antifungal medications within last 1 month.
- ❖ Patients who had used topical antibiotics or antifungals within last 15 days.

A total of 115 patients with clinically diagnosed foot intertrigo were included in the present study based on the selection criteria. Detailed history including the presenting illness, systemic diseases, past history and family history were taken. In particular, history regarding predisposing factors such as hyperhidrosis, frequent exposure to wet conditions, occlusion, exercise and athletic activities, trauma, immobility, occupation, previous use of medications were taken.

A complete clinical examination including detailed dermatological examination of the foot lesions with respect to their anatomical location, distribution and morphology, and other associated dermatoses were performed. All the data collected were entered in a proforma given in the annexure.

Relevant investigations such as complete haemogram and blood sugar levels were performed to screen for patient's immune status and to rule out diabetes mellitus.

Wood's lamp examination was performed in all cases of erythrasma to look for characteristic coral red fluorescence in toe web spaces.

Specimen were collected from the superficial and deeper areas of the lesions by sterile techniques and were subjected to direct microscopic examination (KOH mount and Grams stain), fungal culture and bacterial culture. Antibiotic susceptibility test was done by disc diffusion technique.⁶⁹

SPECIMEN COLLECTION:

The affected area was cleaned with sterile swab moistened with normal saline and allowed to dry. Scrapings from the lesions were taken with the scalpel blade or with forceps. This material was used for KOH examination and for Gram staining. Specimens from deeper area of the lesions were collected with sterile swabs moistened with sterile water and were sent for culture.

MYCOLOGICAL STUDIES:

Direct Examination of the skin scrapings mounted with 10% KOH

The scrapings from the lesions were placed on a clean glass slide to which 1-2 drops of 10% KOH was added and was covered with a coverslip. It was warmed slightly over a low flame, and kept for 20 minutes. Once skin material was digested, the coverslip was pressed down to squash the fragments and render them transparent, and the excess KOH was blotted off using a blotting paper. This preparation was examined under low power and high power magnification for fungal elements.

Culture for Fungi

A part of the specimen was inoculated onto two Sabouraud's Dextrose Agar (SDA) plates. One was incubated at 37°C and other was kept at room temperature, for four weeks. Culture plates were examined every week for growth of any fungi. If any growth was obtained, it was further analyzed for and colony characteristics, pigment production and the morphology was studied under a microscope in lactophenol cotton blue (LPCB) mount of the growth.

Germ Tube Test

A smear was prepared from the suspected colony of candida stained by grams stain and observed for gram positive oval budding cells. If candida was present, a germ tube test was performed to identify *Candida albicans* species.

BACTERIOLOGICAL STUDIES:

Gram Stain Preparation

Smear from the clinical specimen was prepared and stained with Grams stain to look for any gram positive or gram negative bacteria.

Culture for Bacteria

The culture media used were nutrient agar plate, blood agar plate, chocolate agar plate and McConkey's agar plate. The specimens were inoculated on these media and incubated for 24 hours at 37°C. The blood agar and chocolate agar were incubated in carbon dioxide incubator. Smears were prepared from the colonies and stained by Grams stain. The colonies grown on the plate were smeared and stained by Gram's stain method. In addition, relevant biochemical tests were done for identification of the organism.

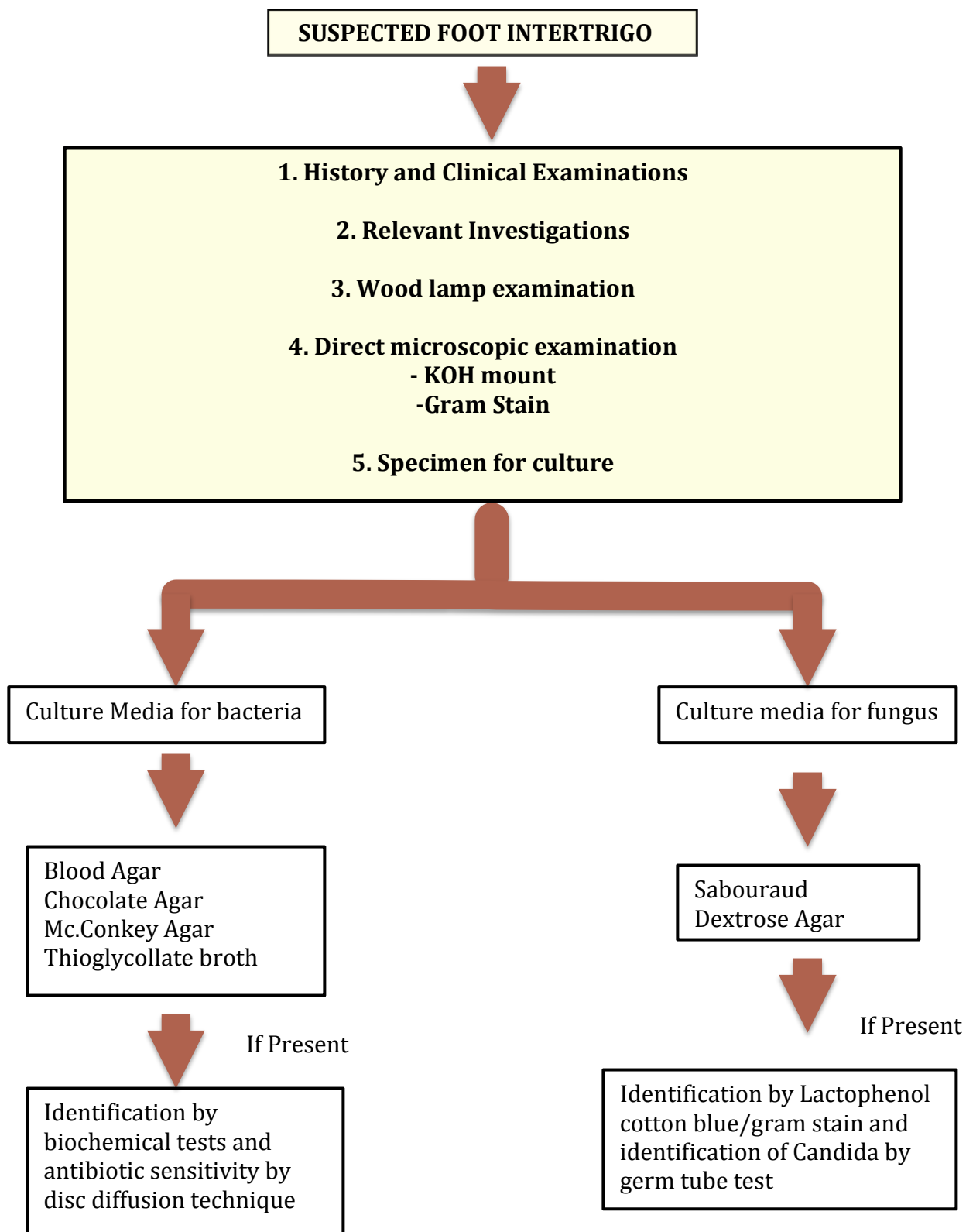
Antibiotic Sensitivity Testing

All the organisms isolated were tested for antibiotic sensitivity by Kirby Bauer's Disc Diffusion Technique. The results were interpreted as sensitive, moderately sensitive and resistant.

STATISTICAL ANALYSIS:

Data was compiled in Microsoft excel after coding and was analyzed using SPSS 20 version software. Qualitative data was represented by frequencies and proportions and analyzed.

FIGURE 6: APPROACH TO CASE OF FOOT INTERTRIGO



OBSERVATION AND RESULTS

OBSERVATION AND RESULTS

Table 4: Age distribution of subjects with Foot Intertrigo

		Frequency	Percent
Age	<10yrs	3	2.6%
	11 to 20 yrs	5	4.3%
	21 to 30 yrs	20	17.4%
	31 to 40 yrs	18	15.7%
	41 to 50 yrs	30	26.1%
	51 to 60 yrs	24	20.9%
	61 to 70 yrs	9	7.8%
	>70yrs	6	5.2%
	Total	115	100.0%

Mean age of the subjects 44.71 ± 17

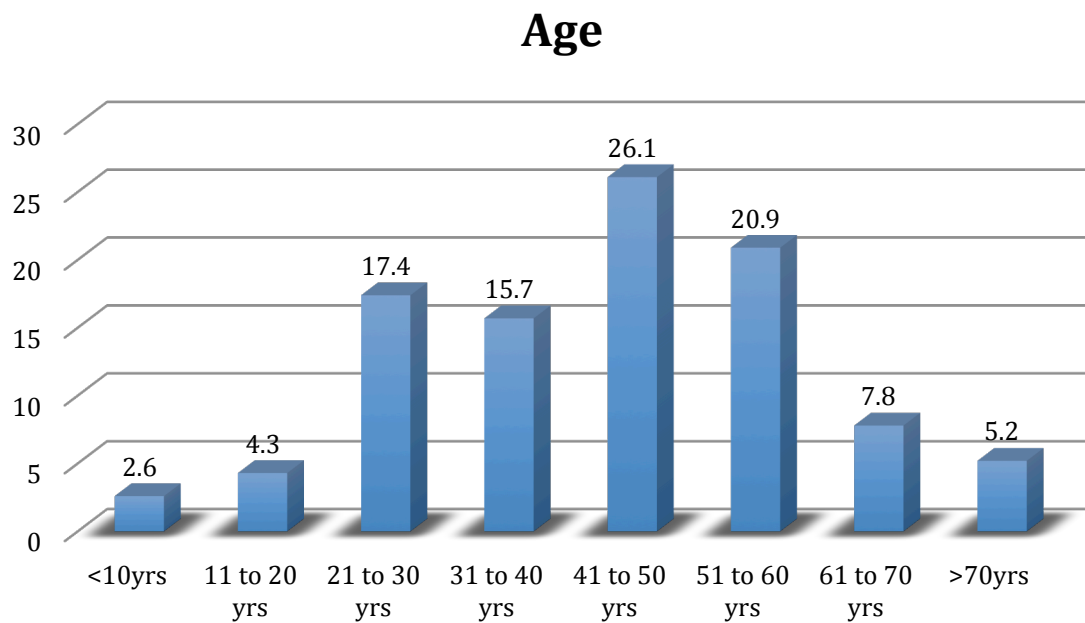


Figure 7: Bar diagram showing age distribution of subjects with Foot Intertrigo

In the present study, majority of subjects were in the age group 41 to 50 yrs i.e. 26.1%, 21% in 51 to 60 yrs age group. Least was found in age group < 10 yrs. Youngest

was 4 years old and oldest was 86 years. Mean age was 44.71 ± 17 . Foot Intertrigo was common in Middle age group.

Table 5: Sex distribution of subjects with Foot Intertrigo

		Frequency	Percent
Sex	Females	57	49.6
	Males	58	50.4
	Total	115	100.0

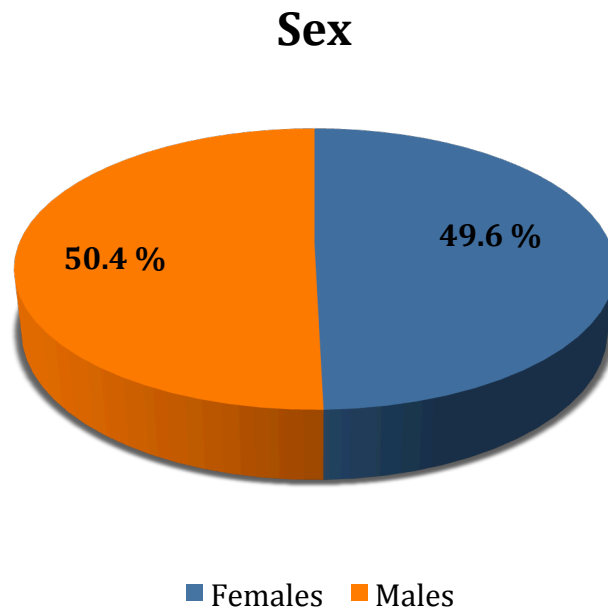


Figure 8: Sex distribution of subjects with Foot Intertrigo

There were total of 57 females and 58 males. There was no predilection towards either sex.

Table 6: Distribution of subjects with Foot Intertrigo based on Occupation

		Frequency	Percent
Occupation	Agriculture	22	19.1%
	Housewife	39	33.9%
	Student	9	7.8%
	Maid	9	7.8%
	Daily laborer	8	7.0%
	Retired/ Unemployed	9	7.8%
	Businessman	6	5.2%
	Skilled work	13	11.3%
	Total	115	100.0%

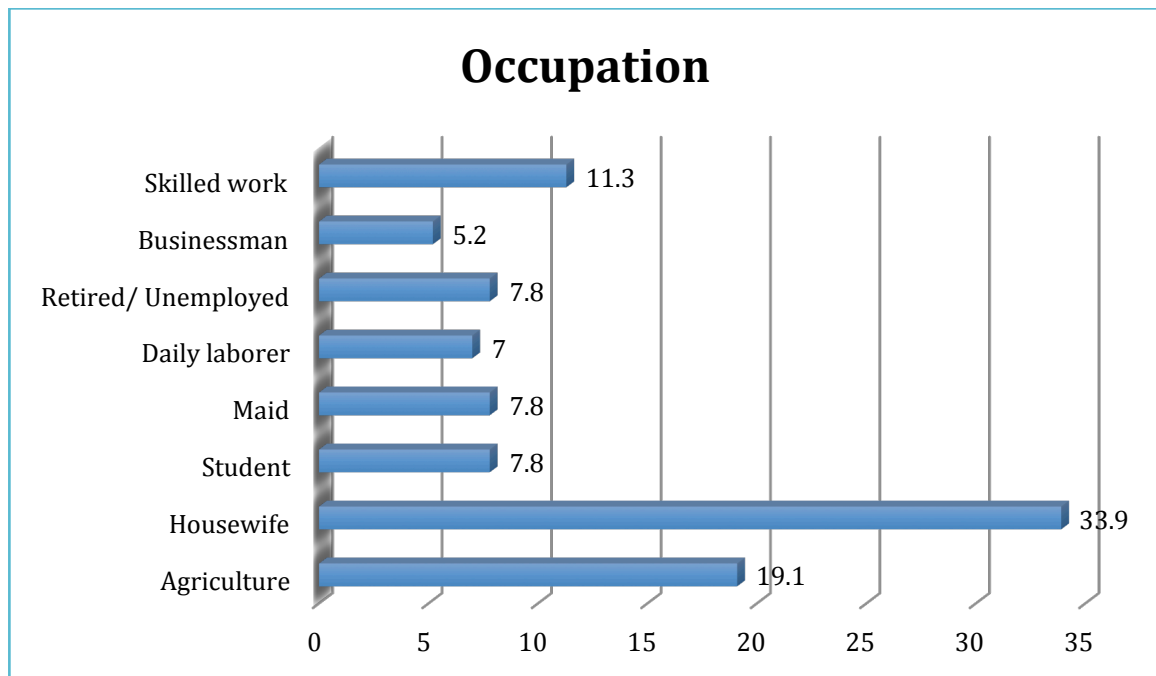


Figure 9: Bar diagram showing Distribution of subjects with Foot Intertrigo based on Occupation

Foot Intertrigo was common among Housewives (33.9%) and agriculturists (19.1%).

Table 7: Onset of Foot Intertrigo

		Frequency	Percent
Onset	Acute	26	22.6 %
	Insidious	89	77.4 %
	Total	115	100.0

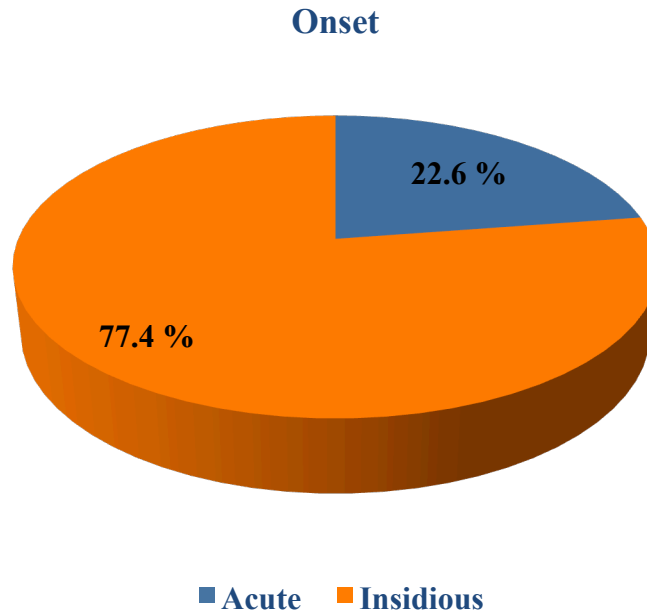


Figure 10: Pie diagram showing Onset of Foot Intertrigo

Majority i.e. 77.4% of subjects had insidious onset and 22.6% had acute onset.

Table 8: Duration of disease in Foot Intertrigo subjects

		Frequency	Percent
Duration of disease	< 6 months	32	27.8
	6 months to 1 year	12	10.4
	1 year to 2 year	27	23.5
	2 year to 5 year	31	27.0
	> 5 years	13	11.3
	Total	115	100.0

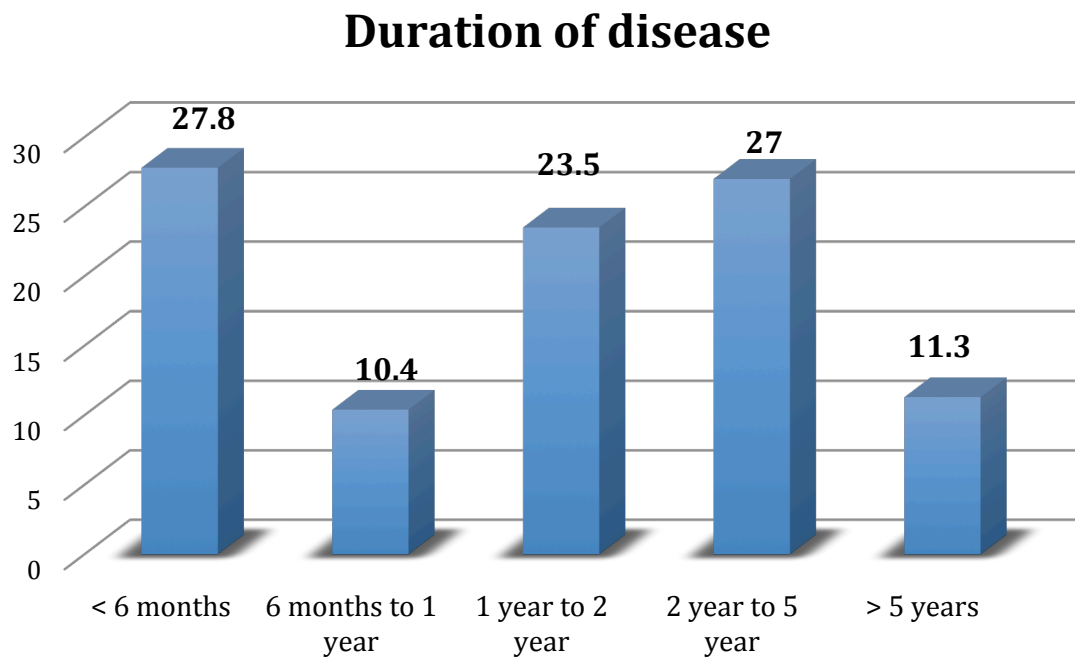


Figure 11: Bar diagram showing duration of disease in subjects of Foot Intertrigo

In the study 27.8% had disease for < 6 months, 27% had disease for 2 to 5 years of duration.

Table 9: Number of predisposing factors among Subjects

		Frequency	Percent
No Predisposing factors		23	20
Number of Predisposing factors	1	73	63.5
	2	17	14.8
	3	2	1.7
	Total	115	115

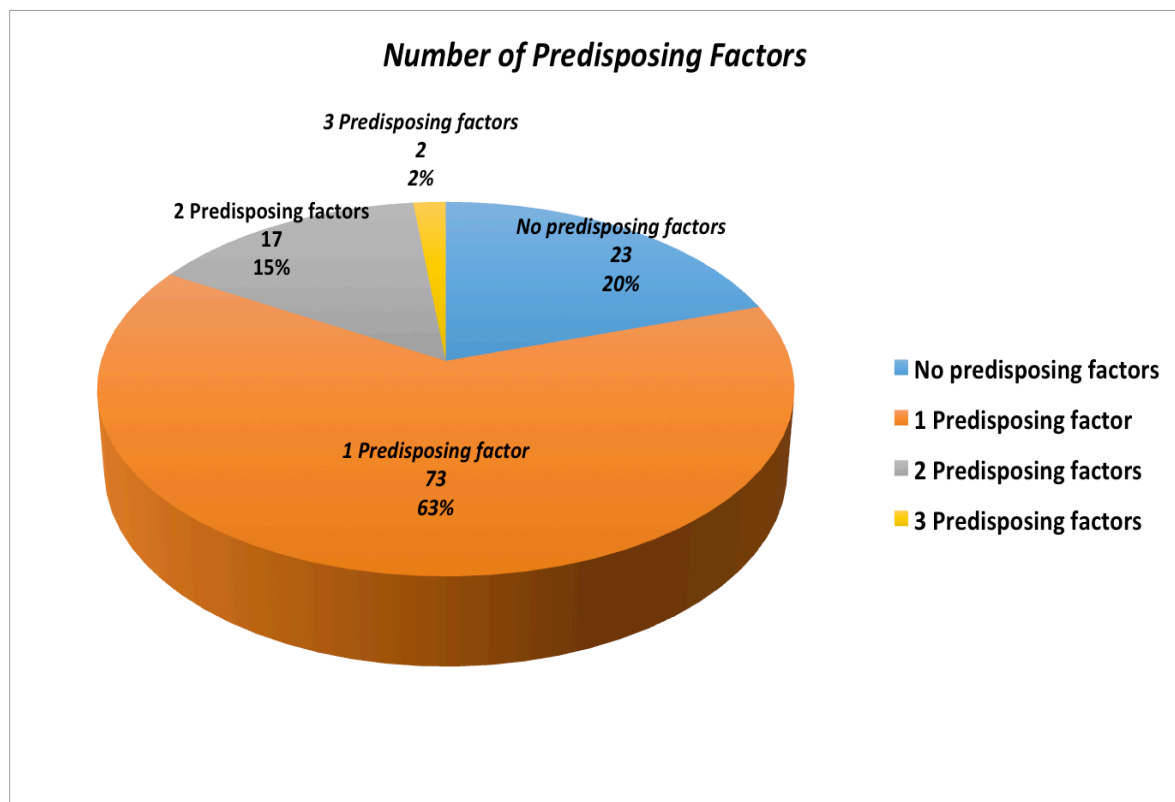


Figure 12: Pie diagram showing Number of Predisposing Factors in subjects of Foot Intertrigo

No predisposing factor was seen in 23 (20%) of the cases. Of the remaining 92 cases, 1 predisposing factor was seen in 73 (63.5%), 2 predisposing factors in 17 (14.8%) and 3 factors in 2 cases (1.7%).

Table 10: Predisposing factors among Subjects

Predisposing factors	No. of Cases	Percentage
Wet conditions	57	49.6%
Hyperhidrosis	10	8.7%
Poor Hygiene	13	11.3%
Occlusive Foot wear	22	19.1%
Foot Deformity	5	4.3%
Obesity	4	3.5%
Immobility	4	3.5%
Athlete	1	0.9%

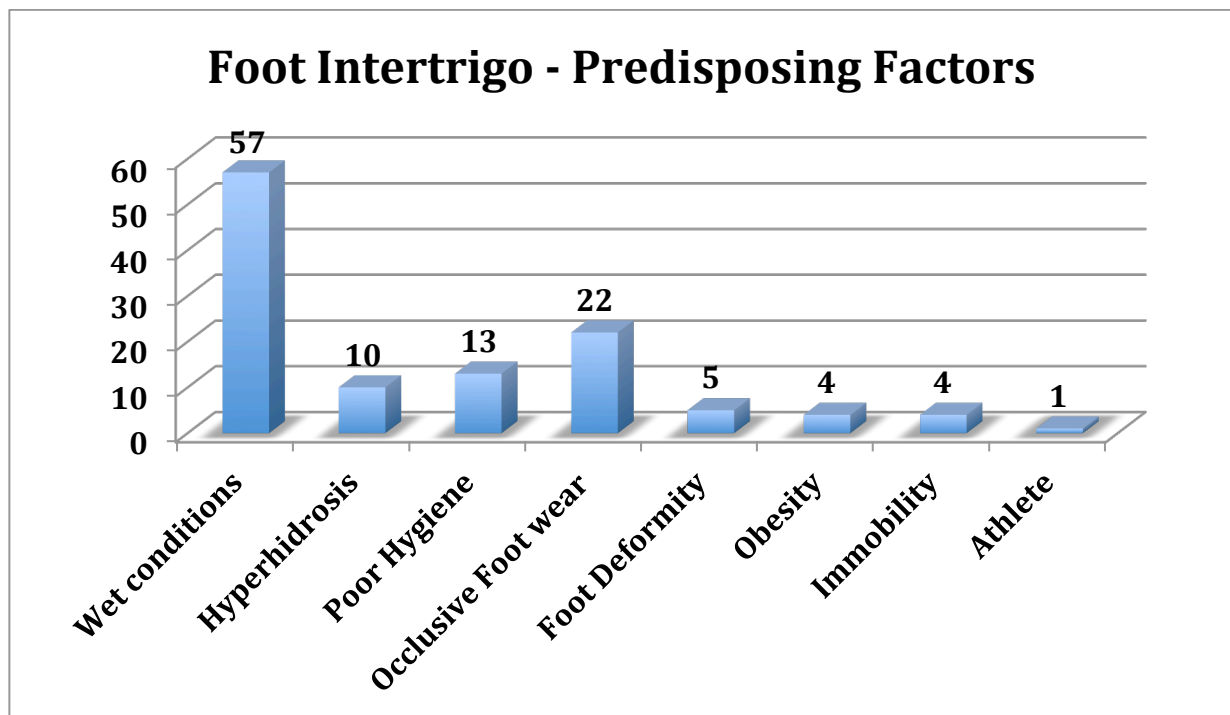


Figure 13: Bar diagram showing Distribution of Predisposing factors in Foot Intertrigo

Most common predisposing factor was Wet conditions in 49.6%. 19.1% had occlusive foot wear as predisposing factor. Poor hygiene and Hyperhidrosis was seen in 11.3% and 8.7% cases respectively.

Table 11: Distribution of associated conditions among the subjects

Associated Condition	Number of cases	Percentage
Diabetes Mellitus	28	24.3%
Hypertension	6	5.2%
Superficial Dermatophytosis	22	19.1%
Intertrigo at other sites	11	9.5%
Eczema	10	8.7%
Hyperkeratotic dermatoses	8	7%
Onychomycosis	3	2.6%
Trophic Ulcer	3	2.6%
Pyoderma	4	3.5%
Cellulitis	5	4.3%
Hansens	3	2,6%
Lipodermatosclerosis	3	2.6%
Psoriasis	2	1.7%
Paronychia	2	1.7%
Pitted Keratolysis	2	1.7%
Diabetic Bullae	2	1.7%
Erythrasma	1	0.9%
Miscellaneous	9	7.8%

Most common associated condition found along with Foot intertrigo were Diabetes mellitus in 28 (24.3%), followed by Superficial dermatophytosis seen in 22 cases (19.1%), and other sites Intertrigo seen in 11 cases (9.5%). Eczemas were seen in 10 (8.7%) cases and Hyperkeratotic deratoses seen in 8 (7%) cases. One of the major complications of Foot Intertrigo, Cellulitis, was seen in 5 (4.3%) cases.

Table 12: Number of Symptoms in Foot Intertrigo subjects

		Frequency	Percent
Asymptomatic		46	40 %
Number of Symptoms	1	47	40.9 %
	2	20	17.4
	3	2	1.7
	Total	115	100.0

46 patients (40%) were asymptomatic at the time of presentation. 47(40.9%) had 1 symptom, 20(17.4%) had 2 symptoms and 2 (1.7%) of them had 3 symptoms.

Table 13: Presenting complaints in Foot Intertrigo subjects

	Number of cases	Percentage
Asymptomatic	46	40%
Symptoms	69	60%
• Burning sensation	16	13.9%
• Itching	42	36.5%
• Pain	23	20%
• Scaling	10	8.7%

In the study only 60% had symptoms, 13.9% had burning sensation, 36.5% had itching, 20% had pain and 8.7% had scaling.

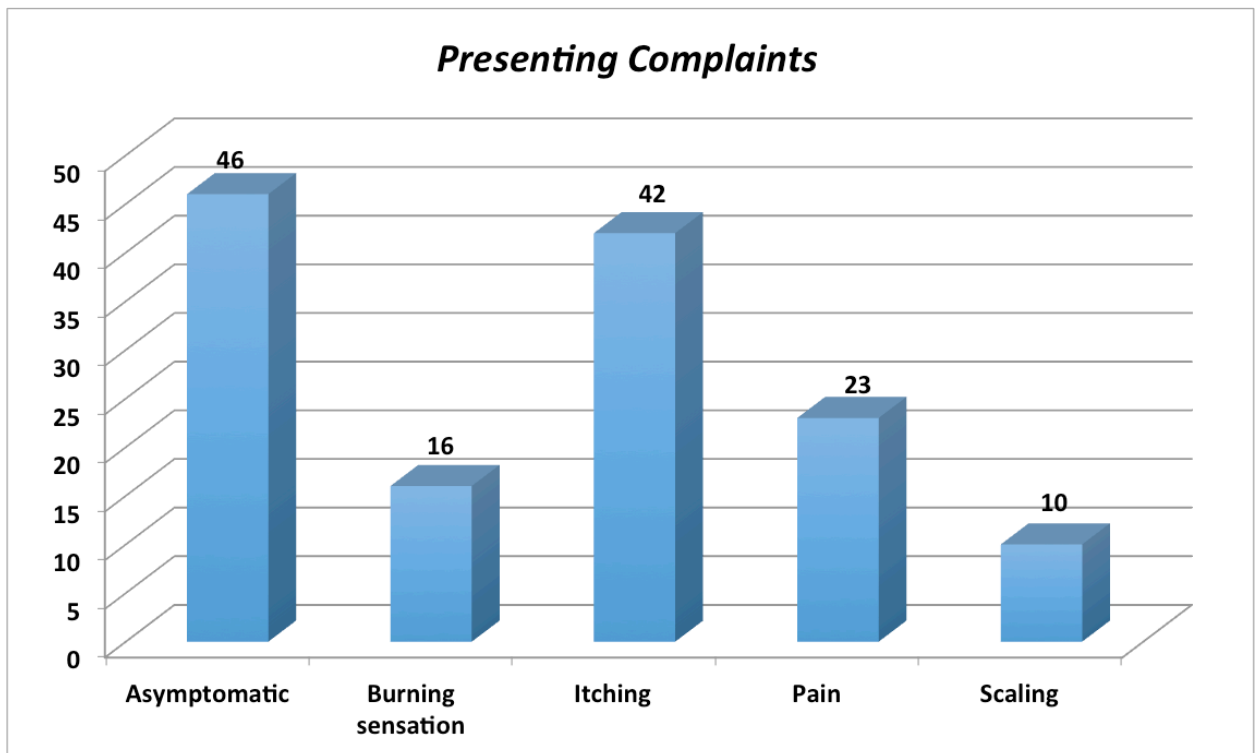


Figure 14: Bar diagram showing Distribution of Complaints in Foot Intertrigo subjects

Table 14: Primary lesions among the subjects

Primary lesions	Present	Absent
Papule	6 (5.2%)	109 (94.8%)
Vesicle	10 (8.7%)	105 (91.3%)
Pustule	10 (8.7%)	105 (91.3%)
Bullae	3 (2.6%)	112 (97.4%)
Papule	7 (6.1%)	108 (93.9%)
Macule	1 (0.9%)	114 (99.1%)

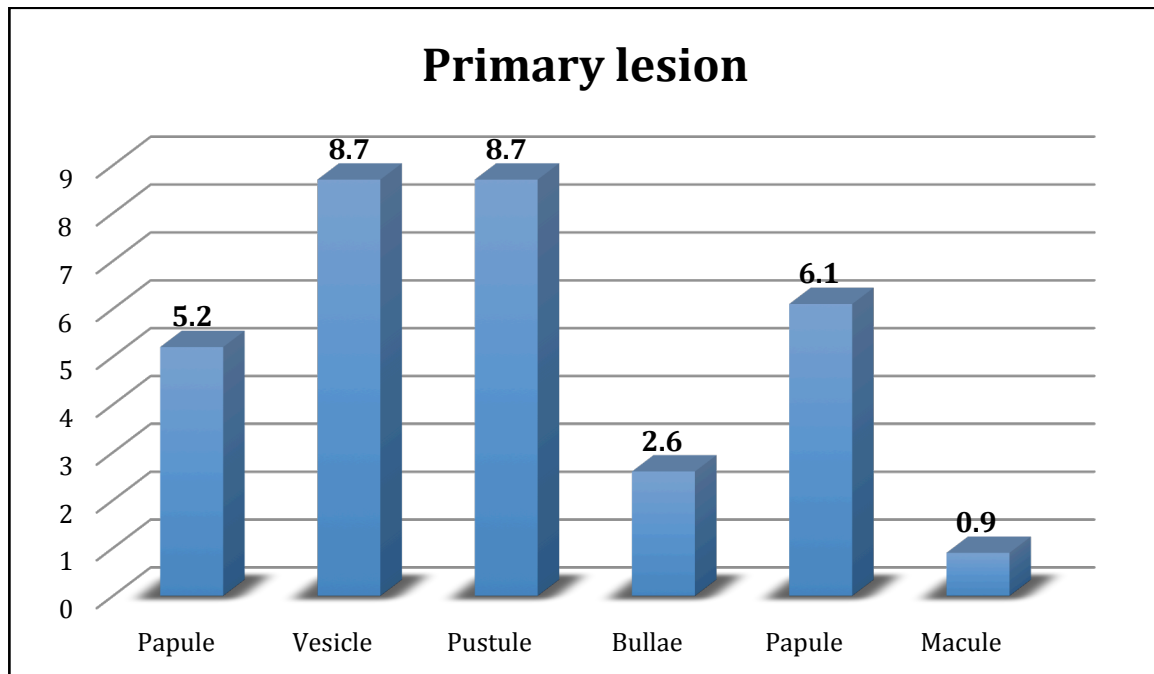


Figure 15: Bar diagram showing Primary lesions among the subjects

In the study most common lesion among Intertrigo was Vesicle and Pustule in 8.7%.

Least common primary lesion was macule.

Table 15: Foot Intertrigo – Clinical Presentation

CLINICAL PRESENTATION	No. of Cases	PERCENTAGE
MACERATION	83	72.2 %
SCALING	44	38.2%
DISCHARGE/EXUDATION	22	19.1%
ULCER/EROSION	16	13.9%
FISSURING	12	10.5%
CRUSTING	8	6.9%

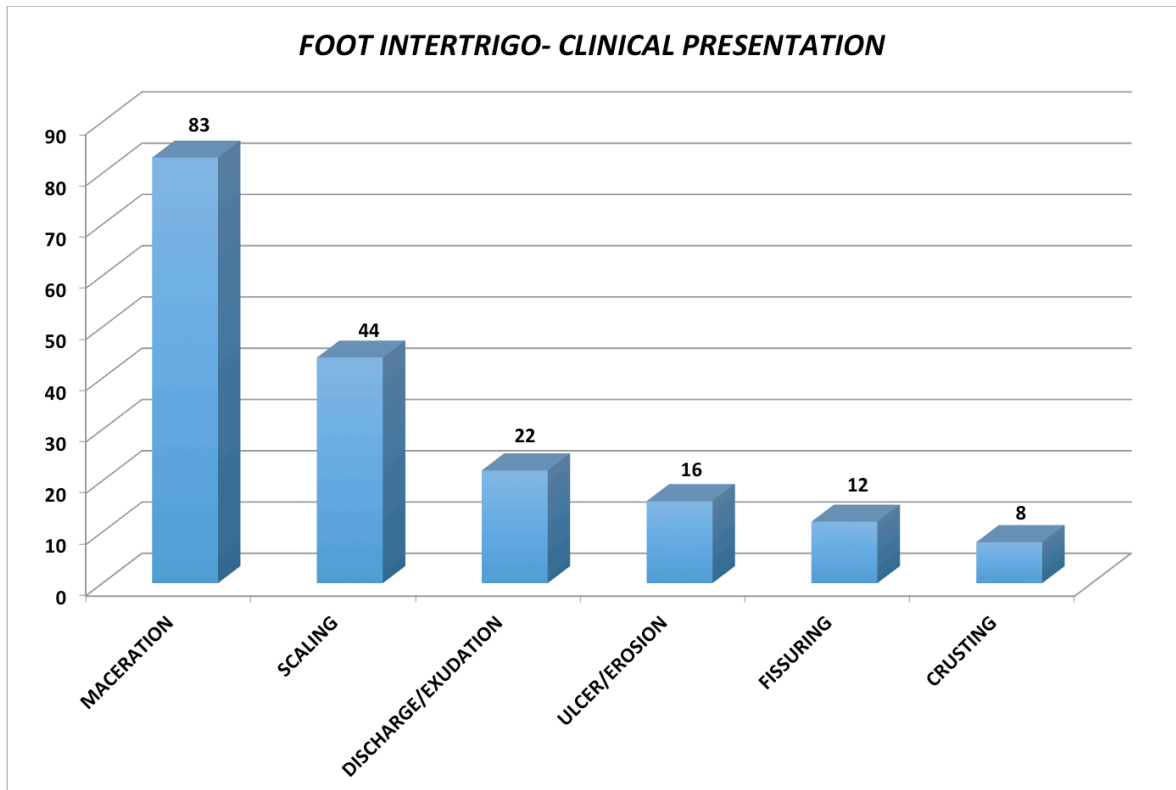


Figure 16: Distribution of Clinical Presentation in Foot Intertrigo

Most common clinical presentation was maceration seen in 83 cases(72.2%) followed by scaling in 44 cases(38.2%). Discharge/exudation was seen in 22 (19.1%), Ulcer/erosion in 16 (13.9%), Fissuring in 12(10.5%) and Crusting in 8 (6.9%) cases.

Table 16: Color of the lesions among the subjects

Color changes	No. of cases	Percentage
Erythematous	34	29.6%
White lesions	56	43.5%
Greenish discolouration	4	3.5%
Hyperpigmentation	7	6.1%

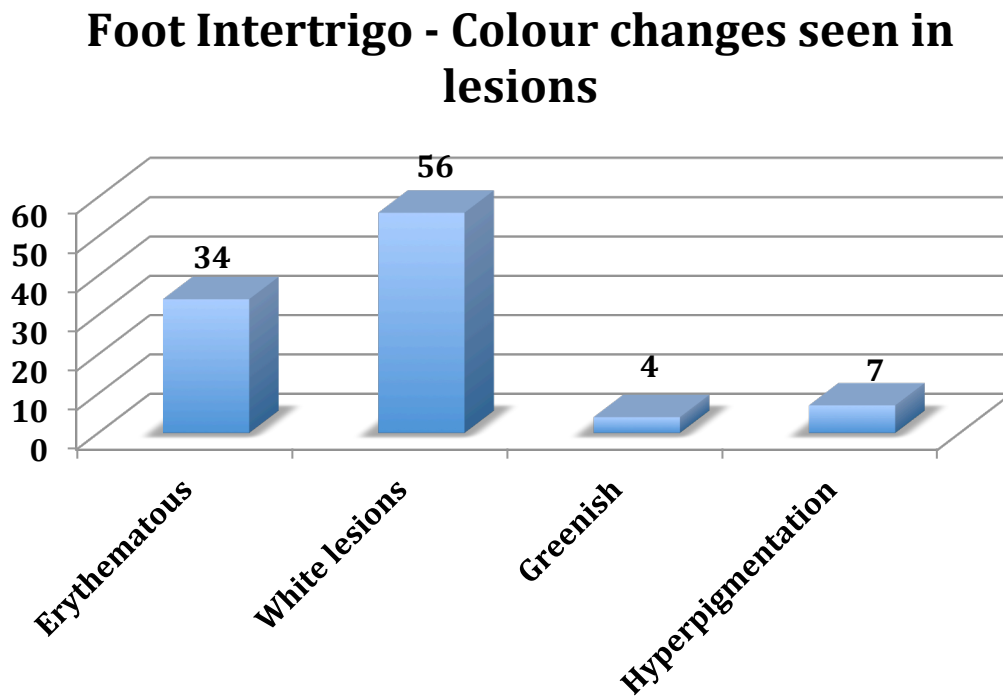


Figure 17: Bar diagram showing Color of the lesions in Foot Intertrigo

Most of the lesions were white in color in 43.5% and 29.6% were Erythematous lesions.

Greenish discolouration was seen in 4% and Hyperpigmentation in 7 %.

Table 17: Foot Intertrigo – Sites of Involvement

TOE WEB SPACE	RIGHT FOOT	LEFT FOOT	TOTAL
1 ST TOE WEB SPACE	12	15	27
2 ND TOE WEB SPACE	17	15	32
3 RD TOE WEB SPACE	45	48	93
4 TH TOE WEB SPACE	75	79	154
TOTAL	149	157	

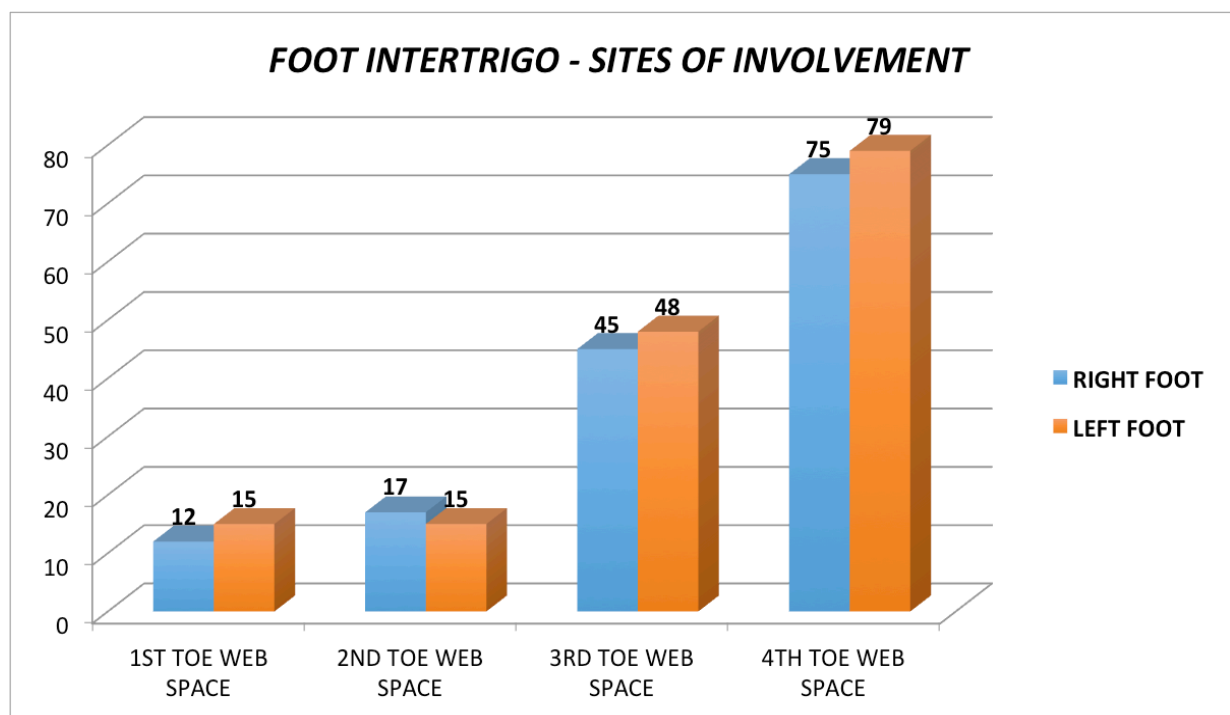


Figure 18: Foot Intertrigo- Sites of Involvement

Most common site of Involvement of foot intertrigo was the 4th Toe web space. In 98 subjects (85.2%), 4th toe web space was involved either unilateral or bilateral. Next common site was 3rd toe web space. The intertriginous lesions were extending to digitoplantar sulcus and sole in 21 cases, extending to dorsal surface in 10 cases and extending to lateral surface in 1 case. Bilateral involvement was seen in 62 subjects (53.9%) and unilateral involvement seen in 53 subjects (46.1%).

Table 18: Foot Intertrigo: Number of Toe web spaces involved

INVOLVEMENT	NUMBER OF CASES	PERCENTAGE
1 WEB SPACE	30	26.1%
2 WEB SPACES	44	38.2%
3 WEB SPACES	11	9.5%
4 WEB SPACES	18	15.6%
5 WEB SPACES	2	1.7%
6 WEB SPACES	2	1.7%
7 WEB SPACES	1	0.8%
8 WEB SPACES	7	6.1%

Intertrigo of toe web spaces was seen in more than 1 site in 85 cases (73.9%). Intertrigo affecting 2 web spaces was commonest, seen in 44 cases (38.3%). All of the web spaces were involved in 7 cases (6.1%).

Table 19: Foot Intertrigo- KOH findings

		Frequency	Percent
KOH Mount	Hyphae	16	64%
	Yeast cells	9	36%
	Total	25	

Among the 25 subjects positive for KOH mount 64% showed hyphae and 36% showed Pseudo hyphae.

Table 20: Foot Intertrigo - Growth on fungal culture

Fungal culture		No. of Cases	Percent
Candida Species	C.albicans	8	50%
	C.tropicalis	1	6.25%
Dermatophytes	Microsporum nanum	1	6.25%
Non Dermatophytic Fungi	A. niger	4	25%
	A.flavus	1	6.25%
	Fusarium Sp.	1	6.25%
Total		16	100 %

Foot Intertrigo - Fungal Culture

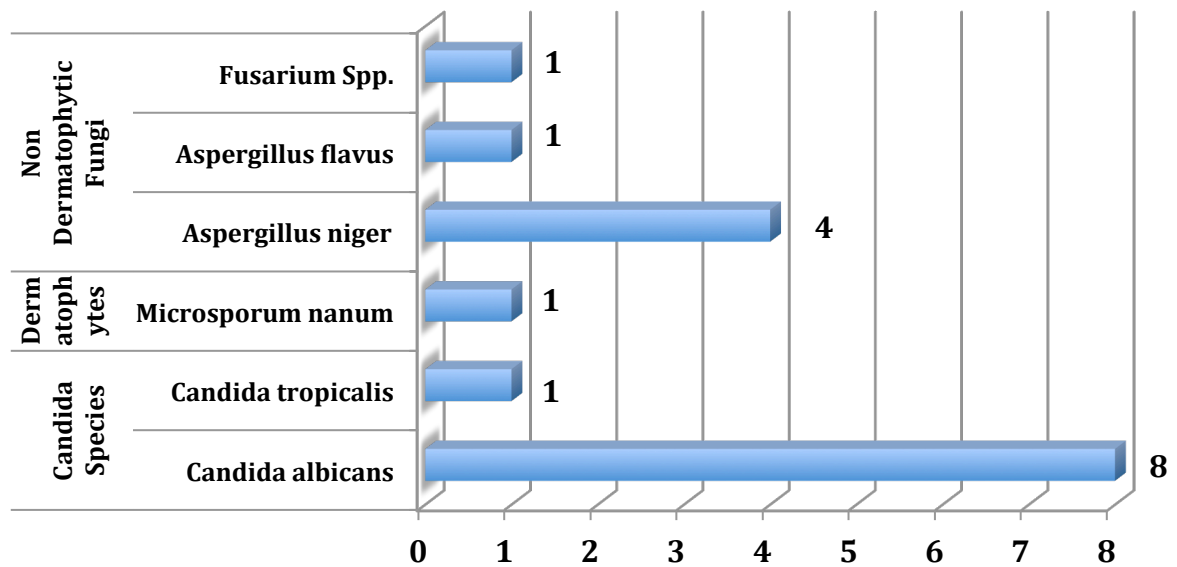


Figure 19: Foot Intertrigo – Growth on Fungal culture.

Among the 16 subjects who showed positive for Fungal culture 50% were C.albicans, 25% were A.niger and 6.5% were A.flavus, C.tropicalis, Fusarium and Microsporum nanum.

Table 21: Gram staining findings among subjects

		No. of Cases	Percentage
Gram's staining	Pus cells	64	55.7%
	Gram Positive Cocci	82	71.3%
	Gram Positive Bacilli	20	17.4%
	Gram Negative Bacilli	36	31.3%
	Gram positive Yeast cells	9	7.8%

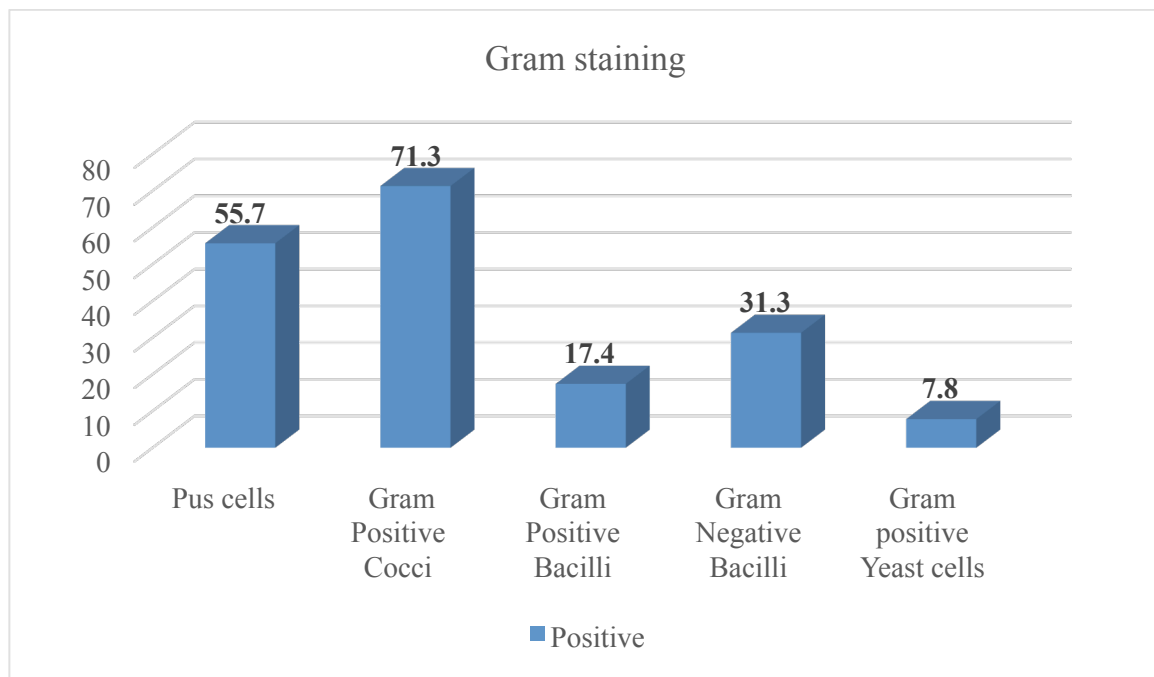


Figure 20: Bar diagram showing Gram staining findings

Gram staining showed majority were Gram positive cocci (71.3%), and 31.3% were Gram negative bacilli. Gram positive bacilli were seen in 17.4%.

Table 22: Foot Intertrigo- Growth on Bacterial Culture

BACTERIAL CULTURE	No. of Cases n=115			No. of Cases
CULTURE POSITIVE	100	Commensal	Micrococci	18
			CoNS	16
		Pathogenic	Gram Positive	31
			Gram Negative	58
NO GROWTH	15			
TOTAL	115			

In total of 115 cases, Bacterial growth was seen in 100 cases, and there was no growth in 15 cases. Commensals such as micrococci were seen in 18 cases and Coagulase negative Staphylococci were seen in 16 cases. Of pathogenic bacteria 31 were gram positive and 58 cases were Gram negative.

Table 23: Bacterial pathogens isolated in Culture.

BACTERIAL CULTURE	No. of Cases	ORGANISM	No. of Cases
GRAM POSITIVE	31	Methicillin Sensitive Staphylococcus aureus	24
		Methicillin Resistant Staphylococcus aureus	4
		Alpha Hemolytic Streptococci	1
		Beta Hemolytic Streptococci	1
		Enterococcus	1
GRAM NEGATIVE	58	Pseudomonas aeruginosa	14
		Acinetobacter Species	12
		Escherichia coli	10
		Klebsiella Species	9
		Enterobacter Species	7
		Proteus Species	3
		Citrobacter koserii	2
		Providencia stuartii	1

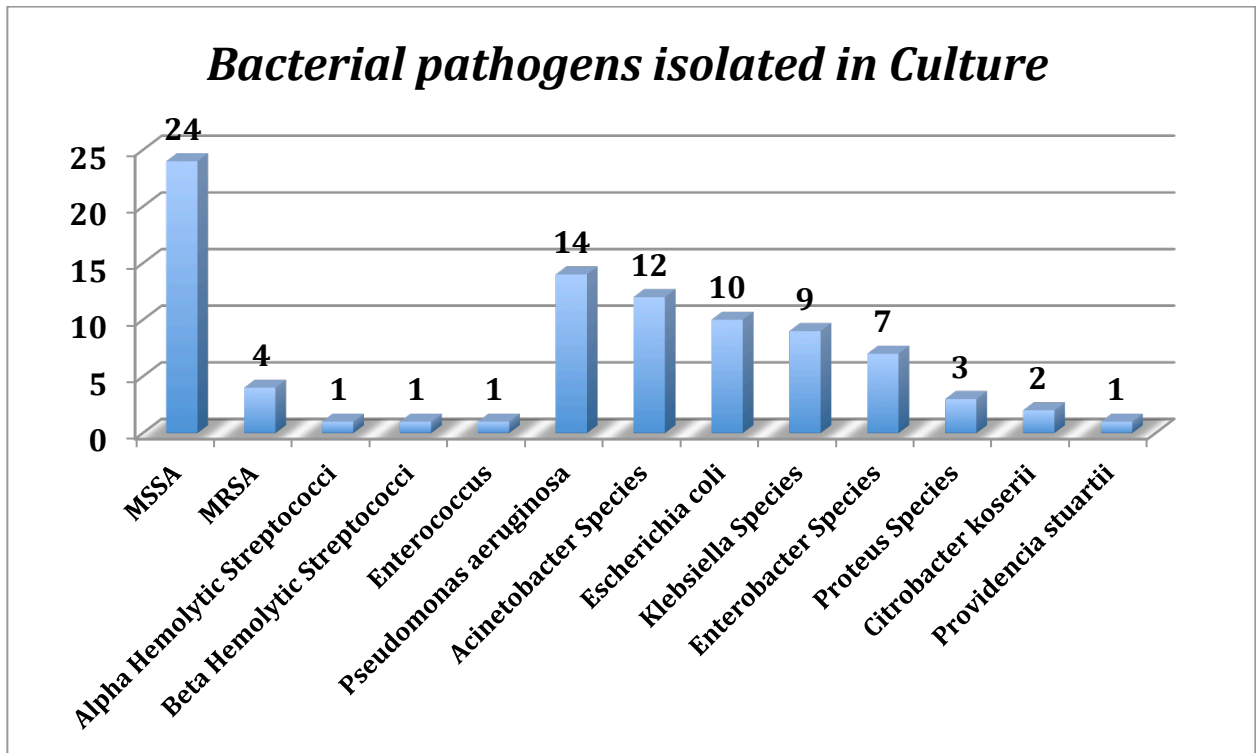


Figure 21: Foot Intertrigo Growth on Bacterial Culture

In the present study, most common bacteria isolated was MSSA which was isolated from 24 cases(20.9%). Pseudomonas aeruginosa was isolated from 14 cases (12.2%) and Acinetobacter in 12(10.4%). E.Coli was isolated in 10(8.7%), Klebsiella species in 9(7.8%), Enterobacter species in 7(6.1%) and MRSA in 4 (3.5%) cases.

Table 24: Foot Intertrigo- Mixed Infections

MIXED INFECTIONS	No. of Cases	Percentage
GPC (clusters) + GPC (chains)	1	2.9%
GNB + GPC	3	8.8%
2 or more GNB	13	38.2%
GPC + Fungal Infection	8	23.5%
GNB + Fungal Infection	9	26.5%
TOTAL	34	

In total of 34 mixed infections, infections with 2 or more Gram negative bacilli constituted about 38.2% of mixed infections. Gram positive cocci in clusters with Gram positive cocci in chains was seen only in 1 case.

Incidence of fungal infection with Gram positive bacterial infection was seen in 8 cases and Fungal with gram negative bacterial infection seen in 9 cases (26.5%).

Table 25: Final diagnosis in patients of Foot Intertrigo

		Frequency	Percent
Final diagnosis	Mixed Toe web Infection	34	29.6 %
	Bacterial Foot Intertrigo	32	27.8 %
	Simple Foot Intertrigo	29	25.2 %
	Fungal Foot Intertrigo	8	7.0 %
	Contact dermatitis	6	5.2 %
	Miscellaneous • Soft corn- 3 • Palmoplantar Psoriasis-2 • Scabies-1	6	5.2 %
	Total	115	100.0

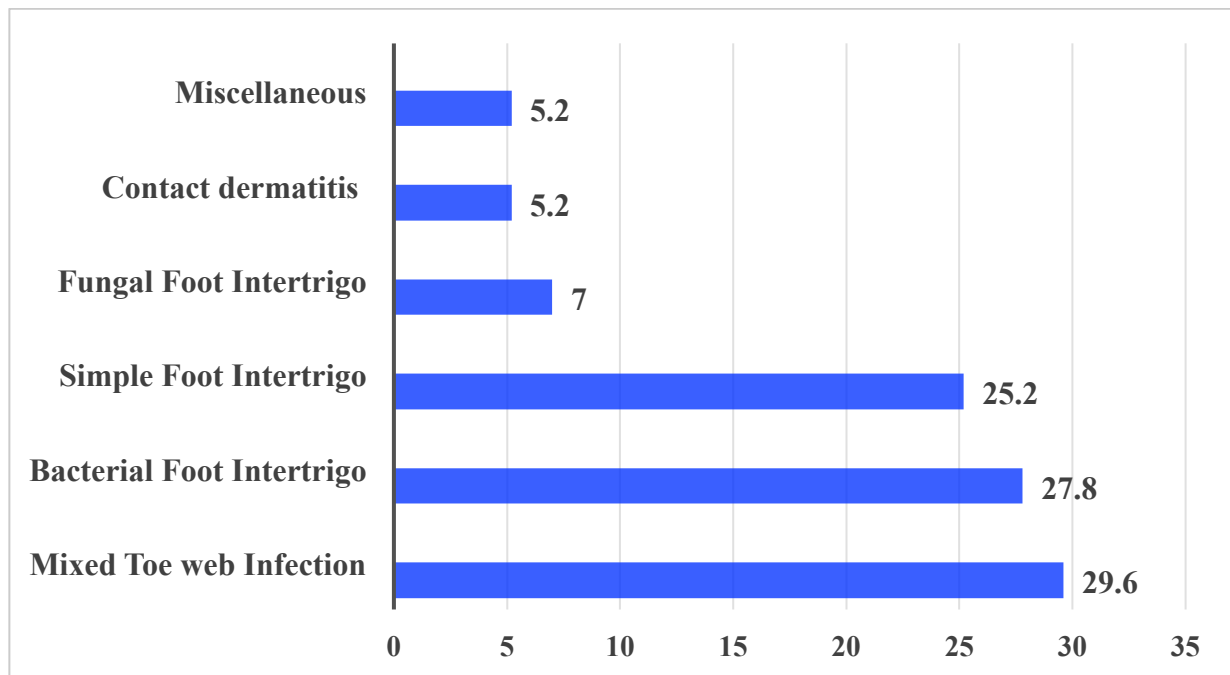


Figure 22: Bar diagram showing Final diagnosis in patients with Foot Intertrigo

Foot Intertrigo was most often due to mixed infections (29.6%), followed by bacterial (27.8%) and simple Intertrigo (25.2%). Fungal foot intertrigo was seen in 7 %.

PHOTOGRAPHS:



Figure 23: Foot Intertrigo of 3rd and 4th toe web spaces with erosions and maceration



Figure 24: Foot intertrigo of 3rd toe web space showing white maceration



Figure 25: Foot Intertrigo of 4th toe web infection showing maceration and greenish discolouration



Figure 26: Foot intertrigo of 4th toe web space showing maceration and greenish discolouration due to Pseudomonas aeruginosa infection



Figure 27: Foot Intertrigo of 4th toe web space extending to plantar surface



Figure 28: Toe web intertrigo with crusting involving the digitopantar sulcus



*Figure 29: Toe web intertrigo affecting the 3rd and 4th toe web spaces in a case of
Hansens and Trophic ulcer*



*Figure 30: Foot Intertrigo affecting all 4 toe web spaces with involvement of
digitoplantar sulcus*



Figure 31: Bilateral foot intertrigo affecting all web spaces with fissuring



Figure 32: 3rd and 4th web intertrigo with pitted keratolysis



Figure 33: A case of diabetic bullae with maceration, erosion and discharge affecting 4th toe web space



Figure 34: Toe web infection of the 1st toe web space with purulent discharge



Figure 35: Interdigital tinea pedis showing scaling of the 4th toe web space.

Figure 36: Case of tinea pedis with extension of scaling to plantar aspect of foot- KOH showed hyphae in this case



Figure 37: Concomitant tinea corporis in the same patient.

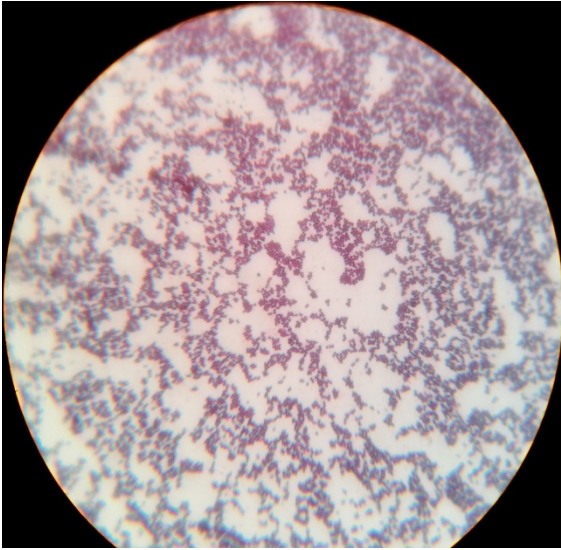


Figure 38: GPC in clusters on Grams stain



Figure 39: Staphylococcus aureus growth on blood agar

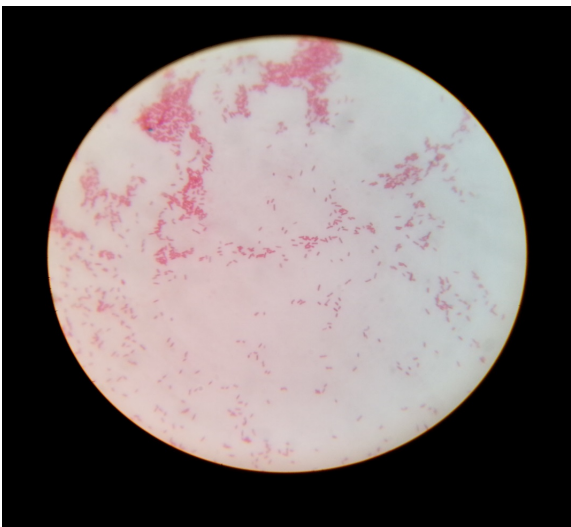


Figure 40: GNB on grams stain

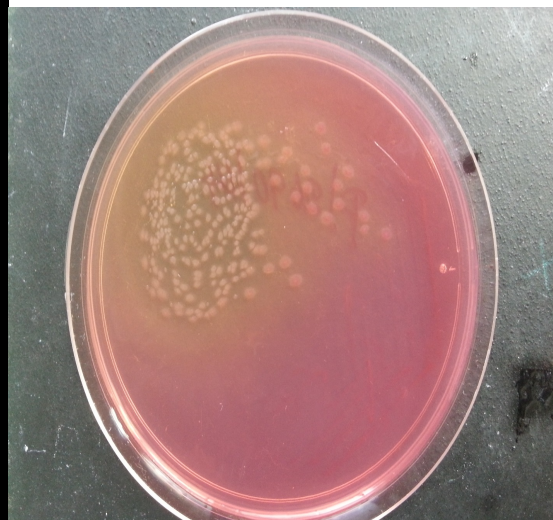


Figure 41: Growth of Pseudomonas aeruginosa on MacConkey agar



Figure 42: Growth of Microsporium nanum on SDA

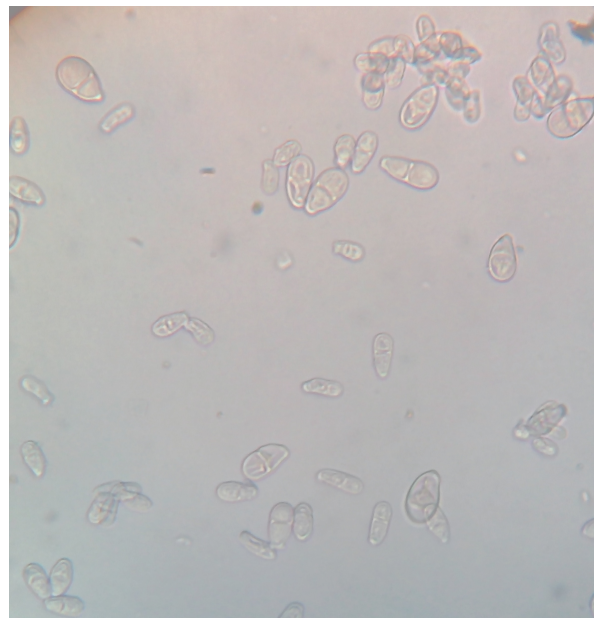


Figure 43: Microscopic picture of Microsporium nanum on LPCB mount

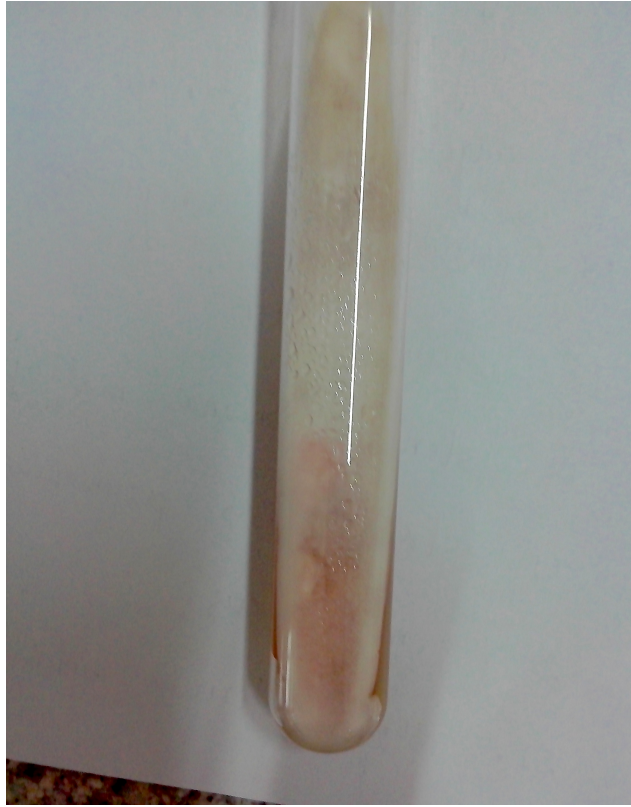


Figure 44: Growth of fusarium on SDA

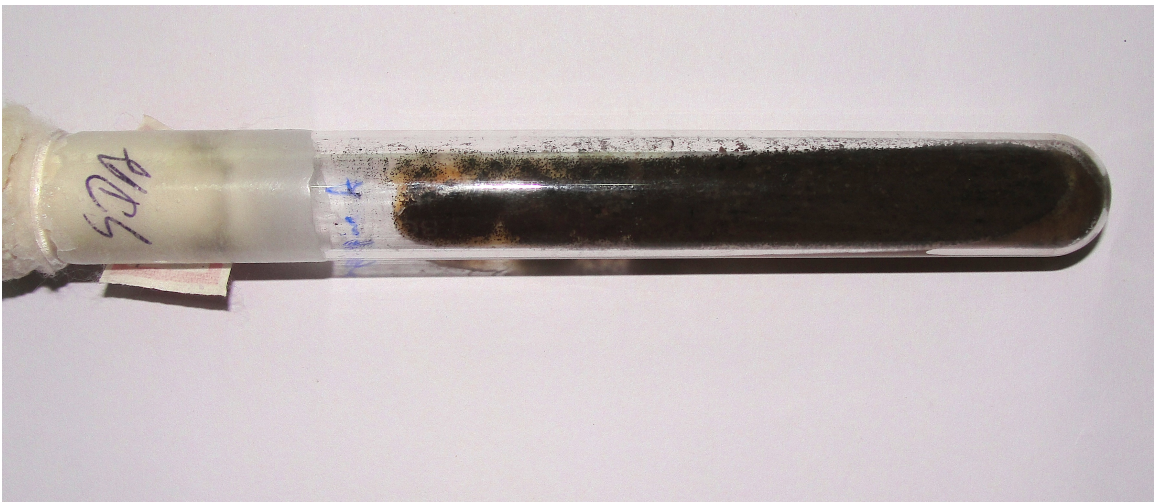


Figure 45: Growth of Aspergillus niger on SDA

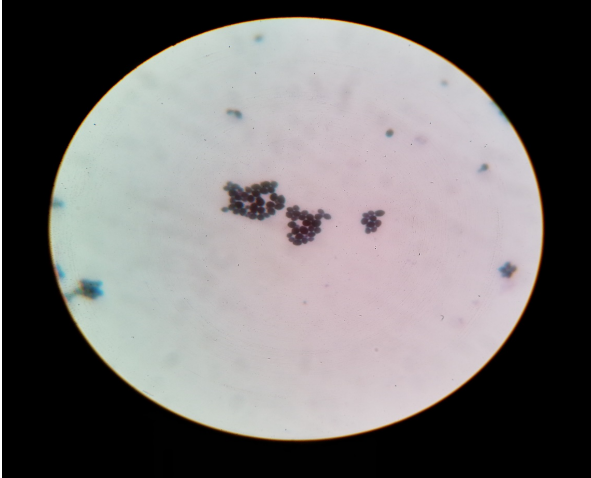


Figure 46: Gram positive budding yeast cells on gram stain

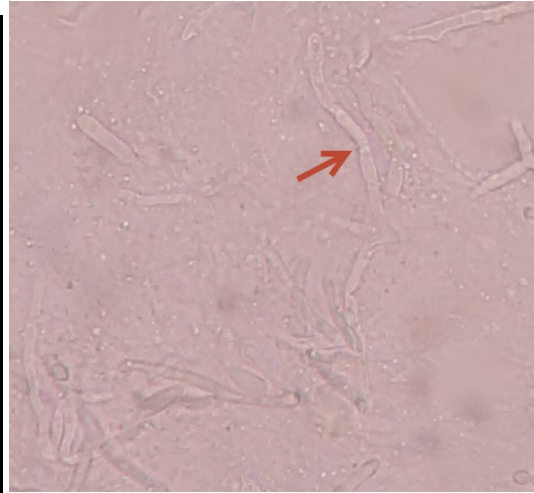


Figure 47: KOH mount showing hyphae



Figure 48: Growth of Candida tropicalis on SDA

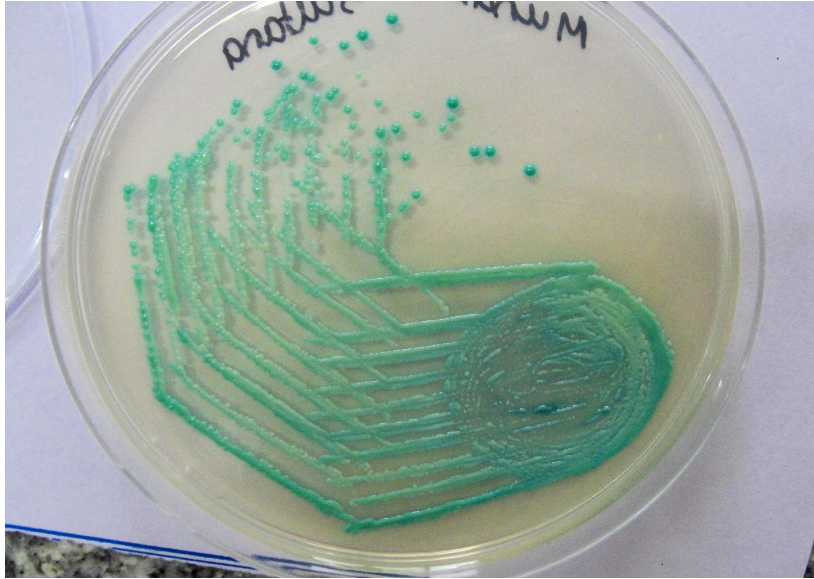


Figure 49: Growth of Candida albicans on SDA

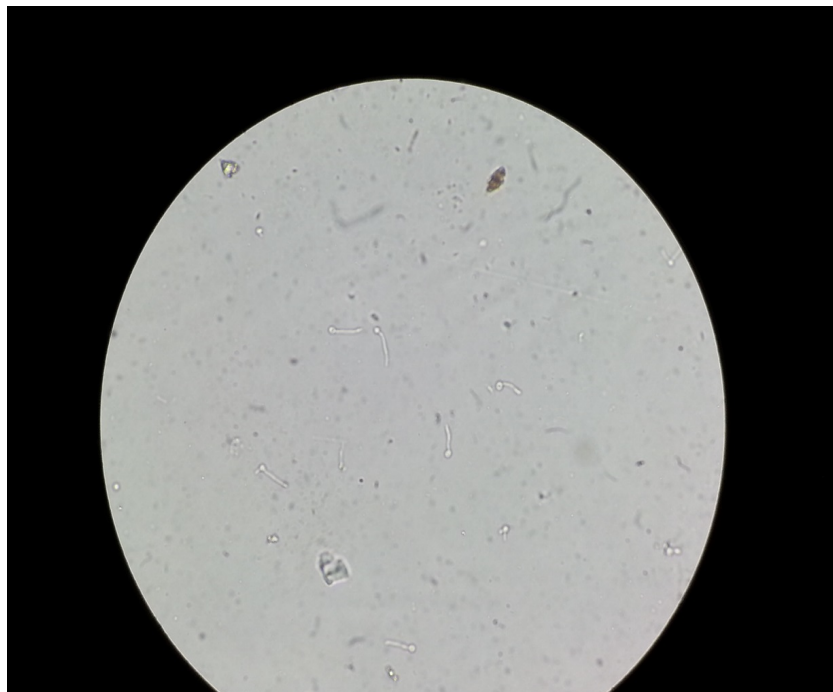


Figure 50: Germ tube formation by Candida albicans

DISCUSSION

DISCUSSION

Foot intertrigo is a relatively common and troubling disorder especially in a hot and humid weather or occluded conditions. Its etiology is variable ranging from a simple intertrigo due to various noninfectious mechanical factors to intertrigo complicated with bacterial or fungal infection. Whether infectious agents play a primary role in intertrigo or simply are common secondary agents is controversial.^{15,70}

Differential diagnosis based on a clinical features alone is difficult because the initial phase of the infection is often common to interdigital tinea pedis, candidal intertrigo, and bacterial intertrigo.^{7,8}

There are no comprehensive studies relating to microbial aetiology and predisposing factors of foot intertrigo. Hence, clinical and microbiological studies are required to assist in the selection of appropriate treatment and the prevention of important complications of foot intertrigo.

In the present study, a total 115 patients with foot intertrigo were enrolled. Detailed history was taken; clinical examination was done along with microbiological investigations. Predisposing factors, preexisting co-morbidities and other associated conditions were enquired about and noted. The following observations are made and are compared with other similar studies.

In the present study, the most common age group affected with foot intertrigo were in age group of 41 to 50 years in 26.1% of cases, and 51 to 60 years in 20%. Mean age of the subjects was 44.71 ± 17 . This finding is similar to the observations made by

Lestringant et al.⁷¹ (45.76 years), Karaca et al.⁸ (49.6 years), Ahmed et al.⁷² (32.6 years), and Jing-Yi Lin et al.³ (59 years).

Foot intertrigo was less commonly noted in children in the present study, may be due to less exposure to the predisposing factors commonly wet conditions as reported by Ahmed et al.⁷²

In our study, a total of 57 females (49.6%) and 58 males (50.4%) were affected, indicating that there was no predilection towards either sex, however male preponderance was observed in other studies, may be due to increased use of occlusive foot wear among the males for professional and extraprofessional reasons or during sports.^{3,7} In our study, females were also almost equally affected and majority of them were housewives and maids. Exposure to wet conditions, during household work, could be the predisposing factor in these cases.

Majority of the patients affected with foot intertrigo in the present study were housewives (33.9%) similar to Ahmad et al.⁷² and Lestringant et al.⁷¹ studies. This can probably be attributed to the constant exposure to excessive moisture and irritants like soap, detergents while doing household chores such as cooking, cleaning, washing clothes and vessels.⁷²

Agriculturists (19.1%) were the second most commonly affected group in our study. In South Asian countries like India, where a lot of agriculturists still depend on conventional ways of farming without use of machinery, they are often required to keep their feet constantly immersed in water and also exposure to various irritants like mud, fertilizers, pesticides during their work, might initiate damage in the stratum corneum, which in conjunction with moisture, may trigger maceration of the toe web spaces.⁷³

Intertrigo is often a chronic disorder with an insidious onset but acute discomfort may be present when there is bacterial infection.^{4,21} In the present study, majority i.e. 77.4% of subjects had insidious onset and 22.6% had acute onset.

Since foot intertrigo lesions are usually simple, mild and asymptomatic,⁴ they are often neglected till complications arise or they develop severe symptoms secondary to either infection, inflammation or other complications. In the present study, 27.8% had foot intertrigo for < 6 months, 27% for 2 to 5 years and 11.3% had it for more than 5 years with the duration ranging from 2 weeks to 15 years. Majority of the foot intertrigo subjects who were enrolled in the study were ignorant or negligent and 22.6% of them had history of previous inadequate treatment.

In the present study most common predisposing factor was chronic exposure to wet conditions seen in 49.6% of subjects, followed by frequent use of occlusive foot wear in 19.1% of subjects, similar to the observations made by Karaca et al.⁸ and Lestringant et al.⁷¹ However exposure to wet conditions among our subjects was mostly due to occupational exposure and household chores, when compared to other studies which was secondary to attending swimming pools, public baths, saunas, and ablutions.^{8,71}

The other frequent predisposing factors seen in this study were poor hygiene in 11.3% of cases and hyperhidrosis seen in 8.7% which is similar when compared to another study.⁷ Other less frequently reported predisposing factors were deformity such as clawing of toes in 4.3% and obesity in 3.5% of subjects. Increased occlusion, stubby toes, along with warm and humid environment of toe web clefts in such individuals causes increased retention of sweat and moisture.^{42,71}

Individuals with foot intertrigo and co-existing comorbidities like diabetes mellitus and obesity are at increased risk of developing cellulitis.^{4,14} In our study, type 2 diabetes mellitus was noted in 24.3% of the subjects similar to other studies.^{71,72} In the present study, cellulitis was noted in 5 patients (4.3%). *Staphylococcus aureus* was isolated from the toe web lesions in 3 patients with cellulitis and *Pseudomonas aeruginosa* in 2 patients similar to the findings in a study by Björnsdóttir et al.⁷⁴ Concomitant fungal infection of other body areas was seen in 25 cases (21.7%), tinea corporis being the most common, similar to Ahmed et al.⁷² where it was observed in 25% of the cases.

Toe web space intertrigo has varied presentation, ranging from mild, asymptomatic lesions to painful inflammatory lesions with profuse malodorous discharge, maceration, and erosions. In the present study, only 60% had symptoms and remaining 40% were asymptomatic. Among the symptomatic patients, itching was the most predominant symptom seen in 36.5% of patients. Less reported complaints included pain (20%), burning sensation (13.9%), and peeling of skin or scaling (8.7%). These findings are similar to studies by Aste et al.⁷ and Jing-Yi Lin et al.³ who found burning, pain and itching as the predominant symptoms.

Bilateral toe web space involvement was more common in our study group seen in 62 cases (53.9%). Most commonly affected toe web space was the 4th toe web space seen in 98 subjects (85.21%), followed by the 3rd toe web space. These findings are in concordance with previous similar studies.^{8,71,72}

Extension of the lesions to the digitoplantar sulcus and sole was seen in 21 cases (18.2%) similar to Aste et al.⁷

In the present study, maceration of the toe web space was the most common clinical presentation seen in 83 cases (72.2%) followed by scaling, seen in 44 cases (38.2%). Maceration of the toe web space causes damage to the stratum corneum, hence predisposing to secondary fungal and bacterial infections, which may in turn complicate simple frictional intertrigo to mixed infection of toe web spaces.^{4,14} Other common findings included exudation in 19.1%, erosions in 16%, fissures in 10.5% and vesicles and pustules in 8.7% of the cases.

Whitish discolouration of lesions was seen in 56 cases (43.5%), erythema in 24 cases (29.6%), and hyperpigmentations in 6.1%. Greenish discolouration was noted in 4 cases (3.5%) and these were associated with gram negative toe web infection and 3 of them were associated with *Pseudomonas* infection. Lestringant et al. also observed similar association of bluish-green pigmentation, in *Pseudomonas aeruginosa* infection of the toe web spaces which is due to the pyocyanin and pyoverdine pigments produced by the organism.⁷⁵

Final diagnosis was made, after obtaining the results of bacteriological and mycological studies. Based on the etiology, foot intertrigo was subdivided into the following categories:

Simple intertrigo: It was diagnosed when no infectious organisms were identified by either culture or direct microscopic examination or when the culture media grew skin commensals from the toe web lesions.^{4,14,15} This was seen in 29 cases (25.2%).

Bacterial intertrigo: When a single bacterial pathogen was isolated from the toe web lesions, it was categorized as bacteria intertrigo. There were a total 32 Bacterial intertrigo cases (27.8%).

Fungal intertrigo: This was diagnosed when KOH mount showed fungal elements and/or when culture grew fungi including yeasts, dermatophytes and non-dermatophytic fungi. This was seen in 7% of the cases.

Mixed toe web infections: When more than one bacteria or bacteria along with fungus, were isolated concomitantly, they were categorized as mixed toe web infections.

In our study, majority of the patients with foot intertrigo had mixed toe web infections which was seen in 34 cases (29.6%), followed by bacterial intertrigo seen in 32 cases (27.8%), simple intertrigo in 29 cases (25.2%), fungal intertrigo in 8 cases (7%), contact dermatitis in 6 cases (5.2%). Miscellaneous causes of intertrigo included 3 cases of soft corn, 2 cases of palmoplantar psoriasis where fissuring and scaling extended to involve interdigital toe web spaces and 1 case of scabies.

The mixed toe web infection rate in the literature is around 22.6% to 75%^{7,76-78} where as frequency of mixed toe web infections in our study was 29.6%. Most common mixed infection was by combined gram negative organisms which was seen in 13 cases (38.2%) followed by mixed gram negative and fungal infection seen in 9 cases (26.5%). The most common concomitant pathogens were *Acinetobacter* sp. with *Escherichia coli*. Similarly the most common concomitant pathogen in Jing-Yi Lin et al.³ was *Pseudomonas aeruginosa* combined with other gram negative or gram positive bacteria,

while in the study by Karaca et al.⁸ it was dermatophytes and coagulase-negative staphylococci.

Out of the 115 cases, bacterial culture was positive in 100 cases. Among these, Micrococci were isolated in 18 cases and Coagulase-negative staphylococci in 13 cases and were categorized as skin commensals. Gram positive pathogens and gram negative pathogens were isolated in 31 (26.9%) and 58 (50.4%) cases respectively. This is in concordance with other studies where increased frequency of gram negative pathogens were isolated.^{3,8} Most frequently isolated pathogens in our study were MSSA (20.9%), *Pseudomonas aeruginosa* (12.1%) followed by *Acinetobacter* sp. (10.4%) and *Escherichia coli* (8.7%). In the study by Karaca et al,⁸ the most common pathogen was coagulase-negative staphylococci (17.9%) followed by *Pseudomonas aeruginosa* (16.7%). Similarly *Pseudomonas aeruginosa*, often together with other gram-negative bacteria or *Staphylococcus aureus*, was isolated as the most common etiologic agent in other studies.^{3,7,76,77}

Most common isolate	Present study	Karaca et al. ⁸	Aste et al. ⁷	Ahamad et al. ⁷²	Lestringant et al. ⁷¹
1	<i>Staph. aureus</i> (24.3%)	<i>P.aeruginosa</i> (16.7%)	<i>P.aeruginosa</i> (46.4%)	<i>Staph.aureus</i> (83.4%)	<i>P.aeruginosa</i> (26.7%)
2	<i>P.aeruginosa</i> 12.2%	<i>Staph. aureus</i> (11.9%)	<i>E.coli</i> (13.8%)	<i>P.aeruginosa</i> (10%)	Coliforms (24.4%)
3	<i>Acinetobacter</i> (10.4%)	<i>C. minutissimum</i> (11.9%)	<i>Staph aureus</i> (8%)	<i>Proteus</i> sp. (3.3%)	<i>B streptococcus</i> (6.7%)

In our study KOH was positive for fungal elements in 25 cases and out of which growth on fungal culture was seen in 64% (16/25) cases. This rate of KOH positive-culture negative frequency is in concordance with a similar study by Karaca et al.⁸ where only 41.6% of KOH positive cases grew positive fungal culture. The differences in the results of KOH examination and culture growth, may be explained by the presence or absence of infecting agent in the particular specimen examined. The culture results also depend upon, whether the nutritional and other requirements like optimum temperature for growth etc. are adequately met or not.⁷⁹ Of the 16 cases, isolated fungal growth was seen 6 cases and mixed growth of fungal along with bacterial pathogens were noted in 10 cases.

In our study, 9 candida species (7.8% of 115), 1 dermatophyte (0.8%) and 6 non-dermatophytic fungi (5.2%) were isolated. *Candida albicans* was the most commonly isolated fungal pathogen seen in 6.8% of cases. Among the positive fungal culture, 50% were *Candida albicans*, 25% were *Aspergillus niger* and 6.5% each were *Aspergillus flavus*, *Candida tropicalis*, *Fusarium* sp. and *Microsporum nanum*. In a similar study by Lestringant et al.,⁷¹ the predominant fungi isolated were *Candida* sp. (57.7%), *Aspergillus* sp. in 31%, *Fusarium* sp. in 11.1%. Dermatophytes were isolated in only 4.4% of the cases.

In concordance with our study, other similar studies have reported a low frequency of dermatophyte isolation in fungal culture^{7,8} This is probably because dermatophytes are frequently not recovered from severe cases of toe web intertrigo and,

in general, recovery rate of fungi from clinically abnormal interdigital spaces ranges from 7.5% to 61%.^{42,80}

An important finding in our study was the isolation of non dermatophyte moulds (NDM) like *Aspergillus* sp. and *Fusarium* sp. in pure cultures in 37.5% of all the positive fungal cultures. Though commonly considered as contaminants, they have been reported to colonize damaged tissues and cause secondary tissue destruction.⁸¹

Also several reports in literature have described toe web intertrigo resulting from *Fusarium* sp.^{51, 71, 82} in concordance with the present study.

CONCLUSION

CONCLUSION

Foot intertrigo is a chronic and troubling disorder which occurs due to a complex interplay between various local predisposing factors, host factors, bacterial and fungal organisms. It can be simple intertrigo, where only mechanical and physical factors play a role or it may be primarily or secondarily infected by various bacterial and fungal pathogens resulting in mixed toe web infections.

Clinically, although foot intertrigo can be mild and asymptomatic, it can progress to potentially life threatening complications like cellulitis. Thus, early diagnosis and prompt treatment is essential. Also, clinical manifestations can be similar in majority of the cases with maceration being the most common presentation, hence, diagnosis cannot be made based only on clinical features, making appropriate microbiological studies imperative.

Dermatophytes, although most often are responsible for initiating the damage to stratum corneum, they are not frequently isolated from the interdigital intertriginous lesions, hence patients do not respond to antifungal treatment alone. Bacterial and fungal overgrowth is imminent under suitable conditions like occlusive environment, moisture, heat, friction along with a damaged stratum corneum.

Therapeutic approaches should not concentrate solely on antifungal therapy as often prescribed by the clinicians, but should address the predisposing factors, preventive measures along with appropriate topical or systemic antibacterial and antifungal agents.

Recommendations for further work:

- Long term multi centric prospective studies are needed to evaluate the response to therapy, recurrence, long term complications associated with foot intertrigo and its management.

SUMMARY

SUMMARY

- A total of 115 patients with clinically diagnosed foot intertrigo who presented to the department Dermatology R. L. Jalappa hospital and research centre, attached to Sri Devaraj Urs Medical College, Tamaka, Kolar between January 2013 and June 2014 were enrolled in the study.
- Detailed history was taken, thorough clinical evaluation and relevant laboratory investigations were done.
- Among 115 cases, 58 were males and 57 were females
- The youngest of study subject was 4 year old and eldest was 80 year old. Mean age in the study group was 44.71 ± 17 years. It was most common in the age group 41 to 60 years.
- Among the subjects with foot intertrigo, 39 were housewives, 22 were agriculturists and 13 skilled workers. Students, maids and unemployed subjects were 9 each. Daily wage labourers and businessmen were 8 and 6 respectively.
- Onset was acute in 26 and insidious in 89 cases.
- Duration of disease was less than 6 months in 32, 6 months to 1 year in 12, 1 year to 2 years in 27, 2 years to 5 years in 31, and more than 5 years in 13 of them.
- There were no predisposing factors noted in 23 cases. 1 predisposing factor seen in 73, 2 factors seen in 17 and 3 factors in 2 cases.
- Most common predisposing factor was exposure to wet conditions seen in 57. occlusive foot wear was noted in 22, poor hygiene in 13, hyperhidrosis in 10, foot deformity in 5, obesity and immobility in 4 each and 1 person was athlete.

- Of 115 cases, 46 were asymptomatic. Itching was most common symptom seen in 42. Pain was noted in 23, burning sensation in 15 and scaling in 10.
- Most common associated condition was diabetes mellitus in 28 followed by superficial dermatophytosis in 22 cases. Intertrigo at other sites were seen in 11 and eczema in 10 cases. Cellulitis, a complication of Intertrigo, was seen in 5 cases.
- Maceration was the commonest morphological presentation seen in 83 cases, followed by scaling in 44, discharge or exudation in 16. Fissuring was seen in 12 and crusting was seen in 8 cases
- Foot intertrigo was bilateral in 62 and unilateral in 53 people. Most commonly affected toe web space was 4th toe web space followed by 3rd toe web space.
- Whitish discoloration of lesions as seen in 56 cases, erythema in 34 cases, hyperpigmentation was seen in 7 people, greenish discoloration in 4.
- On KOH mount, 25 samples were positive for KOH mount of which 16 showed hyphae and 9 showed yeast cells.
- Fungal culture showed growth in 16 cases of which *Candida albicans* was isolated in 8 cases, *Aspergillus niger* was isolated in 4 cases and *Candida tropicalis*, *Microsporum nanum*, *Aspergillus flavus* and *Fusarium* species were isolated in 1 sample each.
- Bacterial culture was positive in 100 cases. Commensals like Micrococci and CoNS were excluded. Gram positive bacterial pathogens were isolated in 31 cases and Gram negative in 58 cases. Most commonly isolated organism was MSSA in 24 cases. *P.aeruginosa* was isolated in 14 cases, *Acinetobacter* in 12, *E coli* in 10

- and Klebsiella spp. in 9.
- In total of 34 mixed infections, concomitant infection with two or more gram negative organisms constituted 13 cases of mixed infections. Gram positive with Gram positive infection was seen only in 1 case.
 - Concomitant infection of fungal with gram positive bacteria was seen in 8 cases and fungal with gram negative bacteria seen in 9 cases.
 - Most common type of Intertrigo was **mixed toe web intertrigo** seen in 34 cases, followed by **single organism bacterial intertrigo** in 32 cases. **Simple intertrigo was seen in 29 cases, isolated fungal infection** was seen in 8 cases, **contact dermatitis** was seen in 6 cases. Miscellaneous causes of intertrigo included 3 cases of **soft corn**, 2 cases of **palmo-plantar psoriasis**, 1 case of **scabies**.

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ANNEXURES

PROFORMA

Date:

Name:

Age:

Sex:

Address:

OP/IP no:

Occupation:

Presenting complaints and duration:

- Skin lesions
- Erythema/redness
- Itching
- Burning
- Pain
- Discharge
- Constitutional symptoms
- Others

History of presenting illness:

SKIN LESIONS

- Flat/Elevated
- Onset: Sudden/Insidious
- Progression: Rapid/Gradual
- Initial site of onset
- Skin lesions anywhere else on the body
- Aggravating/Predisposing factors:
 - Trauma
 - Hyperhidrosis
 - Type of footwear:
 - Frequent bathing habits
 - Swimming pool /common baths
 - Exposure to wet conditions
 - Poor hygiene
 - Athletic or recreational activities
 - H/O Immobility of limbs
 - Splints / brace / artificial limbs
- Relieving factors
- Other Associated diseases:

TREATMENT HISTORY: Topical:

Systemic:

FAMILY HISTORY: H/O fungal infections in contacts

PAST HISTORY: H/O similar complaints in the past

PERSONAL HISTORY:

GENERAL PHYSICAL EXAMINATION:

BUILT: well/moderate/poor

NOURISHMENT: well/moderate/poor

OTHERS: Pallor / icterus / clubbing / cyanosis / Pedal edema / Lymphadenopathy

Nails:

Mucous membranes:

CUTANEOUS EXAMINATION:

- **SITE-** Toe web spaces only: 1st 2nd 3rd 4th Unilateral/Bilateral
 Extension to dorsum
 Extension to plantar surface
- **PRIMARY LESIONS-**
 Macule Vesicle
 Papule Bulla
 Plaque Pustule
- **SECONDARY CHANGES**
 Scaling Exudation
 Maceration Crust
 Erosions/Ulcer
- **MORPHOLOGY**
 Borders- well defined/ ill defined
 Color- Erythematous / Pigmented / White
- **ANY DEFORMITIES OF FOOT / CROWDING OF TOES**
- **OTHER CUTANEOUS LESIONS ELSE WHERE:**

SYSTEMIC EXAMINATION:

PROVISIONAL DIAGNOSIS:

INVESTIGATIONS:

- **RBS**
- **WOOD'S LAMP EXMN**
- **KOH EXAMINATION**

- **GRAM STAIN**

- **BACTERIAL CULTURE**

- **FUNGAL CULTURE**

FINAL DIAGNOSIS:

Consent:

I, _____, have been explained the need for the study and possible interventions and investigations necessary by the doctor in the language I fully understand.

I understand that my photographs or details may be used in the study, which may be published at a later date. I willfully give consent to be a part of the study.

Date

Signature

KEY TO MASTER CHART

Sex	M	Male	Complaints	AS	Asymptomatic	Secondary lesions	M	Maceration
	F	Female		I	Itching		S	Scaling
OCCUPATION	HW	House wife		B	Burning		ER	Erosion
	AGRI	Agriculturists		P	Pain		DC	Discharge
ONSET	A	Acute	Associated Disease	S	Scaling		CR	Crusting
	I	Insidious		DM	Diabetes mellitus		FZ	Fissuring
Predisposing Factors	HH	Hyperhidrosis		T.Corporis	Tinea Corporis	Borders	U	Ulcer
	PH	Poor Hygiene		T.Pedis	Tinea Pedis		EX	Exudate
	OB	Obesity		T.Cruris	Tinea Cruris	colour	WD	
	W	Wet conditions		CD	Contact Dermatitis		ID	
	IM	Immobility		CICD	Cumulative irritant contact dermatitis		WH	white
	OF	Occlusive footwear		HKD	Hyperkeratotic Dermatoses		ERY	erythema
Prior Treatment	TAB	Topical antibiotic	Site			KOH	GR	Green
	TAF	Topic antifungal		L1	Left 1st toe web space		HP	Hyperpigmentation
	TS	Topical steroid		L2	Left 2nd toe web space	Gram stain	H	Hyphae
	OAB	Oral antibiotics		L3	Left 3rd toe web space		YC	Yeast cells
	AM	Alt Medicine		L4	Left 4th toe web space			
Past History				R1	Right 1st toe web space		PC	Pus cells
	DM	Diabetes Mellitus		R2	Right 2nd toe web space		GPC	Gram positive cocci
	SC	Similar complaints		R3	Right 3rd toe web space		GNB	Gram negative bacilli
	HRFT	Hansens released from treatment		R4	Right 4th toe web space		GPB	Gram Positive bacilli
	HMDT	Hansens on MDT		XPS	Extending to plantar surface			
			Primary Lesions	XDS	Extending to Dorsal surface			
				MC	Macule			
				PL	Plaque			
				PP	Papule			
				VS	Vesicle			
				PU	Pustule			
				BL	Bullae			

SI No.	Name	Sex	Age	OP/IP Number	Occupation	Onset	Predisposing	Treatment prior	Past history	HOPI		Associated disease	Site
										Complaints	Duration(mth)		
1	Kadar Pasha	M	60	849035	Shop keeper	I	HH, PH		AS		6	Erythrasma	L4
2	Manjunath	M	32		Electrician	I	IM	TAB,OAB	DM	B	12	DM,LCV	L4 R4
3	Sugunamma	F	63	837056	HW	I	W	AM	SC	I	24		L3 L4 R3 R4
4	Shankrappa	M	38	895282	AGRI	I	W		AS		72	LDS,STASIS DERMATITIS	R3
5	Suresh kumar	M	30	895582	PRIEST	I	W		AS		4	T.corporis	R4
6	MANJULA DEVI	F	50	899940	HW	I	W	TAF	DM	B	84	DM	L3 L4 R3 R4
7	SWARNA	F	44	899872	HW	I				B	36	HTN, SJS-TEN	L3 L4 R3 R4
8	GOWRAMMA	F	45	900148	HW	I	W		AS		18		R2 R3 R4
9	VENKATARATHNAM	F	29	901819	MAID	I	W		AS		24		L4 R4
10	SUBBANNA	M	42	902890	AGRI	A	W	TAF	SC	I, S	6	T.CORPORIS PEDIS	L4 R4
11	RAMAKKA	F	65	877007	HW	I	PH, IM		CA.	AS	30		L4
12	NARAYANA SWAMY	M	80	907857	AGRI	I			DM	AS	60	LIPDERMATOSCLEROSIS DM	L4 R4
13	SUNDAR RAJ G	M	75	K22617	RETIRED	I				AS	36		L4 R4
14	SHANKAR NARAYAN	M	52	871106	AGRI	I	W	TAF	SC	I	8	T.CORPORIS, CRURIS, PEDIS	L3 L4 R3 R4
15	SHARADA IYER	F	47	845130	TEACHER	I	W	TAF	DM,SC	I	120	DM	L4 R4
16	VENKATARATHNAM	M	66	916217	AGRI	I	W, FD		DM	I	48	DM	L4
17	RAVINDRA BABU	M	35	916318	AGRI	A				I	0.5		L2 L3 L4 R2 R3 R4
18	KARTHIK REDDY	M	21	767202	STUDENT	I	OB, OF			P	2		L1
19	IQBALUNISSA	F	58	K5342	HW	I	OB, W		DM	AS	12	DM	L1
20	SYEDA KUBRA	F	27	782764	HW	I				P	18		L3 L4 R3 R4
21	NIRMALA BAI	F	42	874869	HW	I	W	TAF		AS	24	HKD	R 3 R4
22	JAYAMMA	F	58	919468	HW	I	PH, W	AM, TAB		AS	8	PARONYCHIA	L3 L4 R3 R4
23	DR.KUMAR	M	40		DOCTOR	I	OF	TAF		AS	1		R4
24	NARASIMHA N	M	38	924450	AGRI	I	W	TCS		AS	12	STASIS DERMATITIS	L4 R4
25	ADINARAYANA SWAM	M	85	929261	RETIRED	I	IM, W			I	18	INFECTED ECZEMA	L4 R4
26	SATISH R	M	39	930062	TEACHER	I	OF, HH			P	12		L4 R4
27	VEERABHANDRAIAH	M	47	900133	AGRI	I	W			P	24	HKD	L3 L4 XPS
28	SHYAMALA T V	F	23	931070	HW	I	W			I	19		L3 L4 R3 R4
29	VENKATARAJU	M	24		PROFESSIONA	I	OF,	TAF		I	26	T.PEDIS	R1 R4
30	BHAGYALAKSHM	F	40	866861	HW	I	W		AS		24		L3 L4 R3 R4
31	SHYLAJA	F	26	934836	STUDENT	I	OF,			I	24		L3 L4 R3 R4
32	POORNIMA	F	14	935234	STUDENT	I	OF, HH		AS		5		L4 R4
33	NARAYANAPPA	M	60	925256	AGRI	I	PH		HRFT	I	36	TROPHIC ULCER	L3 L4 R3
34	NARAYANA SWAMY B	M	45	942108	AGRI	I		TAF	DM	AS	18	STASIS DERMATITIS, DM	R3
35	PADMAMMA	F	30	945923	MAID	I	W			I	18	T.CORPORIS	R3 R4

36	AYAZ PASHA	M	28	945936	MENT WORK	I			SC	I	12	INFECTED ECZEMA	L3 L4 R3 R4 XDS
37	RATHNAMMA	F	50	946659	HW	A	W			I	7	DLE T.PEDIS	R4 XPS XDS
38	CHOWDAMMA	F	60	939160	HW	I	W		SC	AS	36	ONYCHOMYCOSIS	LL1 L2 L3 L4 R1 R2 R3 R4
39	KRISHNAPPA	M	70	947445	AGRI	I	W		HRFT	AS	48		L4
40	PRAMILA	F	35	K48011	HW	I	W	TAF		AS	24		R4
41	MUNISHWARAMMA	F	50	946956	MAID	I	W			AS	24	FINGER WEB INTERTRIGO,CICD	L4 R4
42	AMBAREESH	M	26	948617	WAITER	I	W, OF			I	120	HKD	L4 R4 X SOLE
43	KRISHNAMM	F	68	947304	HW	I				I	60	XEROSIS	L4
44	VENKATESH MURTHY	M	56	952307	AGRI	I	W			I	180	ONYCHOMYCOSIS	L3 L4 R3 R4
45	SRIDEVI B P	F	53	952802	HW	I	W			I, S	60	PSORIASIS	R2
46	SRIRAMAPPA	M	57	952841	AGRI	I			HTN	I	12	T.CORPORIS, T.FACIAE, FINGER WEB INTERTRIGO,HTN	R2 XPS
47	VENKATESHAIAH	M	42	953834	MENT WORK	I	W	AM		I	40	T.CRURIS, CD	L4 R4
48	NAGARAJ	M	53	957593	AGRI	I			DM	AS	120	DM, CELLULITIS	R3 R4
49	ANANDA KUMAR	M	47	930425	PRIEST	I	W, OF			AS	120		L4 R3
50	PARVATHAMMA	F	52	955104	HW	I	W			AS	48		L4 R4
51	PADMA	F	65	963116	HW	I	W		HTN	I	39	STASIS DERMATITIS	L3 L4 R1 R2 R3
52	SHANAZUNISSA	F	48	963240	HW	A	PH	TAF,TCS		I, S	1	T.CORPORIS	L3 L4
53	PADMAVATHI	F	48	886241	HW	A	W			P	2	DIABETES-NEW	R4
54	NETHRA M	F	20	971959	HW	A	W			I	1	CICD	L1 L2 L3 L4 R1 R2 R3 R4 X PS
55	SINDHU S	F	37	974260	HW	A	W			P, B, I	6		R3 R4 XDS
56	BEERANNA	M	59	976161	POLICE	I	OF		HTN	P	120	HTN	L4 R4
57	RUKMANGADHA	M	30	942812	BUSINESS	I	OF, HH			AS	126		L4 R4
58	NAYEEM TAJ	F	42	980686	HW	I	PH	TAF		I	18	T.CORPORIS, T.PEDIS(INTERDIGITAL)	L1 L2 L3 L4 R1 R2 R3 R4 X PS XDS
59	SAVITHRAMMA	F	50	978181	MAID	A			DM	AS	0.3	DM BL	L2 L3 L4 X DS
60	NARAYANA SWAMY	M	45	942502	DRIVER	I	OF		DM	AS	24	LIPDERMATOSCLEROSIS DM	R3
61	GOPAMMA	F	56	981790	HW	A			DM	AS	3	DM	L4 R4 X DS
62	JEEVENDRA	M	25	964389	JNEMPLOYED	I	W, PH		MRtd	B, P	24	FINGER WEB INTERTRIGO, MR	L1 L2 L3 L4 R1 R2 R3 R4 X PS
63	VENKATA REDDY	M	60	983869	AGRI	I	W			AS	18		L4 R4
64	SUBRAMANI	M	40	986057	MENT WORK	I	W		HRFT	AS	60	HANSENS-RFT, TROPHIC ULCER	R1 R2
65	VENKATARAMANAPP	M	86	K5687	RETIRED			TAF	DM,SC	P, F, SWELLING	0.2	DM, CELLULITIS	L3 L4
66	KAMALAKSHI	F	30	980283	HW	A			DM	B	3	GESTATIONAL DM	R4
67	MD MUNAWAR	M	60	980652	TIRED TEACH	I	OF		DM	AS	36	DM, PLANTAR KERATODERMA	L1 L2 L3 L4 R1 R2 R3 R4 X PS
68	GANGAMMA	F	32	989856	HW	I	W			AS	12		L4 R3 R4
69	SHASHIKALA	F	37	989803	TEACHER	I	W, HH		DM	I	24	FINGER INTERTRIGO, DM	L3 L4
70	GOWRAMMA	F	38	814450	MAID	I	W		DM	AS	60	DM, FINGER WEB INTERTRIGO	L3 L4 R4
71	LAKSHMAIAH	M	80	982480	AGRI	I				I	72	SEBORHOEIC KERTOSIS	L3
72	BORE GOWDA	M	46	989190	CLERK	I	OF		DM	I, B	48	DM	L3 L4 R4
73	PEDDA REDDY	M	35	990775	AGRI	I	W			AS	24	CORN	L4
74	KRISHNAMMA	F	38	824397	MAID	I	W			AS	40	T.CRURIS, FZ FEET	L1 XPS
75	GIRIJA	F	27	991124	HW	I	HH	AM		AS	24	CD	L4 R4
76	RAMAPPA	M	60	991574	RETIRED	I	FD		HMDT	AS	40	HANSENS ON MDT, TROPHIC ULCER	L1 L3
77	RATHNAMMA	F	60	991575	MAID	I	W			AS	48	PHN	L4 R4

78	HEMANT RAO	M	13	992487	STUDENT	A	OF			I	1	T.PEDIS	L2 L3 L4 XPS
79	ANUSHA	F	18	992730	STUDENT	A	OF, HH	TAF		B, P	1	PARONYCHIA	R4
80	LEELAVATHI	F	45	970800	MAID	A	W	TAF		I, S	2	T.CORPORIS PEDIS	L3 L4 R3 R4 XPS
81	CHANNAMMA	F	56	992070	HW	I	W			P, I	36	NEW DM, INGUINAL INTERTRIGO	L3 L4 R3 R4
82	PREMAKUMARI	F	56	994654	HW	A	PH			P	6	INGUINAL INTERTRIGO	L3 R3 R4
83	JYOTHI MARY KURIYA	F	22	965957	STUDENT	I	OF			I, S	24	CICD	L1 R1 X PS
84	MUNIRAJU	M	48	994284	MENT WORK	I		AM		I	36	INFECTIVE ECZEMA	L3 XDS
85	MUNIRAJAPPA	M	44	997233	LABOURER	A	PH			B	6	PYODERMA	L4
86	KANNAIAH SHETTY	M	65	898029	RETIRED	A	OF		DM HTN	P	1	DM HTN	R4
87	ASHWATHAMMA	F	56	997889	HW	A	W		DM	P, I	0.5	DM,GROIN INTERTRIGO	L3 L4
88	BHADRACHALAM	M	58	999474	AGRI	A	W			I, S	6		L4 R3 R4
89	MD GHOUSE	M	49	999445	BUSINESS	I	OF, HH, ATH			AS	48		L4
90	GULNAZ BEGUM	F	50	1002275	HW	I	W			I, S	180	ECZEMA, FINGER INTERTRIGO	L3 L4 R2 R3 R4 XPS XDS
91	UBBAVI	F	5	1004110	STUDENT	I	W,PH		ATOPY, SC	I, S	18	ECZEMA	L3 L4 R3 R4 XDS
92	SOMU	M	27	998031	LABOURER	A	PH	TS	SC	I, B	1	T.CORPORIS, T.PEDIS	L1 L2 L3 L4 R1 R2 R3 R4
93	SRIRAMULU NAIDU	M	70	1004518	RETIRED	I			HRFT	AS	180	HANSENS-RFT, HKD	L3 L4 R3 R4 X SOLE
94	VIKAS	M	21	1008000	WATCHMAN	A	OF HH			AS	5	PITTED KERATOLYSIS, PYODERMA	L3 L4 R3 R4 XPS
95	ABEEBUNISSA	F	50	997576	HW	A	W		VITILIGO	AS	2	VITILIGO	L4 R4
96	MUNI REDDY	M	50	1003430	AGRI	I	W		DM	P, B	18	DM, DIABETIC FOOT, DIABETIC BL	R1 R4
97	DEVARAJ	M	43	1008822	BUSINESS	A	OF			AS	1	CORN, LSC, NEW DM	L4 R3 R4
98	ANJANAPPA	M	50	1009438	AGRI	I			DM	AS	24	DIABETIC FOOT, DM BULLA, CELLULITIS	R3 R4
99	NIDARSHAN REDDY	M	4	1010905	STUDENT	A	PH			I	2	SCABIES	L2 L3 L4 R2 R3 R4
100	BHAGYALAKSHMI	F	70	1015085	HW	I	W			AS	48	ONYCHOMYCOSIS	L4 R2 R3 R4 XPS
101	LAKSHMI	F	36	N1034	HW	I			PSORIASIS	S	24	PSORIASIS	L4
102	ABIDA JOHN	F	53	N13024	HW	I	OB	TAF	SC	I, S	40	T.CORPORIS, NEW DM	L4 R4
103	MAMATHA	F	30	N11758	TEACHER	I	HH	TSA		P	28	HKD	L3
104	ARAVIND KUMAR	M	39	989828	CLERK	I	OF		DM	P	36	DM	L3 L4 R3
105	ZARINA BEGUM	F	40	N13890	HW	I	W		SC	P, B	144	NEW DM	L1 L2 XPS
106	KRISHNA SHETTY	M	54	N13940	AGRI	A			DM	B, P	3	DM, GROIN INTERTRIGO, LEFT LEG CELLULITIS	L3 L4 R3 R4
107	SRIHANUMAPPA	M	75	N3645	RETIRED	I				AS	48		R4
108	LUDHIYA	F	6	N19521	STUDENT	A	PH			P	1.5	PYODERMA	L1 L2 XDS XPS
109	MUNAWAR SULTANA	F	43	N15731	HW	I	W	TS, SA		I, B	4	T.PEDIS	L1 L2 L3 L4 R1 R2 R3 R4 X PS
110	HEMNARAYAN	M	27	N16706	SALESMAN	I	OF			AS	36		L4
111	SHAIKH ABDUL	M	24	N21454	BUSINESS	A				B	2	FURUNCULOSIS, ORAL CANDIDIASIS	L2 L3
112	PADMAMMA	F	45	1018528	HW	I	W, IM, OB	TAF		P	40	VARICOSE VEINS	R4
113	HEMAVATHI	F	49	N25314	HW	I		TAF TS		B, P	12	GLUTEAL INTERTRIGO	L3 L4
114	VENKATESHAPPA	M	60	N37947	AGRI	I	W			P	12	CELLULITIS	L4 L3
115	RAJINI	F	20	N32376	MAID	I	W			I	24		L4 L3 L2 L1 R4 R3 R2 R1 XPS XLS

Examination												
Primary lesions	Secondary Changes	Borders	Color	Deformities/Crowding	RBS	Woods Lamp	KOH	Fungal Culture	Gram stain	Bacterial culture		FINAL DIAGNOSIS
MC	S,	WD	HP		84	CRIF			PC,GPB,GPC	MSSA		BACTERIAL FI
PL	M	ID	Wh		160				PC,GPB,GPC	MRSA, K.PNEUMONIAE SS PNEUMONIAE		MIXED TWI
PL	S, ER	WD	Ery		78				GPC	MICROCOCCI		CD
PL	M,	WD	Wh		82				GPB	MICROCOCCI		SIMPLE FI
PL	M	wd	wh		103	H	<i>A. niger</i>	PC,GPB, GPC	MSSA			MIXED TWI
PL	M	WD	GR		137/201				PC,GNB,GPB,GP	P.AERUGENOSA, ENTEROBACTER SPP.		MIXED TWI
VS	ER, EX	ID	ERY		108				PC,GNB,GPB,PC	P.AERUGENOSA, P.MIRABILIS		MIXED TWI
PL	M,	WD	WH		98				GPB GPC	MICROCOCCI		SIMPLE FI
PL	M	ID	WH		70				GNB GPB PC	E.COLI ACINETOBACTER SPP.		MIXED TWI
PL	S	ID	ERY		94				GNB PC	PROVIDENCIA STUARTII(AMP-C PRODUCER)		BACTERIAL FI
PL	S,	WD	HP		108				GNB GPC	P.AERUGENOSA		BACTERIAL FI
PL	M	WD	WH		162/198				GNB , PC	P.AERUGENOSA, ACINETOBACTER		MIXED TWI
PL	M	ID	WH		120				GPB GPC	CONS		SIMPLE FI
PL	S	ID			100	H	<i>A. niger</i>	GPC GNB		K.OXYTOCA, CITROBACTER KOSERI		MIXED TWI
PL	M	WD	WH		96/128				GPC	MICROCOCCI		SIMPLE FI
PL	M	ID	WH	+	140/190				GNB GPB PC	ENTEROBACTER (AMP-C)		BACTERIAL FI
PL	M	ID	ERY		93				PC,GPC	MICROCOCCI		SIMPLE FI
PL	M	WD	WH		84				GPC	CONS		SIMPLE FI
PL	M, S	WD	HP		119/194					NO GROWTH		SIMPLE FI
PL	M,	ID	ERY						PC GNB	K.PNEUMONIAE SS PNEUMONIAE, ACINETOBACTER, ENTEROBACTER		MIXED TWI
PL	M	ID	ERY		76				GNB PC	K PNEUMOIAE PNEUMONIAE(AMP- C), ACINETOBACTER		MIXED TWI
PL	M	WD	WH		105					NO GROWTH		CD
PL	M	WD	WH		108				GNB PC	P.AERUGENOSA		BACTERIAL FI
PL	S,	ID			93				GPC	CONS		SIMPLE FI
PL	S, M	ID	WH		88				GNB PC	E COLI(AMP-C)		BACTERIAL FI
PL	M	WD	WH		75				GPC PC	CONS		SIMPLE FI
PL	FZ	WD	ERY		118	H	<i>A.flavus</i>	GPC GPB PC	MSSA			MIXED TWI
PL	M, S	WD	ERY		84				GPC GPB GNB PC	E COLI P.AERUGENOSA		MIXED TWI
PL	M, S	ID			98	H			GPC PC	CONS		FUNGAL FI
PL	M	ID	WH		118				GPC	MICROCOCCI		SIMPLE FI
PL	M	WD	WH		114				PC,GNC	ENTEROBACTER SPP., ECOLI(ESBL)		MIXED TWI
PL	M	WD	WH		84				GPC	MICROCOCCI		SIMPLE FI
PL	M, S	WD	WH	+	130	H	<i>Microsporium nanum</i>					FUNGAL FI
PL	M	WD	WH		96/132				PC	NO GROWTH		SIMPLE FI
PL	M	WD	WH		82	H			PC GNB	ACINETOBACTER		MIXED TWI

VS	ER, S	ID	ERY,HP		90			PC GPC GNB	ACINETOBACTER	BACTERIAL FI
PL	S	ID			132	H		PC GNB	ACINETOBACTER	MIXED TWI
PL	S, M	ID			122			GPC GPB	MICROCOCCI	SIMPLE FI
PL	M	WD	WH		112			GPC	MICROCOCCI	SIMPLE FI
PL	M	ID	WH		73			PC GNB	ENTEROBACTER (AMP-C), K.OXYTOCA(AMP-C)	MIXED TWI
PL	M	ID	WH		81			GPC	MICROCOCCI	CD
PL	S	ID			78			GNB GPB	ACINETOBACTER	BACTERIAL FI
PL	M	WD	WH		96			GPB GPC	MICROCOCCI	SIMPLE FI
PL	S, M	ID	WH		103			GPC	CONS	SIMPLE FI
PL	S, FZ	WD	HP					GPC PC	MICROCOCCI	MISCELLANEOUS
PL	M, S	WD	WH		210	H			NO GROWTH	FUNGAL FI
VS, PL	S, M	WD			129				NO GROWTH	CD
PL	M, ER	WD	WH		338/441			PC, GPC	MSSA	BACTERIAL FI
PL	M	WD	WH		98			GPC	MICROCOCCI	SIMPLE FI
PL	M	WD	WH		112				NO GROWTH	SIMPLE FI
PL	M	ID	GR		100	YC	<i>C.albicans</i>	PC GPC GNB GPC	K.OXYTOCA(AMP-C) P.AERUGENOSA	MIXED TWI
PL, PP	M, S	ID	WH		98	H		GPC GPB GNB PC	K.OXYTOCA, P.AERUGENOSA, PROTEUS VULGARIS	MIXED TWI
PL	M	WD	HP		247			GPC PC	CONS	SIMPLE FI
VS, PL	S, FZ, HK	ID			96			GPC	NO GROWTH	CD
PU, PP	CR, EX, S	WD	WH		88	YC	<i>C.albicans</i>	SCANTY PC, YE	P.AERUGINOSA	MIXED TWI
PL	M, S	WD	WH		93			PC GNB	K.PNEUMONIAE SS PNEUMONIAE, P.MIRABILIS	MIXED TWI
PL	M	ID	WH		112			GPC PC	MSSA	BACTERIAL FI
PL	S	ID			86	H		GNB GPC	MSSA	MIXED TWI
BL	CR, ER, S, FZ	ID	ERY		400			GPC GNB PC	E COLI(AMP-C), ACINETOBACTER	MIXED TWI
PL	S M	ID	WH		108/140			GPC GNB PC	ECOLI, MSSA	MIXED TWI
PL	DC, M	ID	WH		100/184			GPC	ALPHA HEMOLYTIC STREP VIRIDANS	BACTERIAL FI
PL	M, DC	WD	GR		76			GNB GPC	P.AERUGENOSA	BACTERIAL FI
PL	M	ID	WH		90			GPC	CONS	SIMPLE FI
PU	ER, U, FZ	ID	WH		88			PC GPC	P.AERUGENOSA	BACTERIAL FI
PL	M, DC	ID	ERY		150/220			PC GPC GNB	P.AERUGENOSA, ENTEROBACTER SPP.	MIXED TWI
PU	M, DC		WH		90	YC	<i>C.albicans</i>	G+ BUDDING YEAST CELLS		FUNGAL FI
PL	M, S	ID			160/200	YC	<i>C.albicans</i>	GPC, G+ BUDDING	MSSA	MIXED TWI
PL	M		WH		77			GPC	MSSA	BACTERIAL FI
PL	M, DC	WD	ERY		127/238	YC	<i>C.albicans</i>	PC, G+ BUDDING YEAST CELLS		FUNGAL FI
PL	M	WD	WH		89	H	<i>Fusarium</i>	GPC GPB GNB PC	ENTEROBACTER(AMP-C)	MIXED TWI
PL	M	WD	WH		92			GPC PC	MICROCOCCI	MISCELLANEOUS
PL	M	ID	WH		128/226			GPC GPB	MSSA	BACTERIAL FI
PL	M	WD	WH		88			GPC	CONS	MISCELLANEOUS
PL	M, S, FZ	WD	WH		95			GPC	CONS	SIMPLE FI
PL	M	ID	WH		90			GPB GPC	CONS	CD
PL	U, ER	WD	ERY	+	92			GPC	MICROCOCCI	SIMPLE FI
PL	M	ID	WH		86			GPC	MSSA	BACTERIAL FI

PP	S				70	H		GPC	MSSA		MIXED TWI
PU	DC, U		ERY		76			GPC PC	MSSA		BACTERIAL FI
PL	M, S	ID			87	H		GPC PC	MSSA		MIXED TWI
PL	M	WD	ERY		102/260	YC	<i>C.albicans</i>	G+BUDDING YEAST CELLS			FUNGAL FI
PL	M,FZ	WD	GR					GPC GNB PC	E COLI ACINETOBACTER SPP.		MIXED TWI
PP, VS	S	ID	ERY		92			GPC PC	MSSA		BACTERIAL FI
PU	CR, DC	ID	ERY		81			GPC PC	MSSA		BACTERIAL FI
PU	M, DC		ERY		102			GPC PC	CONS		SIMPLE FI
PL	M, DC	ID	ERY		119/263			GPC	CONS		SIMPLE FI
PL	M, ER	WD	ERY		126/184			GPC PC	MRSA, B HEMOLYTIC STREP		MIXED TWI
PL	M, S		WH		92	H	<i>A.niger</i>	GPC PC	ENTEROCOCCUS		MIXED TWI
PL	M	WD	ERY		132			GPC PC	MICROCOCCI		SIMPLE FI
VS, PP	CR, S, DC		ERY		86			GPC PC	MRSA		BACTERIAL FI
VS, PL	S FZ	ID			78			GPC PC	MSSA		BACTERIAL FI
PL	M, DC	ID	ERY		82	H		GPC PC	MSSA		MIXED TWI
PL	M, FZ	ID		+	104			GPC	CONS		SIMPLE FI
PL, PP	M, S	WD			84			GPC	MICROCOCCI		SIMPLE FI
PL	M, S	ID	WH		93			PC	NO GROWTH		SIMPLE FI
BL, PL	U, S, DC	WD	ERY		90			GPC PC	MSSA		BACTERIAL FI
PL	M	WD	WH		130/230			GPC	MSSA		BACTERIAL FI
BL	ER, M	WD			598			GPC PC	MSSA		BACTERIAL FI
VS, PU	CR	ID	ERY		71			GPC	MICROCOCCI		MISCELLANEOUS
PL	M, S	WD	WH		130	H	<i>A.niger</i>	GNB PC	ECOLI, P.AERUGENOSA		MIXED TWI
PL	S	WD	HP		88			GPC	CONS		MISCELLANEOUS
PL	M, S	ID	WH		213			GPC	CONS		SIMPLE FI
PL	M, S	WD	WH,ERY		88				NO GROWTH		MISCELLANEOUS
PL	M, DC	ID	WH		132/183	YC	<i>C.albicans</i>	G+ BUDDING YEAST CELLS			FUNGAL FI
VS	ER, DC	WD	ERY		210			GNB PC	CITROBACTER KOSERI DIVERSUS(AMP-C)		BACTERIAL FI
PU	M, CR, GR, DC	WD	ERY		140/278			GPC GNB PC	P.AERUGENOSA		BACTERIAL FI
PL	M	WD	WH		86			GNB PC	K.PNEUMONIAE SS PNEUMONIAE		BACTERIAL FI
VS PU	ER, EX, CR	ID	ERY		70			GPC GNB PC	E.COLI, MSSA		MIXED TWI
PP PUS	S, FZ, DC	ID	ERY		98	YC	<i>C.albicans</i>	G+BUDDING YEAST CELLS	MRSA		MIXED TWI
PL	M	WD	WH		144			GNB PC	ACINETOBACTER		BACTERIAL FI
PL	ER, EX	ID	ERY		104	YC	<i>C.tropicalis</i>	G+BUDDING YEAST CELLS , GPC			FUNGAL FI
PL	M	WD	ERY		87			GPC GNB PC	ACINETOBACTER		BACTERIAL FI
PL	M	WD	ERY		98			GNB PC	E.COLI		BACTERIAL FI
PL	M	WD	WH		88			PC,GPC	MSSA		BACTERIAL FI
PL	S, FZ	WD	ERY		79			GPC PC	MSSA		BACTERIAL FI