“A CLINICAL STUDY OF FUNGAL INFECTIONS IN DIABETIC FOOT”

By

Dr. VIVEK H

DISSERTATION SUBMITTED TO SRI DEVARAJ URS ACADEMY OF HIGHER EDUCATION AND RESEARCH CENTER, KOLAR, KARNATAKA

In partial fulfillment of the requirements for the degree of

MASTER OF SURGERY

IN

GENERAL SURGERY

Under the Guidance of

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Signature of the candidate

Dr.VIVEK.H

LIST OF ABBREVIATIONS

DM	Diabetics Mellitus
HTN	Hypertension
V C A M	Vascular cell adhesion molecule
I C A M	Intercellular adhesion molecule
HbA1c	Glycosylated Haemoglobin
ABI	Ankle-Brachial Index
IPC	Intermittent pneumatic compression
G-CSF	Granulocyte colony-stimulating factor
TMA	Trans-Metatarsal Amputation
10% KOH	10% Potassium Hydroxide
SDA	Sabouraud's Dextrose Agar
LPCB	Lacto-phenol Cotton Blue Mount
BUN	Blood Urea & Nitrogen
Pus C/S	Pus Culture & Sensitivity
LEA	Lower extremity amputation

ABSTRACT

BACKGROUND

India today leads the world with its largest number of diabetic subjects in any given country. Presently 62 million Indians are diabetic and these numbers are on the rise.
Among the several chronic complications of uncontrolled diabetes foot ulcer is one.
Diabetes is the leading cause of non-traumatic lower extremity amputation in developing countries with risk of lower limb amputation being 15 to 46 times higher.

Diabetic wound infections are predominantly polymicrobial, predominantly aerobic, Gram positive cocci (especially *Staphylococcus aureus*) with high incidence of anaerobic species and **fungal infection**. Studies have shown that fungal infection might be regarded as a risk factor of foot ulcer. Treatment of fungal infection in diabetic patients might reduce the disability, morbidity and mortality in diabetic patients

AIMS AND OBJECTIVES:

1. To determine the occurrence of fungal infection in diabetic foot ulcers.
2. To study the clinical outcome of diabetic foot ulcers associated with fungal infection

MATERIALS AND METHODS:

A total of 70 Diabetic Foot patients treated in OPD of general surgery or admitted to R. L. JALAPPA HOSPITAL AND RESEARCH CENTRE, TAMAKA, KOLAR attached to SRI DEVARAJ URS MEDICAL COLLEGE satisfying the inclusion and exclusion criteria of the study, formed the study population. Detailed history, Clinical evaluation & necessary investigations were done.

Fungal isolation from the ulcer done by 10% KOH study and SDA (Sabouraud's dextrose agar) culture methods. Fungal positive patients were treated with anti-fungal agents. The outcome of the disease studied based on ulcer progression, wound healing and tissue loss/amputations.

Study design: Descriptive study

Sample size: 70.

Study place: Dept. of Surgery, R.L.J.H, Kolar

OBSERVATION AND RESULTS:

In present study occurrence of fungal infection of diabetic foot ulcers was found to be 19% of which *Candida species* was the commonest.

Fungal infection was more commonly seen in long –standing (4-7 months), non healing ulcers on antibiotic therapy. Treating these proven cases of fungal infection showed significant improvement in wound healing and reduction in tissue loss.

CONCLUSION

The present study signifies the need of a mycological evaluation of a non-healing diabetic ulcer of a longer duration, with poor progression despite antibacterial therapy & foot care and introduction of prudent antifungal treatment for proven fungal infections in diabetic foot ulcers and thus, to consider fungal infection as a significant risk factor in diabetic foot ulcers.

KEY WORDS: Diabetic foot ulcer; Fungal infection; 10% KOH mount; SDA (Sabouraud's dextrose agar)

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INTRODUCTION

INTRODUCTION

"Diabetes is a wonderful affliction, being a melting down of the flesh and limbs into Urine…..The patients never stop making water….is chronic, and it takes a long period To form, but the patient is short-lived……for the melting is rapid, the death speedy."

--Aretaeus the Cappadocian.

Diabetes Mellitus refers to a group of common metabolic disorders that share the phenotype of hyperglycemia. Diabetes is one of the major problems of this generation with worldwide dimension. It is the disease that involves many organs and systems of the body, notably the eyes, the kidney, the blood vessels and peripheral nerves. The worldwide prevalence of DM has risen dramatically over the past 2 decades, from 30 million cases in 1985 to 177 million in 2000. Based on current trends, >360 million will have diabetes by the year 2030.[1]

According to *Modi et al*, overall incidence of diabetes in India is 1.1%. India today leads the world with its largest number of diabetic subjects in any given country. It is said that presently 62 million Indians are diabetics and these numbers are likely to increase to 57.2 million by the year 2025. This will be 1/6 th of the world's total diabetics. Current prevalence rates are 12.1% in the urban Indian adult population.[2] An estimate shows that nearly 1 million Indians die due to **Diabetes** every year. The average age on onset is 42.5 years.

India is already the ***Diabetes capital*** of the world [3.] Out of the types, the incidence of Type-2 DM is very high amongst Indians with 94-98% of Indian diabetics belonging to this group. The number of detected cases of DM reflects only the tip of the iceberg, because in India there is a large number of undetected cases than detected cases.[4]

The death in each year is due to its complications, which are common in age group of 40-60 years affecting both sexes equally. The complications are more prevalent among the lower socioeconomic status because of negligence, illiteracy and poverty.

Among the several chronic complications of uncontrolled diabetes, foot ulcer is one. Approximately 15% of individuals with DM develop a foot ulcer (great toe or MTP areas most common).[1,2] Diabetes is the leading cause of non-traumatic lower extremity amputation in developing countries. The rick of lower limb amputation is 15 to 46 times higher in diabetics than who are not [5,6.] It is mentioned by specialists that 20 out of 100 diabetic Indians undergo lower limb amputation.

The reasons for foot being the commonest site for complications in diabetics, are that foot is the most vulnerable part of the body for injury and also the most neglected. Secondly it is the site of preference for neuropathy and Ischemia.

Diabetic ulcers occur due to three factors:

1. Trophic changes resulting from peripheral neuritis.
2. Atheroma of the arteries resulting in ischaemia.
3. Excess of sugar in the tissues, which lowers resistance to infection, including fungi.[7]

AIMS & OBJECTIVES

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REVIEW
OF
LITERATURE

HISTORY

Diabetes is one of the many common diseases known to affect the mankind from antiquity. It has been described in the ancient literature of **Egyptian** medical Journal (1500 B.C) has recorded the passing of frequent and large quantities of urine.

Diabetes mellitus was anciently-known to **Indian** and **Arabian** physicians some 5,000 year ago. ***Charaka*** (2 nd century A.D.) has mentioned the sweetness of urine and polyuria in his "Charaka Samhita". ***Sushruta***, the great ancient Indian surgeon, has mentioned about this disease, its symptomatology, etiology and classification which holds good even today. He coined the name 'Madhu-Meha'- meha of honey. It is the **Greeks** who called it as *Diabetes* (passing like a fountain or through a siphon).

The other historical milestones are [1,2,8:]

- Avicenna (980-1027 A.D.) gave a good account of diabetics and the first description of diabetic gangrene.
- Morton (1637 – 1678) made clear the hereditary characteristics.
- Thomas Willis (1613 – 1675) writes that the urine of diabetic was sweet.
- Matthew Dibson (1745 – 1784)proved that sweetness of urine is due to sugar
- **Cullen** (1709- 1790) added the adjective '*Mellitus*' to the disease in order to distinguish from diabetes *insipidus* in which there is no sugar in the urine.
- **Fehling** (1850) reported his quantitative test for sugar in urine
- **Pyrce** (1887) described association of foot ulcer, neuropathy and vascular Disease[12]
- Charles Best and Fredrick Banting (1922 – 1936) discovered Insulin from the Pancreas
- Able (1926) prepared the crystalline insulin

Wound healing:

"Wound healing consists of a complex but very orderly array of overlapping phases in which highly specialized cells interact with an extracellular matrix to lay down a new framework for tissue growth and repair".[9]

History of wound healing:

The treatment and healing of wounds are some of the oldest subjects discussed in the medical literature and probably earliest problems of human race.[10] Empirically the ancient physicians of Egypt, Greece, India and Europe developed gentle methods of treating wounds by removing foreign bodies, suturing, covering wounds with clean material and protecting injured tissue from corrosive agents.

The theory of the *"three healing gestures"* was formed more than 4000 years ago, with earlist writing recorded on a clay tablet from 2200BC. The tablet described the three gestures as *washing wound, making plasters and bandaging the wound*.[12] The modern era of gentle wound care started in the mid sixteenth century when Ambrose Pare, the great French army surgeon, who during the battle of Villaine, applied milder agents like digestive solution of egg yolk, rose oil, honey and turpentine to amputation stump with dramatic results.

One of the early writings relating the concept of wounds was by James Carrick Moore, a member of Surgeon's Company of London in 1789. In his dissertation Moore states "When any accident or disease injures the human frame, it was early observed, that the body possessed within itself, a power of alleviating or remedying the evil.

John Hunter, William Steward Halsted, Alexis Carrel and other great clinical biologist demonstrated that minimizing tissue injury produces rapid and effective healing leading to the "minimal interference" concept of wound care. If the surgeon can remove all impediments, normal wound healing processes will produce the best possible result. In 1893, Lister extended the early studies of Koch and Pasteur and demonstrated the evidence of bacterial growth in wounds and ability of this growth to lead to abscess formation or invasive infection, sepsis and gangrene.

Later Semmelwies, Ehrlich, Fleming and Florey also realized that bacteria by asepsis, antiseptics and antimicrobials heralded a new era in wound management. World War Ist resulted in rapid discoveries surrounding the care of wounds, the foremost among those being the use of extensive debridement.[11]

James Paget has given some scientific knowledge to their handling of wounds, particularly those resulted from war. In the early 1900's Carrel and his associates made investigations with the scientific approach to wound healing. Later Carrel (1916), Harvey and Howe's (1930) studied incised wounds and contributed to the knowledge of wound healing. It was not until the 1960's that concept of moist wound environment for healing was considered. In addition to protecting the wound from infection, the moist environment would help to facilitate debridement, minimize inflammation, reduce pain and diminish scarring.

MEDICAL MYCOLOGY

INTRODUCTION

There are more than 50,000 species of fungi, but most are beneficial to humankind. Some fungi greatly enhance our quality of life by contributing to the production of food and spirits. Other fungi have served medicine by providing antibiotics (eg, Penicillin) and immunosuppressive drugs (eg, cyclosporine) . Fortunately, only a few hundred species of fungi have been implicated in human disease, and 90% of human infections by fungi can be attributed to a few dozen species.[13]

Fungal infections are mycoses. Most pathogenic fungi are exogenous, their natural habitats being water, soil and organic debris. In India, the fungal infections are known since the ancient civilization and have been mentioned in Atharva Veda (Mycetoma is described as Padavamikam ie Anthill foot).

GENERAL CHARACTERISTICS

All fungi are eukaryotic organisms, and each fungal cell has at least one nucleus and a nuclear membrane, endoplasmic reticulum, mitochondria and secretory apparatus. Most fungi are obligate or facultative aerobes. They are chemotrophic, secreting enzymes that degrade a wide variety of organic substrates into soluble nutrients. All fungi have an essential rigid cell wall that are composed hugely of carbohydrates as well as glycoproteins and lipids.
The surface components of the cells wall mediate attachment of the fungus to host cells.

MORPHOLOGY

Yeasts – Yeasts are single cells, usually spherical to ellipsoid in shape and varying in diameter from 3 to 15 µ (Microns). Most yeasts reproduce by budding (asexual) some species produce buds that characteristically fail to detach and become elongated; continuation of the budding process then produces a chain of elongated yeast cells called **Pseudohyphae.**

The yeasts are ubiquitous in the environment being found on fruits, vegetables and other plant materials (exogenous). Some live as normal inhabitants in and/or on the human body (endogenous).

Molds – Growth in the mold form occurs by production of multicellular filamentous colonies. These colonies consist of branching cylindrical tubules called **Hyphae**, varying in diameter from 2 to 10µ. The mass of intertwined hyphae that accumulates during active growth is a **Mycelium.** Some hyphae are divided into cells by cross-walls or septa, typically forming at regular intervals during hyphal growth. Hyphae that penetrate the supporting medium and absorb nutrients are the vegetative or substrate hyphae. In contrast, aerial hyphae project above the surface of the mycelium and usually bear the reproductive structures of the mold.

Dimorphic Fungi – These fungi have two types of morphology at different temperature i.e yeast forms at body temperature (37 ^{o} C) and filamentous form at room temperature (25 ^{o} C). Most of the true pathogenic fungi are dimorphic in nature.

CLASSIFICATION [13,45]

Based on the basis interaction between the host and the fungus, the symbiotic host-parasite relation of the fungi can be divided into **3** modes:

1. **Commensalism** – The fungus which neither gets benefit not harmed by the host-parasite relationship.
2. **Mutalism –** The fungus takes benefits from the host-parasite relationship.
3. **Parasitism -** The host is harmed by the host-parasite relationship.

The **Fungal diseases** in man can be classified according to the site of primary involvement as follows:

1. **Superficial Mycoses** – The infection is limited to the outermost layer of the skin and its appendages.
2. **Cutaneous Mycoses –**The infection extends deeper into the epidermis and it also invades hair and nails.
3. **Subcutaneous Mycoses –** The infection is due to the low degree of pathogenic organism. It involves the dermis, subcutaneous tissues, muscles and fasciae.
4. **Systemic Mycoses –** The infection originates primarily at one site like lungs and later on disseminates systemically to secondary body sites.
5. **Opportunistic Mycoses –** Because of increasing use of immunosuppressive therapy and AIDS epidemic, Opportunistic mycoses has been introduced. The infectious agents normally are of low pathogenic potential, which produce disease only under unusual circumstances, mostly involving host debilitation.

The fungal infections are not usually transmitted sexually like the viral, bacterial or parasitic diseases. However, balanoposthitis caused by Candida species is supposed to be transmitted by sexual contact. Piedra is also taken into this category and higher rates have been reported among homosexuals in Denmark.

This classification of fungi is based on the mechanism and spores that result from sexual reproduction. The major taxonomic groups are listed below.

A. **ZYGOMYCETES:** Sexual reproduction results in a zygospore; asexual reproduction occurs via sporangia. Examples: Rhizopus, Absidia, Mucor.
B. **ASCOMYCETES:** Sexual reproduction involves a sac or ascus, producing ascopores. Asexual reproduction in via conidia. Molds have septate hyphae. Eg: Ajellomyces (Blastomyces,Histoplasm), Arthroderma (Microsporum, Trichophyton), and Yeast genera such as Saccharomyces.
C. **BASIDIOMYCETES:** Sexual reproduction results in 4 progeny basidiospores supported by a club-shaped basidium. Hyphae have comples septa. Eg: Mushrooms, Filobasidiella Neoformans (Anamorph, Cryptococcus Neoformans).
D. **DEUTEROMYCETES:** This is an artificial grouping of the imperfect fungi for which a teleomorph or sexual reproduction has not been discovered. The anamorphic state is characterized by asexual conidia. When a sexual cycle is discovered, a species is reclassified to appropriately reflect its phylogeny. Examples: Coccidioides Immitis, Paracoccidioides Brasiliensis, Candida Albicans.

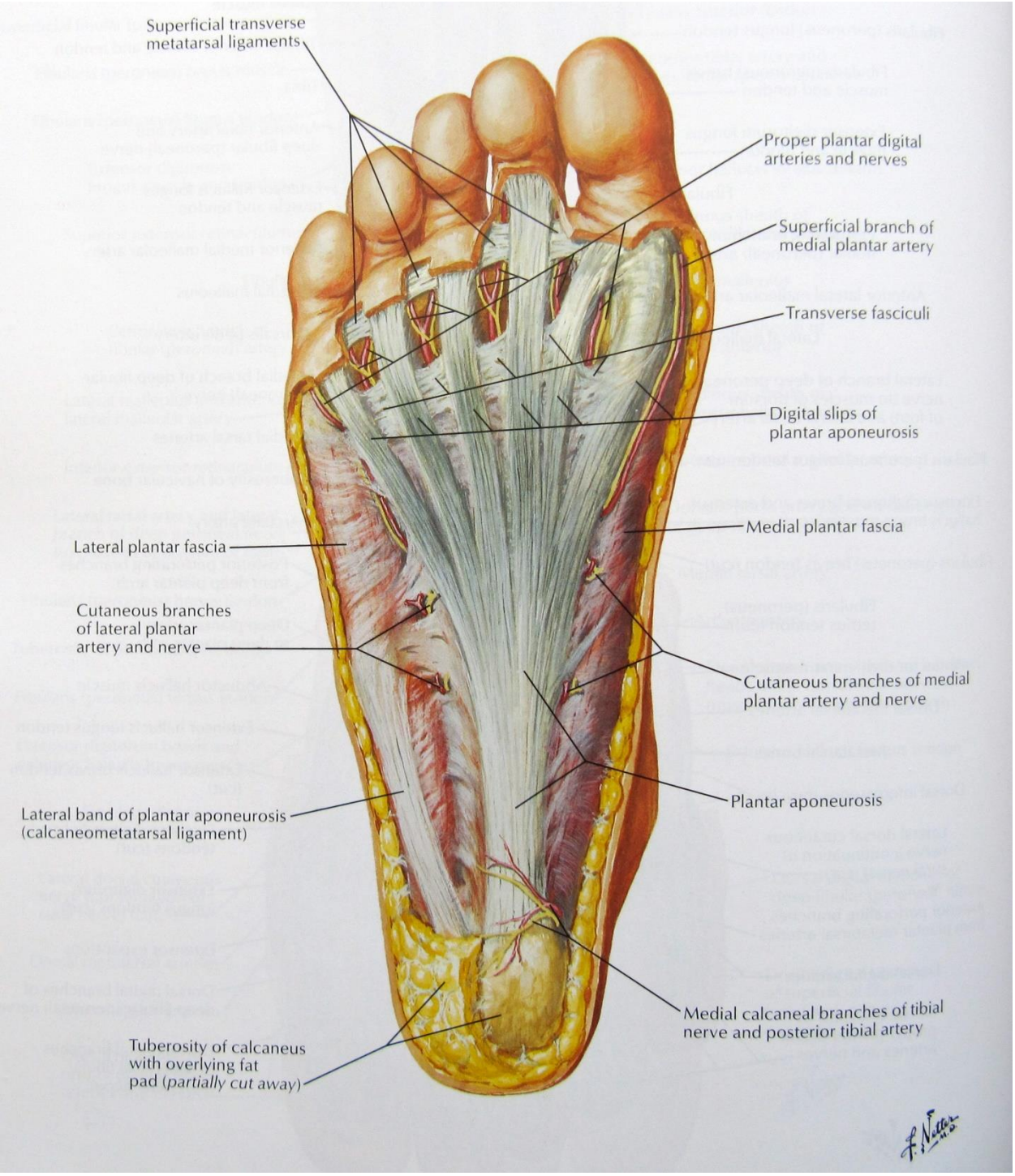
SURGICAL ANATOMY OF FOOT

SKIN: The skin of dorsum of the foot is thin and highly flexible, containing hair follicles, sweat glands and scanty sebaceous gland. Hairs are sparse and thick. The plantar skin is 5mm thick especially over those points which bear weight viz. heel, ball of big toe and lateral margins of the sole[16].

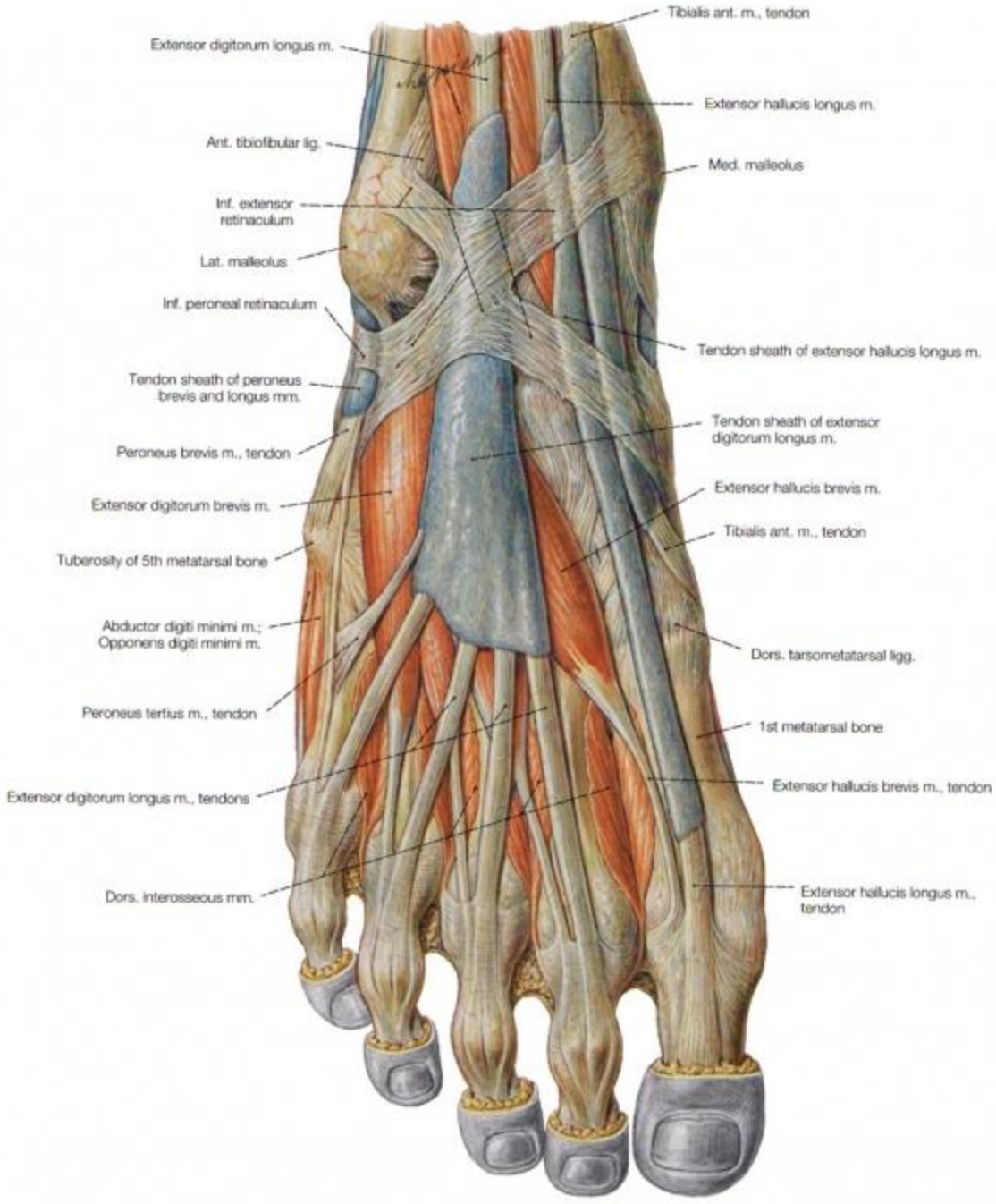
Hypodermis is not part of the skin but attaches the skin to underlying structures. Hypodermis is composed of loose areolar connective tissue most of this collagenous and elastic fibers running parallel to the surface of the skin. Hypodermis is well supplied with blood vessels and nerve endings. Tactile sensation is exceptionally good in the sole.

SUBCUTANEOUS TISSUE: The subcutaneous tissue in the sole is more fibrous, tough and stingy. Fibrous septa divide the tissue into small loculi which are filled with fluid fat under tension, this makes a shock absorbing pad.

DEEP FASCIA: Deep fascia on the dorsum of the foot (*fascia dorsalis pedis*) is the thin layer continuous above with the inferior extensor retinaculum. At the sides of the foot, it blends with plantar aponeurosis, anteriorly it ensheathes the dorsal tendons. The deep fascia covers the whole length of the sole. It spreads out over the sole and is inserted by five slips into each of the five toes. It lies superficial to the vessels, the nerves, the muscles and tendons. It consists of 3 portions, relatively thin *medial* and *Lateral* parts, a very dense and strong *intermediate* part. This thickened strong central part is known as **plantar aponeurosis**.



Anatomy of the foot (Plantar aspect)



Anatomy of the foot (Dorsal aspect)

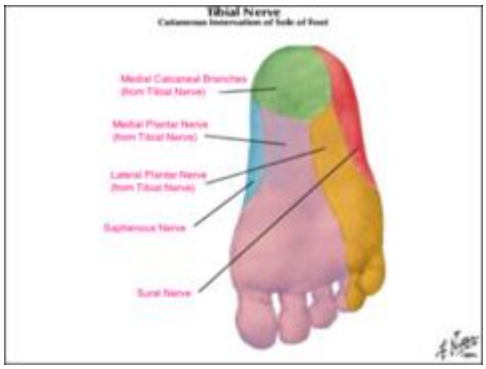
NAILS: The adult nails of the toes are flattened elastic structures of a horny texture.

The root of the nail is implanted into a groove in the skin. The exposed part is the body of the nail, the skin is attached to the undersurface of the nail forming the 'Hyponychium'. The root of the nail is overlapped by a skin fold, the stratum Corneum of which is prolonged distally as a thin cuticular fold the 'eponychium'.The 'germinative zone' together with the subjacent corium forms the nail bed.

NERVE SUPPLY: Cutaneous nerves are arranged in the following way:

DORSUM: The Saphenous nerve – supplies the medial border of the foot up to the ball of great toe. The **Superficial peroneal** – Skin over entire dorsum except lateral and medial borders and the cleft between the 1st & 2nd toes. The **Deep peroneal** supplies the cleft between the 1st and 2nd toes. The **Sural nerve** supplies the lateral border of the foot upto the tip of little toe. The **digital branches** of the **medial & lateral plantar nerves** supply the distal parts of dorsum of the toes.[9, 15, 17]

SOLE: The **medial plantar nerve** supplies the 3 ½ digits on the medial side of the foot. The **lateral plantar nerve** supplies 1 ½ dights. The **Medial calcanea**l branch of the posterior tibial nerve supplies the skin under the heel.



Cutaneous innervation of the foot

VASCULATURE OF FOOT: [14,15]

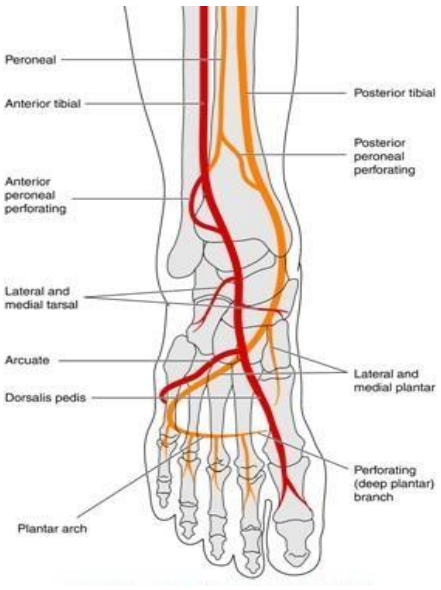
All the arterial supply of the foot is derived from the **Popliteal artery.**

The **anterior tibial** artery enters the extensor compartment of the leg and continues in the foot as the **dorsalis pedis** artery.

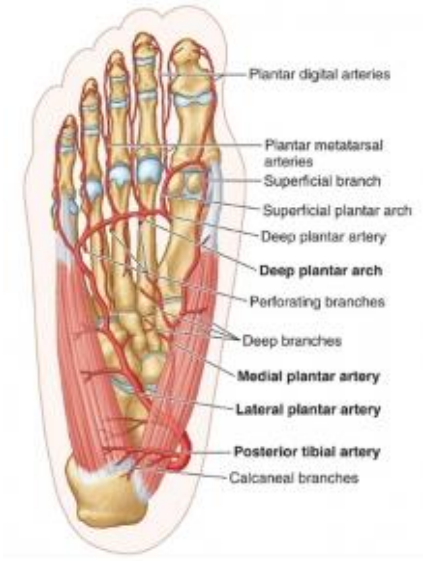
The **posterior tibial artery** divides into **Medial plantar artery**, supplying the medial side of the foot & to the big toe, and **Lateral plantar artery** crosses the sole obliquely on the lateral side of the nerve, just deep to the first layer of the sole, towards the base of the fifth metatarsal bone.

The plantar arch curves convexly forwards across the bases of 4th, 3rd & 2nd metatarsals and is joined in the proximal part of the first intermetatarsal space by the dorsalis pedis artery.

From the convexity of the plantar arch, plantar **metatarsal arteries** run forwards and bifurcate to supply the four web spaces and digits. The veins accompanying the perforating arteries take most of the blood from the sole and from the interosseous muscles to the dorsal venous arch.



(A)Dorsum of foot



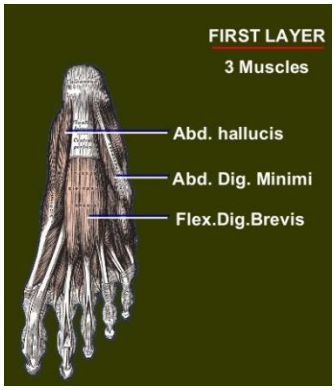
(B) Plantar aspect of foot

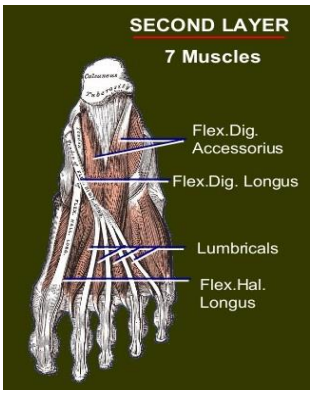
ARTERIAL SUPPLY OF FOOT

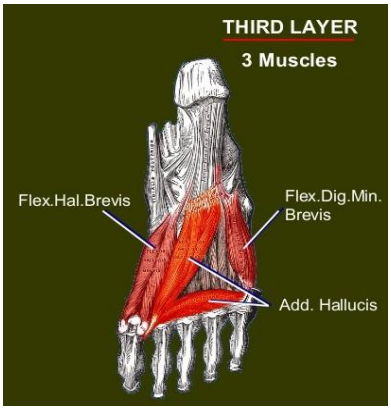
MUSCLES OF THE FOOT

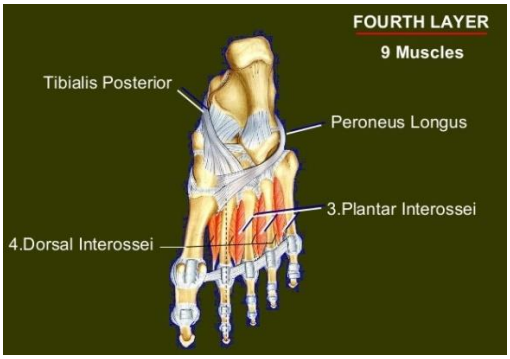
The muscles in the extensor group are located anteriorly in the leg. They include **Tibialis Anterior, Extensor Hallucis longus,** laterally are the **Peroneal muscles.** The flexors are in the posterior compartment of the leg. Beneath the plantar fascia, the muscles in the sole of the foot are categorized into four layers.

Muscles in the **first** layer include Flexor digitorum brevis, Abductor halluces and Abductor digiti minimi. **Second** layer includes tendons of Flexor hallucis longus & Flexor digitorum longus, Flexordigitorum accessories muscle(quadrates plantae) and the Lumbricals. In the **third** layer are Flexur hallucis brevis, Adductor hallucis and Flexor digiti minimi brevis**.** **Fourth** layer includes four dorsal interossei and three plantar interossei and tendons of peroneus longus & tibialis posterior [14,15,17]









BONES OF THE FOOT

The bones of the foot are the tarsal bones, metatarsals and the phalanges.

The **Tarsal** bones are as follows*:* ***Calcaneum*** is the largest bone of the foot and forms the prominence of the heel, it articulates with talus above and cuboid in front. ***Talus* c**arries the whole body weight. It lies on weight bearing calcaneum below the tibia**.** ***Navicular*** bone can be seen and felt on the medial border of foot. ***Cuboid*** bone is wedge shaped, narrowest laterally and broadest medially where it articulates with lateral cuneiform. ***Cuneiform*** bones – all the three are wedge shaped, medial is largest & intermediate the smallest.

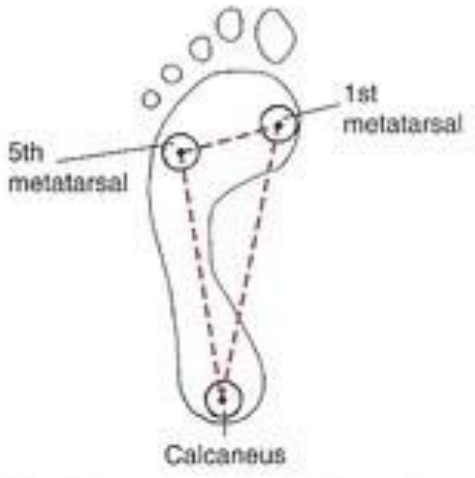
The **Metatarsal** bones resemble metacarpals of the hand, each possessing a head distally, a shaft and a base proximally. There are five metatarsals and they are numbered from medial to lateral side. Each toe has three **Phalanges** except the big toe, which possess only two.

ARCHES OF FOOT [17]:

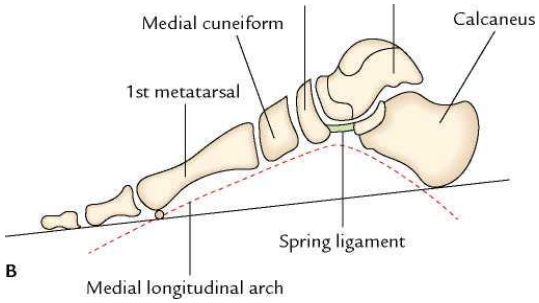
The Medial Longitudinal Arch formed by the three cuneiform bones articulating posteriorly with navicular and anteriorly with metatarsal bones. This is completed along with body of Talus &medial tubercle of the Calcaneum.

The Lateral Longitudinal Arch is formed by 4^{th} & 5^{th} metatarsal bones, Cuboid and lateral half of Calcaeum bone.

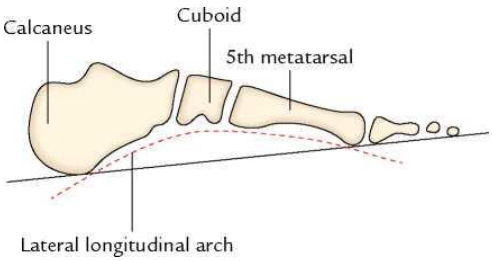
The Transverse Arches include the anterior (complete), which is formed by the heads of the 5 metatarsal bones and the posterior (half-dome), which is formed by the greater parts of the tarsus & metatarsus, which is completed by similar arch in opposite foot.



Weight bearing areas (Right foot)



Medial arch



Lateral arch

Bones forming arches of foot

PATHOGENESIS OF DIABETIC FOOT

Diabetes mellitus primarily affects the vessels & nerves causing vasculopathy and neuropathy.

VASCULAR DISEASE

Diabetes mellitus affects both large and small vessels, changes occurring both in the tunica media and intima with deposits of platelets, lipids, cholesterol and calcium. The atherosclerotic process appears at a young age and advances more rapidly and is common in the both sexes. The vessels commonly involved are tibial & peroneal arteries, small vessel, and arterioles and collateral vessels.

The diabetic patient responds to infection by developing vascular thrombosis and increased extra-vascular tissue tension and the local production of tissues destructive enzymes, derived from phagocytic lysosomes which further leads to local thrombosis and small vessels occlusion. And hence a vicious cycle continues in an ischemic diabetic limb.

Diabetic atherosclerosis has also been described as extraordinarily diffuse, multi-segmental, and often Involving collateral vessels. Intimal atherosclerosis and medial calcific stenosis (MCS) are both commonly found in diabetic.[19]

The main whole blood cell types typically found in the evolving atheroma are the monocytes and lymphocytes. A number of adhesion molecules or receptors for leucocytes expressed on the surface of arterial endothelial cells participate in the recruitment of leucocyte to the nascent fatty streak. Receptors of particular interest include – vascular cell adhesion molecule (VCAM – 1), Intercellular adhesion molecule (ICAM-1) and P selectin.[20]

RISK FACTOR FOR DIABETIC MICROVASCULAR DISEASE [21]

- Genetic predisposition
- Family history
- Smoking
- Hypertension
- Hypertriglyceridemia
- Age
- Duration of diabetes
- Autoimmune drugs
- Hypercholesterolemia
- Beta blockers.

NEUROPATHY

Its incidence parallels the duration and severity of hyperglycemia in both Type- 1 and type- 2 diabetic mellitus . Vascular and metabolic factor are both implicated in the pathogenesis of diabetic neuropathy. Nerve biopsies in diabetic neuropathy suggest that focal nerve fibre loss and ischemic injury is more prominent in type- 2 than type- 1 diabetes.

PATHOPHYSIOLOGY OF DIABETIC NEUROPATHY [1,22]

1. Hyperglycema: Increased polyols - Sorbitol and Fructose.
2. Hyperosmolarity – Edema of nerve
3. Reduced myoinositol – Altered myelin synthesis & Low Na-k ATPase activity.
4. Occlusion vasanervorum.

Chronic painful diabetic neuropathy is difficult to treat but may respond to antidepressants (TCA such as amitriptyline, desipramine, imipramine, or selective Serotonin norepinephrine reuptake inhibitors such as duloxetine) or anticonvulsants (gabapentin, pregabalin, carbamazepine, lamotrigine), of which duloxetine and Pregabalin has been approved by U.S Food & Drug Administration (FDA).[1]

Major predisposing factor for diabetic foot infections is foot ulceration, which Is usually related to peripheral neuropathy. Peripheral vascular disease and various Immunological disturbances play a secondary role.[23]

DIABETIC FOOT INFECTION

Infections in the foot can be considered to be minor and common, or major and fortunately rarer. Foot infections are either acute or chronic and arise as a result of direct inoculation or haematogenous spread. With a significant proportion of population remaining barefoot, minor skin trauma is a frequent cause of local infection. In diabetics, combination of vascular insufficiency, neuropathy & poor cellular function, infection can be extremely difficult to treat.

Minor infections of include fungal infections, verrucas, infected blisters, infected bursitis & ingrowing toe-nails associated with paronychia. **Fungal** infections are relatively uncommon and can be important as they can cause generalized discofort.

Major infections of foot are common in Diabetes. These may be superficial, often associated with ulceration. Deeper infection may involve soft tissue only with abscess formation or can involve bones (osteitis or osteomyelitis), also local joints (pyogenic arthritis).

The bacteriology of diabetic foot infections are predominantly polymicrobial, with a high incidence of anaerobic species.[24] *Candida* was the predominant isolate among fungal pathogens.[25] Fungal foot disease was found in 48% of entire diabetic population. C.*parapsilosis, C. albicans and C. tropicalis* were the most common causes of these infections.[26] Also includes Zygomycosis (Mucormycosis) due to organisms of the genera Mucor, Absidia and Rhizopus have been reported mostly in immunosuppressed patients. Direct inoculation into the skin accounts for the infections since these fungi are ubiquitous in soil.[27]

DIABETIC FOOT DEFORMITIES [1,2,7,22]

Diabetic foot problems are at increased risk of infection & ulceration, and trauma (trivial) secondary to neuropathy and microvascular changes, can lead to collapse of the foot, also known as Charcot neuroarthropathy. This process has three stages (Eichenholtz), which takes upto 18 months to run its course: Stage 1- Fragmentation; Stage 2 –Coalescence; and Stage 3 -Bone consolidation.

TOES AND WEBS

- Cock up deformity seen in great toe
- Hammer toe

Varus deformity of toes

DISTAL FOOT : Following deformities can occur:

- Hallux valgus [1,2,7,22]
- Distal foot calluses

HEEL: Because of bony prominences, heel is the site of neurotropic ulcers. Ulcer & patches of gangrene can develop heel lesion after leads to leg or thigh amputations.

DIABETIC GANGRENE

Diabetic gangrene is due to three factors [32] :

- Trophic changes resulting from peripheral neuritis.
- Atheroma of the arteries resulting in ischemia
- Excess of sugar in the tissue which lower resistance to infection

The neuropathic factor impairs sensation and thus favors the neglect of minor injuries and infections, so that inflammation and damage to tissues are ignored. Thick callosities develop on sole and are means whereby infection gains entry. Infection involving fascia, tendon and bone can spread proximally via subfascial planes.

CHARECTERISTICS OF DIABETIC ULCER [10,28]

The ulcer is usually full thickness, therefore extracellular matrix components are initially absent. The most common site is on the foot, especially over bony prominences and on the heel. An adequate description of ulcer characteristics (i.e, depth, appearance and location) is necessary for selection of appropriate treatment. In addition, it must be determined whether the ulcer is the result of neuropathy, ischemia or typically both.

DIABETIC ULCERS are classified as: 1. Ischemic and 2. Neurotrophic

Ischemic Ulcers: Yellow punched out necrotic ulcer, margin sharp, well vascularised, On debridement no bleeding from center, would bleeds from periphery, severe pain, more during night, Pain relieved on siting or dangling the feet. Prognosis for healing is poor. Treatment is by meticulous foot care and to control the risk factors.

Neuropathic Ulcers: Caused by pressure. Located in weight bearing areas. Has a pink granulation base, circular, generally white fibrotic rim surrounded by hyperkeratotic tissue. There is a good vascular supply, so foot is often warm with a inflammatory reaction around the ulcer. Musculoskeletal deformity tends to occur with callus formation. Also known as mal perforans ulcer, plantar ulcer, trophic ulcer and perforation or pressure ulcers.

CLASSIFICATION:

University of Texas Diabetic Wound Classification System[29]

Stage	Grade 0	Grade 1	Grade 2	Grade 3
A	Pre or post Ulceration Lesion Completely epithelialized	Superficial Wound, not Involving tendon, Capsule or bone	Wound Penetrating to tendon or capsule	Wound penetrating to bone or joint
B	With infection	With Infection	With Infection	with infection
C	With ischemia	with Ischemia	With Ischemia	with Ischemia
D	With infection and ischemia	With infection and ischemia	With infection and ischemia	with infection and ischemia

Wagner Ulcer Classification System[30]

Grade	Lesion
0	No open lesions; may have deformity or cellulitis
1	Superficial diabetic ulcer (partial or full thickness)
2	Ulcer extension to ligament, tendon, joint capsule, or deep fascia without abscess or osteomyelitis
3	Deep ulcer with abscess, osteomyelitis, or joint sepsis
4	Gangrene localized to portion of forefoot or heel
5	Extensive gangrenous involvement of the entire foot

WOUND HEALING

Wound healing involves two distinct processes

- **Regeneration :-** When healing takes place by the proliferation of parenchymal Cell & usually results in complete restoration of the original tissues.
- **Repair : -** When healing takes place by proliferation of connective tissue elements resulting in fibrosis and scarring. It involves further 2 processes granulation tissue formation and contraction of the wound.[31,32,33]

CYTOKINES IN WOUND HEALING[31]

CYTOKINE	SYMBOL	SOURCE	FUNCTION
Platelet derived Growth factor	PDGF	Platelets & Macrophages	Fibroblasts proliferation & Angiogenesis
Transforming Growth factor	TGF –B	Platelets, Neutrophils & Macrophages	Fibroblast proliferation, Collagen metabolism, Indirect angiogenesis & Activation of other growth Factors
EPIDERMAL GROWTH FACTOR	EGF	Platelets	Stimulates epithelial cells, Fibroblast proliferation & Granulation tissue formation
Insulin growth Factor	IGF -1	Fibroblasts	Stimulates synthesis of Collagen &fibroblast Proliferation
Fibroblast growth factor	----	Macrophages and other tissue platelets	Fibroblast and epithelial cell proliferation, matrix deposition, wound contact and angiogenesis.

FACTORS AFFECTING WOUND HEALING [33, 34]

GENERAL FACTORS:

1 . Age

2 . Nutrition –

a) Proteins for Neoangiogenesis, Collagen synthesis, wound remodeling

b) Vit A – Neoangiogenesis, epithelialization

c) Vit C – Co–factor in hydroxylation of praline & lysine, Normal fibroblastic function,

d) Vit E – Anti –oxidants, stabilizes cell membranes

e) Vit B complex - co – factor for enzymes in metabolism

f) Zinc - Associated with RNA & DNA Polymerase

g) Copper - Collagen cross–linkages

h) Iron – Hydroxylation of lysine & praline in collagen synthesis

i) Manganese – Actives enzymes in glycosylation of pro-collagen.

3. Corticosteroids and Cytotoxic drugs

4. Anaemia, Uraemia, Jaundice, Hypoalbumenemia, Metabolic diseases

5. Malignant diseases

6. 7. HIV and immunocompressive diseases

LOCAL FACTORS :

1. Infection and hematoma

2. Presence of necrotic tissue and foreign body

3. Poor blood supply

4. Tissue tension and large defect

5. Venous or lymph stasis

6. Recurrent trauma

7. Site of wound – Wounds over joints and back has poor healing

CLINICAL PRESENTATION

SIGNS AND SYMPTOMS:

A. VASCULAR INSUFFIENCY [7, 33]

Intermittent claudication	Cold feet
Nocturnal pain	Rest pain
Absent pulses	Blanching on elevation
Dependent rubor	Atrophy of subcutaneous fatty tissues
Loss of hair on foot and toes	Thickened nails
Gangrene	Blue toe syndrome
Acute vascular occlusion	Miscellaneous.

In acute vascular occlusion, the FIVE "Ps" in the lower extremity are –

1. Pain - Sudden onset
2. Pallor - Waxy
3. Paresthesia - Numbness
4. Pulselessness below the block
5. Paralysis

B. NEUROPATHY [1,7,22,33]

Parasthesia	Radicular pain
Hyperasthesia	Hypoesthesia
Loss of vibratory and position sense	
Heavy callus formation over pressure points	
Trophic ulcers	Anhydrosis
Radiographic changes	Demineralisation
Osteolysis	Charcot's joint.

INVESTIGATIONS

The following investigations are done for the diagnosis and treatment in the diabetic foot:-

1. To demonstrate the extent and severity of the disease process.
2. To screen diabetic patients for peripheral vascular insufficiency.
3. To confirm and control the inter-current diseases interfering with the healing process.

URINE EXAMINATION [32,33,34,35]

Albumin : Albuminuria present in 25% patients.

Sugar : Dip test for sugar

Shows yellow, red or green colour.

Glycosuria:- Result from hyperglycemia when blood glucose level reaches 180 gm% (Renal threshold). Renal threshold increase in age and in diabetic nephropathy.

Ketonuria: Detected by Rotheras acetone test. First sign to be recognized in ketosis.

BLOOD INVESTIGATIONS

a) **Fasting blood sugar:**

Hyperglycemia is most decisive indication of diabetes. It is estimated by Folin Wu or Somgy's nelson method. Fasting blood sugar more than 120mgm% is indicative of diabetes.

b) **Post prandial blood sugar:** After overnight fast, the patient is given breakfast of 100gms of carbohydrates or 100 gms of glucose load. Then venous blood is checked for glucose level every half hour for two hours. If it exceeds 180mgm% is indicative of diabetes mellitus.

c) Oral glucose tolerance test:[36] Unrestricted carbohydrate diet for 3 days before the test. Sample of blood and urine are taken prior to the test and 100 gms of glucose in water is administered orally. Blood and urine samples are taken at ½ hourly intervals for 2-3 hours and examined quantitatively for glucose. This gives glucose tolerance curve.

In normal subject fasting blood sugar is 80-120 mg% and peak valve is not above 180mg%. In diabetes the blood glucose may rise to a high peak value of 300mg% or even more subsequently slow fall sets in, so that many hours may elapse before the fasting blood sugar levels is regained and urine samples contain no sugar.

d) Glycated Haemoglobin:[36]

Glycated haemoglobin provides an accurate and objective measures of glycaemia control over a period 3 months. The rate of formation of HBA1c is directly proportional to the ambient blood glucose concentration; a rise in 1% corresponds to approx. average increase of 2 mmol/L in blood glucose.HBA1c reflects the changes in glycaemia control occurring in the month before measurement.

PUS CULTURE & SENSITIVITY

Pus from infected area is cultured for microorganisms and their sensitivity to various antibiotics is tested.

RADIOLOGY OF THE FOOT[28,33]

X–ray of the foot should be taken if there is any suspicious infection deep in the foot , e.g. abscess or osteomyelitis. In osteomyelitis, there will be destruction of bone, commonly seen at metatarsophalangeal joint. Sequestrum and subperiosteal new bone formation are common.

NON INVASIVE EVALUATION[37,39]

The non-invasive techniques give an accurate assessment of anatomic and physiologic vascular status. Fall in the ankle pressure may be used as an objective test for assessing and following the course of obliterative arterial diseases.

1) PHOTOPLETHYSMOGRAPHY:

Preferred procedure for obtaining digital recordings. The pressure at which the waveform obliterates corresponds to digital systolic pressure. Also measures volume changes in lower limb. Sensitivity (63% - 100%) Specificity (63%).

2) SEGMENTAL PRESSURE:

Pressure cuffs of 12cms width are placed in different position and by listening with the Doppler probe over one of the pedal vessels, the pressure at the level of the inflated cuff can be obtained.

3) TOE PRESSURE:

They provide a highly accurate method for determining the success in the healing of an ulcer or in minor amputation. A toe pressure of 20-30 mm of Hg below which healing is doubtful.

4) WAVEFORM EVALUATION:

The waveform in the normal state shows a rapid systolic upstroke and a peaked appearance. Detonation of wave form seen just proximal to an obstructing lesion, distal to the lesion the wave form is with a loss of normal rapid systolic upstroke and the slope is same as that of down stroke.

5) ANKLE BRACHIAL INDEX [37,38,39]

Since the ankle systolic blood pressure varies with the central systolic aortic pressure, it is convenient to normalize the values by dividing the ankle pressure by the brachial blood pressure.[38] Values are the lowest when there is complete occlusion and highest when there is minimal atheromatous change. It constitutes an effective means of following up the patient's course. Consistent decrease indicates advancing disease or failure of reconstruction. Spontaneous rise indicates development of collaterals. The higher systolic pressure at the ankle is divided by the brachial pressure to give ankle brachial index (ABI).[39]

Normal ABI is more than 1.0. Vascular disease is confirmed if it is less than 0.9. ABI correlates well with the symptomatology as follows:

Symptom	**Likely ABI**
No symptoms	> 0.9
Claudication	0.8 – 0.5
Rest pain	0.5—0.2
Gangrene	< 0.3

6) DUPLEX SCANNING WITH ULTRASOUND ANALYSIS (DOPPLER STUDY)

The recorded Doppler signal is used in two ways-

- To measure segmental systolic pressure
- To provide flow velocity wave form patterns for analysis.

This combines B- mode anatomic capabilities of revealing the location and amount of vessel lumen and stenosis can be recorded with Doppler derived velocity recordings.

INVASIVE TECHNIQUES

1) RADIONUCLIDE BONE SCINTIGRAPHY :

- Bone scanning using technetium 99m phosphonates are useful in identifying early osteomyelitis.
- Gallium accumulates in areas of active inflammation – sequential scan is useful in monitoring the response to treatment for osteomyelitis.

2) COMPUTED TOMOGRAPHY :

- Well suited for imaging complex articulations and numerous soft tissue structures.
- Can identify and characterize the extent of soft tissue infection.

3) MAGNETIC RESONANCE IMAGING:

- Detects and displays bone marrow alterations in osteomyelitis.
- Displays the contrast between soft tissue, medullary tissue and cortex with clarity.

4) ANGIOGRAPHY[39] :

Angiography remains the 'Gold Standard' for evaluating patients with arterial disease for surgery. A good angiographic study requires a skilled radiographer, image intensification, rapid cassette chnager or cine film, contrast media and state of art equipment. The percutaneous transfemoral (Seldinger) technique is commonly used.

Anatomic evaluation of the vascular supply to the leg and foot, possibility of large vessel stenosis or occlusion superimposing on distal diabetic vascular disease are most important indications for angiography.

COMPLICATIONS: Local bleeding and haematoma, Thrombosis, peripheral embolization, renal toxicity, Idiosyncratic or allergic reaction to contrast like nausea, vomiting, itching, hypertension, laryngeal edema and bronchospasm.

5) DIGITAL SUBTRACTION ANGIOGRAPHY (DSA) [37,39]

Digital subtraction angiography is visualization of vessels using digital flurographic techniques for image enhancement.

PRINCIPLE: If two x-ray beams of different proton energy impinge on tissue, the lower energy beam will be relatively more attenuated by substances having high atomic numbers (i.e. bone and iodine) due to photoelectric effect, various tissues could therefore be subtracted from final image.

ADVANTAGES:

1. DSA accomplishes significantly better contrast resolution.
2. Highly sensitive screening technique for carotids and lower limb vessels.
3. When compared to conventional angiography, cost is less.
4. Digital subtraction angiography can be performed routinely on out-patient basis.
5. Digital subtraction angiography can demonstrate small reconstituted vessels distal to an obstruction which could not be seen on a catheter cut-film study.

ANGIOGRAPHIC INFORMATION

An ideal Angiography should answer 4 vital questions:

1. The exact site and extent of the stenosis/occlusion.
2. The ‘Run-In’. Is the state of arteries proximal to the stenosis normal or not?
3. The ‘Run-off’. Arterial bypass surgery is only feasible if a distal artery is open beyond the block.
4. State of collateral circulation.

TREATMENT

STRATEGIES FOR SAVING THE DIABETIC FOOT

- Correction of vascular risk factors
- Improve the circulation
- Regular foot Inspection
- Treatment of foot ulcers
- Prescribing special shoes
- Treatment among medical disciplines
- Patient Education

IMPROVING CIRCULATION

A regular walking program will improve circulation in 80 % of the patients. Drugs like pentoxifylline can be used. The ultimate treatment for claudication is vascular surgery to by-pass narrowed arteries or endarterectomy. Transcutaneous angioplasty is also useful.

The surgical approach to aortoiliac occlusive is aortoiliac endarterectomy, bypass grafting and extra-anatomic reconstructions. In the patients with simple occlusion of the superficial femoral artery, autogenous saphenous vein bypass from the common femoral artery to the popliteal artery is done. For patients with significant disease in the above knee popliteal artery, a bypass to the distal popliteal artery should be done.

PRIMARY TREATMENT OF FOOT ULCERS

EVALUATION

1. Clinical appearance & Depth of penetration
2. Radiological appearance.
3. Metabolic control
4. Debridement
5. Bacterial culture (aerobic & anaerobic)
6. Parenteral antibiotics
7. Avoidance of soaking the feet & foot care

TEAMWORK[40]

The team members involved in the case of diabetic foot care includes,

- Primary physician to educate about foot care.
- Nurse educator to educate about diabetic and foot care and to examine the foot.
- Podiatrist : To detect and correct diabetic foot lesions.
- Vascular Surgeon: For revascularization
- Pedothist/Orthotist: For prophylactic shoe-fitting.
- Physiotherapist rehabilitation: To apply cast & rehabilitation.
- Home care nurse: To improve the clinical course of patient at home.

PATIENT EDUCATION: The patient should be taught about good diabetic control, foot-care, dangers of smoking and alcohol. By this the patient can prevent injury to insensitive foot and detect lesions earlier. Maintenance of record of investigations.

WOUND MANAGEMENT

WOUND CARE AND DRESSING

The concept of moist wound healing has been examined and gradually accepted by wound care clinicians during the last 40 years, and has led to the development of hundreds of dressings that support a moist wound environment. Wound dressings exhibit various fluids-handling mechanisms: absorption, gelling, retention and moisture vapour transmission.

Common categories of wound dressings are as follows:

Absorbers: Super-absorbent dressings in a variety of types including Iodine gel, Sodium Chloride impregnated and clear acrylic. Exudate is absorbed into the dressing matrix. In the case of some, the fluid can be expressed from the dressing under pressure.

Accessories – Wound Care: Includes elastic skin closures, protective barriers, skin preparation's and disposable measuring guides.

Bandages: Plaster of Paris and cotton or poly stretch bandages as well as straps.

Composite Dressings: Dressings with three layers. Absorbs drainage and prevents bacteria from entering the wound.

Calcium Alginate Dressings: Calcium Alginate dressings are derived from seaweed and available as sheets or ropes for packing deep wounds. Following absorption, the exudates interacts with the dressing material to form a gel.

This is a typical attribute of alginates: carbohydrate polymers gel according to the proportion of uronic acid units in their composition. They absorb moderate to heavy wound drainage and are capable of absorbing many times their own weight but not recommended for lightly draining wounds.

Compression dressings

Collagen Granules

Absorbant dressings

Cleansers & Debriders: Wound Cleansers / Debriders cleanse a wound before dressing application. They contain ingredients that absorb and/or deodorize the wound, also used to remove dead tissue while keeping the healthy tissue intact.

Collagen Dressings: Collagen dressings are used for moderate to heavily draining wounds to enhance healing and tissue repair. Collagen dressings can be used on burns, pressure ulcers, scrapes, cuts and also dermatologic conditions.

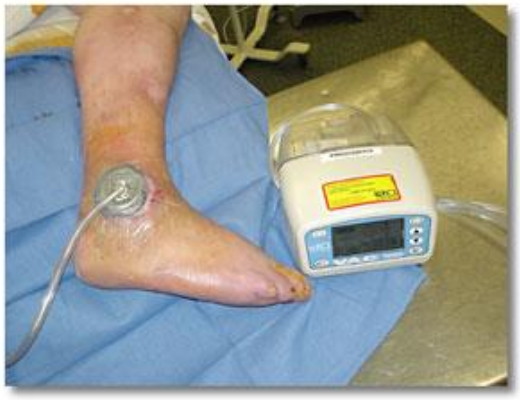
Compression Dressings: Compression dressings apply controlled pressure to areas of the body to help control swelling and promote circulation, encouraging wound healing. Where venous ulceration occurs, the patient needs assistance in achieving a good venous blood return. Compression therapy has two main functions: to counteract venous hypertension and to control edema. Intermittent pneumatic compression [41] (IPC) therapy is administered through a boot-shaped device which, by means of a pump, is inflated and deflated to achieve alternating, dynamic compression of the encased limb.

Foam Dressings: Foam dressings are highly absorbent and generally made from hydrophilic polyurethane foam, offering a moist environment to insulate and cushion the wound. They require a cover dressing and tape. Comfortable and gentle, they will not stick to wounds or leave residue. They can also be used for heavily draining wounds.

Hydrocolloid Dressings: Hydrocolloid Dressings are soft, absorptive, adhesive wafers that became gel-like as they absorb and are best used on lightly to moderately draining wounds. They are waterproof and impermeable to bacteria and contaminates. Easy to apply but not recommended for infected wounds.

Hydrogels: Hydrogels are transparent and water-based preparations. They hydrate tissue and facilitate debridement using the body's own enzymes. Because of the relatively high water content, there is limited drainage and/or absorption.

Hydrocolloid dressing



Negative pressure therapy

Unna boots

Non-adherent Dressings: Non-adherent dressings are available non-impregnated or impregnated and discourage foreign matter from becoming lodged in the wound bed. They can be used on skin tears, donor sites and skin grafts.

Silicone Gel Sheeting: Silicone Gel Sheeting have been designed for temporary use in the management of both existing and new hypertrophic scars and keloids.

Silver Dressings: Silver coated highly absorbent alginate dressings remains effective for 3 days and has a 7 days wear-time. With a highly absorbent alginate to provide a moist wound environment, high absorbency and the antimicrobial power of silver delivered within the dressing and to the wound bed. Used for pressure, venous and diabetic ulcers and donor and recipient graft sites.

Topical negative pressure therapy: Suction drainage of wounds has been used for many years. The integrated vacuum-assisted closure technique is claimed to improve perfusion, reduce oedema and promote granulation tissue formation. The removal of exudates, particularly the more viscous forms, also removes bacteria and protease enzymes – both barriers to healing.

Transparent Dressings: Tranparent Adhesive Dressings maintain a moist environment which is permeable to the oxygen and vapor and impermeable to bacteria and contaminants can be applied on blisters, superficial wounds or wounds with light amount of drainage. They allow evaluation of wound progress without removal of dressing.

Unna Boots: Unna Boots are named after a German dermatologist, Paul Gerson Unna. Unna Boots are typically used to treat edema, ulcers and sores. Unna Boots uses a zink oxide paste which helps ease skin irritation and promote healing. Unna Boots can be worn up to 7 days, without changing. It helps keep the wound area moist. As the Unna Boot dries, it forms a semi-rigid cast, providing support.

RECENT ADVANCES IN DIABETIC ULCER MANGEMENT

OZONE THERAPY[1,42]

Ozone (03), contains a large excess of energy with molecular weight of 48 and density one and a half times that of oxygen. Ozone/oxygen mixtures exert significant antimicrobial activity. Ozone has a wide range of action. At a concentration of 1 mg per liter of water at 1 ^{o}C, ozone rapidly inactivates Coliform bacteria, Staphylococcus aureus, and Aeromonas hydrophilia and is influenced by P^{H} and temperature. Higher doses are tissue toxic. At dosage concentrations used in external therapy, ozone essentially inactivates all bacterial species (aerobic & anaerobic bacteria, facultative species), spores and cysts are neutralized as well. Alsoeffective against mycotic organisms.

MAGGOT DEBRIDEMENT THERAPY[43] :

Maggot debridement therapy is an inexpensive and efficient adjunct for treatment of diabetic foot wounds. Researchers have proven that maggots are useful in debriding wounds, controlling wound odor and reducing bacterial burden in wounds. Maggots are the fly larvae and are available in medical grade from commercial laboratories in the United States and Europe. This treatment requires proper staff training and patient acceptance.

GRANULOCYTE COLONY STIMULATING FACTOR (G-CSF) [44] :

A Preliminary randomized controlled study showed that adding subcutaneous injections of recombinant granulocyte colony-stimulating factor (G-CSF) to local wound care and antibiotic therapy led to significant and rapid infection resolution with better outcomes in diabetic patient with severe foot infections.

SURGICAL MANAGEMENT

The aim of the treatment is not merely healing but walking on a healed foot or residual foot. Rehabilitation plays an important role after amputation. The following factor forms the basis of therapeutic decision making in the presence of ischemia.

1. Extent of the gangrene.
2. Status of vessels for revascularization.
3. Individual results of arterial reconstruction.
4. Available amputation level.
5. Quality of the amputation program & prosthetic rehabilitation.
6. Individual patient factors (prior amputation, stroke, poor eyesight and others).

DEBRIDEMENT [28,33,39]

Debridement may be definitive in patients with good blood flow. Minor areas of necrosis over angular prominences resulting from an episode of excessive walking can be allowed to demark and separate.

TYPES OF AMPUTATION MINOR [28,33,39]

TOE AMPUTATION : This is one of the most frequently performed peripheral amputations in the diabetic patients and is usually indicated for digital ischemia , joint sepsis and /or osteomyelitis or soft tissue loss secondary to recurring ulceration. Primary advantage of toe amputation is that minimal tissue is removed.

RAY AMPUTATION : Generally if a toe amputation is precluded because of an advancing process at the phalangeal- metatarsal crease, or because of distal metatarsal head involvement, a ray amputation is indicated. But proximal dependent rubor, gangrene infection or cellulitis are clearly contraindications to the Ray amputation.

TRANSMETATARSAL AMPUTATION : Provided that planter skin is uncompromised, the indications are infection, gangrene of multiple toes or metatarsal heads and extension of the infection process proximal to the metatarsal phalangeal crease, but not up to the distal & middle third junction of the forefoot.

SYMES AMPUTATION : If the infectious or gangrenous process excludes a Transmetatarsal amputataion, then the next level is at ankle is Symes amputation. It is removal of foot with the calcaneum and cutting of tibia and fibula just above the ankle joint with retaining heal flap.

MAJOR AMPUTATIONS [28,33,39]

BELOW KNEE AMPUTATION: When a distal amputation site is precluded below Knee amputation is done. Contraindications to this are infectious or gangrenous Process that extends to within 5 cm from the tibial tuberosity or that involves the proposed skin flaps (especially the posterior flap).

ABOVE KNEE AMPUTATION: The flap length should not be cut more than 1 to 1.5 times the bone length. Bone is sectioned at such a length so as to allow closure without tension. Vascular structures are dissected in the subsartorial canal, bleeding vessels ligated. Approximating flexor to extensor and abductors to adductors, muscles are sutured. The subcutaneous layer is closed and skin with interrupted sutures.

GUILLOTINE AMPUTATION: Guillotine amputation is in which the limb is cut off in one transverse plane without formation of flaps. All the tissues of the limb are divided at same level and the bone end is left exposed on the cut surface. Major vessels and nerves should be clearly cut with a sharp blade at a sufficiently high level so that they retract into healthy muscle. It is indicated whenever there is presence of gross sepsis as infection spreading is controlled. Disadvantage is that re-amputation must be performed at higher level in order to cover the bone end with soft parts.

PROSTHESIS AND REHABILITATION

PROSTHESIS FOR ABOVE KNEE AMPUTATION[28]

The ideal length of tibial stump for optimal function with prosthesis (measured from knee joint line) should be 15- 18cms depending on the patients height. Most below knee amputees using prosthesis should be fitted with patellar tendon bearing (PTB) prosthesis. With this type of limb, all the body weight is borne by the knee and the patients controls the prosthesis with own knee movement, and poses only minor restrictions on the range of knee movement. The posterior aspect of the stump upto the popliteal fossa and medial & lateral para tibial areas and the medial condylar flare also carry significant load.

PROSTHESIS FOR ABOVE KNEE AMPUTATION

The shortest length of above knee stump for effective use of prosthesis is 8cms, measured from the origin of adductor longus on the pelvis to the cut end of femur. The ischial tuberosity is the key weight bearing part of the above knee stump, although sockets should be total contact type. In above knee stumps, brims are of various sizes and are designed to locate the tuberosity positively on the posterior flap seating. A recent development is the ischial containment socket also known as 'CAT-CAM' or contour adducted trochanteric-controlled alignment method socket. This socket lies less on the ischial tuberosity for weight bearing, but stabilizes the stump in the coronal plane by providing a bony lock between the medial aspect of ischial tuberosity and the greater trochanter.

The other prosthesis available are soft retaining suction socket which is worn next to the skin without the usual stump sock. Most above knee prosthesis are of carbon fibre endoskeletal construction usually with one piece cosmetic cover.

PROSTHESIS FOR SYMES AMPUTATIONS

The standard prosthesis for Symes amputation consists of closely fitting compliant lever which is usually made up of polyethylene foam. Normally the socket is designed for end weight bearing, but extend proximally to the level of patellar tendon to avoid excessive movements over the shaft of the tibia during walking.

REHABILITATION

The major aim when considering a patient for possible amputation is to remove a painful and /or infected and therefore dangerous limp or part of a limp and to restore mobility and activity of the patient.

The achievement of the second aim begins preoperatively when the nature of the operation is discussed. In a patient having a below knee amputation the importance of quadriceps function must be emphasized. The greatest contribution to rehabilitation is made by the surgeon, who carefully and meticulously performs the operation because a major factor delaying rehabilitation is problems with the amputation wound.

The conventional conservative approach has been to await healing of the wound completely an then refer the patient to a specialist in limp fitting and rehabilitation who undertakes the subsequent management often at an another center. At the other extremes all amputations are performed by a single group of surgeons, immediate fitting of prosthesis is undertaken and a coordinated program of aggressive rehabilitation is begun.

DIABETIC FOOT CARE GUIDELINES

To avoid serious foot problems that could result in losing a toe, foot or leg, be sure to follow these guidelines:

- ✓ **Inspect your feet daily**, Check for cuts, blisters, redness, swelling, or nail problems. Call your doctor if you notice anything.
- ✓ **Wash your feet in lukewarm water**. Keep your feet clean by washing daily.
- ✓ **Be gentle when bathing your feet**. Wash them using a soft washcloth or sponge. Dry by blotting or patting.
- ✓ **Moisturize your feet** – but not between your toes. Use a moisturizer daily to keep dry skin from itching or cracking.
- ✓ **Cut nails carefully** – and straight across. Also, file the edges. Don't cut them too short, since this could lead to ingrown toenails
- ✓ **Never trim corns or calluses.**
- ✓ **Wear clean, dry socks**. Change them daily.
- ✓ **Avoid the wrong type of socks**. Avoid tight elastic bands (they reduce circulation)
- ✓ **Wear socks to bed**. If your feet get cold at night, wear socks. NEVER use a heating pad or hot water bottle.
- ✓ **Shake out your shoes and inspect the inside before wearing.**
- ✓ **Keep your feet warm and dry.**
- ✓ **Never walk barefoot.**
- ✓ **Take care of your diabetes.** Keep your blood sugar levels under control.
- ✓ **Don't smoke**, Smoking restricts blood flow in your feet.
- ✓ **Get periodic foot exams**. See your foot and ankle surgeon on a regular basis for an examination to help prevent the foot complications of diabetes.

MYCOTIC INFECTION

SUPERFICIAL MYCOSES

PITYRIASIS VERSICOLOR: Pityriasis Versicolor is a chronic mild superficial infection of the stratum corneum caused by Malassezia Globosa, M.Restricta, and other members of the ***M furfur*** complex. Discrete, serpentine, hyper or hypopigmented maculae occur on the skin, usually on the chest, upper back, arms or abdomen.

TINEA NIGRA: Tinea Nigra (or **Tinea Nigra Palmaris**) is a superficial chronic and asymptomatic infection of the stratum corneum caused by the dematiaceous fungus *Hortaea (Exophiala) Werneckii*. This condition is more prevalent in warm coastal regions and among young women. The lesions appear as a dark (brown to black) discoloration, often on the palm. Microscopic examination of skin scrapings from the periphery of the lesion will reveal branched, septate hyphae and budding yeast cells with melaninized cell walls.

PIEDRA: ***Black piedra*** is a nodular infection of the hair shaft caused by Piedraia hortai. ***White piedra,*** due to infection with trichosporon species, present as softer, larger, yellowish nodules on the hairs. Axillary, public, beard, and scalp hair may be infected. Treatment for both types consists of removal of hair and application of a topical antifungal agent. Piedra can be seen as endemic in tropical under developed countries.

CUTANEOUS MYCOSES

Cutaneous mycoses are caused by fungi that infect only the superficial keratinized tissue like skin, hair, and nails. The most important of these are the ***Dermatophytes***, a group of about 40 related fungi that belong to three genera: ***Microsporum, Trichophyton and Epidermophyton*** . Dermatophytoses are among the most prevalent infections in the world.

Being superficial, Dermatophyte (ringworm) infections have been recognized since antiquity. In skin they are diagnosed by the presence of hyaline, septate, branching hyphae or chains of arthroconidia. Dermatophytes are classified as ***Geophilic, Zoophilic and Anthropophilic*** depending on whether their usual habitat is soil, animals, or humans.

CLINICAL CLASSIFICATION OF THE DERMATOPHYTES[46]

CLINICAL NAME	SITE OF LESION	ORGANISMS MOST FREQUENTLY ISOLATED
Tinea capitis, Epidemic	Scalp	*Microsporum audouini, Trichophyton Tonsurans (U. S.), Trichophyton Violaceum, Microsporum ferrugineum*
Tinea capitis, Nonepidemic	Scalp	*Microsporum canis, Trichophyton Verrucosum, Micro Sporum gypseum (rare)*
Tinea favosa (favus)	Scalp, torso	*Trichophyton schoenleinii, Trichophyton Violaceum*
Tinea barbae	Beard	*Trichophyton rubrum, Trichophyton Verrucosum*
Tinea corporis	Arms, legs, Torso	*Trichophyton rubrum, Microsporum Canis, Trichophyton mentagrophytes*
Tinea cruris	Gentitocrural Folds	*Trichophyton rubrum, Trichophyton Mentagrophytes, Epidermophyton Floccosum*
Tinea pedis and Manus	Feet, hands	*Trichophyton rubrum, Trichophyton Mentagrophytes*
Tinea unguium	Nails	*Trichophyton rubrum, Trichophyton Mentagrophytes, Epidermophyton*

SUBCUTANEOUS MYCOSES

The fungi that cause subcutaneous mycoses normally reside in soil or on vegetation. They enter the skin or subcutaneous tissue by traumatic inoculation with contaminated material. In general, the lesions become granulomatous and expand slowly from the area of implantation.

SPOROTRICHOSIS: Sporothrix schenckii, is a thermally dimorphic fungus that lives on vegetation. It is associated with a variety of plants-grasses, trees, moss, and bushes. Following traumatic introduction into the skin, S. schenckii causes **sporotrichosis,** a chronic granulomatous infection. The initial lesion develops as a granulomatous nodule that may progress to form a necrotic or ulcerative lesion. Meanwhile, the draining lymphatics become thickened and cord-like. Multiple subcutaneous nodules and abscesses occur.

CHROMOBLASTOMYCOSIS: It is a subcutaneous mycotic infection caused by traumatic inoculation by any of five recognized fungal agents that reside in soil and vegetation. All are dematiaceous fungi, having melaninized cell walls: *Phialophora verrucosa, Fonseecaea pedrosoi, Rhinocladiella aquaspersa, Fonsecaea commpacta,* and *Cladophialophora carrionii.*

The infection is chronic and characterized by the slow development of progressive granulomatous lesions that in time induce hyperplasia of the epidermal tissue. Cauliflower like nodules with crusting abscesses eventually covers the area. Small ulcerations or “black dots” of hemopurulent material are present on the warty surface.

MYCETOMA: It is a chronic subcutaneous infection by any of several saprophytic species of fungi or Actinomycetous bacteria that are normally found in soil. The clinical features defining mycetoma are local swelling and interconnecting, often draining sinuses that contain granules, which are microcolonies of the agent embedded in tissue material. An **actinomycetoma** is a mycetoma caused by an Actinomycete; a **Eumycetoma (Maduromycosis, Madura foot)** is a mycetoma caused by a fungus.

Mycetomas occur only sporadically outside the tropics and are particularly prevalent in India, Africa, and Latin America. The granule color, texture, and size and the presence of hyaline or pigmented hyphae (or bacteria) are helpful in determining the causative agent. Itraconazole, Ketoconazole, and even Amphotericin-B can be recommended for madurella infections.

ENDEMIC MYCOSES:

Each of the four primary systemic (dimorphic) mycoses – **Coccidioidomycosis, Histoplasmosis, Blastomycosis, and Paracoccidiodomycosis** are geographically restricted to specific areas of endemicity. The fungi that cause Coccidioidomysis and Histoplasmosis exist in nature in dry soil or in soil mixed with guano, respectively. Each of these four mycoses is caused by a thermally dimorphic fungus, and most infections are initiated in the lungs following inhalation of the respective conidia. Only a few infections lead to disease, which may involve dissemination from the lungs to other organs. With rare exceptions, these mycoses are not transmissible among humans or other animals.

OPPORTUNISTIC MYCOSES [1,45,46]

Patients with compromised host defenses are susceptible to ubiquitous fungi to which healthy people are exposed but usually resistant. As members of the normal microbial flora, **Candida** and related yeasts are endogenous opportunists. Other opportunistic mycoses are caused by exogenous fungi that are globally present in soil, water, & air.

CANDIDA AND RELATED YEASTS:

Several species of the yeast genus Candida are capable of causing candidiasis. They are members of the normal flora of the skin, mucous membranes, and gastrointestinal tract. Candida species colonize the mucosal surfaces of all humans during or soon after, and the risk of endogenous infection is ever present. Candidiasis is the most common systemic mycosis, and the most common agents are C.albicans, C.tropicalis, C.glabrata, C.guilliermondii, and C.dubliniensis. The widespread use of fluconazole has precipitated the emergence of more azole-resistant species eg. C.krusei and C.lusitaniae.

Clinical manifestations of Candidiasis:

A. CUTANEOUS AND MUCOSAL CANDIDIASIS:

Oral thrush can occur on the tongue, gums, lips or palate. It is a patchy to confluent, whitish pseudomembranous lesion composed of epithelial cells, yeasts, and pseudohyphae. Risk factors include AIDS, treatment with corticosteroids or antibiotics, high levels of blood glucose, poor hygiene and cellular immunodeficiency.

Yeast invasion of the vaginal mucosa leads to **vulvovaginitis,** characterized by irritation, pruritus and vaginal discharge. **Intertriginous** infection occurs in moist, warm parts of the body such as the axillae, groin and intergluteal or inframammary folds. Interdigital involvement between the finger common in homemakers, bartenders and cooks. Candidal invasion of the nails and around the nail plate causes **Onychomycosis.**

B. SYSTEMIC CANDIDIASIS:

Candidemia can be caused by indwelling catheters, surgery, intravenous drug abuse, or damage to the skin or gastrointestinal track. Patients with compromised phagocytic defenses may develop occult lesions in kidney, skin (macula-nodular lesions), eye ,heart, and meninges.

C. CHRONIC MUCOCUTANEOUS CANDIDIASIS:

Most forms of this disease have onset in early childhood, are associated with cellular immunodeficiencies and endocrinopathies and result in chronic superficial disfiguring infections of any or all areas of skin or mucosa.

Fungal infection of **diabetic foot ulcers** were caused by 8species of *Candida* [47]. The most common causative agent was C. parapsilosis [26]. It was followed by C. tropicalis, C. albicans and C. glabrata. The remaning 4 species [C. krusei, C.kefyr, C,famata (Torulopsis candida) and C. lipolytica] were isolated with an equally low frequency.

CRYPTOCOCCUS NEOFORMASNS

Cryptococcus neoformans is a yeast that is characterized by a thick polysaccharide capsule. It occurs worldwide in nature and can found in very large numbers in dry pigeon feces. Cryptococcosis is usually associated with immunosuppression, AIDS, or malignancy but can also occur in apparently normal hosts. In culture, *C neoformans* produces a whitish mucoid colony in 2-3 days. Microscopically, in culture or clinical material, *C neoformans* is a spherical budding yeast (5 – 10 um in diameter), surrounded by a thick capsule. Specimens are examined in wet mounts,both directly and exacerbations. Combination therapy of Amphotericin B and Flucytosine [48] has been considered to be the standard treatment for cryptococcal meningitis.

ASPERGILLOSIS

Aspergillus species are ubiquitous saprophytes in nature and aspergillosis can be seen worldwide. *A.fumigatus* is the most common human pathogen, but many others, including *A. flavus, A, niger* and *A, terreus* may cause disease. This mold produces abundant small conidia that are aerosolized.

Clinical Findings :-

A. ALLERGIC FORMS – In home atopic individuals, development of IgE antibodies to the surface antigens of aspergillus conidia elicites an immediate asthmatic reaction upon subsequent exposure. This phenomenon is characteristic of - **Allergic bronchopulmonary aspergillosis,** which is clinically defined as asthma, recurrent chest infiltrates, eosinophilia, and both type- 1 (immediate) and type- 2 (Arthus) skin test hypersensitivity to aspergilus antigen.

B. ASPERGILLOMA - Aspergilloma occurs when inhaled conidia enter an existing cavity, germinate and produce abundant hyphae in the abnormal pulmonary space. Some patients are asymptomatic; others may develop cough, weight loss, fatigue, and hemoptysis.

C. INVASIVE ASPERGILLOSIS – High risk patients are those with lymphocytic or myelogenous leukemia and lymphoma, bone marrow transplant recipients, and especially individuals on corticosteroid therapy. Symptoms include fever, cough, dyspnoea and hemoptysis. Hyphae invade the lumen and walls of blood vessels, causing thrombosis, infarction and necrosis.

Aspertgilloma is treated with Itraconazole or Amphotericin B [1, 48] and surgery. Invasive aspergillosis requires either the native or lipid formulation of Amphotericin B or Voriconazole, often supplemented with cytokine immunotherapy.

MUCORMYCOSIS

Mucormycosis (Zygomycosis) is an opportunistic mycosis caused by a number of molds which are ubiquitous in nature, thermo – tolerant saprophytes. The leading pathogens among this group of fungi are species of the genera Rhizopus, Rhizomucor, Absidia, Cunninghamella and Mucor.

The major clinical from is **Rhinocerebral mucormycosis**, which results from germination of the sporangiospores in the nasal passages and invasion of the hyphae into the blood vessels, causing thrombosis, infarction and necrosis. The disease can progress rapidly with invasion of the sinuses, eyes, cranial bones and brain.

Management consists of aggressive surgical debridement , rapid administration of Amphotericin B and control of the underlying disease.

LABORATORY DIAGNOSIS OF FUNGAL INFECTION[49]

The laboratory diagnosis of fungus infection is made by microscopic examination of materials from the lesions and by morphological studies of fungal isolates.

Tissue specimens, such as skin scrapings are generally as wet mounts after treatment with **10% potassium hydroxide (KOH).** The alkali digests cells and other tissue materials enabling fungus elements to be seen clearly. The **Periodic Acid Schiff (PAS)** and **Methanamine silver** are valuable methods for the demonstration of fungal elements in tissue sections.

The commonest culture media used in mycology are **Sabouraud's Glucose Agar (PH 5.4**), **Czapek-Dox Medium and Corn Meal Agar.** The addition of antibiotics prevents bacterial contamination. Cyclohexamide (actedione) incorporated in the medium inhibits many contaminants moulds. Cultures are routinely incubated in parallel at room temperature (22^0 C) for weeks and at 37^0C days. Identification is based on the fungus and of its colony. Growth characteristics useful for identification are the rapidity of growth, colour & morphology of the colony on the obverse and pigmentation on the reverse. Small bits of fungus colonies may be teased onto a slide and mounted in **Lactophenol cotton blue** for microscopic study . **Slide culture** privides a useful technique for the study of fungus morphology.

Biochemical and serological tests seldom employed in mycology.

Fungus tag-encoded FLX amplicon pyrosequencing (fTEFAP), a universal fungal identification method,bacterial tag-encoded FLX amplicon pyrosequencing (bTEFAP), a universal bacterial identification method and a new **quantitative polymerase chain reaction (qPCR)** wound pathogen panel are used recently for rapid identification of yeast in tissue samples.

The universal fTEFAP and bTEFAP methods take 24 hours to return but are able to detect the relative contribution of any bacteria of yeast in a chronic wound diagnostic sample.[50,51]

Most fungi occur in nature and grow readily on simple sources of nitrogen and carbohydrate. Traditionally, Sabouaud's agar, which contains glucose and modified peptone (P^H 7.0), has been used because it does not readily support the growth of bacteria. However, other media, such as inhibitory mold agar, have facilitated the recovery of fungi from clinical specimens.

ANTI-FUNGAL AGENTS[1,48]

1. ANTIBIOTICS – a) Polyenes- Amphotericin B, Nystatin, Hamycin, Natamycin.
   b) Heterocyclic Benzofuran- Griseofulvin.
2. ANTIMETABOLITE – Flucytosine.
3. AZOLES – a) Imidazoles (Topical): Clotrimazole, Econazole, Miconazole, Oxiconazole.
   (Systemic): Ketoconazole,
   b) Triazoles (systemic): Fluconazole, Itraconazole, Voriconazole.
4. ALLYLAMINE- Terbinafine.
5. Other TOPICAL AGENTS – Tolnaftate, Undeylenic acid, Benzonic acid, Quiniodochlor, Ciclopirox olamine, Butenafine, Sodium thiosulfate.

MATERIALS AND METHODS

MATERIALS AND METHODS

Source of Data:

All eligible cases attending OPD of General Surgery and admitted to surgery wards in R.L.J. Hospital, Kolar for the treatment of Diabetic foot Ulcer during the period of study from January 2015 to December2015 were included.

METHOD OF COLLECTION OF DATA

The data for this study was collected from the seventy subjects fulfilling the inclusion /exclusion criteria, admitted in R.L.J. Hospital and patients attending Surgery OPD for Diabetic foot ulcer care during the study period from January 2015 to December2015 , using a standard proforma specially designed for the study.

Study Design : Descriptive study

Sample Size : 70

Study Period : January 2015 to December 2015

Inclusion criteria:

Patients having ulcer of Grade 1, 2 and 3 according to WAGNER'S classification.

Exclusion criteria:

1. Venous ulcer and Arterial ulcer.
2. Patients receiving systemic Anti-fungal treatment for other disorders.
3. Patients on dialysis for Chronic Renal Failure.
4. Patients with immunocompromised status.

STATISTICAL ANALYSIS:

The collected data is entered into an excel format and analysis will be done using descriptive statistical methods like proportion for qualitative data, mean SD for quantitative data and the significance of association (difference of proportion) by using Chi square test.

P value ≤ 0.05 will be considered as statistically significant.

COLLECTION OF SAMPLES

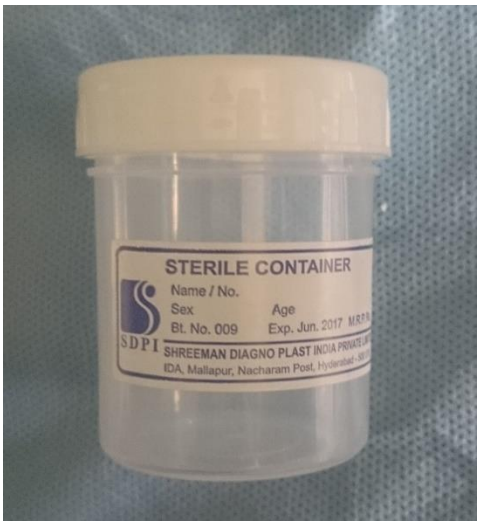
70 random cases were selected from the study group either admitted in surgery wards or attending surgery OPD for Diabetic Foot Ulcer management. Cases were selected based on inclusion and exclusion criteria included in our study. Consent was taken from all the subjects.

A proforma was developed to record the medical history, examination details and investigation reports. Medical history was taken for all subjects. Details regarding type of diabetes, its duration, treatment, compliance and personal habits were recorded. Meticulous examination was done including description of the of the Ulcer (site, size, shape, grade, classification) and as per the proforma of the study and tests performed to check for neuropathy and ischemia. Necessary investigation reports required for the study were documented in the proforma. Investigations required were recent fasting and post prandial blood sugar, Glycated hemoglobin, Arterial/Venous doppler if vascular pathology is suspected.

Sample Collection: Ulcer was cleaned with sterile normal saline before collecting the samples. Tissue for the study were taken from the depth of the ulcers including the edges, consisting mostly the granulation tissue and the necrotic slough found on the ulcer bed. Average size of the tissue block collected measured around 0.5 x 0.5cms.

Transport Media: Tissue samples were collected in sterile cotton swab or plastic bottles (Autoclavable) containing approx. 4-5ml of normal saline in it. These swabs / bottles were autoclaved at 120^0 C for 10 min before they were used for tissue collection from the ulcers under aseptic precautions, then the bottles were sealed and labeled and were taken to Microbiology Lab within an hour of sample collection for further processing.

Sterile Swab



Sterile Bottle

PROCESSING OF SAMPLES

Tissue blocks received in the Microbiology lab, were taken out from the Sample collection bottles and were Triturated (m: churning) using a Sterile Triturer and then the tissue will be ready for processing.

The prepared tissue sample was then subjected to the following processes:

1. PRELIMINARY EXAMINATION

KOH 10% Preparation

PROCEDURE: A part of Triturated tissue specimen is placed on a clean slide and 10% KOH is added to it and left in the incubator at 37^0 C for ½ hour. Cover slip is placed over it and is examined under the microscope.

The Alkali digests the keratin tissue and other tissue and other tissue materials enabling the fungus elements to be seen clearly (As shown in above picture – Arrow represents branching fungal filament.)

CONTENT: Potassium Hydroxide 10 gm dissolved in 100 ml of Distilled Water.

Careful examination is required to differentiate from the keratin tissue elements and aerosols which appear filamentous like fungus, but differentiating features will be branching filaments with/ without septations. First, examination is done under the low power with reduced light and looked for fungal elements. The size, branching, septa, presence and distribution of spores etc is noted which will be later confirmed under high power.

2. CULTURAL PRODCEDURE

Triturated tissue samples are inoculated onto 4 tubes containing Sabouraud's Dextrose agar with antibiotics- Chloramphenicol (2 of them with Actidione). **Tissue materials are placed deep into the medium on a slant using a thick needle or a scalpel. One pair of culture tubes (SDA + AB with and without Acitdione) is then incubated** at Room Temperature ($25 – 30^{0}$ C) and the other pair at 37^{0}C respectively. Growth on culture is examined twice in a week upto **4-6** weeks before it was declared negative.

INTERVENTION TO SUBJECTS

Swab samples are taken from the superficial ulcer for KOH preparation. If positive, tissue sample for Sabaroud's dextrose agar culture and sensitivity done and whenever indicated patient are instituted for Anti-fungal therapy.

Sometimes fungal filaments may not be seen on 10% KOH preparation, but growth will be seen on SDA culture media. Then, Anti-fungal therapy was started for such patients. Type of fungal infection, its morphology was later confirmed with SDA culture.

The other supportive wound care measures like Glycemic control with Insulin/Oral hypoglycemic agents, Anti-biotic as per Pus Culture & Sensitivity, relieving of pressure over the affected part of the foot were continued and the regression of the wound was studied.

Dressing of the wound was done regularly either using special dressing materials and locally applying agents like solutions and Creams, the decision of which was done by the senior staffs of corresponding units in the Department of surgery. Any surgical intervention (Wound debridement/SSG/Secondary suturing/ Disarticulation/Amputations) required during the course of the study, based on ulcer progression was done so as per advice by the senior doctors treating the ulcer.

FOLLOW UP

Each ulcers were followed upto 3 months or till the ulcer healing (which ever occurred earlier) from the time of inclusion in the study and documented regarding the progression of ulcer based on ulcer grade, healing of ulcer based on requirement of any method of surgical intervention and whether patient had to undergo any amputation.

Fungal positive patients who were started on Anti-fungal therapy were followed up every 15 days till the time of ulcer healing or 3 months (earliest among them). The other fungal negative ulcers were followed once a month.

Progression of ulcer in patients on anti-fungal therapy was documented based on ulcer grade as compared to that at the time of inclusion in the study, duration of ulcer healing and any surgical intervention required during the course of ulcer healing. Finally, whether amputation was required for any of the diabetic foot ulcer patients.

The collected data is entered into an excel format and analysis will be done using descriptive statistical methods like proportion for qualitative data, mean SD for quantitative data and the significance of association (difference of proportion) by using Chi square test.

P value ≤ 0.05 will be considered as statistically significant.

OBSERVATIONS
AND
RESULTS

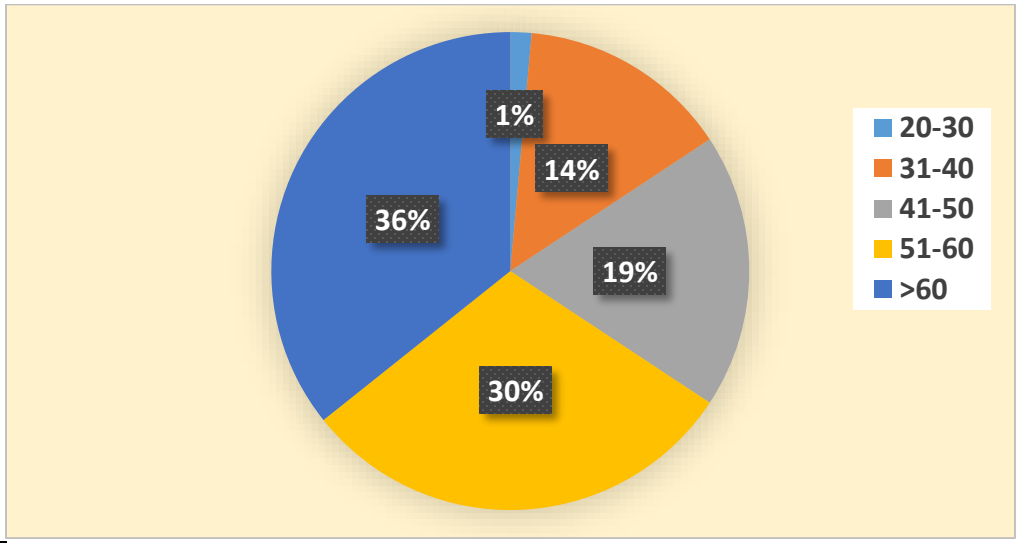
RESULTS

Study Design: A descriptive clinical fungal isolation study consisting of 70 patients with diabetic foot ulcers is undertaken to study the incidence of fungal isolation and its correlation with various clinical features.

Table 1: Age distribution of patients studied

Age (in years)	Frequency	Percent (%)
20-30	1	1.4
31-40	10	14.3
41-50	13	18.6
51-60	21	30.0
>60	25	35.7
Total	70	100.0

Graph 1: - Graph showing Distribution of participants according to Age group

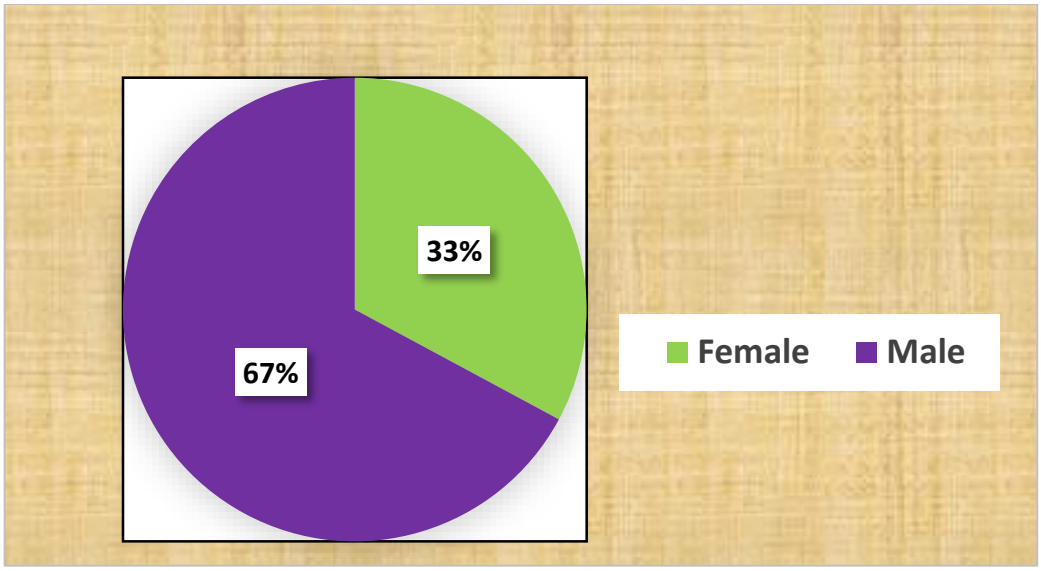


A total of 70 diabetic foot patients were included in the study. Most of the patients studied were above 60 years (36%) followed by 51-60 years (30%), 41-50 years (19%), 31-40 years (14%) and 20-30 years (1%).

Table 2: Gender distribution of patients studied

Gender	Number of Patients	%
Male	47	67.1%
Female	23	32.9%
Total	70	100.0%

Graph 2:- Gender distribution of patients studied

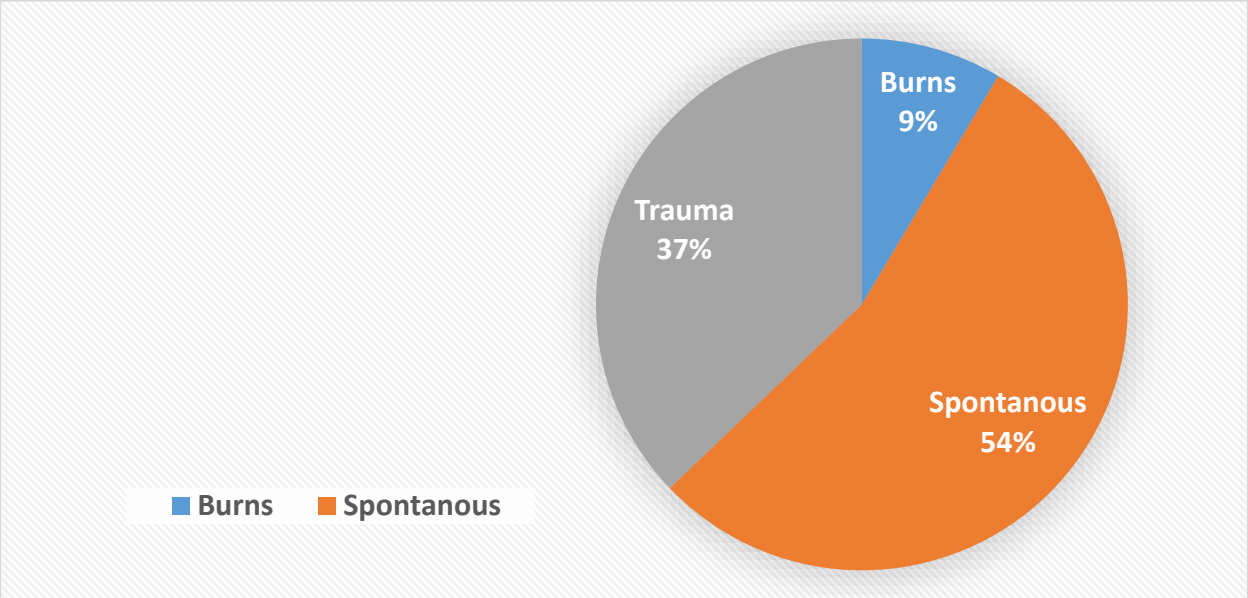


Of the 70 patients studied, 47 were males forming 67.1% of cases and 23 were females forming 32.9%.

Table 3: - Distribution of participants according to Mode of onset

Mode of onset	Frequency	Percent
Burns	6	8.6
Spontanous	38	54.3
Trauma	26	37.1
Total	70	100.0

Graph 3:- Distribution of participants according to Mode of onset

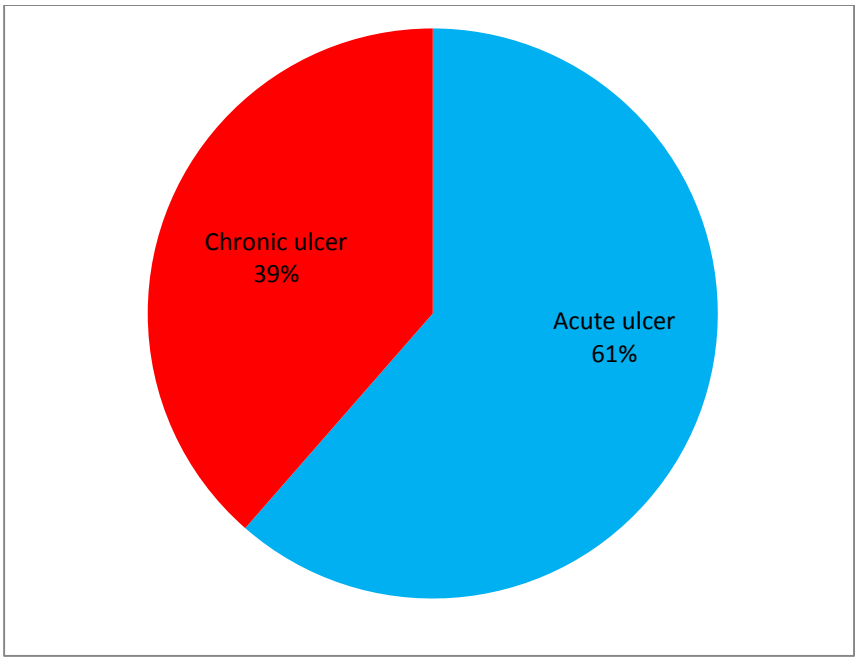


Majority of foot ulcers include in our study were spontaneous in onset (54.3%) followed by secondary to trauma (37.1%) and burns (8.6%). The prevalence of diabetic foot ulceration secondary to trauma is more frequent in older patients, may be due to bare foot walking habits in our country.[54]

Table 4: Distribution of participants according to ulcer type

Ulcer	Frequency	Percent
Acute ulcer	43	61.4
Chronic ulcer	27	38.6
Total	70	100.0

Graph 4:- Distribution of participants according to ulcer type

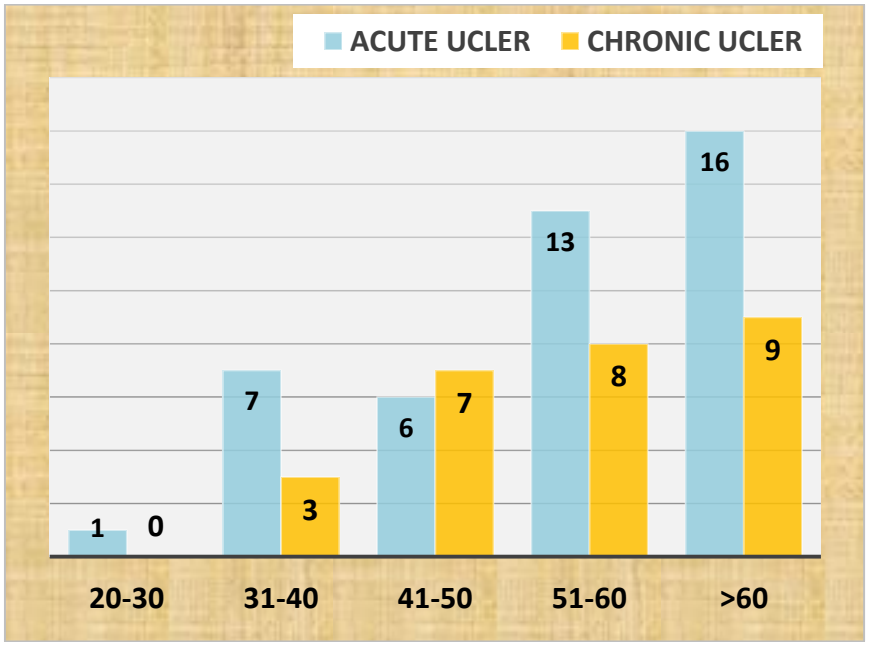


In a total of 70 cases studied, most of the ulcers were acute (61%) than chronic (39%).

Table 5: - Distribution of participants according to age group among acute ulcer and chronic ulcer

Age (in years)	ACUTE ULCER	CHRONIC ULCER	P value
20-30	1	0	0.683 NS
31-40	7	3	
41-50	6	7	
51-60	13	8	
>60	16	9	
Total	43	27	

Graph 5:- Distribution of participants according to age group among acute ulcer and chronic ulcer.

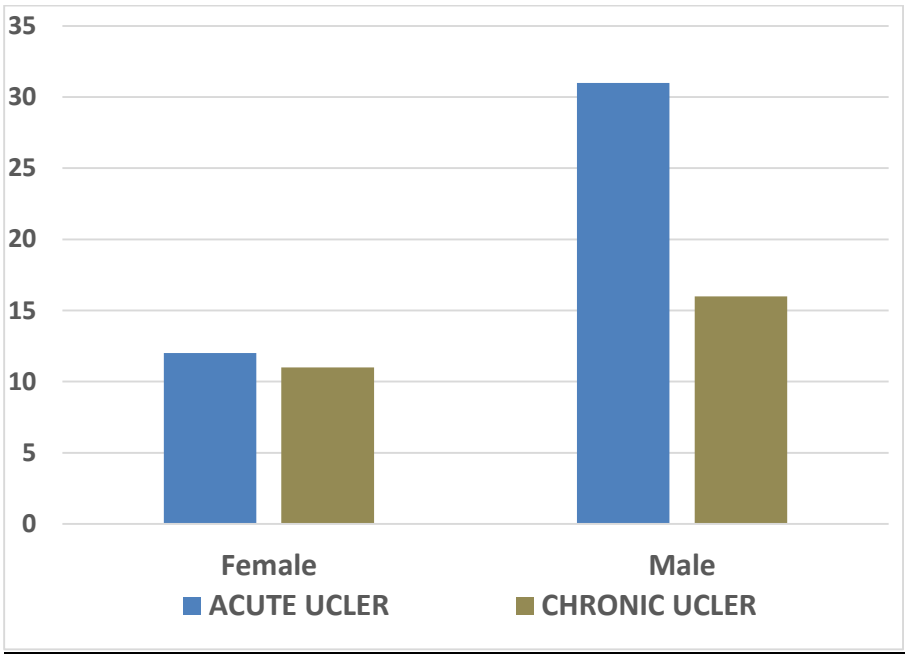


There is no significant difference among acute ulcer and chronic ulcer between various age group.

Table 6: - Distribution of participant according to sex among acute ulcer and chronic ulcer

SEX	ACUTE	CHRONIC	P VALUE
Female	12	11	P value - 0.304
Male	31	16	
Total	43	27	

Figure 6: - Graph showing Distribution of participant according to sex among acute ulcer and chronic ulcer

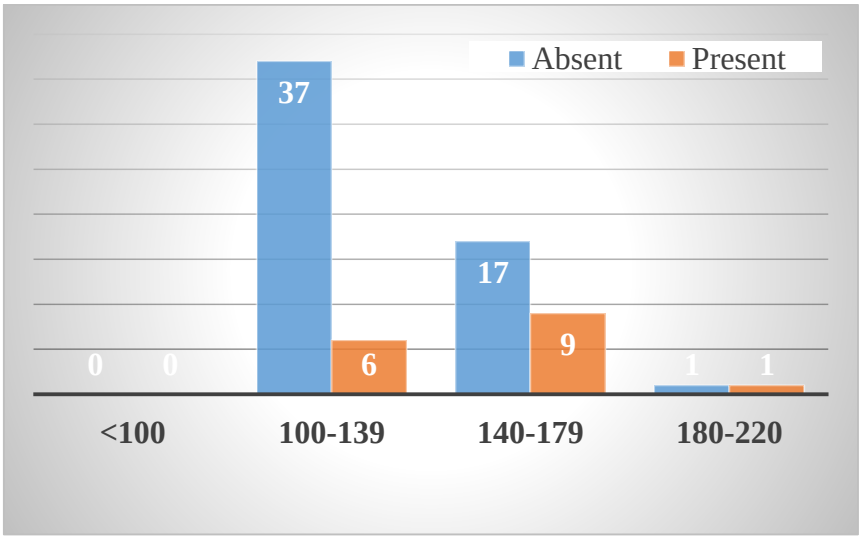


There no is significant difference among acute ulcer and chronic ulcer between male and female.

Table 7: - Distribution of patients according to blood sugar level

	Fungal infection		P value
Blood sugar level (mg/dl)	**Absent**	**Present**	
<100	0	0	0.048 NS
100-139	37	6	
140-179	17	9	
180-220	1	1	

Graph 7: - Distribution of patients according to blood sugar level

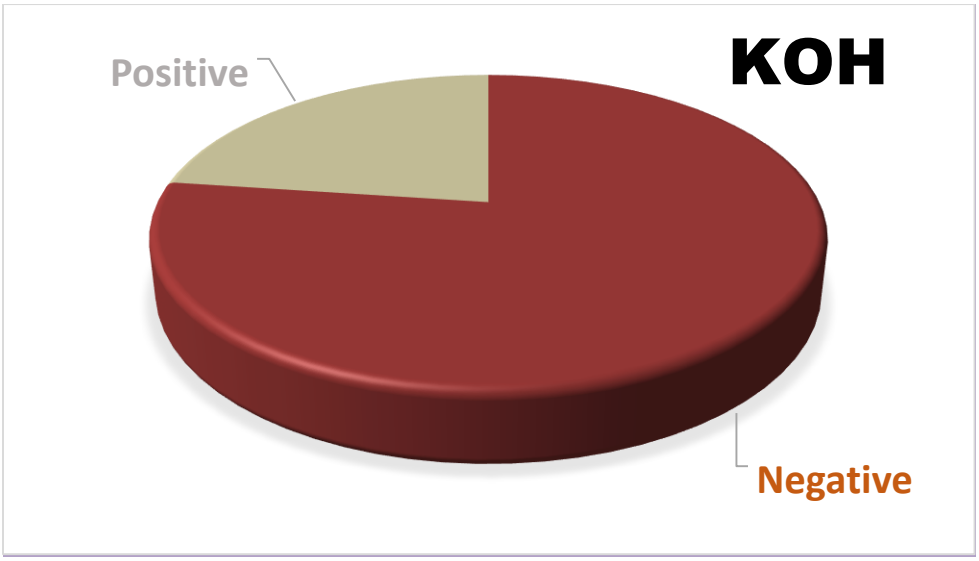


Fungal infected diabetic ulcers were most commonly seen with patients having blood sugar levels between 140 to 179 mmhg and are statistically significant. This may be because of the fact that mild to moderate hyperglycemic state favors the growth of fungal infection in diabetic foot ulcers.[18]

Table 8: - Distribution of patients according to KOH results

KOH	Frequency	Percent
Negative	54	77.1
Positive	16	22.9
Total	70	100.0

Graph 8: - Distribution of patients according to KOH results

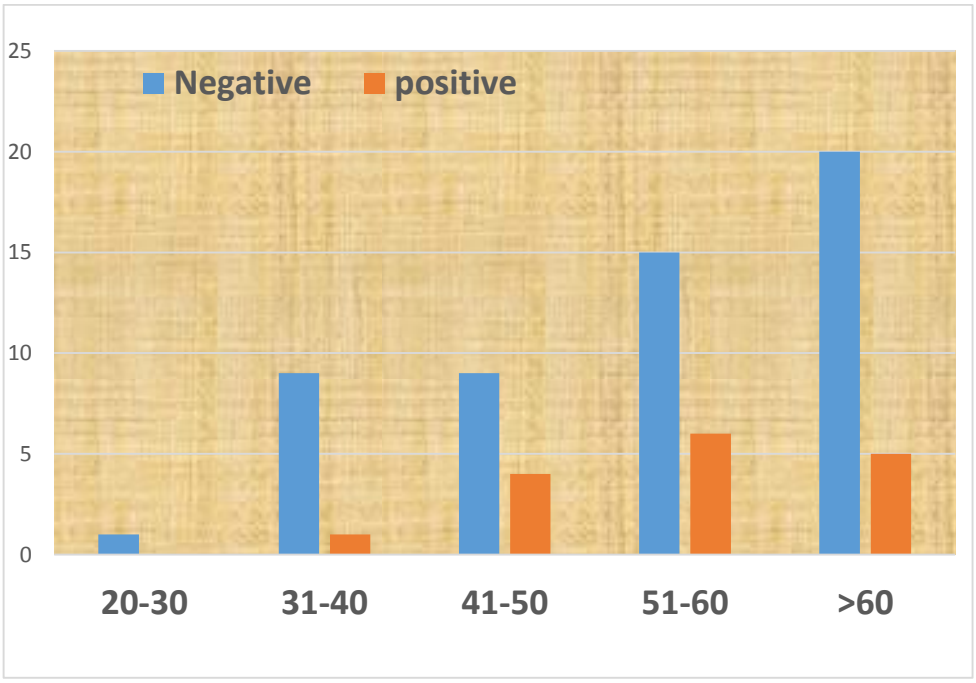


Of the 70 patients who were subjected for KOH mount 54 patients were negative forming 77.1% and 16 were positive forming 22.9% of cases.

Table 9: - Distribution of patients according to age group among KOH positive & Negative

Age (in year)	KOH		P value
	Negative	Positive	
20-30	1	0	0.699 NS
31-40	9	1	
41-50	9	4	
51-60	15	6	
>60	20	5	
Total	54	16	

Graph 9:- Distribution of patients according to Age group among KOH positive & Negative

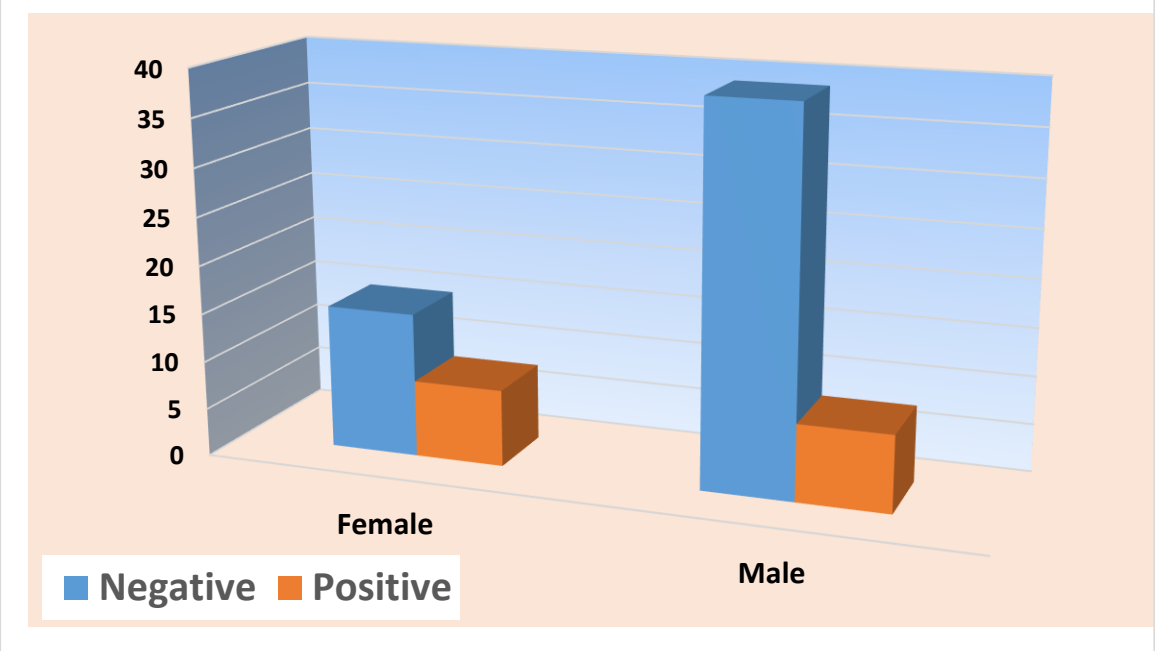


There is no significant difference among KOH positive & Negative between various age group.

Table 10: - Distribution of patients according to sex among KOH positive & Negative

SEX	KOH		P value
	Negative	Positive	
Female	15	8	0.96 NS
Male	39	8	
Total	54	16	

Graph 10 :- Distribution of patients according to sex among KOH positive & Negative

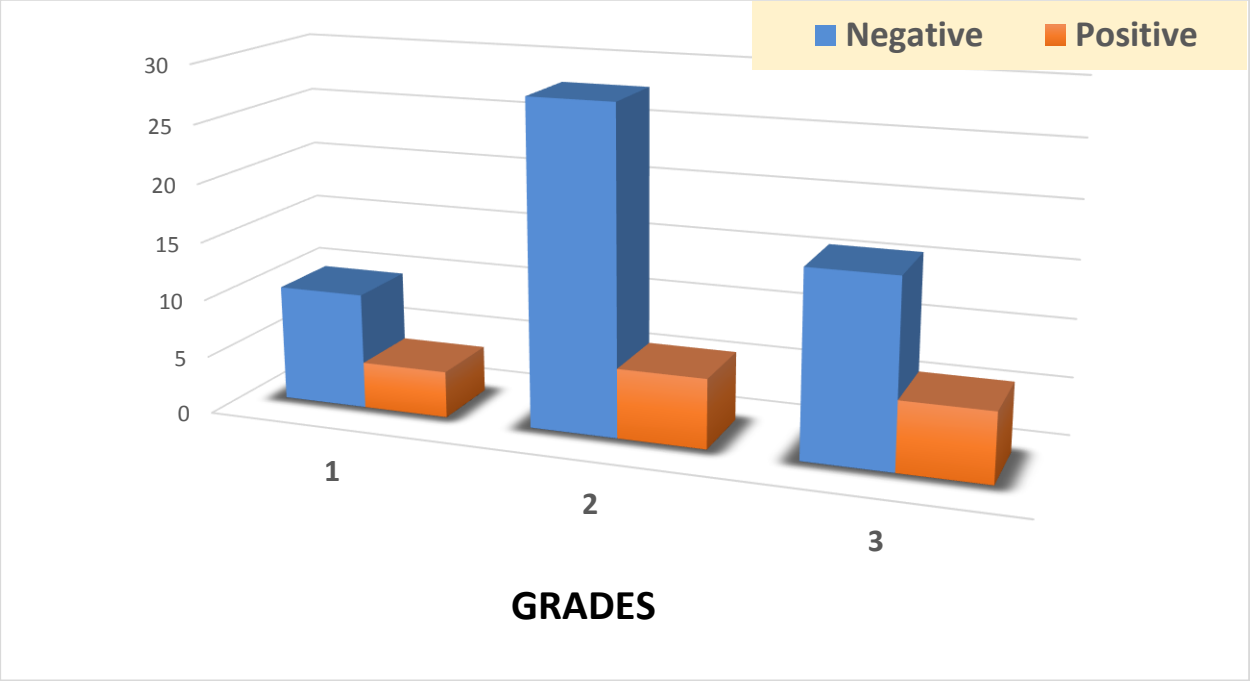


There is no significant difference among KOH positive & Negative between Male and Female.

Table 11: - Distribution of participants according to grade among KOH positive & Negative

	KOH		P value
GRADE	Negative	Positive	
1	10	4	0.499
2	28	6	
3	16	6	
Total	54	16	

Graph 11 :- Distribution of participants according to grade among KOH positive & Negative

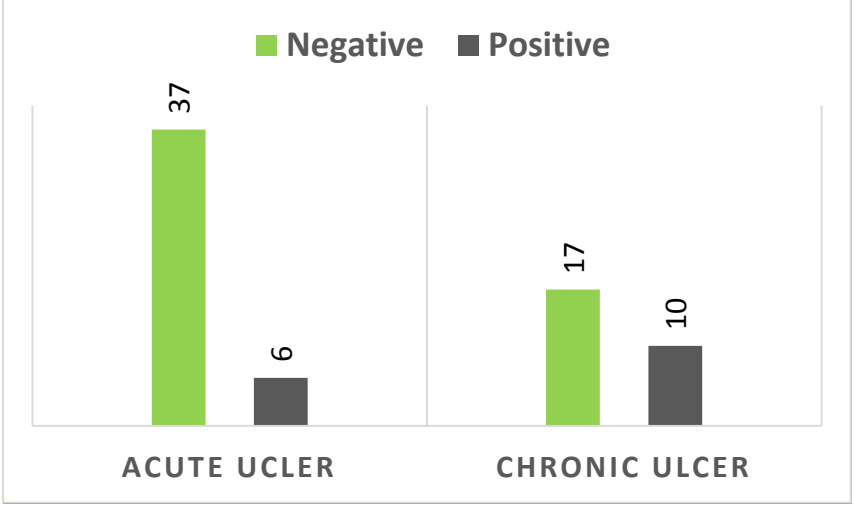


There is significant difference among KOH positive & Negative between various grades.

Table 12 : - Distribution of participants according to type of ulcer among KOH positive & Negative

Type of ulcer	KOH		P value
	Negative	Positive	
ACUTE	37	6	0.025
CHRONIC	17	10	
Total	54	16	

Graph 12 :- Distribution of participants according to type of ulcer among KOH positive & Negative cases.

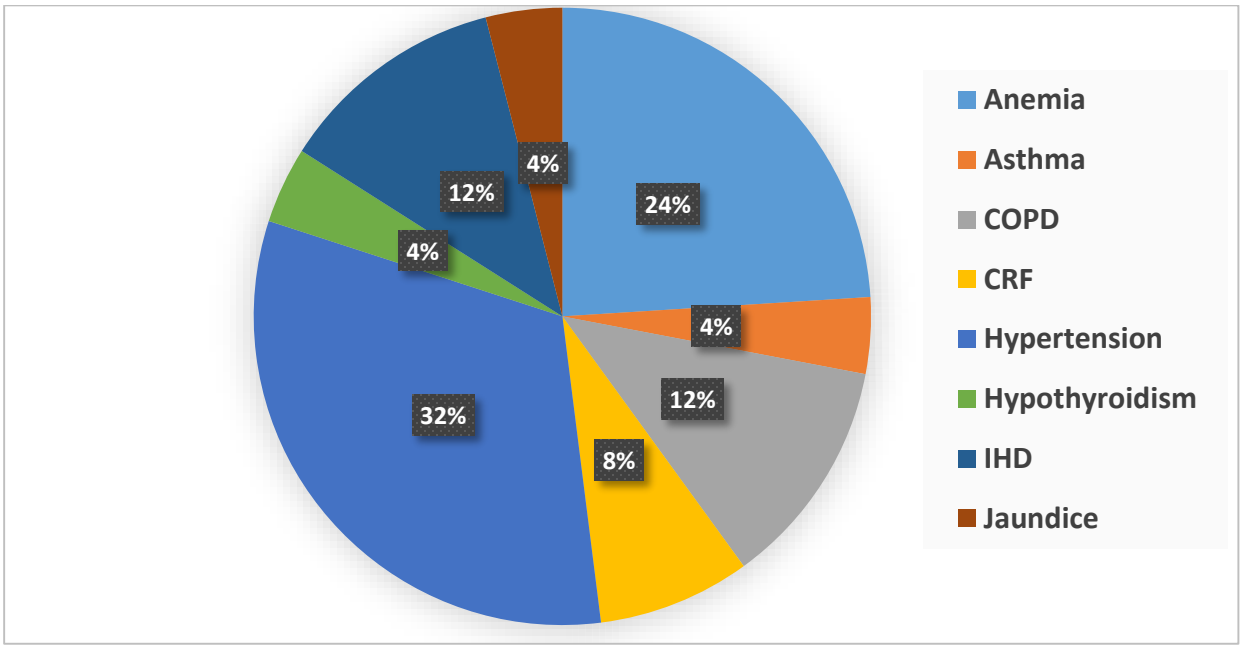


There is significant difference among KOH positive & Negative between Acute and chronic ulcer. KOH positive cases were most frequently noted in chronic ulcers than acute ulcers.

Table 13: - Co-morbidities associated with the patient.

CO-MORBIDITES	Frequency	Percent (%)
Anemia	6	24
Asthma	1	4
COPD	3	12
CRF	2	8
Hypertension	8	32
Hypothyroidism	1	4
IHD	3	12
Jaundice	1	4
Total	25	100.0

Graph 13:- Co-morbidities associated with the patient.

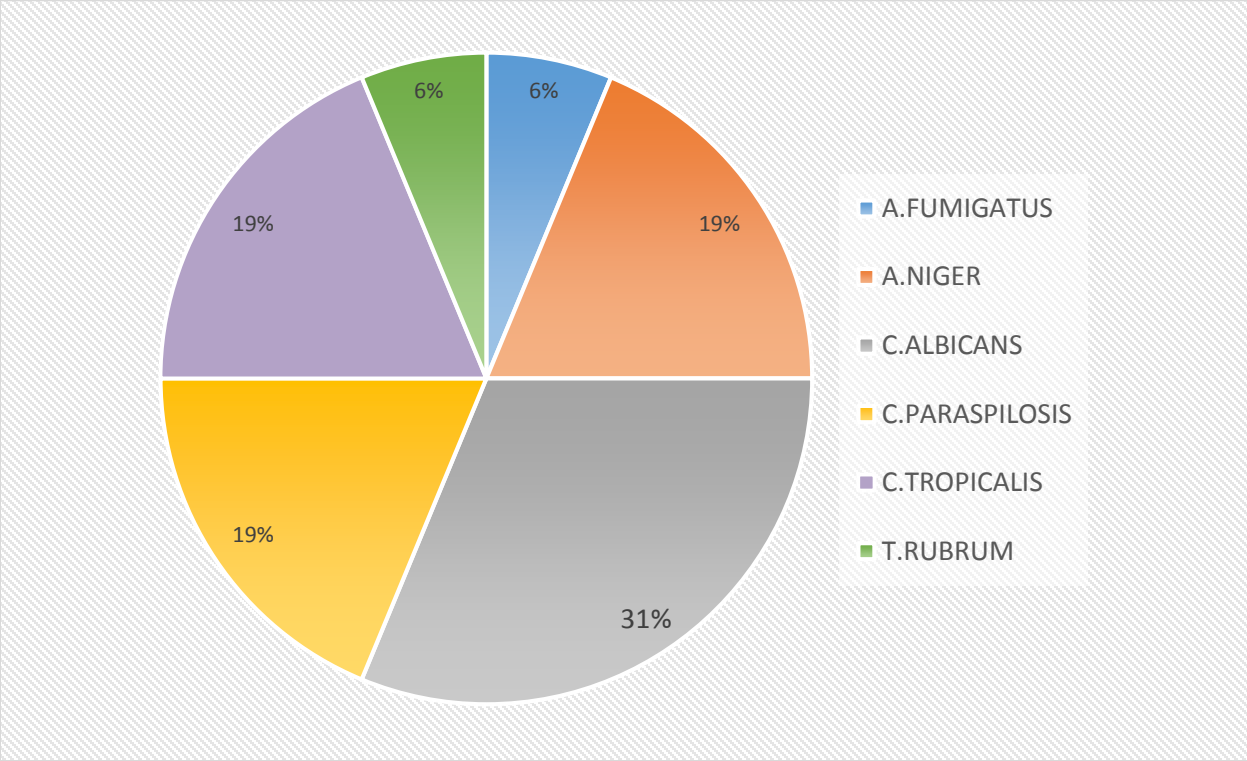


Out of 70 cases studied, 25 cases were found to have comorbidities with 8 (32%) cases of Hypertension, 6 (24%) cases of Anaemia, 3 (12%) Cases of IHD and COPD each, 2 (8%) cases of CRF, 1(4%) case of Asthma and Jaundice each.

Table 14: - Fungal isolates.

SDA C/S	Frequency	Percent
A.FUMIGATUS	1	6.25
A.NIGER	3	18.75
C.ALBICANS	5	31.25
C.PARASPILOSIS	3	18.75
C.TROPICALIS	3	18.75
T.RUBRUM	1	6.25
Total	16	100.0

Graph 14:- Fungal isolates.

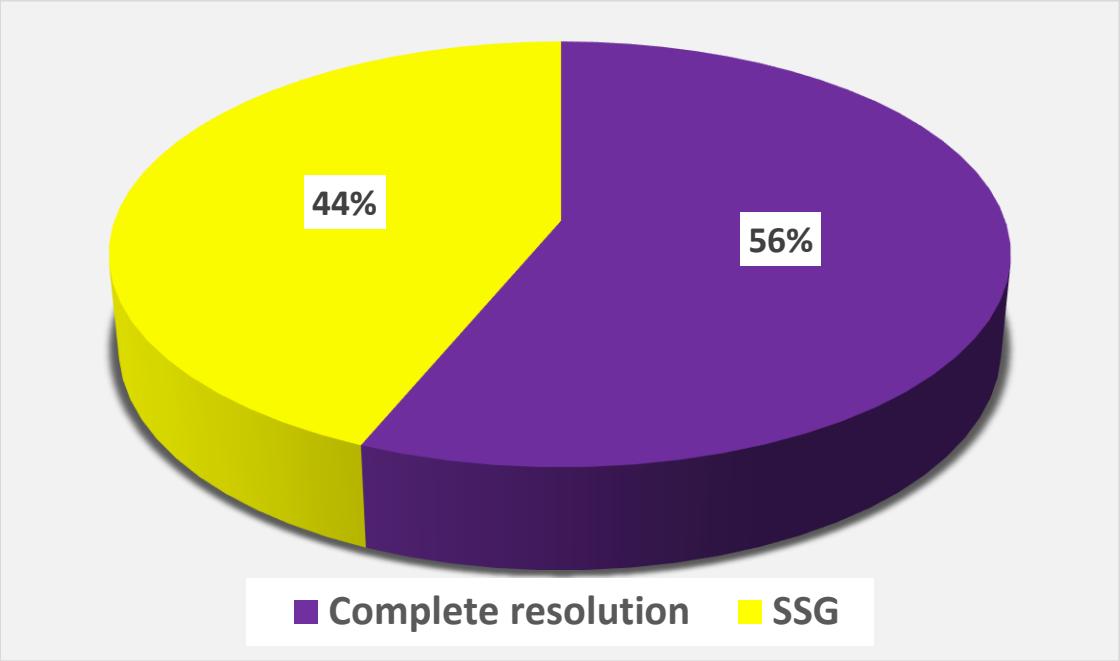


In 70 cases studied total of 16 cases were fungal infected. Most common fungus to be identified was C.Albicans in 5 cases (31%), followed by 3 cases each of C.Paraspilosis, C.Tropicalis & A.Niger, 1 (6%) case each of A.Fumigatus and T.Rubrum.

Table 15: - Outcome of ulcer after anti-fungal therapy

OUTCOME	Frequency	Percent
Complete resolution	9	56.25
SSG	7	43.75
Complications	0	0
Total	16	100.0

Graph 15:- Outcome of ulcer after anti-fungal therapy



Out of 70 diabetic foot subjects, 16 cases were found to be fungal infected. These patients after undergoing anti-fungal therapy, SSG was done for 7 cases and remaining 9 diabetic foot ulcers contracted completely without any complications.

FUNGAL ISOLATES

Out of 70 subjects studied, 16 (22.9%) of them had fungal infection which was confirmed by growth in Sabaroud's dextrose agar medium.

The list of all fungus isolated in our study and their characteristics are as follows:

1. **CANDIDA: 11 Cases**

Candida Albicans - 5

Candida Paraspilosis - 3

Candida Tropicalis - 3

Characteristics of the growth were as follows:

10% KOH Preparation – Yeast like cells seen

Gram Stain – Gram +ve, spherical, budding yeast like cells, approximately 3-6 µm in size, Pseudohyphae can be seen.

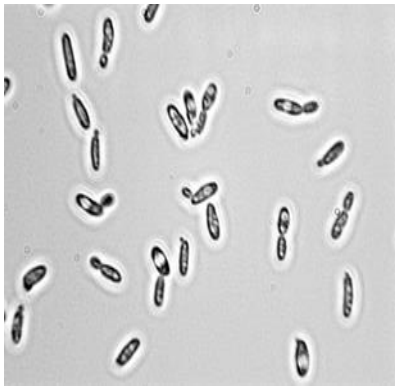
SDA Culture – MACRO: Cream, pasty colonies. Growth were seen at 37^{o}C in SDA tube without Actidione, Growth can be seen within 24hrs of inoculation.

MICRO: Gram Stain - Gram +ve, spherical, budding yeast, like cells Gram +ve along with Pseudohyphae.

Microscopic morphology of Candida species

Candida Albicans

Candida Paraspilosis



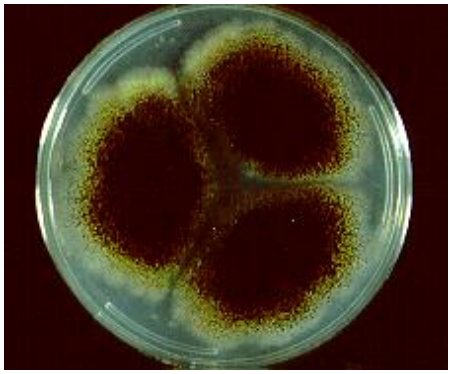
Candida Tropicalis

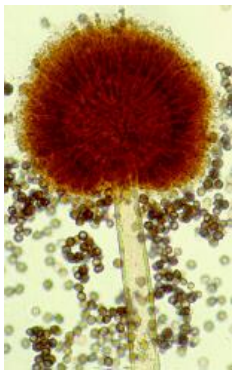
2. **<u>ASPERGILLUS</u> : 4 Cases**
   Aspargillus Niger - 3
   Aspargillus Fumigatus - 1

Characteristics of the growth were as follows:

<u>10% KOH</u> – Septate fungal filaments seen in all types.

<u>Aspergillus Niger</u>; SDA Culture – Growth was seen in 2-6 days. Growth were seen at room temperature in SDA tube without Actidione. **MACRO :** Black powdery colonies.



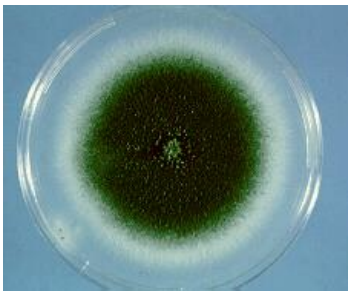


Microscopic morphology of Aspergillus Niger

<u>**A**spergillus Fumigatus</u>; SDA Culture - Growth occurred in about 4 – 6 days

Growth were seen at room temperature in SDA tube with Actidione.

MACRO : Bluish – green, powdery colonies seen.



Microscopic morphology of Aspergillus Fumigatus

3. <u>Tricophyton Rubrum</u> - 1 Case

Characteristics of the growth were as follows:

SDA Culture – Very stunted, white to cream, downy to glabrous colony with a pale yellow-brown reverse pigment.

Microscopic morphology of Tricophyton Rubrum

DISCUSSION

DISCUSSION

The present study was conducted at the department of general surgery, Sri Devaraj Urs Medical College, Kolar. This is a descriptive study of 70 cases who were treated for Diabetic foot Ulcers during the period of study from January 2015 to December2015.

1: Age distribution of patients studied (Table 16)

	Bansal *et al* [24]	Raja *et al* [52]	**PRESENT STUDY**
Mean age ± SD (in years)	57.04 ± 11.63	57.6	**55.8±13.05**
Age group	51 -70	51 – 70	**< 60**

Table 16: Age distribution

Subjects included in other studies also belonged to the same group with similar mean age, showing that foot complications are common in the elderly patients. All the patients included in our study were of Diabetes Mellitus Type II and 68 % of them had diabetic history > 5 years.

The present study shows that majority of the Diabetic foot patients were above 60 years (36%) followed by 51-60 years (30%), 41-50 years (19%), 31-40 years (14%) and 20-30 years (1%). This confirms the understanding that there is gradual rise in incidence of the diabetic foot infection as age advances because of increased number of co-morbid factors like atherosclerosis, hypertension, anaemia, CRF, conditions requiring steroid therapy and personal habits like smoking and alcoholism.

2: Gender distribution of patients studied (Table 17)

	Bansal *et al* [24]	**Raja *et al* [52]**	**PRESENT STUDY**
Male	79%	61%	**67%**
Female	21%	39%	**33%**

Table 17: Gender distribution

Of the 70 patients studied 47 were males forming 67.1% of cases & 23 females forming 32.9%. Similar finding are obtained in some studies like Bansal *et al* [24], 71% of the patients were males and 21 females. In other studies like Raja *et al* [52], 61% were males.

Men are more likely to have some of the independent predictors for diabetic foot ulceration such as peripheral arterial disease, Outdoor working, cigarette and alcohol use, and peripheral neuropathy. Peripheral neuropathy may be a particularly important factor in gender differences in diabetic foot ulcers. Sensory neuropathy is the most common type of neuropathy associated with diabetic foot ulceration and men have nearly twice the odds of having insensate neuropathy as women and have nerve conduction abnormalities that are more severe. The increased odds of sensory neuropathy in men are entirely due to the effect of height. Additionally, hormonal factors may play a role since women (particularly of reproductive age) may have additional neural protection afforded to them, due to better endothelial function in microvasculature. Despite having lower incidence rates of disease, several studies suggest women have higher mortality rates associated with diabetes-related lower extremity amputations.[52]

3: Duration of ulcer

The duration of ulcers in the present study ranged from 2 months to 2 years with Mean ± SD : 3.76 ± 1.39 months. Majority belonged to 4-7 months (43%) similarly, Emilija *et at* [25] study also had mean duration of ulcer to be 3-4 months.

4: Blood sugar levels of patient's studied and fungal isolation

Present study shows the correlation between fungal infection and Blood sugar levels. According to the present study patients with high blood sugar levels (140-179 mg/dl) are more vulnerable of developing fungal infected diabetic foot ulcer.

5: Grades of diabetic foot ulcers

Most of the ulcers included in present study belonged to Wanger's Grade II (48%) & Grade III (32 %) and were spontaneous in onset. However, other studies like Bansal *et al* [24] (Grade III – 35.9 %, Grade IV – 44.66 %) and Emilija *et at* [25] (Grade III – 36.4%, Grade IV – 31.87%) have encountered with higher number of Grade III & IV ulcers.

6: Fungal isolates (Table 18)

	Bansal *et al* [24]	Emilija *et al* [25]	**PRESENT STUDY**
Total subjects	103	509	**70**
Fungal isolates	9%	4.5%	**23%**
Most Common	Candida (29%)	Candida	**Candida (68.75%)**
2nd common	Aspergillus (21%)	-	**Aspergillus (25%)**

Table18: Fungal isolates – comparison with other studies

As shown in Table 18, fungal isolation was more in present study than compared to other studies like in Bansal *et al* [24] with 9% fungal growth and, candida & Aspergillus being commonest isolates in decreasing order. However Emilija *et al* [25] grew only Candida species.

7: Morphological classification of fungi

Based on morphology, all the fungal isolates obtained in our study can be classified as Yeasts and Molds. Yeasts were 11 in number (68.75%) and Molds were 5 (31.25%)

Among yeasts, Candida was the commonest – 11 no.s (8%) and among Molds, the highest incidence was Aspergillus species – 4 no.s (80%). Similar results obtained in other studies are shown in Table 19.

Table 19: Morphological classification of fungi – comparison

	Bansal *et al* [25] (%)	Seema nair *et al* [55] (%)	**PRESENT STUDY (%)**
Yeasts	50	66	**68.75**
Highest – candida	92	82	**100**
Molds	50	34	**31.25**
Highest – Aspergillus	42	65	**80**

Bansal *et al* [25] study had equal number of Yeasts and Molds isolated (50% each), but in Seema nair *et al* [55] study, yeasts (66%) were more than molds (34%) which is same as in our study where Molds were isolated more compared to Yeasts. However among Yeasts, Candida was the commonest to be grown, in all the studies including the present one and Aspergillus among Molds showed highest incidence in all.

8: Outcome

After treating the fungal infected diabetic foot ulcer, SSG was done for 7 cases and in remaining 9 diabetic foot ulcers, discharge was reduced, granulation tissue was well noticed and wound contracted completely by around 15 days.

9: Co-morbidities

Out of 70 cases studied, 25 cases were found to have co-morbidities with 8 (32%) cases of Hypertension, 6 (24%) cases of Anaemia, 3 (12%) cases of IHD and COPD each, 2 (8%) cases of CRF, 1(4%) case of Asthma and Jaundice.

This study is also supported by another study by Donald *et al* [23] in which it was noticed that comorbidities like anaemia, jaundice, COPD, CRF and hypothyroidism as a risk factors will increase the rate of fungal infection as well duration of ulcer in diabetic foot and he also observed that the cause of this was because of reduced immune competence and wound healing factors.

10: Mortality

Of the 70 diabetic foot ulcer patients studied, 16 patients developed fungal infection and no mortality was seen in this study.

On studying the factors influencing fungal growth in diabetic foot ulcers, it was seen that it was more common in Males, in the age group of above 60 years, and in ulcers of Wagner's Grade II & III, occurring spontaneously. However there were no significant correlation among these factors and fungal positivity. But duration of Diabetes and Blood sugar levels showed significant association with fungal infection.

However, in present study, it was found that ulcers of duration between 4 – 7 months showed a significant association with fungal positivity. This was probably due to the fact that, non-healing ulcers of longer duration on antibiotic therapy, show selective and immunomodulating actions on the microbial environment of the ulcers, favoring growth & replication of other commensals including fungus which are otherwise non-infective.

On follow-up, it was also noticed that ulcers with fungal infection on antifungal therapy had a good prognosis & course of ulcer progression, with significantly less amount of tissue loss (P value – 0.004**) and requiring only conservative management for ulcer healing.

Similarly, studies by Emilija *et al* [25] and Seema Nair *et al* [55], have shown poor treatment outcome without administration of specific therapy in fungal infected ulcers, thus justifying the introduction of systemic or local antifungal therapy in patients with verified fungal ulcer infections.

Statistical Method:

Descriptive and inferential statistical analysis has been carried out in the present study. Results on continuous measurements are presented on Mean ± SD (Min-Max) and results on categorical measurements are presented in numbers (%). Significance is assessed at 5% level of significance.

Chi-square test has been used to find the significance of study parameters on categorical scale between two or more groups.

Statistical Software:

The statistical software namely SAS 9.2, SPSS 15.0, Stata 10.1, MedCalc 9.0.1, Systat 12.0 and R environment ver.2.11.1 were used for the analysis of the data and Microsoft Word and Excel have been used to generate graphs, tables etc.

CONCLUSION

CONCLUSION

- ✓ Incidence of fungal infection in this study is 23%.
- ✓ Commonest fungi isolated were Candida species.
- ✓ Fungal infection was more commonly seen in long-standing (4-7 months), non-healing ulcers on antibiotic therapy.
- ✓ Grade II & III Diabetic foot ulcers are most vulnerable for fungal infection.
- ✓ Patients with Blood sugar levels between 140 – 179mg/dl are more susceptible for fungal infection.
- ✓ Treating these 16 proved cases of fungal infection with anti-fungal agents (systemic/ Local), showed improvement in disease status and a significant reduction in tissue loss, with faster and more conservative wound healing process.
- ✓ Though fungal infections are rare in diabetic foot, if present has to be treated with Anti-fungal therapy to prevent complications like chronicity, osteomyelitis and need of amputations.
- ✓ The present study signifies the need of a mycological evaluation of a non-healing diabetic ulcer of a longer duration, with poor progression despite antibacterial therapy and foot care and introduction of prudent antifungal treatment for proved fungal infections in Diabetic foot ulcers.
- ✓ We believe that our results will create awareness among clinicians, of the need to treat fungal infections of diabetic foot ulcers, as well as encourage further research into these infections.

SUMMARY

SUMMARY

- Foot infections are the major cause of morbidity in people with diabetes. Devitalized tissue is the site where the micro – organisms responsible for the non-healing ulcers inflict damage. Numerous studies have been carried out on the bacteriology of diabetic foot ulcers. Though there are a few reports on fungal pathogens in diabetic foot infections, there is a paucity of published work on the incidence of fungal pathogens in deep tissues.

- Randomly selected 70 cases of diabetic foot ulcers were studied for the occurrence of fungal infection. Majority of patients in present study were Males (67%) with a mean age of 55.8 years.

- In present study, the ulcers included were both acute & chronic type, of duration 5 days to 2 years (Mean duration of ulcer – 4 to 7 months) and most of them were Wagner's Grade II (48%) & Grade III (32%) types.

- Ulcers were managed regularly with appropriate dressings, Glycemic control, Anti-fungal treatment as per pus Culture & Sensitivity, relieving of pressure over the affected part of the foot was continued. Any surgical intervention required during the course of the study, based on ulcer progression was implemented.

- 16(23%) cases showed positive for fungal infection. Commonest fungi isolated were Candida (68.75%), followed by Aspergillus (31.25%). Anti-fungal therapy was started in these patients [Oral Fluconazole 150 mg OD weekly for about 6 weeks to 6 months depending on the progression of the ulcer along with Topical anti-fungal (Fluconazole/ Ketoconazole)].

- Progression of each ulcer was studied from the time of inclusion, both during hospital stay and after discharge, for 6 months and analyzed based on healing of ulcer, surgical intervention required and any tissue loss. Also factors influencing the fungal infection in diabetic ulcers in present study were analyzed.

- In present study, it was noticed that Ulcers with fungal infection on anti-fungal therapy had a good prognosis & course of ulcer progression, with significantly less amount of tissue loss (P value – 0.004) and requiring only conservative management for ulcer healing.

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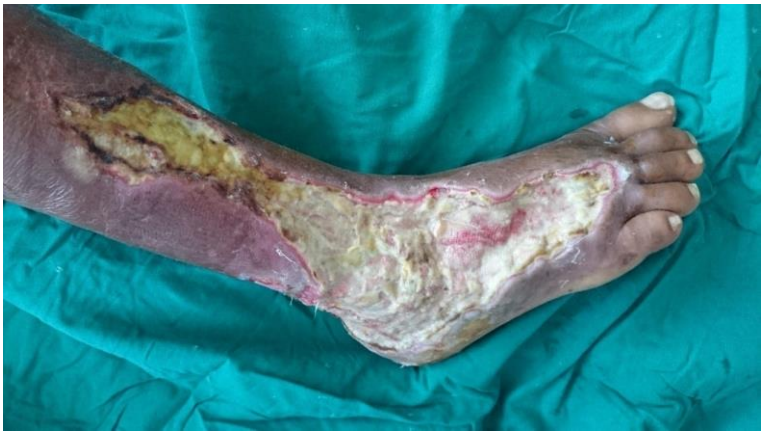

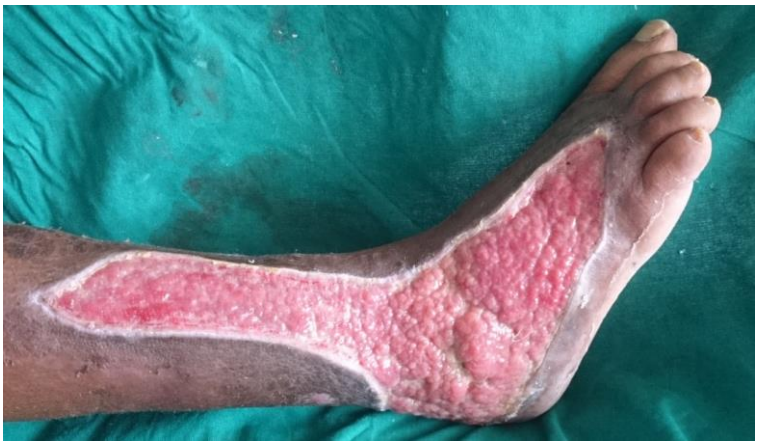

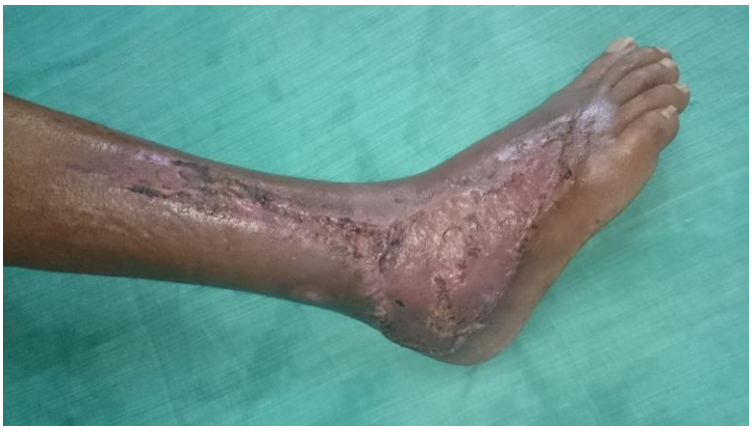

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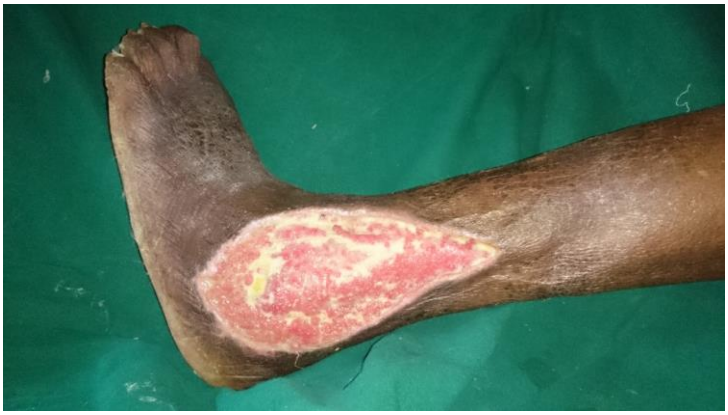

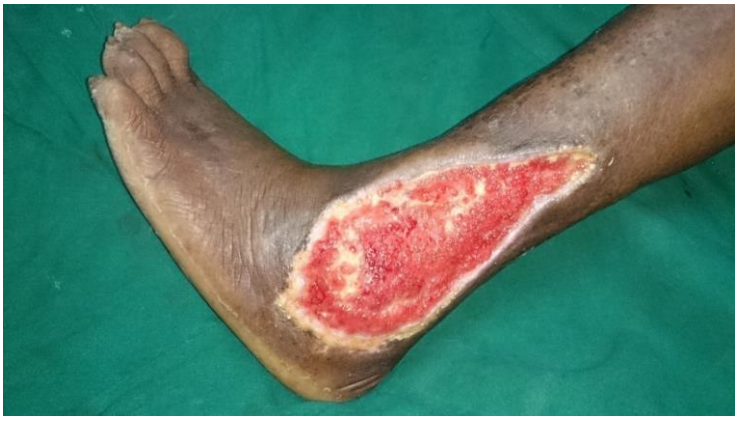

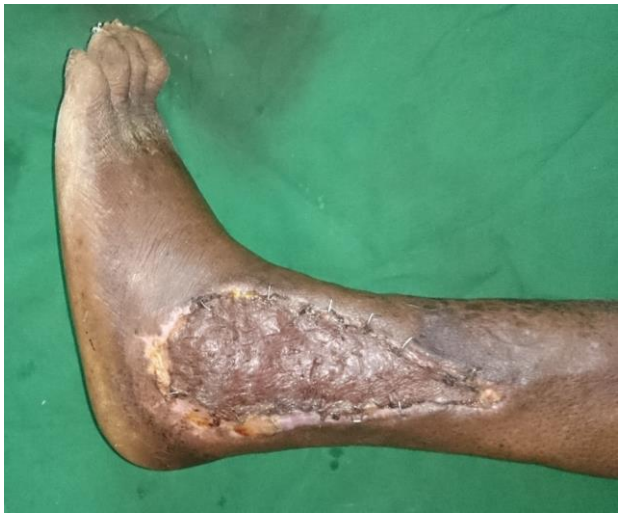

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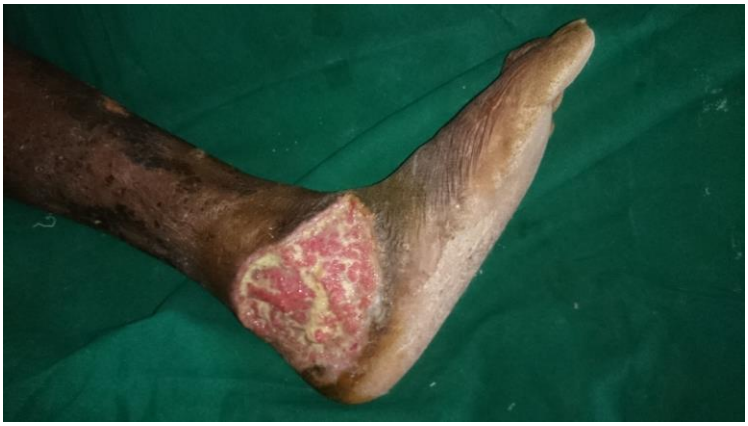

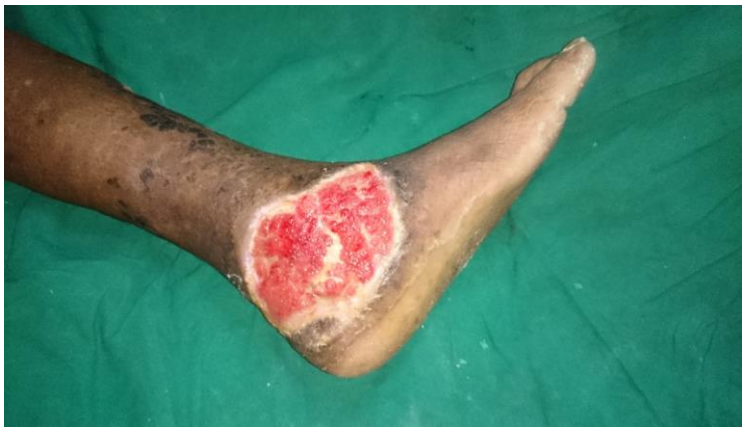

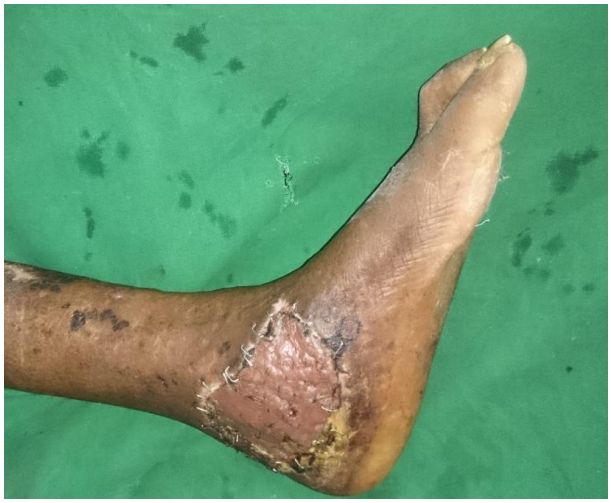

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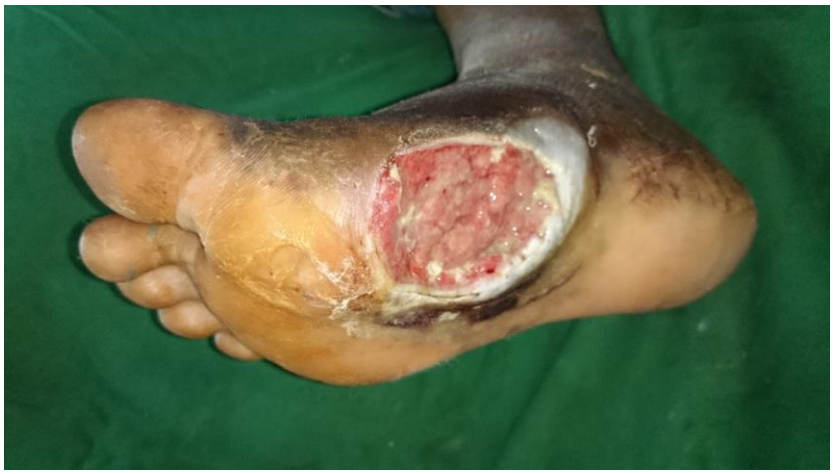

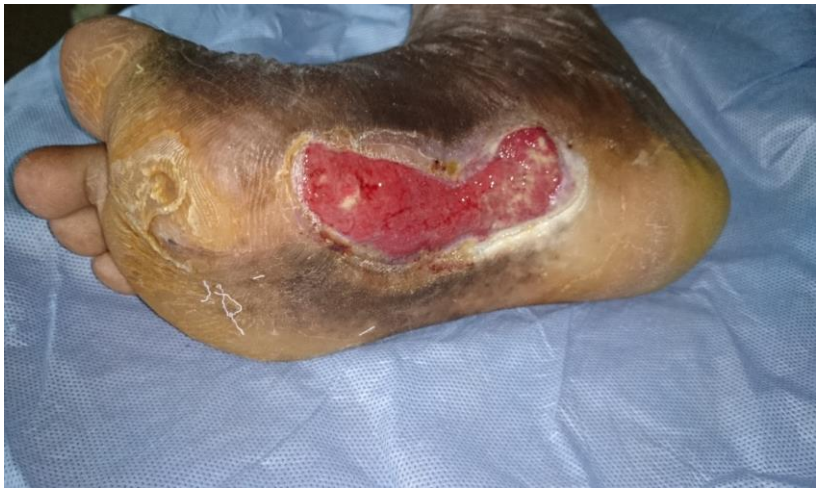

WOUND BEFORE AND AFTER ANTI-FUNGAL THERAPY

ANNEXURE 1: PROFORMA

PROFORMA

Case number –

Date of study –

Hospital No (OP/IP) –

Name: -

Age: -

Sex: - M / F

Address:-

Occupation:-

COMPLAINTS - Ulcer over _____ foot since ____ days /months/years

HISTORY OF PRESENT ILLNESS

1) Site –
2) Duration –
3) Onset –
4) Pain – Yes / No
5) Discharge – No / Yes, Purulent / serous / serosanguinous
6) Discoloration - Yes / No
7) Swelling of limbs – Yes / No
8) Weakness – Yes / No
9) Fever – Yes / No
10) Polyuria / Polyphagia / Polydypsia – Yes / No
11) Loss of appetite & Loss of weight – Yes / No
12) Inability to walk – Yes / No
13) Claudication pain – Yes / No
14) Rest pain – Yes / No
15) Vision – Normal / Abnormal

DIABETIC HISTORY

1) Age of onset -
2) Duration of diabetes -
3) Type of diabetes – TYPE 1 or TYPE II
4) Controlled or Uncontrolled
5) Type of Treatment – INSULIN / OHA/OR BOTH

Past History

Asthma, Epilepsy, Tuberculosis , Hypertension ,

Varicose veins, Arterial insufficiency, Any other illness – Y / N

Family history ;

Personal History;

.Diet ; Veg / Non-veg / Mixed

Addiction - Alcohol / Smoking / Tobacco / Betel nut / Any other.

General Physical Examination;

Appearance - Normal / Toxic

Built - Good / Moderate / Poor

Nourishment - Thin / Obese / Emaciated

Dehydration - present / absent

Pallor - Present/ Absent ,

Cyanosis -Present/ Absent

Clubbing - Present / Absent

Pedal edema- Present / Absent

Temperature - Febrile / Afebrile,

Pulse ; / min

BP ; mmHg

Local Examination; **ULCER**

Site –

Shape –

Size -

Number -

Edge –

Margin –

Floor –

Base –

Induration – Present / Absent

Type –

Surrounding skin – Normal / Edematous / Gangrenous

Rise in Local Temperature - Yes / No

Local Tenderness - Tender / Non tender

Any callosities / Soddening - Yes / No

Any Abscess/ Gangrenous changes - Yes / No

REGIONAL LYMPHADENOPATHY – Yes / No

PERIPHERAL NEUROPATHY – Yes / No

PERIPHERAL PULSES OF ALL LIMBS – Yes / No

VENOUS DRAINAGE OF THE LIMBS –

REFLEXES – Normal / Abnormal

JOINT MOVEMENT – Normal / Abnormal

DEFORMITIES – Claw Toe / Hallus Valgus / Hammer Toe / NIL

Systemic examination :

1 .CVS :

2 .RS :

3. CNS

4. P/A :

GRADING – Grade 1 / Grade 2 / Grade 3

Routine Investigations;

Haemoglobin: g/dl

HBA1c: %

GRBS : mg/dl

PPBS : mg/dl

FBS : mg/dl

Doppler Study :

KOH REPORT :

SDA - CULTURE AND SENSITIVITY REPORT :

SUMMARY

GRADE OF ULCER –

FUNGAL GROWTH, IF ANY -

ANTI –FUNGAL THERAPY – Systemic -

- Local -

SURGICAL INTERVENTION DONE –

OUTCOME OF DISEASE –

ANNEXURE 2: MASTER CHART

SERIAL NO	NAME	AGE	SEX	OP / IP no	DOS	MOO	DURATION	BSL	GRADE	CO-MORBIDITES	KOH -Ve	KOH +Ve	SDA C/S	TOPO
1	S G	55	M	60737	03-01-2015	Trauma	2M	133	2		Negative			
2	V k	75	F	70404	03-01-2015	Spontanous	7M	157	3		Negative			
3	A R	50	M	90235	17-01-2015	Spontanous	5M	156	2	Anaemia		Candida species	C.ALBICANS	SSG
4	R K	52	M	98729	21-01-2015	Trauma	20D	110	1		Negative			
5	S r	35	M	84487	28-01-2015	Trauma	1M	126	3	Asthma	Negative			
6	B k	59	M	97271	28-01-2015	Burns	9M	194	2	Hypertension	Negative			
7	K L	53	M	83889	09-01-2015	Spontanous	4M	185	1	CRF		Dermatophytic fungi	T.RUBRUM	Complete resolution
8	R V	60	M	107725	09-02-2015	Trauma	2Y	170	3	Anaemia	Negative			
9	K N	59	M	107866	09-02-2015	Spontanous	1M	128	2		Negative			
10	M G	62	M	83882	01-03-2015	Trauma	20D	133	3	Hypertension	Negative			
11	T S K	65	M	110496	16-02-2015	Spontanous	2M	129	3		Negative			
12	R	50	M	53369	23-02-2015	Burns	25D	122	3	Hypertension	Negative			
13	R M	60	M	114583	23-02-2015	Trauma	12D	138	1		Negative			
14	C H	60	M	108809	03-03-2015	Spontanous	7M	148	2	Jaundice		Budding Yeast cells	C.ALBICANS	SSG
15	V	85	F	31249	03-03-2015	Spontanous	6M	156	1	Anaemia		Budding Yeast Cells	C.ALBICANS	Complete resolution
16	A	65	M	122621	19-03-2015	Spontanous	2M	170	1	CRF	Negative			
17	B	35	M	123952	18-03-2015	Burns	12D	166	2		Negative			
18	R	73	M	129286	03-04-2015	Trauma	17D	134	1	COPD	Negative			
19	P	70	F	122963	14-03-2015	Spontanous	20D	126	3	IHD		Fungal elements seen	A.FUMIGATUS	SSG
20	L	65	F	121621	31-03-2015	Spontanous	5M	132	2	Hypothyroidism	Negative			
21	R K	46	M	55199	01-04-2015	Spontanous	20D	140	2		Negative			
22	M A	65	M	133298	14-04-2015	Trauma	4M	161	2		Negative			
23	R S	60	F	124365	15-04-2015	Spontanous	45D	116	3			Budding Yeast Cells	C.ALBICANS	Complete resolution
24	K K N	58	M	134632	15-04-2015	Spontanous	2M	138	2		Negative			
25	P	52	F	139109	27-04-2015	Spontanous	2Y	124	2			YEAST CELLS SEEN	C.ALBICANS	Complete resolution
26	R	57	M	140137	29-04-2015	Trauma	7M	116	2		Negative			
27	L	32	F	101242	07-05-2015	Trauma	15D	130	1		Negative			
28	F	54	F	73475	08-05-2015	Spontanous	4M	136	2		Negative			
29	S	45	F	148140	20-05-2015	Spontanous	1Y	156	2		Negative			
30	K R	70	M	147384	20-05-2015	Spontanous	20D	115	3		Negative			
31	P K	75	F	145407	21-05-2015	Trauma	5M	149	1		Negative	Budding Yeast	C.TROPICALIS	Complete resolution
32	S S	51	M	145810	23-05-2015	Trauma	20D	118	2	Hypertension	Negative			
33	M	56	M	148189	23-05-2015	Spontanous	2M	130	3	Anaemia	Negative	Septate fungi	A.NIGER	SSG
34	T V	56	M	145563	23-05-2015	Burns	5D	168	2		Negative			
35	S	32	F	149653	25-05-2015	Trauma	10D	136	2		Negative			
36	A R	68	M	151110	18-05-2015	Spontanous	1M	122	3		Negative			
37	S G B K	73	M	149613	30-05-2015	Spontanous	6M	138	2		Negative			
38	R	58	M	1021425	02-06-2015	Spontanous	17D	116	2		Negative			
39	N R	65	M	148655	02-06-2015	Spontanous	22D	128	1		Negative			
40	S R	29	M	153647	13-06-2015	Trauma	2M	140	2	COPD	Negative			
41	R	40	M	155782	13-06-2015	Spontanous	20D	138	3		Negative			
42	N	43	M	157915	15-06-2015	Spontanous	4M	118	3		Negative			
43	S	60	F	159348	20-06-2015	Burns	10D	130	2		Negative			
44	S	65	F	162344	23-06-2015	Spontanous	15D	145	2	Anaemia	Negative			
45	D R	65	M	161550	24-06-2015	Spontanous	5D	167	2			Budding Yeast	C.TROPICALIS	SSG
46	M	65	M	160695	29-06-2015	Trauma	7D	126	1		Negative			
47	M	40	M	159323	02-07-2015	Spontanous	4M	144	3		Negative			
48	P N	65	M	167043	15-07-2015	Spontanous	1Y	131	2		Negative			
49	V	36	M	173817	26-07-2015	Spontanous	12	155	3		Negative			
50	V	48	F	177337	04-08-2015	Spontanous	5M	168	3			Fungal elements seen	A.NIGER	Complete resolution
51	D S	52	M	179093	07-08-2015	Trauma	25D	138	3		Negative			
52	R	75	F	181411	10-08-2015	Spontanous	3M	130	2		Negative			
53	K V	42	F	182468	13-08-2015	Trauma	10D	138	3		Negative			
54	P	35	F	51812	24-08-2015	Spontanous	20D	140	2		Negative			
55	T R	75	M	24845	30-08-2015	Spontanous	2M	134	2		Negative			
56	L V	65	F	196128	21-09-2015	Trauma	25D	145	3			Septate fungi	A.NIGER	Complete resolution
57	M	60	F	199089	23-09-2015	Trauma	2M	137	2		negative			
58	V M	45	M	200882	29-09-2015	Spontanous	3M	140	2	Hypertension	negative			
59	S	37	F	202922	03-10-2015	Burns	15D	156	1		Negative			
60	J R R	65	M	202333	05-10-2015	Spontanous	20D	135	2	Hypertension	Negative			
61	R	60	F	204091	09-10-2015	Trauma	4M	131	3	IHD		Budding Yeast	C.PARASPILOSIS	Complete resolution
62	H B	65	F	193743	09-10-2015	Trauma	12D	138	3	Anaemia	Negative			
63	V M	42	M	7319	10-10-2015	Trauma	20D	127	2		Negative			
64	S P	43	M	183716	16-10-2015	Spontanous	6M	153	1	IHD	Negative			
65	G	45	M	208639	17-10-2015	Trauma	2M	128	1		Negative	Yeast cells seen	C.PARASPILOSIS	Complete resolution
66	A	34	M	217376	23-11-2015	Spontanous	7M	148	2			Budding Yeast	C.TROPICALIS	SSG
67	M	50	M	226130	28-11-2015	Spontanous	4M	154	2	COPD		Oval budding yeast	C.PARASPILOSIS	SSG
68	S	50	M	225218	03-12-2015	Trauma	2M	136	1	Hypertension	Negative			
69	M Z	75	F	229814	07-12-2015	Trauma	4M	140	3		Negative			
70	N	76	M	237209	24-12-2015	Trauma	2M	131	2	Hypertension	Negative			

ANNEXURE 3: KEY TO MASTER CHART

KEY TO MASTER CHART

M	-	MALE
F	-	FEMALE
IP/OP	-	In patient / Out patient
DOS	-	Date of study
MOO	-	Mode of onset
BSL	-	Blood sugar level
D	-	Days
M	-	Months
Y	-	Years
KOH –ve	-	KOH negative
KOH +ve	-	KOH positive
SDA	-	Sabouraud's Dextrose Agar
C/S	-	Culture & Sensitivity
CRF	-	Chronic renal failure
COPD	-	Chronic obstructive lung disease
IHD	-	Interstitial heart disease
SSG	-	Split –skin grafting
IOFU	-	Intervention on follow up