# STUDY OF TINEA PEDIS IN DIABETIC VERSUS NON-DIABETIC PATIENTS

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## ABSTRACT

**Background :** The aim of this work is to study the clinical features and causative fungi of tinea pedis in diabetic and non-diabetic patients.

**Result :** Tinea pedis was estimated to be the second most common skin disease in the United States, after acne. Up to 15% of the U.S., population may have tinea pedis. Across Europe and East Asia, prevalence rates reach 20 %.

**Methods:** The Complete history taking regarding: age, sex, occupation, residency, history of diabetes and diabetic profile (fasting blood sugar and post prandial).and Clinical examination of the feet

**Aim of the study :** The aim of this work was to study the clinical features and causative fungi of tinea pedis in diabetic and non-diabetic patients

Conclusion : Tinea pedis is more frequent in tropical climates and may be associated with use of occlusive footwear. Males are more often affected than females. It is mostly affecting the web space between the fourth and fifth toes. Children do not often develop tinea pedis. Patients with atopic dermatitis or immuno suppressive disorders may be predisposed to developing tinea pedis


Key words : **tinea , diabetic non-diabetic**

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## INTRODUCTION

Tinea pedis is the term used for a dermatophyte infection of the soles of the feet and the interdigital spaces. Tinea pedis has afflicted humanity for centuries, so it is perhaps surprising that the condition was not described until Pellizzari did so in 1888. The first report of tinea pedis was in 1908 by Whitfield and Sabouraud, believed that tinea pedis was a very rare infection caused by the same organisms that produce tinea capitis. The first reported case of tinea pedis in the United States was noted in Birmingham, Alabama, in the 1920s. World War (I) troops returning from battle may have transported trichophyton rubrum to the United States.[1]

Tinea pedis was estimated to be the second most common skin disease in the United States, after acne. Up to 15% of the U.S., population may have tinea pedis. Across Europe and East Asia, prevalence rates reach 20 %.[2, 3]

Tinea pedis is more frequent in tropical climates and may be associated with use of occlusive footwear. Males are more often affected than females. It is mostly affecting the web space between the fourth and fifth toes. Children do not often develop tinea pedis. Patients with atopic dermatitis or immuno suppressive disorders may be predisposed to developing tinea pedis.[4]

Dr. Sabouraud was one of the first to formally categorize this group of dermatophytes and produced a remarkable tome in 1910 (Les Teignes, Libraires de l'Academie de Medicine) which describes culture of the dermatophytes in detail along with extensive photogravure plates, drawings, clinical descriptions and discussion of treatment. Although the taxonomic classification has been modified since that time this rightly earned him a central position in twentieths century medical mycology and a lasting impact in the form of Sabouraud's Agar. This peptone-sugar medium developed by Sabouraud in the late 19th century was revolutionary in enabling fungi to be grown, studied and described under standardized conditions an important advance given the way in which most macroscopic and microscopic features are influenced by the nutritional value of the growth substrate.[5]

Tinea pedis is commonly called 'athlete's foot'. Because it is known to be very common among athletes, the pathogens that cause it have been isolated on the floors of swimming pool dressing rooms and showers. Moreover, studies have shown that infections generally

occur when individuals' feet are exposed to these pathogens.[6]

## Pathophysiology

Dermatophytes can survive solely off of human stratum corneum, which provides a source of nutrition for the dermatophytes and for the growing fungal mycelia." Dermatophyte infections involve three main steps: adherence to keratinocytes, penetration through and between cells, and the development of a host response.[7]

**Table1. Mechanisms by which dermatomycosis occurs and factors involved in the defence of the body against fungi [8]**

Table:1

| |
|---|
| I. **Fungi's aggressive factors** |
| 1. Adhesive factor (ability to adhere) |
| 2. Proteinases |
| 3. Cellular wall components |
| 4. Others |
| II. **Hosts' defensive factors** |
| 1. Horny layer |
| 2. Epidermal Langerhans cells |
| 3. Keratinocytes |
| 4. Epidermis-derived basic protein components |
| 5. Neutrophils (abscess) |
| 6. Lymphocytes (in particular CD4-positive T cells, TH1 type) |
| 7. Macrophages |
| 8. Plasma cells |
| 9. Granulomatous reactions |
| 10. Mechanisms for foreign matter expulsion |
| 11. Others |
| III**. Environmental factors** |
| 1. Trauma |
| 2. UV rays |
| 3. Drugs (steroids, immunosuppressive drugs, etc.) |

## Pathogens

Three species of fungi, Trichophyton rubrum, Trichophyton mentagrophytes, and Epidermophyton floccosum are together responsible for the vast majority of cases of tinea pedis throughout the world**.** The Nondermatophyte causes include Scytalidium dimidiatum, Scytalidium hyalinum, and, rarely, Candida species. Of these keratinophilic organisms, Trichophyton rubrum is the most common pathogen associated with chronic tinea pedis, while other fungal pathogens have also been associated with the disorder .The factors affecting the transmission of these dermatophytic pathogens are dependent on the source of infection, which is usually either human (anthropophilic), animal (zoophilic) or soil (geophilic). The most common anthropophilic dermatophyte infection seen is T. rubrum. It was showed that T. rubrum accounted for over 76% of all dermatophyte infections, including tinea pedis and may account for over 2/3 of all

tinea pedis infections. The spread of infections with this pathogen have been attributed to large population movements during World War II. Outbreaks of infection of glabrous skin have been associated with contact with infected, desquamated skin scales. This may occur in military camps and in factories.[1, 9]

**Clinical syndromes**

Depending on the pathogen and anatomical distribution, tinea pedis may present in a given patient as one of several syndromes. Typically, three variants are seen and include: [9]

**1. Interdigital-type tinea pedis**

Interdigital tinea pedis is the most common type, usually manifests in the interspace of the fourth and fifth digits and may spread to the underside of the toes. Patients often complain of itching and burning sensations on the feet accompanied by malodor. There are generally two types of interdigital tinea pedis. The first is a scaly, dry type called dermatophytosis simplex.. The second type is symptomatic and presents with wet, macerated interdigital spaces..[9]

***Dermatophytosis simplex***

It describes the most uncomplicated form of interdigital tinea pedis. Because of the already existing breakdown of the skin barrier by the dermatophytes, secondary bacterial infections are common. An overgrowth of bacteria can lead to highly inflamed, macerated skin, which is termed “dermatophytosis complex". Interdigital infections was divided into two variants; dermatophytosis simplex and dermatophytosis complex. [10, 11]

**2. Moccasin-type tinea pedis**

The moccasin type is a more severe, prolonged form of tinea pedis that covers the bottom 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. Subungual onychomycosis coexisting with moccasin type dermatophytosis is most often caused by T. rubrum as well.[9]

***Trichophyton rubrum syndrome***
***(***Dermatophytosis Syndrome***)***

Trichophyton rubrum syndrome (TRS) for denomination and define the following obligatory clinical and mycological criteria for TRS. **(A)** Skin lesions at the following four sites: (1) feet, often involving soles; (2) hands, often involving palms; (3) nails; and (4) at least one lesion in another location than (1) (2) or (3), except for groins. **(B)** Positive microscopic analyses of potassium hydroxide preparations of skin scrapings in all four locations. **(C)** Identification of Trichophyton rubrum by cell culture at three of the four locations at least. For diagnosis of TRS the criteria (A) and (B) and (C) have to be fulfilled. [12]

**3.Vesiculobullous tinea pedis**

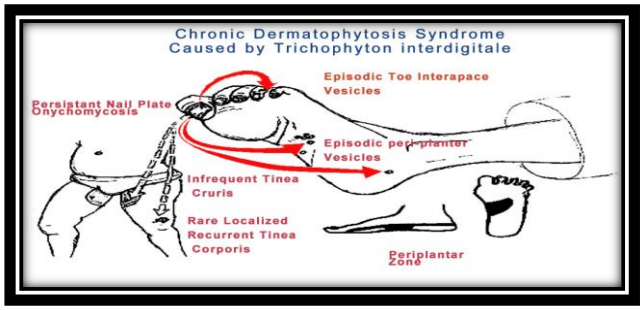
Vesiculobullous tinea pedis is the third type of dermatophyte infection of the feet. ooccasionally a pustular variant may be seen. This type comprises pustules or vesicles on the instep and adjacent plantar surfaces of the feet and is less common. Bacterial infection needs to be considered in the differential diagnosis and ruled out by microscopy and/or culture. 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. KOH preparations of the aspirate should be examined for presence of hyphae.[9]

***T. mentagrophytes (interdigitale) syndrome***

Diagram of the *T. mentagrophytes* (*interdigitale*) syndrome: this syndrome is basically a life-long tinea pedis. Superficial lesions of the surface of the nail plate are usually present. In response to foot stress (heat and occlusion), large pruritic vesicle or bullae appear in the periplantar skin (most commonly noticed in the plantar arch) and on the dorsum of the toes near the nails and the inner sides of the toes. Except in severe cases, the thick plantar skin remains free of lesions.

The arrow ribbons indicate (hypothetically) that the infection of the periplantar skin and toes may be renewed episodically from the nail plate lesions. The interrupted arrow ribbons indicate rare infection of other body sites in patients with TI tinea pedis, again (hypothetically) from nail plate lesions. The small foot diagrams show the periplantar zone in black, which is the usual site of TI vesicles. In contrast, plantar areas left white are the usual site of vesicles in *T. rubrum* tinea pedis.[13]

Figure 1[13]




**4. Acute Ulcerative Type.**

Rampant bacterial co-infection, most often from Gram negatives in combination with T mentagrophytes, produces vesiculopustules and large areas of purulent ulceration on the plantar surface. Cellulitis, lymphangiitis, lymphadenopathy, and fever are frequently associated. The vesiculo-bullous and acute ulcerative types commonly produce a vesicular id reaction, either as a dyshidrotic like distribution on the hands or on the lateral foot or toes.[7]

**Other rare types**

**Scutular favus-like tinea cruris et pedis in a patient with AIDS**

Superficial fungal infections are common in patients with HIV infection. These infections may be widespread or have an unusual form or structure Scutula are yellow cup-shaped crusts classically present in favus of the scalp.[14]

Table 2: **Predisposing Risk Factors for Tinea Pedis**.[9]

| |
|---|
| A. Host Factors |
| 1. Immunosuppression |
| a.) Chemotherapy |
| b.) Immunosuppressive Drugs |
| c.) Steroids |
| d.) Organ Transplant |
| e.) Acquired Immunodeficiency Syndrome (AIDS) |
| 2. Poorly controlled diabetes mellitus |
| 3. Obesity |
| 4. Age |
| B. Local Factors |
| 1. Trauma |
| 2. Occlusive Clothing |
| 3. Public Showering |
| 4. Moist Conditions [14] |

**Transmission of Tinea Pedis**

Transmission of tinea is affected by a number of factors including the type and

duration of exposure to the fungus and the person's degree of innate resistance. Hence, not all people are equally at risk of acquiring the infection. The presence of an inoculum in a place where feet are likely to come into contact with it is essential for the development of the infection. The most influential factor in acquiring the infection was the use of the public swimming pool, in a survey of foot ringworm in coal miners, show a definite correlation between the incidence of the disease and use of pithead baths, on patients in a long-stay hospital found that four out of nine patients who had never walked had tinea pedis. The fungus was isolated from communal socks that were laundered and distributed indiscriminately among the patients, suggesting that infected socks were a source of cross infection. The isolated active fungi from the floors of swimming baths and from the pool itself were isolated. The infection could be acquired by fragments of skin in which the fungus is embedded, or directly by the conidia and hyphae of the saprophytically growing fungus.The importance of a person's inherent resistance to infection, despite the fact that exposure to the inoculum has occurred. This group placed massive inocula in the interdigital spaces of normal feet, and then subjected the feet to warm, moist conditions favourable to the growth of the fungus. However, inducing tinea pedis in this way was unsuccessful. The condition of the feet, the type of foot-wear worn and climate are also suggested as factors antecedent to acquiring infection, Trauma has been suggested as a predisposing factor for a dermatophyte infection, Nevertheless, the presence of fungi has been found on lesion-free feet which suggests a carrier state, or that trauma may not necessarily be a prerequisite to the establishment of infection. Badly ventilated footwear is also associated with increased infection.it was found that boys had a higher rate of infection than girls, who wore lighter,

better ventilated shoes, The role of footwear in tinea infection is also apparent in the study who surveyed the crew of a nuclear submarine. The incidence of clinical disease was almost double in subjects who wore heavy 'steaming boots' (63%) compared with those who wore sandals (37%). Similarly, the occlusive effects of the soldier's army boot on the frequency of tinea pedis among soldiers in tropical climates were reported. Soldiers in high-humidity areas developed tinea pedis more frequently (85%) than those in high temperature, low-humidity areas (32%).[15, 16]

## Diabetes mellitus and fungal infections

Complications of the feet are a major cause of morbidity, mortality, and disability in persons with diabetes. A relatively common disorder, diabetes mellitus affects over 16 million people in the United States. The World Health Organization predicts that by the year 2010 the world's diabetic population will double to over 200 million people. Diabetes affects persons of all ages and in all socioeconomic segments of the population. There are over 625,000 newly diagnosed diabetics entering the health care system in the United States each year. Non-traumatic lower extremity ulcers and amputations associated with diabetes are an important and costly problem. Because of the morbidity and complexities of the disease, total direct health care costs spent for patients with diabetes are higher than for those in the general population. In the United States, patients with diabetes use health care resources at a rate 3 times higher than non-diabetic population. The average number of outpatient visits made to health care providers each year is 15.5 per patient with diabetes compared with 5.5 per patient in the general population. In addition to these direct costs, society faces indirect costs relating to diabetic complications that result in loss of productivity.[17-19]

Diabetes mellitus is a common condition, which frequently has skin manifestations. The attachment of glucose to protein may result in a profound effect on structure and function of that protein, and account for clinical manifestations of the disease.

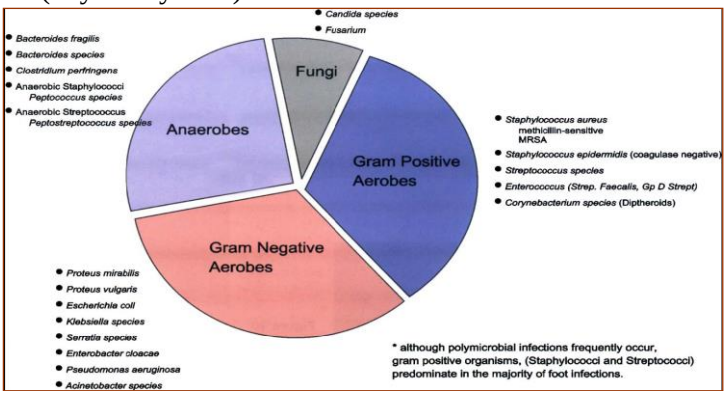
It has been suggested that increased cross-linking of collagen in diabetic patients is responsible for the fact that their skin is generally thicker than that of non-diabetics. Advanced glycosylation products are probably responsible for yellowing of skin and nails. Increased viscosity of blood due to stiff red blood cell membranes results in engorgement of the post-capillary venules in the papillary dermis, detected as erythema of the face, or periungual erythema. It is suggested that these skin changes may eventually be used as a reflection of the patient's current as well as past metabolic status.[20]

Although dermatophyte infections are probably not common in the diabetic population, they are of special concern. Toe web space infections may lead to inflammation and fissuring that can serve as a portal of entry for bacterial infection in a compromised diabetic foot. The oxygen demand of the subsequent inflammation may exceed the ability of the diabetic microcirculation, leading to gangrene. It is for that reason that tinea pedis should be aggressively managed in patients with neurovascular compromise.

The Involvement of the toe-nails by dermatophytes (onychomycosis) is common among elderly diabetics as it is in the population at large. The infection itself is of little consequence, but the nail dystrophy which results may make proper nail care more difficult for the patient. Currently the most promising (but not FDA approved) treatment appears to be itraconazole 400 mg/day for one week a month for 4-6 months.[20]

Candida infection of the web spaces usually involves the 3-4 web space of the hands or the 4-5 web-spaces of the toes. This area has a tendency to retain moisture due to occlusion from apposing surfaces of skin. Presumably, the increased sugar content of the skin encourages the establishment of this infection. The clinical appearance is a white patch of skin, often with central peeling. Toe web space involvement is often mistaken for a dermatophyte infection, but the diagnosis can be confirmed on potassium hydroxide preparation.[20]

Diabetic foot infections are generally considered polymicrobial, because multiple organisms are frequently found in a wound milieu. Staphylococcus and Streptococcus remain the most important organisms causing infection. as in figure 3.[21]




The complex effects of **diabetes and hyperglycemia**, such as **vascular damage, collagen impairment,** and **immunologic dysfunction**, manifest in an assortment of cutaneous disorders. With the increasing incidence of diabetes, primary care physicians will encounter the cutaneous manifestations of diabetes, as well as skin reactions to diabetes treatment, on a regular basis. Because these lesions may indicate poor glycemic control or be the first sign of diabetes, it is important to be able to recognize this diverse variety of skin findings.[22]

**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. Other interdigital inflammatory

conditions need to be included in the differential diagnosis and include erythrasma, impetigo, pitted keratolysis, Candida intertrigo, and Pseudomonus aeruginosa.[23]

***Paradoxically*** tinea pedis was associated with a lower risk of foot ulcer, because tinea pedis needs an intact autonomic nervous system to causes sweating and, warmth, which are .the risk factors for ulceration (figure 4).[23]

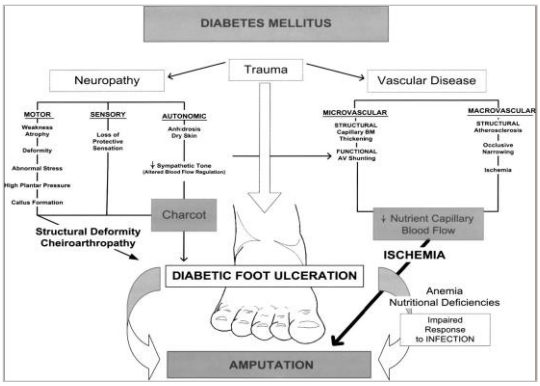



Figure 5 shows complication and risk factors for amputation in DM.[21]

Patients with diabetes seem to have diagnostic, therapeutic and preventive needs with regard to mycotic diseases of the feet that have hitherto been underestimated. The patients themselves are highly interested in these matters. In particular, the frequency of moccasin-type tinea is apparently underestimated, because of the ***low grade of inflammation*** and the ***similarity to dry sebostatic skin often seen in diabetics***. Autonomic neuropathy, such as impaired sudomotor activity, shows a high prevalence (about 60%) and may make substantial contribution to mycotic infection[24, 25]

**Diagnosis of Tinea Pedis**

**Tinea pedis diagnoses include:**

**1. Microscopic examination of scrapings.**

Potassium hydroxide preparation (KOH) Scale is obtained from the site of infection.The active border or the edge of a lesion is suitable for obtaining scale. In blistering lesions, the roof of the vesicle is an appropriate specimen. In pustular lesions, the purulent debris is appropriate. The material is placed on a glass slide and 10% to 15% KOH is added with or without dimethyl sulfoxide (DMSO). If DMSO is added to the KOH, heating is generally not necessary. A fungal stain such as Chlorazol Black E or Parker's blue black ink may be added to highlight the hyphae. A positive KOH will show numerous septate hyphae. A note whether the hyphae are non pigmented or pigmented can be made because certain non dermatophyte infections (e.g., Exophiala wemeckii) have pigmented hyphae.[26]

In vesicopustular type large pustules on his feet that contained hyphae on Gram's stain of the pus and on a potassium hydroxide preparation of the pustule roof. Cultures were negative for bacteria, but grew Trichophyton rubrum in some cases.[27]

**2**. **Fungal culture.** The standard fungal culture medium is Sabouraud's glucose agar. The addition of an antibiotic, such as chloramphenicol, inhibits bacterial overgrowth that may inhibit the growth of pathogenic dermatophytes or

nondermatophyte molds. Media containing cycloheximide are useful when selectively screening for dermatophytes. Appropriate agar choices include dermatophyte test medium (DTM), Mycosel, and Mycobiotic. DTMs have a color indicator that changes the medium from yellow to red in the presence of a dermatophyte. However, the color reaction will obscure the features used to identify the colony morphology of

many organisms. For those media in which a color change indicates the presence of dermatophytes, it is important to follow the manufacturer's instructions in terms of the number of days between culture inoculation and reading.[26]

**3. Skin biopsy.** A skin biopsy and histopathological study are rarely needed to confirm a diagnosis of tinea pedis. Fungal elements within the stratum corneum can usually be identified using periodic acid-Schiff or Gomori methenamine-silver stain but may be sparse or absent in inflammatory or interdigital tinea pedis complicated by secondary bacterial infection. Neutrophils may be noted within the stratum corneum, a finding that should prompt consideration of a dermatophyte infection. In vesicular tinea pedis, spongiotic intraepidermal vesicles are present; in the chronic hyperkeratotic (moccasin) type, hyperkeratosis and epidermal acanthosis usually are present. Both types are associated with an acute or chronic dermatitis that may contain eosinophils.[1]

**4. Confocal scanning laser microscopy (CM)**

Is a noninvasive, painless, real-time, reflectance imaging technique for the skin and other tissues A confocal microscope contains a low-power laser beam that illuminates the targeted area of tissue. Only the light reflected from a thin "section" within tissue is detected, thus allowing for excellent cellular detail that is comparable to that of histologic examination. High resolution is necessary for imaging dermatophyte hyphae; CM has the highest resolution of any live tissue imaging technique. The illumination power during CM does not produce substantial heating or tissue changes. Several recent publications have documented the appearance of different skin disorders with confocal microscopy, including allergic contact dermatitis, folliculitis, sebaceous hyperplasia, psoriasis, and nonmelanoma skin cancers. In addition, reflectance CM has been able to successfully identify hyphal structures in patients with onychomycosis.[28-31]

**6.PCR TEST:** Molecular identification using PCR for the amplification of fungal DNA from tissue is being applied to fungal diagnosis more frequently A test available for broad-spectrum detection of fungal infection is the G test, which tests for (1,3)-,B-D-glucanpresent in most fungal cell walls.. The assay is very sensitive, with a detection limit of 1 pg/ml.," Clinical studies suggest that normal individuals have (1, 3)-, B-D-glucan concentrations of <10 pg/mL in blood, with a mean value of c. 2.7 pg/mL. In contrast, patients with fungal infections typically have *(1,3)-,B-D*glucan concentrations of >20 pg/mL, thus providing a reasonable cut-off between normal and abnormal levels. The test takes 1 h, facilitating rapid diagnosis.[32]

**7. ELIZA TEST:** Commercially available ELISA kits for the detection of mannan and galactomannan, respectively, are available for Candida and Aspergillus antigens and demonstrate good specificity but variable sensitivity. used for early diagnosis invasive fungal infection[33]

**8. When a nondermatophyte mold is a possible pathogen**, as may occur in certain cases of tinea pedis and tinea manuum, media that do not contain cycloheximide are useful. For example, Scytalidium. dimidiatum and Scytalidium. hyalinum can be causative pathogens in tinea pedis and tinea manuum. These organisms generally do not grow on media containing cycloheximide; therefore, the use of non cycloheximide media can be helpful in these circumstances.[34]

**9. Studies for differential diagnosis may include the following:**

a. Bacterial culture to rule out secondary infection

b. Wood's light examination to rule out erythrasma, especially in intertriginous disease and involvement of the scrotum.[34]. An accurate etiologic diagnosis of tinea pedis depends entirely on careful and repeated bacteriologic and mycologic investigation and exclusion of such conditions as tight occlusion of the toe webs and other predisposing factors. It may be possible to suspect on clinical

grounds a pure candidal infection whenever there is a bright red base with overhanging colarette of whitish epithelium. The presence of satellites of vesicles at the periphery is a helpful feature as is also the association of candidal infection elsewhere such as paronychia, angular stomatitis, etc. An uncomplicated dermatophyte infection may be represented by mild scalying and/or erythema. Gram-negative toe web maceration may be suspected in the presence of severe boggy maceration with foul discharge. a greenish tinge of the skin of the interspaces and marked pain A greenish-blue fluorescence by Wood's light may be helpful for the diagnosis of Pseudomonas infection.[34]

## PATIENTS

Fifty diabetic patients and fifty non-diabetic patients suffering from tinea pedis were concluded in this study .The patients were selected from the dermatology and diabetic outpatient's clinics of the Alexandria main University Hospital.

## METHODS

1) **The Complete history taking regarding:** age, sex, occupation, residency, history of diabetes and diabetic profile (fasting blood sugar and post prandial).

2) **Clinical examination of the feet**

- The skin of the web examined for the presence of scaling, erythema, white maceration, erosions, fissures, oedema or discharge.
- The Signs of cellulitis or lymphangitis were looked for.
- All the other type of fungal infections in the feet was recorded.
- The lesion was uni or bilateral.
- The distribution and the type of tinea pedis.

3) **Clinical examination of the whole skin:** The skin of the whole body was examined especially for fungus infection.

4) **Research participants were divided into two groups:**

**GroupA**: included fifty non-diabetic patients with tinea pedis were subjected to:

a. Scraping test using 10%KOH
b. Fungal culture.

**Group B**: Fifty diabetic patients with tinea pedis were subjected to:

a. Scraping test using 10%KOH
b. Fungal culture.
c. fasting blood glucose and 2-hour post load glucose.

6) **Mycological study was included:**

1. Collection of samples.
2. Scraping test using 10%KOH. ***portion of the collected material was placed over a glass slide, a cover glass mounted, then 10% KOH solution was added at the edge of the cover glass allowed to diffuse beneath the cover over the sample,***

***heated gently over a low flame, then examined microscopically***

3. Fungal culture.

► to differentiate and diagnose candida albicans from others were done:

***A)Germ Tube Test(GTT)***

The germ tube test provides a simple, reliable and economical procedure for the presumptive identification of Candida albicans.

**B)Corn meal agar**

It is a relatively simple medium, consisting of an infusion of corn meal and agar. The infusion product contains sufficient nutrients to support the growth of fungal species. **Corn Meal Agar** is used for the cultivation of fungi and the demonstration of chlamydospore production.

**Corn Meal Agar** stimulates sporulation of *C. albicans*, and is useful in suppressing certain other fungal growth. Chlamydospore production is an important diagnostic characteristic used in the identification of *C. albicans*.

***C) Gram stain.*** A Gram stain of a colony grown on routine primary media (e.g. Sabouraud) reveals round to oval budding yeast-like cells or blastoconidia, measuring 3.5-7 by 4-8 µm that retain crystal violet.figure(6)

Table 3: **Comparison of Trichophyton mentagrophytes to other fungi** [35]

| Fungus | Colonial Morphology | Microscopic Morphology Phase Contrast |
|---|---|---|
| Trichophyton mentagrophytes | **Granular form**: Growth rate: moderate;<br>Texture: granular, flat; Thallus color: buff to tan<br>Reverse: pale yellow, tan, or reddish brown<br>**Velvety form**: Growth rate: moderate<br>Texture: velvety, flat, thin, with fine powder<br>Thallus color: white to sandy to butter yellow<br>Reverse: white to tan, rarely reddish brown<br>**Downy form**: Growth rate: moderate<br>Texture: deep, cottony; Thallus color: white<br>Reverse: pale yellow to tan | **Velvety and granular forms:** round microconidia in grape-like clusters spiral hyphae +/- cigar shaped, thin walled macroconidia, narrowly attached to hyphae<br>**Downy form:** pyriform microconidia indistinguishable from *T. rubrum* |
| Trichophyton rubrum | Growth rate: slow to moderately rapid<br>Texture: downy to cottony<br>Thallus color: white to pale pink<br>Reverse: blood red (PDA) to reddish brown<br>Variants: yellow and may produce red pigment.<br>coffee brown soluble pigment unpigmented<br>deeply red, heaped up, folded yellow orange reverse | **few** pyriform, lateral microconidia<br>pencil shaped macroconidia uncommon microconidia form on macroconidia arthroconidia produced from hyphae and macroconidia |
| Trichophyton tonsurans | Growth rate: moderately fast<br>Texture: suede like with radial folds, granular to woolly sometimes powdery or velvety often with abundant mycelium in the medium<br>Thallus color: white to creamy yellow, rose<br>Reverse: lemon yellow to mahogany brown, may have dark diffusing pigment | **Microconidia are numerous** characteristic varying in size and shape from long clavate to broad pyriform, are borne at right angles to the hyphae, which often<br>remain unstained by lactophenol cotton blue. Microconidia are occasionally on match stick-like conidiophores; growth enhanced by thiamine.<br>**Macroconidia are rare** or occasional smooth, thin-walled, irregular, clavate may be present on some cultures. Numerous swollen giant forms of microconidia and chlamydoconidia are produced in older cultures.<br>**Hyphae** are relatively broad, irregular, much branched with numerous septa that may be spiral shape. |

**List of Reagents and Instruments**

**Procedures**

**1. Prepare a Slide Smear:**

**A.** Transfer a drop of the suspended culture to be examined on a slide with an inoculation loop. **B.** Spread the culture with an inoculation loop to an even thin film over a circle of 1.5 cm in diameter, approximately the size of a dime. Thus, a typical slide can simultaneously accommodate 3 to 4 small smears if more than one culture is to be examined.

**C.** Air-dry the culture and fix it or over a gentle flame, while moving the slide in a circular fashion to avoid localized overheating. The applied heat helps the cell adhesion on the glass slide to make possible the subsequent rinsing of the

smear with water without a significant loss of the culture. Heat can also be applied to facilitate drying the the smear. However, ring patterns can form if heating is not uniform, e.g. taking the slide in and out of the flame.

**2. Gram Staining:**

**A.** Add **crystal violet stain** over the fixed culture. Let stand for 10 to 60 seconds; for thinly prepared slides, it is usually acceptable to pour the stain on and off immediately. Pour off the stain and gently rinse the excess stain with a stream of water from a faucet or a plastic water bottle. Note that the objective of this step is to wash off the stain, not the fixed culture.

B. Add the **iodine solution** on the smear, enough to cover the fixed culture. Let stand for 10 to 60 seconds. Pour off the iodine solution and rinse the slide with running water. Shake off the excess water from the surface.

C. Add a few drops of **decolorizer (ethanol or acetone).** so the solution trickles down the slide. Rinse it off with water after 5 seconds. The exact time to stop is when the solvent is no longer colored as it flows over the slide. Further delay will cause excess decolorization in the gram-positive cells, and the purpose of staining will be defeated.

D. **Counter stain solution (safranin)** for 40 to 60 seconds. Wash off the solution with water. Blot with bibulous paper to remove the excess water. Alternatively, the slide may be shaken to remove most of the water and air-dried.

**3. Examine the finished slide under a microscope.**

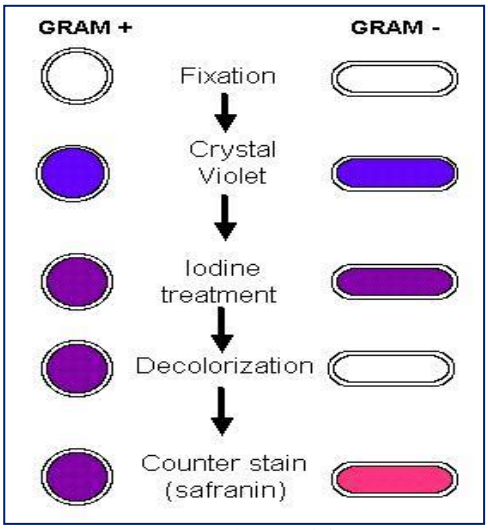



**Figure (6)**

**Statistics**

Monte Carlo test, Pearson's test and the chi square test were used for statistical evaluation, and the program SPSS15.0 was used for all statistical calculations.

## RESULTS

This study was carried out on 100 patients suffering from tinea pedis, they were divided into two groups: group I includes 50 patients (29 males and 21 females) who were non-diabetic, while the other 50 patients (25 males and 25 females) in group II were diabetic.

The following data was collected and analysis:

**1. Demographic data:**

**Table (7): Comparison between diabetic and non-diabetic cases according to demographic data**

| | Non Diabetic | | diabetic | | $\chi^2$ (p) |
|---|---|---|---|---|---|
| | No. | % | No. | % | |
| **Sex** | | | | | |
| Male | 29 | 58.0 | 27 | 54.0 | 0.162 (0.687) |
| Female | 21 | 42.0 | 23 | 46.0 | |
| **Age** | | | | | |
| 30- | 14 | 28.0 | 6 | 12.0 | |
| 40- | 22 | 44.0 | 26 | 52.0 | 4.033 (0.133) |
| 50+ | 14 | 28.0 | 18 | 36.0 | |
| **Occupation** | | | | | |
| Employed | 34 | 68.0 | 27 | 54.0 | 2.060 (0.151) |
| Non employed | 16 | 32.0 | 23 | 46.0 | |
| **Residency** | | | | | |
| Urban | 23 | 46.0 | 21 | 42.0 | 0.162 (0.687) |
| Rural | 27 | 54.0 | 29 | 58.0 | |

$\chi^2$: Chi square test


**Figure 7:** Demographic data of the studied patients group **(sex)**

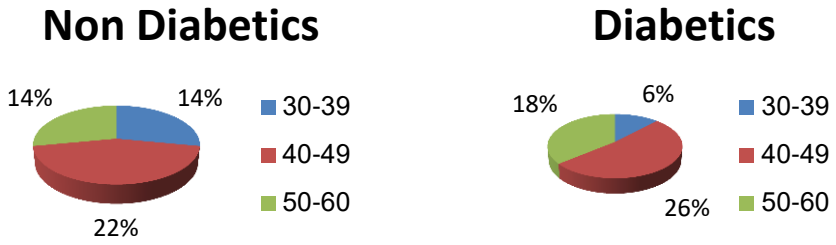



**Figure 8:** Demographic data of the studied patients group**(age)**

**e. <u>Diabetic profile</u>:**

Distribution of the diabetic cases according to diabetic profile was done. It was found that 15 patients (30%) had controlled diabetic profile, and 35 patients (70%) had uncontrolled diabetic profile. The diabetic profile was done for all diabetic patients was fasting blood glucose (normal value 80-130) and 2-hour post load glucose (normal valueupto180). (Table 8)

**Table (8): Distribution of the diabetic cases according to diabetic profile**

| | No. | % |
|---|---|---|
| **Controlled** | 15 | 30.0 |
| **Uncontrolled** | 35 | 70.0 |

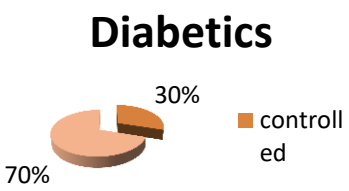



**Figure 9: Distribution of the diabetic cases according to diabetic profile**

**2. Comparison between diabetic and non-diabetic cases according to distribution and site:**

**Table (9): Comparison between diabetic and non-diabetic cases according to distribution and site**

| | Non Diabetic | | diabetic | | |
|---|---|---|---|---|---|
| | No. | % | No. | % | |
| **Distribution** | | | | | |
| Unilateral | 23 | 46.0 | 20 | 40.0 | $\chi^2$= 0.367 |
| Bilateral | 27 | 54.0 | 30 | 60.0 | p = 0.545 |
| **Site** | | | | | |
| 4th IDS | 13 | 26.0 | 10 | 20.0 | |
| 3rd , 4th IDS | 12 | 24.0 | 15 | 30.0 | |
| 3rd IDS | 1 | 2.0 | 0 | 0.0 | MCp = 0.929 |
| Planter surface of the foot | 22 | 44.0 | 22 | 44.0 | |
| The skin of the entire sole, heel and sides of the foot | 2 | 4.0 | 3 | 6.0 | |

$\chi^2$: Chi square test

MCp: p for Monte Carlo test

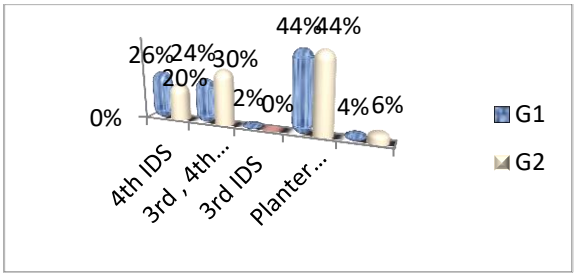



**Figure 10: Comparison between diabetic and non-diabetic cases according to distribution and site**

**3. Comparison between diabetic and non-diabetic cases according to type and causative fungi**

**Table (10): Comparison between diabetic and non-diabetic cases according to type and causative fungi**

| | Non Diabetic | | diabetic | | MCp |
|---|---|---|---|---|---|
| | No. | % | No. | % | |
| **Type** | | | | | |
| Interdigital | 26 | 52.0 | 25 | 50.0 | |
| Maccosian | 2 | 4.0 | 3 | 6.0 | 1.000 |
| Vesicular | 22 | 44.0 | 22 | 44.0 | |
| **Causative fungi** | | | | | |
| Trichphyton rubrum | 2 | 4.0 | 3 | 6.0 | |
| Trichphyton mentogrophytes | 22 | 44.0 | 22 | 42.0 | 1.960 |
| Candida albicans | 25 | 50.0 | 23 | 46.0 | |
| Candida non albicans | 1 | 2.0 | 2 | 4.0 | |

MCp: p for Monte Carlo test

**Figure 11: Comparison between diabetic and non-diabetic cases according to type**

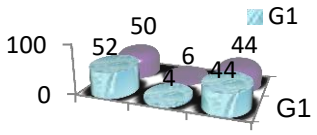
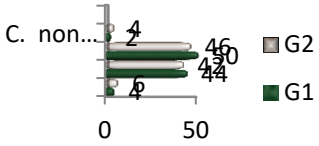



**Figure 12: Comparison between diabetic and non-diabetic cases according to Causative fungi**




**4. Relationship between diabetic profile with distribution, site, type & causative fungi:**

**Table(11): relationship between diabetic profile with distribution, site, type and causative fungi**

| | Diabetic profile | | | | Test of sig. |
|---|---|---|---|---|---|
| | Controlled | | Uncontrolled | | |
| | No. | % | No. | % | |
| **Distribution** | | | | | |
| Unilateral | 5 | 33.3 | 15 | 42.9 | $\chi^2$= 0.397 |
| Bilateral | 10 | 66.7 | 20 | 57.1 | p = 0.529 |
| **Site** | | | | | |
| 4th IDS | 5 | 31.2 | 5 | 14.7 | MCp = 0.030* |
| 3rd , 4th IDS | 5 | 31.2 | 10 | 29.6 | |
| 3rd IDS | 0 | 0.0 | 0 | 0.0 | |
| Planter surface of the foot | 3 | 18.7 | 19 | 55.7 | |
| The skin of the entire sole, heel and sides of the foot | 3 | 18.7 | 0 | 0.0 | |
| **Type** | | | | | |
| Interdigital | 9 | 60.0 | 16 | 45.7 | MCp = 0.006 |
| Maccosian | 3 | 20.0 | 0 | 0.0 | |
| Vesicular | 3 | 20.0 | 19 | 54.3 | |
| **Cusative fungi** | | | | | |
| Trichphyton rubrum | 3 | 20.0 | 0 | 0.0 | MCp = 0.011* |
| Trichphyton mentogrophytes | 3 | 20.0 | 19 | 54.3 | |
| Candida albicans | 9 | 60.0 | 14 | 40.0 | |
| Candida non albicans | 0 | 0.0 | 2 | 5.7 | |

$\chi^2$: Chi square test

MCp: p for Monte Carlo test

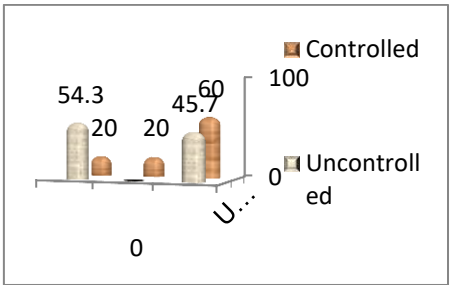



**Figure 13: Relationship between diabetic profiles and the type of tinea pedis.**

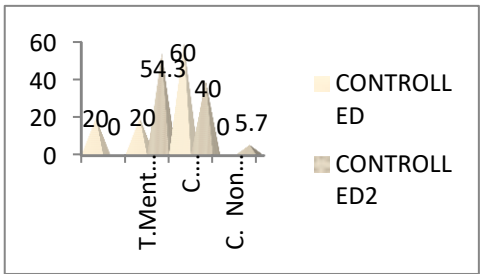



**Figure 14: Relationship between diabetic profiles and The Causative fungi.**

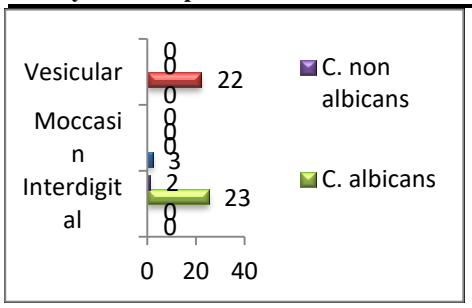



Figure 15: Relationship between the **fungi and the types of tinea pedis in diabetics.**

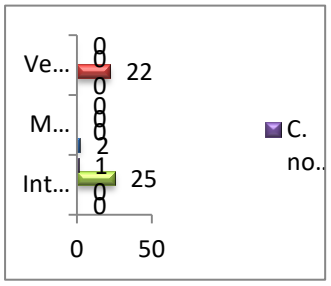



**Figure 16: Relationship between the causative** fungi and the types of tinea pedis in non-diabetics**.**

**Table (12):relationship between type and causative fungi in non-diabetic, diabetic cases and whole sample**

| | Type / Cusative fungi | Interdigital | | Moccasin | | Vesicular | | MCp |
|---|---|---|---|---|---|---|---|---|
| | | No. | % | No. | % | No. | % | |
| Non diabetic | Trichphyton rubrum | 0 | 0.0 | 2 | 100.0 | 0 | 0.0 | <0.001 |
| | Trichphyton mentogrophytes | 0 | 0.0 | 0 | 0.0 | 22 | 100.0 | |
| | Candida albicans | 25 | 96.2 | 0 | 0.0 | 0 | 0.0 | |
| | Candida non albicans | 1 | 3.8 | 0 | 0.0 | 0 | 0.0 | |
| Diabetic | Trichphyton rubrum | 0 | 0.0 | 3 | 100.0 | 0 | 0.0 | <0.001 |
| | Trichphyton mentogrophytes | 0 | 0.0 | 0 | 0.0 | 22 | 100.0 | |
| | Candida albicans | 23 | 92.0 | 0 | 0.0 | 0 | 0.0 | |
| | Candida non albicans | 2 | 8.0 | 0 | 0.0 | 0 | 0.0 | |
| Total | Trichphyton rubrum | 0 | 0.0 | 5 | 100.0 | 0 | 0.0 | <0.001 |
| | Trichphyton mentogrophytes | 0 | 0.0 | 0 | 0.0 | 44 | 100.0 | |
| | Candida albicans | 48 | 94.1 | 0 | 0.0 | 0 | 0.0 | |
| | Candida non albicans | 3 | 5.9 | 0 | 0.0 | 0 | 0.0 | |

MCp: p for Monte Carlo test

## DISCUSSION

Tinea pedis is the term used for a dermatophyte infection of the soles of the feet and the interdigital spaces.
It can be divided into 3 major categories, all of which have different pathophysiologic aspects and different etiological agents.

**1. Interdigital type.**
**2. Plantar moccasin type.**
**3. vesiculobulous type.**

It is also note-worthy that, in the present study, the bilateral distribution of tinea pedis was more common than unilateral in both studied groups (in non-diabetic and diabetic patients).
The bilateral distribution was more in diabetic patients with uncontrolled diabetic profile 20 out of 35 patients (57.1%). This in agreement with Mansour A.& Hamdi I. K.(2007)[23] that bilateral distribution as common in diabetic patients with tinea pedis. This is explained by Poor glycemic control increases the risk of infection by causing abnormal microcirculation, decreased phagocytosis, impaired leukocyte adherence, and delayed chemotaxis. Dermatophyte infections can present a significant threat in persons with diabetes. Diabetic neuropathy in the distal lower extremities creates an ideal environment for dermatophyte infections.[36]

On the contrary, Romano et al.[37] reported that the frequency of cutaneous fungal infections is not higher in diabetics as compared to healthy individuals**.** Underlying neuropathy, angiopathic changes, poor glycemic control and lack

of proper care are the factors responsible for these common infections in diabetics.

Furthermore, in our study, the interdigital type of tinea pedis was commonest type in both studied groups, the vesicular type was the second and the moccasin type was the least.

The interdigital spaces mostly 3rd, 4th was the common site for tinea pedis in both studied groups. This in agreement with study carried out by Wolff K, et al (2005)[38] and Mayser P. ,et al (2004)[39] in which they found that Interdigital tinea pedis was the most common form, involves the toe webs, especially in the third and fourth web spaces. Ingordo V, et al.(2004)[40] had demonstrated that tinea pedis affected the third and fourth clefts. Hainer BL.(2003)[42], Mansour A.& Hamdi I. K.(2007)[23] , Muhsin T.M., et al(1999)[42] , Danon, 2007[43] and Hirschmann JV. (2000)[27] were stated that interdigital type of tinea pedis was the ommonest type but usually manifests in 4th interdigital space.

In regard to study conducted in Alexandria, Zoulfakar G. Sherine (2002)[44] they found that interdigital tinea pedis was the most common form, involves the toe webs, especially in the third and fourth web spaces. Lestringant G.G.,et al (2001)[45] in UAE found that Interdigital tinea pedis was the most common form, involves a third and fourth toe-web was always involved.

It is important to note that, in our study, there is significant relationship between the site and type of tinea pedis with Diabetic profile. The causes of increased infection susceptibility in patients had DM are not yet clear. The higher blood glucose, the greater the deposit of glycated metabolites. Besides the metabolic change, other factors favoring increased infections in DM patients should be mentioned. Among them, chronic vascular and neurological complications, and impaired immune response mostly characterized by reduced neutrophil chemotaxis and phagocytosis in DM compared to non-DM individuals.[46-49] Also Ansel JC., et al. (1997)[50] and Leonhardt JM, etal (2003)[48] Stated that superficial mycoses (*tineas*) were known to cause pruritus and its absence could suggest a compromised response in DM patients. It might be due to impaired superficial innervations caused by diabetic neuropathy, a condition that predisposes infections and traumas. Bub JL., et al.[51] another contributing factor to skin lesions was the presence of macro and microangiopathy seen in DM patients. They cause vascular alterations with increased permeability and reduced vascular response to sympathetic nerve stimuli leading to a reduced ability to respond to thermal stress and/or local hypoxy.

Josh N., et al.(1999)[52] was found that epidermis invasion by spores occured when this natural barrier is compromised and deeper epidermal layers are not able to activate the immune response against infections, as both mechanisms are impaired in the skin of DM patients. Also, increased skin infections, both fungal and bacterial, were detected among type 1 and type 2 DM patients with inadequate metabolic control.

Moreover, Goldin A., et al. (2006)[53] have proved that Dysregulation of glucose, insulin and lipids leads directly to physical signs in skin of patients with diabetes mellitus. Chronically elevated blood glucose leads to non-enzymatic glycosylation (NEG) of cutaneous proteins, which eventually leads to irreversible advanced glycosylation end products (AGEs) Dysregulation of glucose, insulin and lipids leads directly to physical signs in skin of patients with diabetes mellitus. Another study conducted by Baloch G. Hussain, et al.(2008)[54] found that cutaneous manifestations of DM tinea pedis and tniea ungium and others were closely related to uncontrolled diabetes of long duration. To prove this, Buxton (Buxton et aI., 1996)[55] investigated 100 patients with type 1 diabetes mellitus and found that in well-controlled diabetics fungal infections are not commoner than in matched controls. Notably, Lugo Somolinos, et al. (1992)[56] have excluded the possibility of a relation between dermatophyte infections and blood sugar levels in a population of 100 subjects, finding that 31% of diabetics had fungal infections versus 33% of controls. The

most frequent pathology was tinea pedis (interdigital type).

The moccasin type was often misinterpreted as dry skin, which is a well-known symptom of diabetic polyneuropathy. This might explain why moccasin-type tinea pedis was misjudged even by experienced specialist physicians and chiropodists. (Gupta and Humke, 2000[57]; Farkas et aI., (2002)[58].

In immunosuppressed patients or those with diabetes and in patients with more extensive tinea pedis infections, such as vesicobullous, moccasin type, resistant infections or chronic infections, oral therapy should be considered either alone or in combination with topical treatment.[10]

The visculobullous type in our study was the 2nd common type of tinea pedis.Tinea pedis often begins in the web between the fourth and the fifth toes, and progress to involve the webs, subdigital and interdigital surfaces of the toes. Simple scaling infections was caused by dermatophyte invasion of the stratum corneum. In chronic infections of intertriginous spaces, a fissure of the toe webs with soggy macerated white epidermis may be the only clinical sign. These erosive infections are caused by selection and overgrowth of bacteria, with the production of sulfur compounds that leads to inhibition of dermatophytes and accounts for the lower recovery of fungi from the most severe and chronic cases. Chronic infections may also affect the soles, heels and lateral surfaces of the feet resulting in widespread furfuraceous scaly patches or hyperkeratotic plaques, called moccasin-type infection. Chronic plantar infections are usually caused by T. rubrum and found primarily in patients with an atopic background. Acute or subacute infections in the arch and sides of the feet due to an immune response of delayed hypersensitivity to *T. mentagrophytes*, are characterized by episodes of intense inflammation with the formation of vesicles, secondarily infected by bacteria and resulting in the formation of oedematous and painful vesicopustules.. Many predisposing factors have been suggested such as frequent use of swimming pools, poor personal hygiene, frequent washings, occlusive footwear, hyperhydrosis and physical activity, although a real association between any of these and the development of tinea pedis has never been established. However, family history or evidence of tinea pedis and onychomycosis was probably the most important predisposing factor.[59-61]

Tthis explained by Zaitz(1989)[62], seasonal variation in the frequency of *tinea pedis* apparently havingrelations with the time of diagnosis: in our study it is impossible to particularize when the patient was infected,since the onset of the lesions ranged between one week and 40 years. On the other hand, Rio Grande do Sul have well defined seasons, and at the end of the spring especially young people search for medical care, with the purpose to frequent swimming pools. However, the peak incidence in the autumn, as sure as can be, signify that the patients had been infected during the summer, resulting from the communal use of swimming baths and restroom.

In addition, Perea S.,et al (2000)[63] found that trichophyton rubrum was commonest causative fungi of tinea pedis and found that no significant relationship between factors such as concomitant diseases(diabetes, psoriasis, and vascular diseases)and tinea pedis. Eckhard M.,et al(2007)[25] found same result but found The prevalence of positive fungal samples was found to be higher for diabetic participants with less controlled blood glucose levels. Diabetic patients with dermatological foot lesions had older age, longer duration, higher Mean random blood sugar and Mean glycosylated hemoglobin. These results are in agreement with several studies.[37, 64 - 67] and denote that foot skin diseases are moreliable to occur with poor glycemic control.

Roujeau, et al (2004)[68] found interdigital tinea pedis to be a statistically significant risk factor for developing acute bacterial cellulitis. Because dermatophytic infection has also been correlated to a higher rate of gangrene and diabetic ulceration in high-risk patients, this early treatment could serve as an important prevention. Legge Bradford S., et al (2008)[69] found Tinea

pedis is most commonly caused by Trichophyton rubrum, There was no significant difference between the fungi observed in the diabetic versus non diabetic patients. Jang K.A.,et al(2000)[70] was found that Trichophyton rubrum the causative fungi of tinea pedis in Korean patients (1st,2ndIDS,sites of predilection) Occasionally, tinea pedis presents as an inflammatory vesicopustular type,which is usually caused by T.mentagrophytes.

Our results were in agreement with G.G. Lestringant et al (2001)[45] in UAE found that candida albicans and non albicans was commnest causative fungi of interdigital type of tinea pedis. It had postulated that the presence of bacteria and yeasts in toe-webs is a secondary phenomenon. Primary involvement by dermatophytes is always required to initiate the process, by damaging the stratum corneum. Subsequently, because of the antifungal properties of some bacteria, dermatophytes are unable to be cultured.[59, 60, 71] However, previous studies in other countries that share some of the UAE population's characteristics, have shown a prevalence of bacteria and *Candida* spp. over dermatophytes.[72] A similar spectrum in toe-web intertrigo has been reported in a Muslim population living in France.[73] Zoulfakar G. Sherine (2002)[44] in Alexandria found that candida albicans was the main causative fungi in interdigital type while dermatophyte main causative fungi in two other types.

## CONCLUSION

Tinea pedis in diabetic and non-diabetic patients had the following:

There was no significant difference between the two studied groups regarding age (40-49) year-old ,There was no significant difference between the two studied groups regarding sex (male),There was no significant difference between the two studied groups regarding distribution (bilateral). There was no significant difference between the two studied groups regarding site (Interdigital Spaces).There was no significant difference between the two studied groups regarding type (Interdigital). There was no significant difference between the two studied groups regarding causative fungi (Candida).

There was significant relationship between the causative fungi and the types of tinea pedis in all patients. In diabetic group, there was significant difference between the diabetic patients with controlled and uncontrolled diabetic profile. Regarding the site, type and causative fungi of tinea pedis i.e. the vesicular type (trichophyton mentagrophyte as a causative fungi )was the commonest type in patients with uncontrolled diabetic profile Those differ from diabetic patients with controlled diabetic profile that interdigital type (candida as a causative fungi)was the the commonest type.

Correlation was found with control of diabetes, or with blood glucose levels.

## RECOMMENDATIONS

The following steps should be used to prevent the development of Tinea pedis as well as to prevent reinfection:

1. Drying of the feet well, especially between the toes.

2 The use of light and airy shoes particularly in hot humid weather.

3. Wearing of cotton socks and changing them daily or more frequently if they become damp.

4. Making sure that the family is free of infection so that the members will not constantly reinfect each other.

5. Making a habit to wear slippers in steam rooms or dressing rooms of gym as chances are overwhelming that someone walking around has athlete's foot.

- More emphasis needs to be placed on foot care education and regular foot skin examinations, particularly in patients with diabetes, if foot ulcers and amputations are to be prevented in later life.

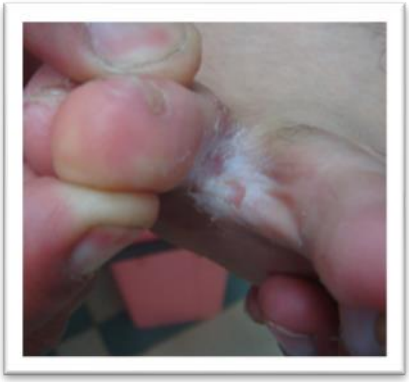


**FIGURE 16**: Interdigital type of tinea pedis

**FIGURE 17: Candida albicans on *Sabouraud's agar***

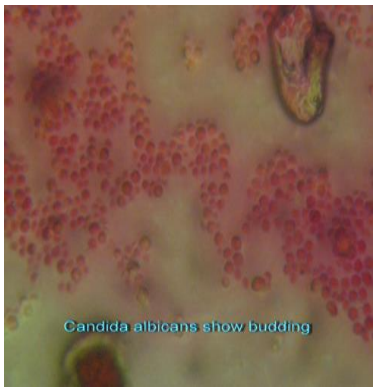



**FIGURE 18 Candida albicans**

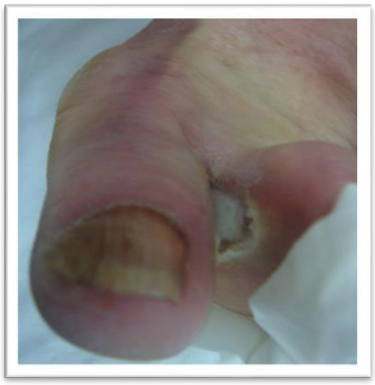


**FIGURE 19 : Candida albicans on : *Sabouraud's agar***

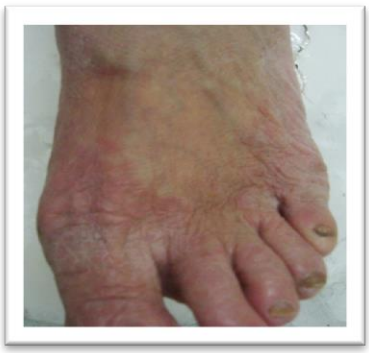


**Figure 20**: Moccasin Tinea Pedis

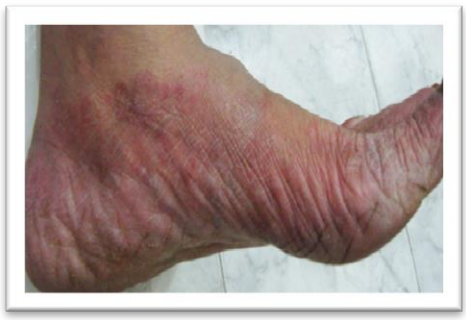
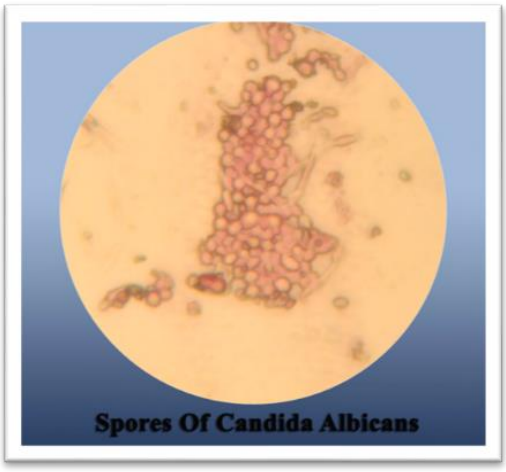


**Figure 21: T. Rubrum on *Sabouraud's agar***



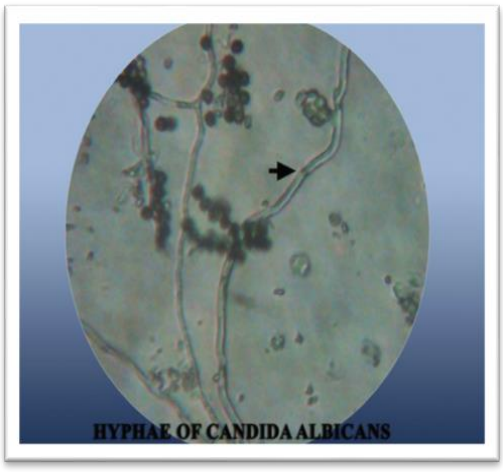



**Figure 22: spores of candida albicans**


**Figure 24: Germ Tube Test of Candida Albicans**

**Figure 23: Hyphae Of Candida Albicans**

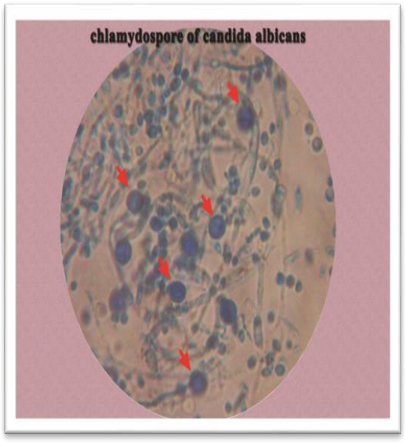



**Figure 25 : chlamydospores of candida albicans**


**Figure 23**


**Figure 24**

**Figure26**:T. Mentagrophyte on Sabouraud's aga r

T. Mentagrophyte on Sabouraud's agar

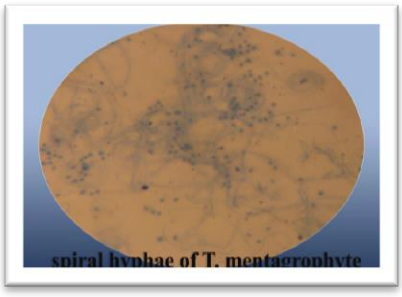
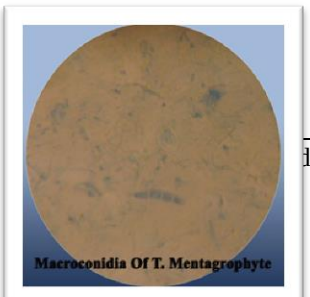
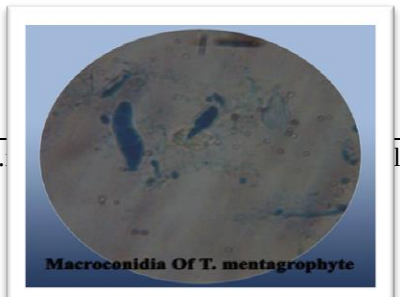



**Figure 27**


Figure 28





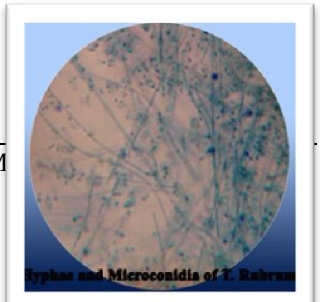
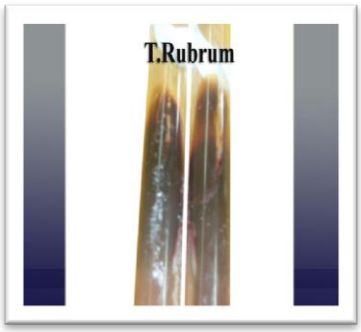



**Figure 29**


**Figur30**




**Figur31**

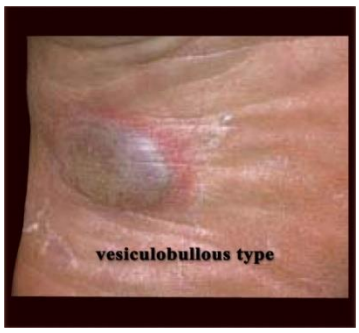



**Figure 32** **Figure33** **Figure34**

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## List of Abbreviations

| Abbreviation | | Full word |
|---|---|---|
| **TP** | **:** | Tinea pedis |
| **GTT** | **:** | Germ Tube Test |

| | | |
|---|---|---|
| **DTH** | **:** | Delayed type hypersensitivity |
| **T** | **:** | Trichophyton |
| **DM** | **:** | Diabetes mellitus |
| **GDM** | **:** | Gestational Diabetes mellitus |
| **DMSO** | **:** | Dimethyl sulfoxide |
| **KOH** | **:** | Potassium hydroxide |
| **DTM** | **:** | Dermatophyte test medium |
| **CM** | **:** | Confocal scanning laser microscopy |
| **MCP** | **:** | P for monte carlo test |
| **APMA** | **:** | American podiatric medical society |
| **G1** | **:** | Group 1 |
| **G2** | **:** | Group 2 |