A PROSPECTIVE STUDY OF ANALYSING PREDICTIVE FACTORS FOR MAJOR LOWER EXTREMITY AMPUTATIONS IN DIABETIC FOOT INFECTION

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CHENNAI

BONAFIDE CERTIFICATE

This is to certify that this dissertation titled **"A PROSPECTIVE STUDY OF ANALYSING PREDICTIVE FACTORS FOR MAJOR LOWER EXTREMITY AMPUTATIONS IN DIABETIC FOOT INFECTION"** submitted by **DR.B.SATHIYASEELAN** to the faculty of General Surgery, The Tamil Nadu Dr. M.G.R. Medical University, Chennai in partial fulfilment of the requirement for the award of MS degree Branch-I General Surgery, is a bonafide research work carried out by him under our direct supervision and guidance from

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DECLARATION

I**, DR.B.SATHIYASEELAN** solemnly declare that the dissertation titled **"A PROSPECTIVE STUDY OF ANALYSING PREDICTIVE FACTORS FOR MAJOR LOWER EXTREMITY AMPUTATIONS IN DIABETIC FOOT INFECTIONS"** has been prepared by me. This is submitted to **The Tamil Nadu Dr.M.G.R. Medical University, Chennai,** in partial fulfilment of the regulations for the award of MS degree (Branch I) General Surgery.

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CONTENTS

INTRODUCTION

Diabetes is a common disease causing lower extremity amputation throughout the world, particularly in India, the Diabetic capital of the world. By 2025, it is estimated that India will have world's majority of diabetics. Diabetes Mellitus is the most important cause of non-traumatic amputations worldwide. Amputations are perhaps the most feared complication of Diabetes.

In 2011 around 8.3% of world population had diabetes. Among these, vast majority were inhabiting in developing nations. It is projected that by the year 2030, around 10% of the human race would become diabetics. The troublesome fact is that this disease is being diagnosed more and more in the younger population of the world. Each year, over one million amputations are being performed to alleviate disease caused by diabetes. This roughly works out as one amputation in the world every 30 seconds. The predisposing factors that lead to amputation are diabetic neuropathy, bony deformities, inconspicuous trauma, and vascular diseases. In the presence of an ulcer, local sepsis and vascular insufficiency are major causes of amputation. The presentation of lesions greatly differ based on socio-economic status, quality of foot care and usage of footwear. It has been projected that one in six diabetics living in developed nations will develop ulceration before they die. This problem is even

more menacing in developing nations. Diabetic foot not only affects the individual but also their family and the community as a whole. It causes great strain on the financial and health care infra-structure of the nation. Another cause of concern is the emergence of type 2 Diabetes in children. These cases will eventually progress to develop micro and macrovascular complications including life-threatening infections at an early age. Around 25% of these cases will be non-healing and upto 28% will end up in amputation. Investing in a scientific foot care techniques and guidelines will be more cost effective in the long run. Amputations alter quality of life and longevity. Amputations are associated with an increased risk of re-amputation and at an increased mortality in first decade after amputation. Early diagnosis and prompt therapy is mandatory. A team approach can reduce the number of amputations. Adequate infra-structure and facilities are essential. However, ignorance on the part of the patients and also the health care provider has made this goal hard to attain.

AIMS AND OBJECTIVES OF THE STUDY

1. To formulate a scoring system using various contributory factors for early detection of unsalvageable diabetic foot in our locality.
2. To analyse those various factors which contribute to major amputations in lower extremity in diabetic foot patients.
3. To study about common bacterial organisms which complicate diabetic wounds and their antibiotic sensitivity pattern using tissue cultures in our locality.

DESIGN OF STUDY

Prospective observational study

PERIOD OF STUDY

March 2015 to September 2016

SELECTION OF STUDY

Inclusion criteria

- All adult patients with diabetic foot infections admitted in Dept. of General Surgery, Govt. Rajaji Hospital, Madurai during the period of study.

Exclusion criteria

- Immunocompromised individuals like HIV, TB, Malignancy.
- Those who are not willing to participate in the study.
- Those who expired during the stay at hospital.

CONSENT

Individual written and informed consent

COLLABORATIVE DEPARTMENT

Department of Microbiology

PARTICIPANTS

All patients from casualty, surgery OPD and ward admitted with diabetic foot infections at Govt. Rajaji Hospital , Madurai

METHODOLOGY

SOURCE AND DATA

All patients diagnosed to have diabetic foot infection.

METHODS OF COLLECTION OF DATA

1. Details of cases
2. Full history
3. Clinical examination
4. Biochemical investigations
5. Radiological investigations
6. Bacteriological tissue culture

CLINICAL EXAMINATION

Complete inspection and palpation of the ulcer especially for presence of gangrene, pulse status of concerned part, ankle brachial index (ABI) and bony involvement.

BIOCHEMICAL INVESTIGATION

1. Hb%
2. TC/DC
3. ESR

4. CRP

5. LIPID PROFILE

6. RENAL FUNCTION TESTS

RADIOLOGICAL INVESTIGATION

X-ray of concerned local part

BACTERIOLOGICAL TISSUE CULTURE

Gram positive and gram negative bacteria and their antibiotic sensitivity pattern.

CARDIOVASCULAR SYSTEM

Echocardiogram

DIABETES MELLITUS - HISTORY

Diabetes as a disease started in approximately 1550BC. Egyptian Papyrus records it as a disease that causes rapid weight loss and frequent urination. Greek physician Aretaeus of Cappodoc IA(81-133AD) described it as a disease in which limbs and flesh melt down meaning "a flowing through". Galen said, Diabetes disease process has an affliction to the kidneys. The term Diabetes was coined by Appllonius Memphites. The term Mellitus was coined by Thomas Willis meaning "honey sweet" in 1675. In1776 Dapson first demonstrated excretion of large amounts of sugar in urine & circulating blood in diabetic patients . The discovery of Glycogenesis in Liver is a landmark in history of Diabetes. Claude Bernard said, diabetes is caused by high glucose synthesis, in 1800's. During 18th and early 19th century glycosuria has been accepted as a diagnostic feature of Diabetes due to metabolic derangement. In 1869-Islets cells were discovered in pancreatic tissue by Paul Langerhans. Pancreas as pivotal in causation of the disease was elucidated by Mering & Minkowski in 1889. Discovery of Insulin and its practical application by Banting and Best 1921 is a major milestone. Roger Hirsworth discovered two types of Diabetes in 1935. In 1950, oral medications of Diabetes came into existence. In 1961,first injection of Insulin was developed by Decton-Dickson. Various researches are going on for the treatment of Diabetes.

REVIEW OF LITERATURE

HISTORICAL ASPECT OF DIABETIC FOOT

In 19th and 20th century, disease of lower limb in diabetic patients was designated as 'gangrene in diabetic foot' or as 'diabetic gangrene'. The significant distinction between gangrene due to Vascular insufficiency and gangrene due to infection in the limb with a normal or near normal blood supply was not made until 1893.

Aseptic surgery improved the survival of amputation flaps. Diabetic foot disease is the major cause of diabetic mortality surpassing hyperglycemia coma at present.

The discovery of insulin has reduced the need of intervention to some extent. The major cause of diabetic foot disease were infection ischemia and neuropathy. The emphasis on preventive care and health education about diabetes should be appreciated.

EPIDEMIOLOGY OF DIABETICS AND DIABETIC FOOT IN INDIA

In 2002 in India about 32million people were affected by diabetes. The international diabetes federation estimated about 40.9million diabetic patients in India and also estimated that it will rise up to 69.9million in 2015.

In India about 3.6percent people are affected by diabetic foot ulcers in clinic population. Factors such as walking on barefoot rituals like walking on fire, ill fitting shoes and chapels and poor knowledge regarding foot care leads to increase incidence of diabetic foot complications in India.

INTRODUCTION TO DIABETIC FOOT

Foot ulcers, microbial infection and Charcot neuropathic Osteoarthopathy are the three major foot complications of diabetes that will mostly lead to gangrene and hence to amputation. As a result they are one of the major causes of hospitalization for diabetic patients which can lead to huge expenditure for the country every year. A multidisciplinary team approach tends to decrease the complications of diabetic foot to major extent.

The pathophysiology of diabetic foot should be well understood to treat or to prevent diabetic foot and its major complications.

WHO DEFINITION OF DIABETIC FOOT

The foot of a patient that has a potential risk of pathologic consequences including infection, ulceration, and/or destruction of deep tissues associated with neurological abnormalities, various degrees of peripheral vascular disease and /or metabolic complications of diabetes in the lower limb.

ETIOLOGY AND RISK FACTORS

The most common precursor for lower limb amputations in diabetic patients is foot ulceration. The various risk factors attributed to diabetic foot are peripheral neuropathy,vascular disease, restricted joint mobility, foot deformities, abnormal foot pressures,minor trauma, previous history of ulceration or amputation and defective vision.

RISK FACTORS FOR ULCERATION

1. Peripheral sensory neuropathy
2. Structural foot deformity
3. Injury and ill fitting shoes
4. Callosities
5. Previous ulcers/amputation
6. Abnormal foot pressures
7. Restricted joint mobility
8. Elevated blood glucose levels
9. Duration of diabetes
10. Impairment of vision
11. Extremes of age
12. Other comorbidities

Peripheral neuropathy in the absence of trauma is the major factor for diabetic foot ulcerations. This was revealed by recent multicentric studies. Around 45-60percent of diabetic ulceration are purely neuropathic, while 30 -45percent have booth neuropathic and ischemic components.

Motor neuropathy will result in atrophy of anterior crural muscles, hence muscle wasting may lead to foot drop,equinus hammertoes, prominent planter metatarsal heads.

Autonomic neuropathy may lead to dry skin which in turn leads to cracking and fissuring which creates a port for entry of bacteria.

Autosympathectomy associated with sympathetic failure, arteriovenous shunting, micro vascular thermo regulatory dysfunction impairs perfusion of normal tissues and microvascular responses to injury. All these processes will subsequently leads to ulceration.

Foot deformities, abnormal biomechanics, congenital disorders, or previous surgery will lead to high focal foot pressures. All these factors leads to vulnerable areas in the foot predisposing to ulceration. Although plantar aspect of foot is commonly affected, footwear irritation may cause ulceration in dorsal and medial aspect of foot. Such deformities might include prior partial amputation, prominent metatarsal heads, hammer toes, Charcot arthropathy, or hallux valgus.

Foot trauma in a patient with peripheral sensory neuropathy is a significant cause of ulceration. Besides puncture wounds and blunt injuries to the foot, Even trivial trauma such as repetitive stress from walking and day to day activities will lead to callus formation. Ill fitting shoes which may lead to trauma to a foot may add insult to injury in turns leads to ulceration.

Peripheral vascular disease as such does not cause ulcers directly in a diabetic foot. But once ulcer develops then arterial insufficiency will lead to prolonged healing and causes an increased risk for amputations. And in addition to that infections are very difficult to treat due to poor oxygenation and poor delivery of antibiotics to the infection site owing to arterial insufficiency. And hence early recognition and aggressive treatment of lower extremity ischemia is therefore vital in lower limb salvage.

Restricted joint mobility is now described as a potential risk for ulcers. Cheiroarthropathy is the stiffening of capsular structures and ligaments which is due to glycosylation of collagen in patients with long standing diabetes mellitus. The subsequent reduction in ankle, subtalar,

first metatarsophalangeal joint mobility has shown to result in high focal pressures with increased risk of ulceration.

MECHANISM OF INJURY

Diabetic foot ulcers have multi factorial etiology. Foot deformities and neuropathy cause skin breakdown in persons with diabetes by the following two mechanisms.

- Injuries due to continuous low pressure (ill fitting shoes).
- Injuries due to chronic repetitive trauma from walking.

Prolonged low pressure over bony prominence (i.e., bunion or hammer toe deformities). Various studies shows shoe trauma in association with loss of protective sensation and concomitant foot deformity, are major precipitating events leading to foot ulceration in diabetes.

Prolonged repetitive moderate stress normally occurs over the sole of

foot and is related to prominent metatarsal heads, atrophied or anteriorly displaced fat pads, structural deformity of the lower extremities, and prolonged walking. Rigid deformities such as hallux valgus, hallux rigidius, hammer toes and limited range of motion of ankle subtalar and metatarsophalangeal joints also may lead to the development of ulceration in diabetic foot patients. Other biomechanical perturbations including partial foot amputations, also will have the same adverse effects in diabetic foots.

ANATOMY OF LOWER LIMB

ANATOMY OF THE SOLE OF THE FOOT

SKIN

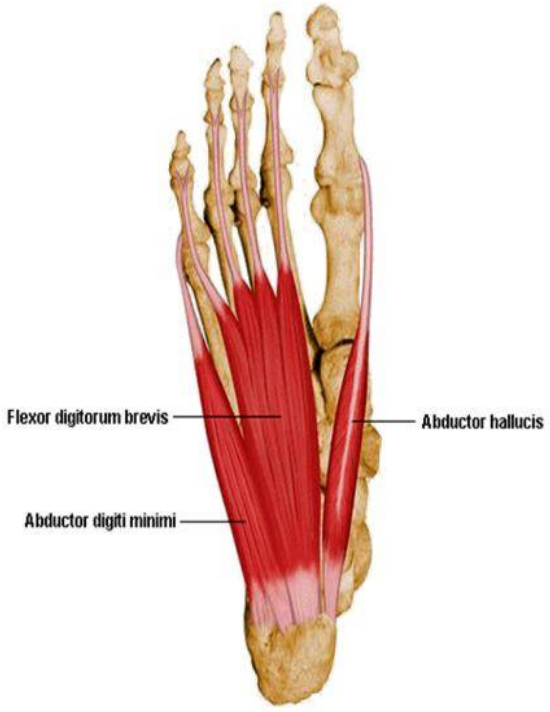
It is thick , firmly attached to the deep fascia by fibrous bands. sweat glands are present in large numbers. The sensory nerve supply to the medial skin of sole is derived from the tibial nerve, lateral part of the sole is innervated by corresponding plantar nerve,and its Medial two thirds is innervated by medial plantar nerve.

DEEP FASCIA

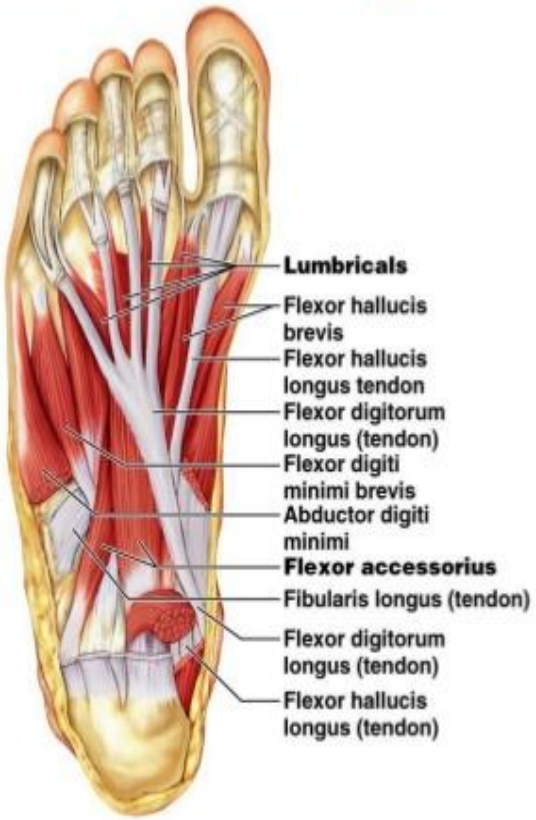
Deep transverse metatarsal ligaments and aponeurosis together constitute deep fascia. Flexor retinaculam extends from the Medial malleolus to the medial surface of calcaneum. It attaches the tendons of deep muscles to the medially in the ankle. The aponeurosis occupies the central area of Sole, triangular in shape and its apex is attached to the calcaneum, and its base attached to the toes. Each slip has two bands, passing superficially to the skin and deeply to the root of toe. Fibrous septa form the fascial spaces of sole, gives firm attachment to the overlying skin. It protects the underlying vessels, tendons, nerves. It helps to maintain the arches in the foot.

1st Layer

- **Three Muscles:**
  1) Abductor hallucis
  2) Flexor digitorum brevis
  3) Abductor digiti minimi



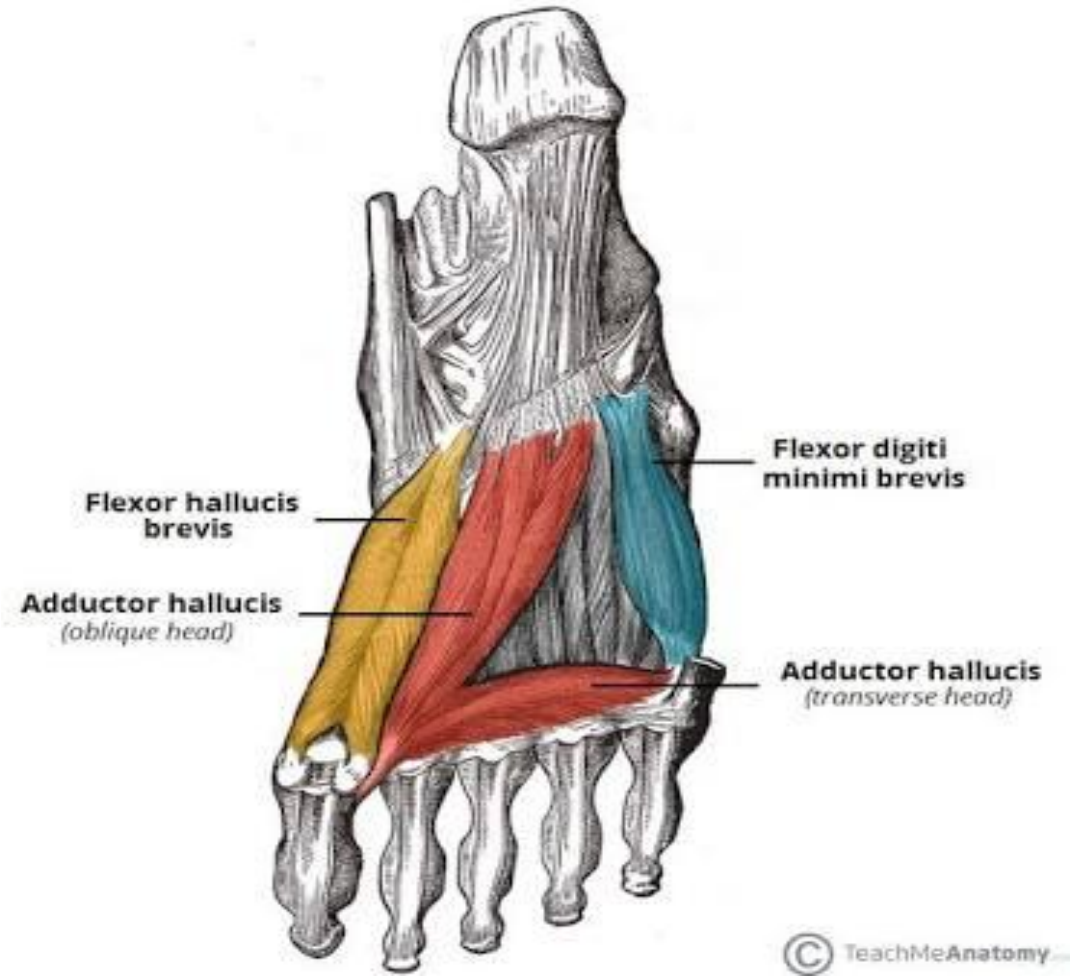
Plantar Muscles: Second Layer



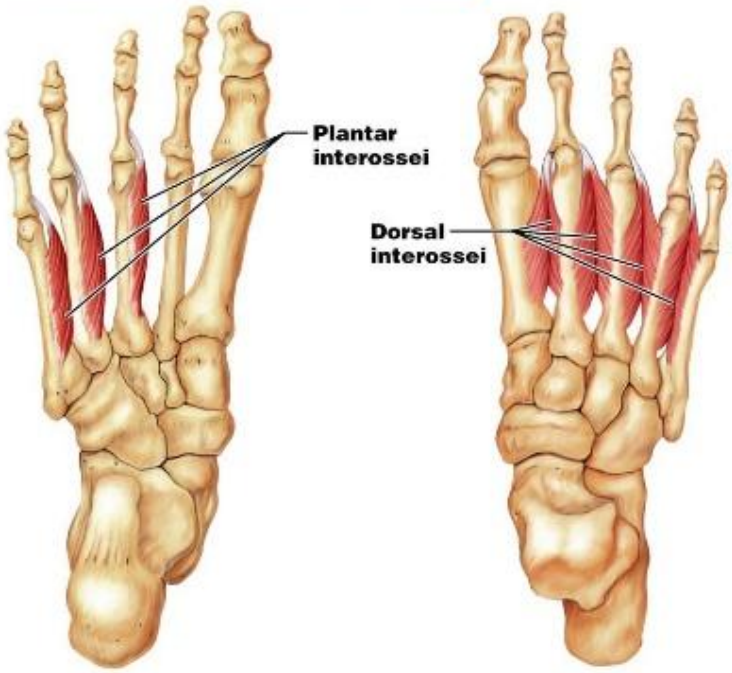
(b) Second layer

Figure 10.25b

THIRD LAYER


Flexor digiti minimi brevis
Flexor hallucis brevis
Adductor hallucis (oblique head)
Adductor hallucis (transverse head)
© TeachMeAnatomy

Plantar Muscles: Fourth Layer



(e) Fourth layer: plantar interossei

(f) Fourth layer: dorsal interossei

Figure 10.25e–f

MUSCLES OF THE SOLE

They are divided into four layers.

I Layer Abductor hallucis

Abductor digiti minimi

Flexor digitorum brevis

II Layer Lumbricals

Flexor hallucis longus

Flexor digitorum longus

III Layer Flexor hallucis brevis

Flexor digiti minimi brevis

Adductor hallucis

IV Layer Interossei

Tibialis posterior

Peroneus longus

ARTERIAL SUPPLY

1.Medial Plantar Artery

Medial side of toe is supplied by MPA, gives off numerous cutaneous, muscular and articular branches.

2.Lateral Plantar Artery

Plantar arch is supplied by LPA. Plantar arch gives digital arteries to lateral side of the small toe and both sides of four lateral toes.

3.Dorsalis Pedis Artery(DPA)

DPA joins with the plantar artery on the lateral aspect and supplies plantararch . Its branches are

-Lateral tarsal artery

-First dorsal metatarsal artery which supplies both side of big toe.

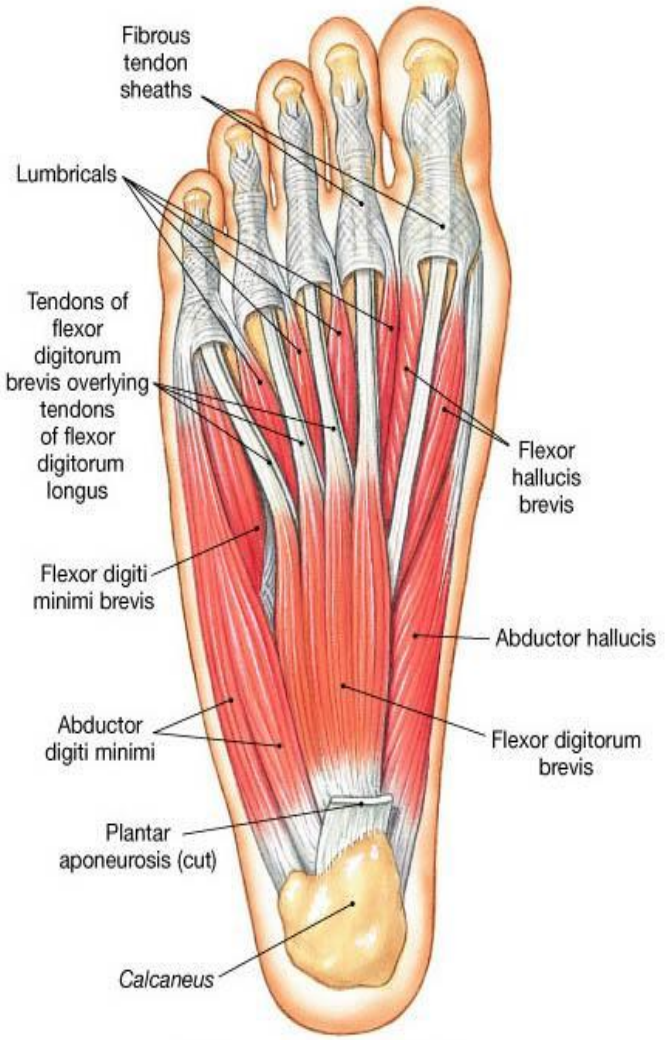
-Arcuate artery supplies metatarsal branches to the toes.

Medial and lateral plantar veins accompany the corresponding arteries and join behind the medial malleolus to form the posterior tibial venae commitanes.

NERVE SUPPLY

1. Lateral Plantar Nerve

Supplies flexor digit minimi, abductor hallucis, quadaratus, plantaris, abductor digiti minimi, second and third and fourth lumbricals and all interossei. It supplies cutaneous twigs to the skin sole on lateral aspect, one and half toes on the lateral aspect and its nail beds and tips.



(d) Plantar view, superficial layer

2.Medial Plantar Nerve

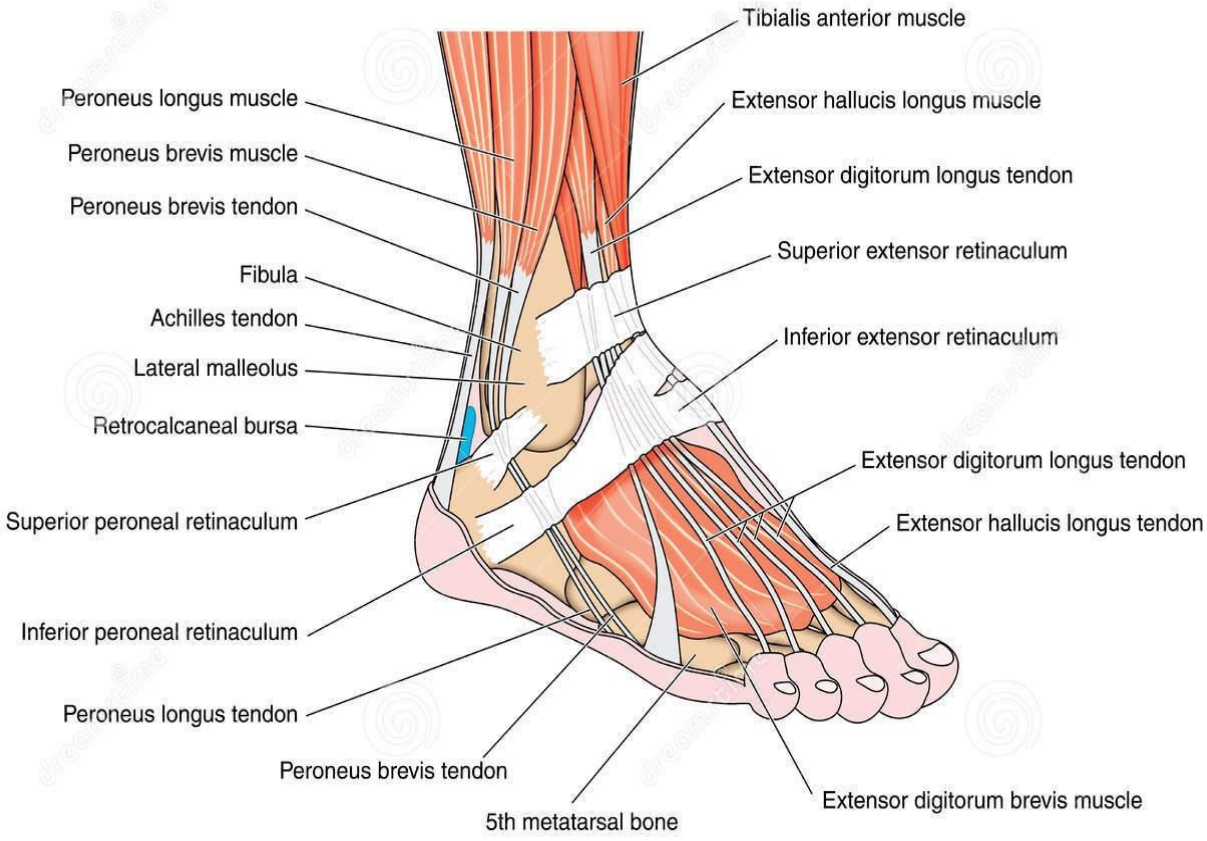
Supplies muscular branches to abductor hallusis, flexor hallusis brevis, flexor digitorum brevis. It also supplies cutaneous branches to medial three and half toes and its corresponding nail beds and tips.

DORSUM OF THE FOOT

SKIN

Skin on the dorsal aspect is freely mobile on the underlying tendons and bones. Sensory nerve supply to the dorsum is derived from the superficial peroneal nerve. It also receives sensory innervations by deep peroneal,saphenous, sural nerves.The skin covering the dorsal surfaces of terminal phalanges, nail beds are supplied by medial and lateral plantar nerves. Knowledge of the spaces of the foot is very important because infections in any of the spaces result in extension along fascia or tendon. The four medial spaces can be approached from inside of the foot by an incision along inner border of first metatarsal bone. The digital vessels arise from dorsal arch pass via intermetatarsal ligaments to the toes. Infection of the toe or web space if not adequately controlled spreads deeper. It reaches the tendon sheaths of long flexors or lumbricals and spread to 3rd layer. Very high pressure build up in the closed area because of pus, edema, and presence of gas forming organisms. This causes tissue necrosis and mechanical pressure on digital vessels leads to gangrene of the foot.

DORSUM OF THE FOOT



ANATOMY OF THE LEG

Superficial fascia

Contains superficial veins like great saphenous vein, short saphenous vein, cutaneous nerves like infrapatellar branch of saphenous nerve, saphenous nerve,lateral cutaneous nerve of calf, superficial peroneal nerve, sural nerve, lymphatics, and small unnamed arteries.

Deep fascia

Extension of deep fascia form the septa divide the leg into three compartments anterior, posterior and lateral.

Anterior compartment

Muscles	Tibialis anterior
	Extensor hallucis longus
	Extensor digitorum longus
	Peroneus tertius
Vessels	Anterior Tibial vessels
Nerve	Deep peroneal nerve

Lateral Compartment

Muscles	Peroneus longus
	Peroneus brevis
Nerve	Superficial peroneal nerve
Vessels	Peroneal vessels

MEDIAL SIDE OF THE LEG

Formed by medial surface of the shaft of tibia. The greater part of this surface is subcutaneous and is covered by skin and superficial fascia. Three muscles are inserted into the upper part of medial surface of the tibia from three compartments of the thigh namely Sartorius, gracilis, and semitendinosus forming Guy ropes.

BACK OF THE LEG

Superficial fascia of the back of the leg contains small and great saphenous veins and their tributaries, several cutaneous nerves,and medial and lateral calcaneal arteries.

Superficial muscles of this area are

- Gastronemius
- Soleus
- Plantaris

Nerve supply to superficial muscles of the back is Tibial nerve.

Posterior group of muscles that are present in deep aspect are

• Popliteus

• Flexor digitorum longus

• Flexor hallucis longus

• Tibialis posterior

Vascular supply to this area by Posterior tibial vessels.

ANATOMY OF THE THIGH

FRONT OF THE THIGH

The superficial fascia of the front of the thigh contains great saphenous vein, cutaneous nerves, vessels, lymphatics and lymph nodes.The upper third of the thigh medially contains the femoral triangle, middle third carries the femoral vessels through the adductor canal. Muscles of the frontal aspect of the thigh are

- Sartorius
- Rectus femoris
- Vastus lateralis
- Vastus intermedius
- Vastus medialis

Nerve supply : Femoral Nerve

MEDIAL ASPECT OF THE THIGH

Muscles

Adductor longus

Adductor brevis

Adductor magnus

Gracilis

Pectineus

Obturator externus

Nerve supply

Obturator nerve

Accessory obturator nerve

Arterial supply

Obturator artery

Medial circumflex femoral artery

BACK OF THE THIGH

Muscles

Semitendinosus

Semimembranosus

Biceps femoris

Nerve supply Sciatic nerve

Vascular supply

Lateral circumflex femoral

Medial circumflex femoral vessels

PATHOPHYSIOLOGY OF DIABETIC ULCER FOOT

The pathophysiology of diabetic foot is due to the following factors

• Neuropathy

• Angiopathy

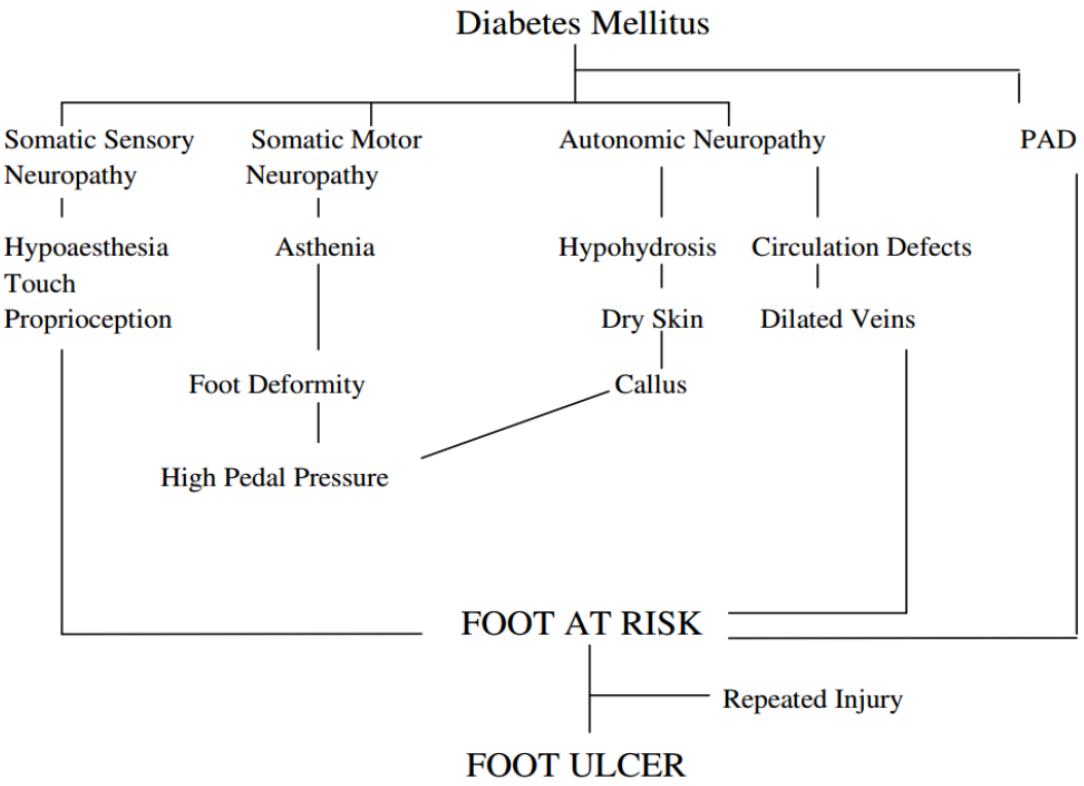
• Mechanical stress

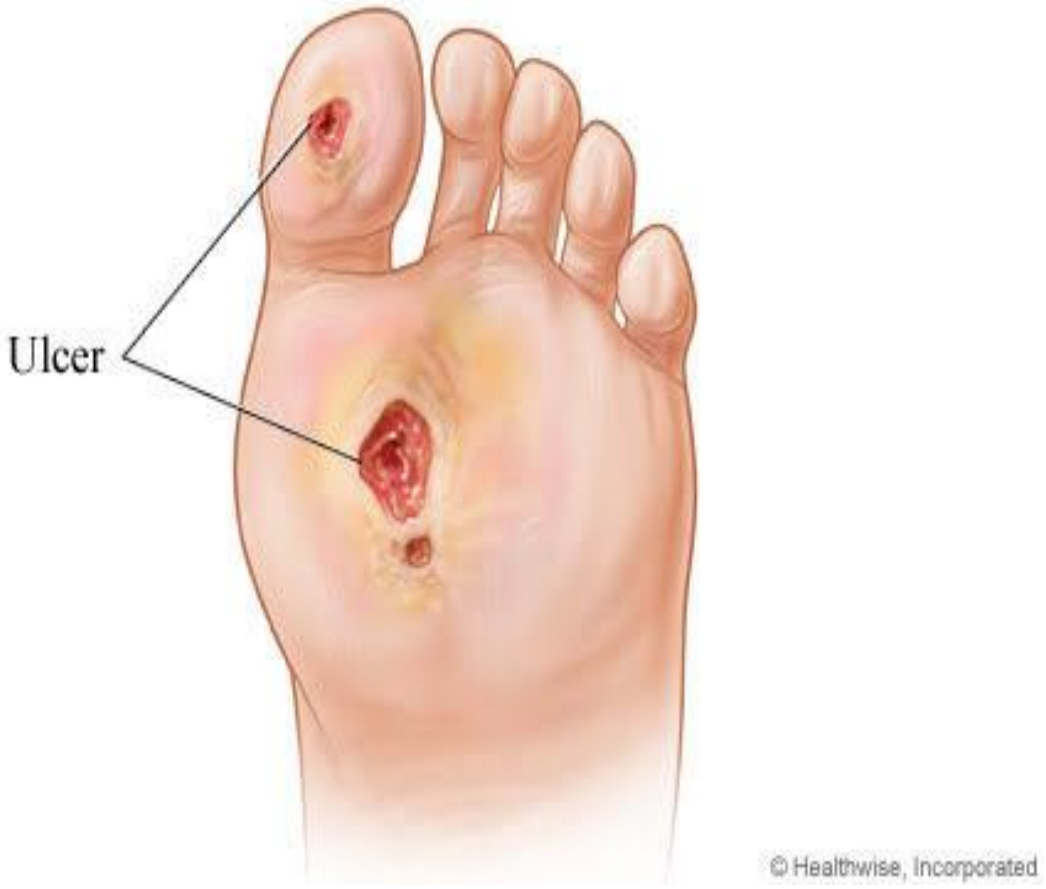
• Faulty wound healing

• Metabolic derangement

• Patient and provider's neglect

PATHWAY TO DIABETIC FOOT ULCER




Ulcer
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DIABETIC NEUROPATHY

Polyneuropathy is one of the commonest complications of Diabetes .Most common among neuropathies in patients with diabetes are chronic sensorimotor distal symmetric polyneuropathy and the autonomic neuropathies.

Sensory deficit starts in the lower limb distally and progresses to involve feet and legs in a ‘stocking’ pattern. This is followed by the upper extremeties in a ‘glove’ like pattern. Diabetes affects autonomic nervous system also. As the disease progresses wasting of the small muscles of the hand and asthenia of the limb occurs. Sensory loss is the main clinical symptom. Some patient may experience tingling, burning pain , shooting pain down the legs. Neuropathic pain is nocturnal and causes insomnia. They may develop postural hypotension, depressive symptoms . A paradoxical feature is that both pain and numbness may co-exist in the same limb, a phenomenon aptly named as ‘painful, painless’ leg. The former is due to C fibre damage and the latter due to A fiber damage. The commonest clinical presentation is reduced sense of vibration in the toes. Ankle jerk is absent and with advancement of disease knee reflex also gets involved. Capillary circulation is an important component, the

alteration of which, can lead to neuropathy. Vasa Vasorum involvement hinders the clearance of metabolic end products from the tissue and also prevents nutrient delivery, thereby producing nerve damage . Autonomic neuropathy leads to hypohidrosis and predisposes to breakage of dry skin. The 'purely' neuropathic foot is actually warm because of abnormal A-V shunting. This abnormal increase in blood flow can lead to neuropathic oedema. The American Diabetologist Association recommends screening for neuropathy, at the time of diagnosis and for autonomic neuropathy five years after initial diagnosis in Type 1 and at the time of diagnosis in Type 2 Diabetes followed by annual screening thereafter.

Diabetic polyradiculopathy is a neurological manifestation characterized by disabling pain along the course of one or more nerve roots. Truncal radiculopathy causes pain over the thorax and abdomen. Involvement of the lumbar plexus or femoral nerve may cause severe pain in the thigh or hip and have diabetic amyotrophy. All these are usually self-limiting and resolve over 6-12 months.

Mononeuropathy is less frequently seen compared to polyneuropathy. It presents with pain and paresis along the distribution of a particular nerve. Involvement of Occulomotor Nerve is most common, heralded by diplopia. Examination shows ptosis, opthalmoplegia with normal pupil reaction to light. 4th , 6th and 7th cranial nerves may be affected. Peripheral mononeuropathies with concomitant affliction of more than one nerves may occur.

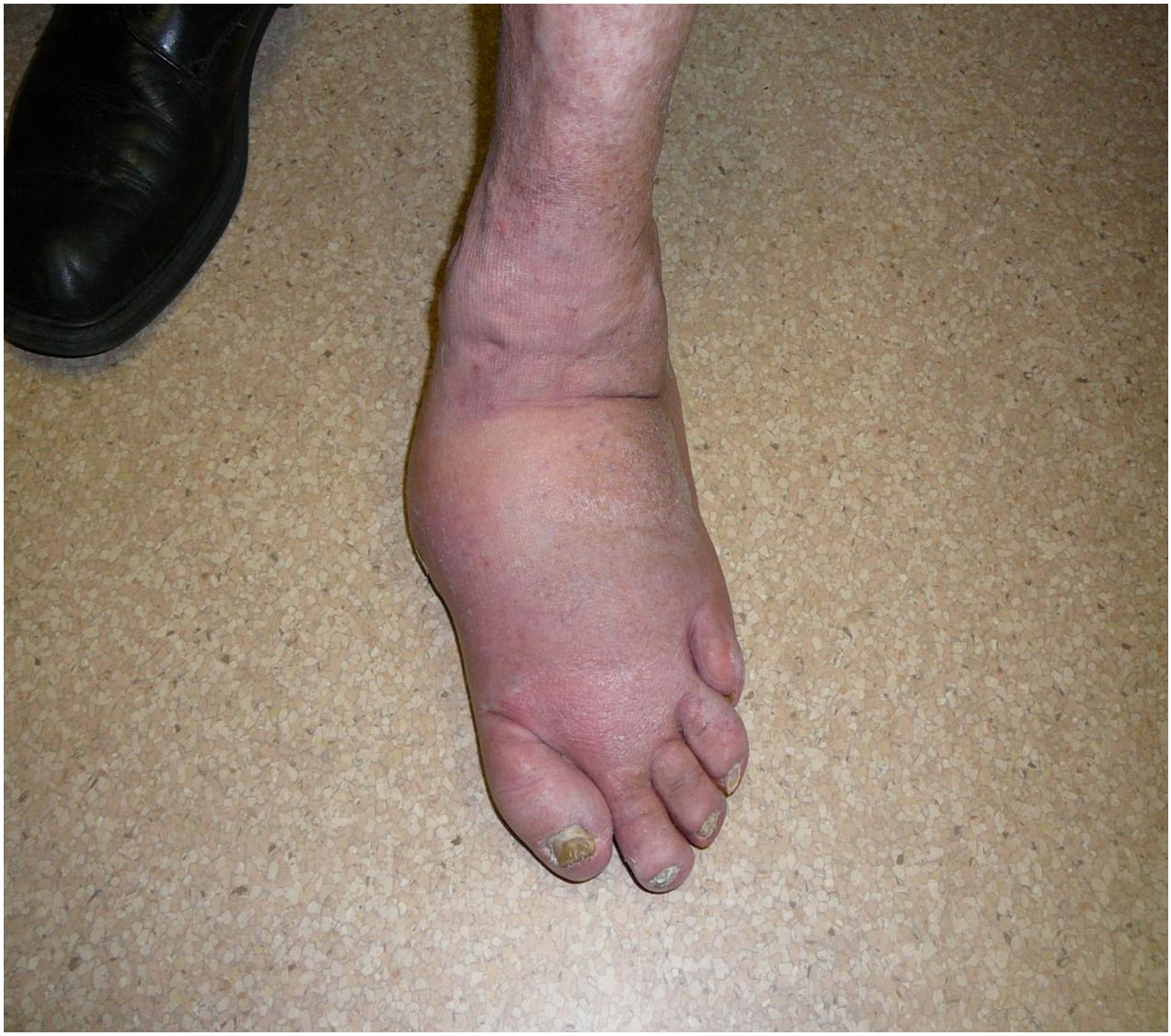
Autonomic neuropathy can involve multiple systems. Cardiovasular effects include tachycardia at rest and orthostatic hypotension. Gastroparesis and bladder voiding abnormalities may be seen. Diabetic cystopathy is the inability to sense bladder fullness and failure to empty the bladder completely. It can cause impotence and sexual dysfunction in both male and female. Increased sweating of the upper extremities and anhydrosis of the lower extremities may occur. Anhydrosis leads drying and crackling of feet which increases chances of ulcer formation. Peripheral neuropathy plays a key role in the events leading on to amputation.

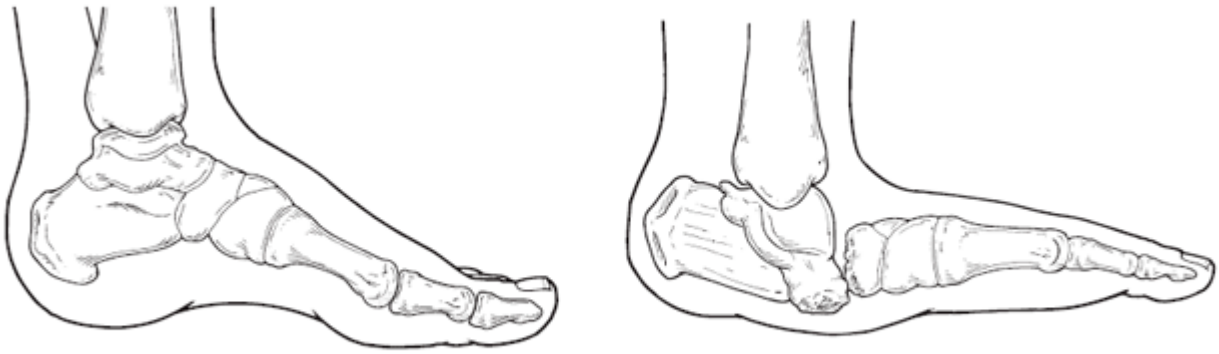
PROPOSED HYPOTHESIS OF DIABETIC NEUROLOGICAL DAMAGE

- Nerve Ischaemia
- Protein kinase c activation
- Free radical injury
- Metabolic pathway hyperactivity eg. Polyol pathway
- Nerve Regeneration abnormalities
- Chronic High circulating glucose levels

CHARCOT FOOT

Charcot neuroarthropathy is manifested by joint dislocations without major trauma and pathological fractures.It is of idiopathic origin. The concept





Normal Foot

Charcot Foot

by Virchow states that bony changes were due to unperceived sub-clinical trauma that are usually not noticed due to insensitivity of the joint. There is reduced bone density in the foot in patients with Charcot neuropathy. The earliest clinical manifestation is swelling of the foot with pain or discomfort. The study by Boykao et al, found relationship between ulcer and charcot deformity, but other foot deformities were not independent ulcer predictors. Acute Charcot foot may be mistaken for gout, cellulitis, and osteomyelitis. Plain radiographs will show bone and joint destruction and loose bodies. Three Phase 99mTc bisphosphonate demonstrates active Charcot process. The treatment given to Charcot foot patients are prolonged immobilization using total contact cast, Charcot restraint orthotic walker(CROW), Schotchcast boot(SCB),and Pneumatic walking braces.

ANGIOPATHY

Diabetes can affect both macro and microcirculation. In patients with Diabetes, atherosclerosis develops at an early age. Medial calcification, Diffuse intimal fibrosis and Atherosclerosis are the most common macrovascular changes observed with Diabetes. The most common risk factors associated to vascular component are dyslipidemia, hypertension, duration of Diabetes, severity of the disease, smoking, Insulin resistance. Moss and colleagues said that current smokers less than 30 years of age were more prone to ulcerate. Cessation of smoking is associated with a decrease the atherogenic process.

Hypertension is almost twice common in diabetics compared to non-diabetics. Arteriosclerosis, specific diabetic microangiopathy and diabetic fibrillosis are the micro vascular changes observed with Diabetes. The typical histological changes are thickening of capillary basement membrane, proliferative changes in arterioles and arteries which include enlargement and proliferation of endothelial cells. Enlargement of endothelial cell is a feature in diabetes leading to small vessel occlusion, causing foot ulceration termed 'small vessel disease' with the presence of palpable pulses in the foot. Increased resting blood flow due to denervated sympathetics causing loss of vasoconstriction, with loss of regulation in circulation in the arterio-venous vessels. A 'capillary steal' phenomenon is induced leading to shunting of blood away from the capillaries leading to reduced skin nutrition. This explains paradoxical ulceration despite increased blood flow.

PERIPHERAL VASCULAR DISEASE

Peripheral vascular disease occurs at an early age in diabetic patients . It is highly likely to involve vessels below popliteal artery. The mechanism by which vascular disease causing lesion of nerves are ischemia caused by occlusion of vessels, altered permeability of capillaries causing osmotic and metabolic derangements. In western countries, vascular alterations is an important factor for foot ulcerations causing major amputations later . Minor trauma and antecedent infections increase blood requirement beyond the

capacity, leading to ischemia and ulceration. Patients presents with intermittent claudication, rest pain and nocturnal pain. Nocturnal pain and rest pain are relieved by keeping legs in dependent position. The circulation is predominantly caters to the splanchnic area during sleep, resulting in decreased perfusion of the lower extremities resulting in ischemic neuritis that disturbs sleep. The features of the ischemic limb are cold feet with absent pulses, delayed venous filling with blanching on elevation. There is loss of hair, thickened nails, and the skin appears shiny. Clinical assessment of the peripheral circulation is extremely useful in the assessment of outcome.

FAULTY WOUND HEALING

• Due to prolonged persistence of the abscess

• Poor granuloma formation

• Presence of bullae, necrobiosis

• Fungal infection of the nail

• High rate of carriage of staph.aureus in the nares and nails

METABOLIC DERANGEMENT

Hyperglycemia in diabetics impair the complement fixation, ketosis impairs the leukocyte function, monocyte mediated immune functions are diminished, and alteration in polymorphonuclear leucocyte function leading to deficient wound healing. Abnormal glucose levels and toxic metabolites play a role..

HAEMATOLOGY

Plasma viscosity, Platelet activity, Haemotocrit , red cells and white blood cells deformability are altered in diabetic patients. These changes profoundly influence the ischemic process.

INFECTION

Infections in the foot are common with diabetics. Uncontrolled infection may progress to amputation. Even with advancement in the treatment, uncontrolled sepsis cause about 60% of the lower extremity amputation . In patients with gangrene due to diabetes amputations is the treatment. Moist gangrene is most common in diabetic patients. Diabetic patients without infection, the prognosis is better than patients with infection. If the bones are involved, the risk of amputation is around eight times than those with involvement of soft tissue alone. Fungal infections are common in the web space in the diabetics. Infections may occur in the nailbed. Preventing cross infection influences the outcome.

The descriptions of the involved region is by

- Severity
- Extent of the involvement
- Clinical appearance
- Location
- Etiology

There are two categories:

• Non-limb threatening

• Limb threatening

• Extensive infections that threaten limb or life require prompt hospitalization and appropriate treatment.

Foot infections occur at the site of trauma or ulceration. If there is a breach in the epidermis, colonisation of the bacteria occur in the dermis and the underlying tissues. Usually the inflammatory signs are absent around the wound. They may present with symptoms like fever, nausea, and fatigue. They may present with unexplained increase in the blood sugar levels. If these features are found, sepsis should be suspected.

The lesions should be thoroughly examined to rule out exposed bones, joints and tendon sheaths. Failing to do so will result in rapid progression and involvement of deeper structures. The spread through tendon sheaths occurs both proximally and distally. Gold standard for diagnosing ulcer with infection is punch or tissue biopsy since the culture of the wound are often misleading and may not represent the organism within the underlying granulation tissue.

The severity of the infections are assessed by

• analyzing depth of the wound,

• presence of ischemia,

• the presence of infection

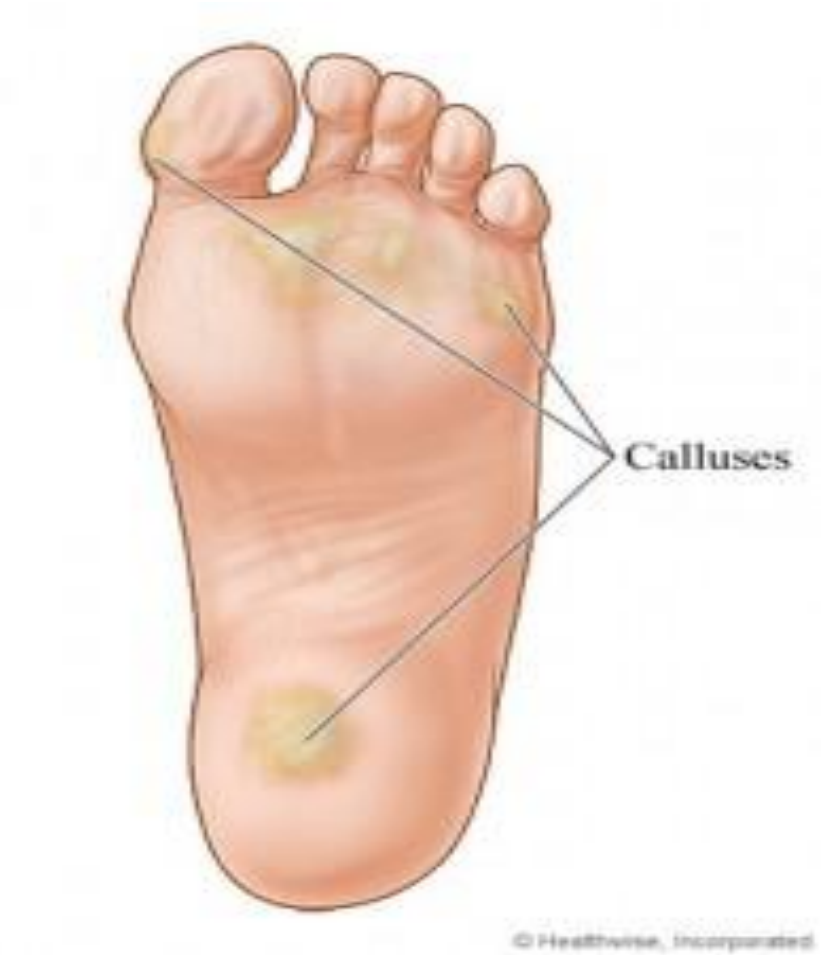
In the presence of osteomyelitis, bone sampling and sensitivity testing become minimum required procedures to prevent progression to more aggressive stages of the disease

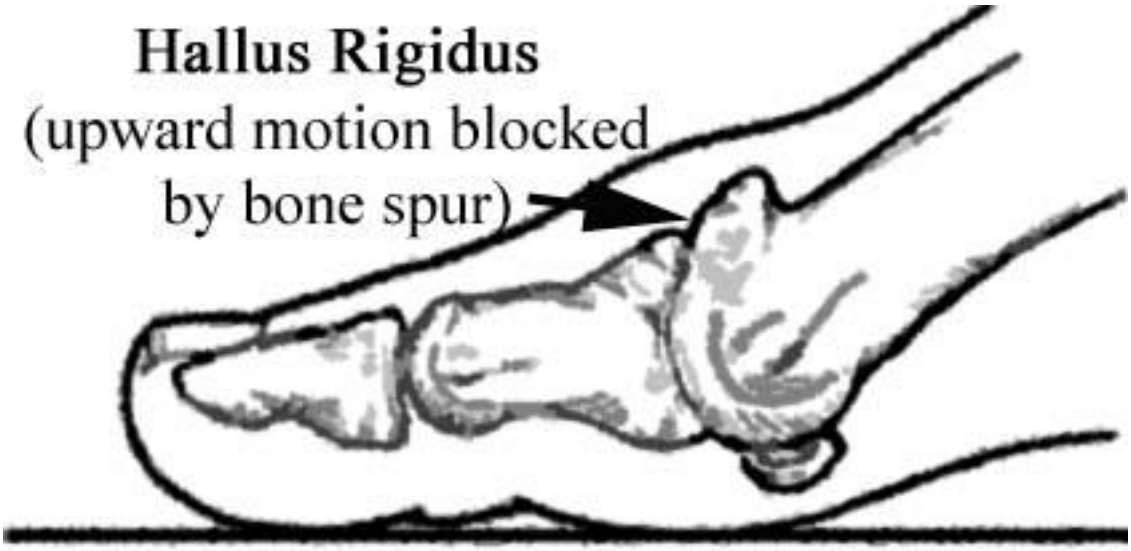
BIOMECHANICS OF FOOT

Biomechanics is a science that deals with effect of forces acting on living tissues. The major cause of ulcer formation in diabetics is loss of pain sensitivity. Most of the non-healing ulcers are not caused by super-added medical conditions, but due to presence of basic biomechanical factors. Progressive elevation of plantar pressure is a reliable predictor of ulcer formation. Biomechanical factors play pivotal role in all phases of management of foot ulcers starting from prevention level to tertiary expert care level.

STRESS AND STRESS CONCENTRATION

The mean pressure acting at the feet of a 100kg individual is about 75 kilopascals. Dynamic pressures are higher compared to static pressures and simple walking. Shearing stress also has a key part in the evolution of ulcers.


Calluses


Hallus Rigidus
(upward motion blocked
by bone spur)

NEUROPATHY AND HIGH PRESSURE

Peripheral neuropathy results in loss of sensations that confer protection against injury. This loss is severe enough that patients may not even perceive severe trauma such as penetrating injury to the feet or even scalding caused by boiling water or burns. Disuse atrophy predisposes to ulceration. High pressure areas that frequently ulcerate include toes and head of metatarsals. Pressure applied repeatedly over the same areas especially those overlying bony prominences in the setting of profound sensory loss causes ulcer formation that starts from within out ie, from bone to the outer soft tissue. Callus are often found on inspection. They have a dark base indicative of an underlying deep ulcer which bleeds and stains the deep surface of the callus.

INTRINSIC FACTORS CAUSING ELEVATED PRESSURE

• Changes in foot architecture like long second metatarsal

• Increased angle of foot arches

• Soft tissue lesions like callus, clawing of toes due to fat pad migration leaving bony prominences exposed

• Limited joint mobility

EXTRINSIC FACTORS CAUSING ELEVATED PRESSURE

• Poor foot wear

• Ill fitting shoes

• Non compliant soles

• Prior surgery

ACTIVITIES CAUSING ELEVATED PRESSURE

• Bare foot walking

• Improper shoes

• Improper foot care

• Altered gait

FOOT ULCER DEFINITION & CLASSIFICATIONS OF DIABETIC FOOT

The International consensus currently defines a " foot ulcer" in the diabetic patients as a "full-thickness wound below the ankle , irrespective of the duration".

Classifications:

The International Working Group On Diabetic Foot recommends use of a uniform classification system to

• Enable universal understanding among health care providers

• Provide accurate assessement of healing potential

• Standardize management protocols

• Eliminate observer variations

• Should be universally acceptable and usable.

Universally used classification is WAGNER'S CLASSIFICATION33

Diabetics possess a life time risk of around 15% for possible ulcer formation.

WAGNER CLASSIFICATION

Grade 0 - No ulcer but high risk foot (deformity or cellulitis)

1- superficial diabetic ulcer

2 - ulcer extends to ligaments, tendons, joints, capsule or deep fascia without abscess and /or osteomyelitis

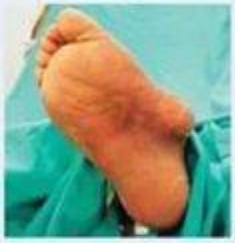
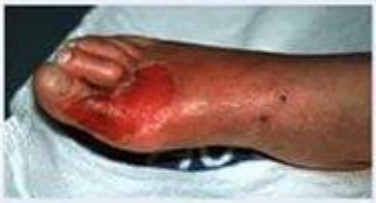
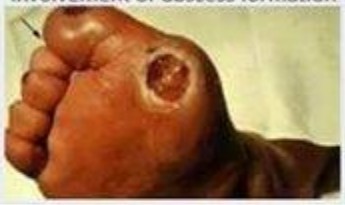
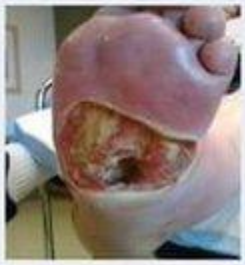
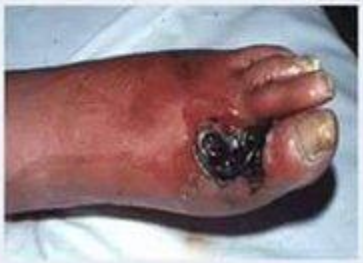
3 – deep ulcer with abscess/ osteomyelitis/ joint sepsis

4 - gangrene localized to portion of fore foot

5 - extensive gangrenous involvement of entire foot

This most valuable grading for the diabetic ulcer foot designed by William Wagner. It is also known as WAGNER-MEGITT'S CLASSIFICATION. This system help to analyze the progress of the patient, both positive and negative outcomes, and to standardize the treatment plan.

Wagner classification of diabetic foot ulcers

Grade 0	Grade 1	Grade 2
No ulcer in a high-risk foot 	Superficial ulcer involving the full skin thickness but not underlying tissues 	Deep ulcer, penetrating down to ligaments and muscle, but no bone involvement or abscess formation 
Grade 3	**Grade 4**	**Grade 5**
Deep ulcer with cellulitis or abscess formation, often with osteomyelitis 	Localized gangrene 	Extensive gangrene involving the whole foot 

CLASSIFICATION – UNIVERSITY OF TEXAS

Aetiology (Stage) included -

Staging

–Stage A: No infection or ischemia

–Stage B: Infection present

–Stage C: Ischaemia present

–Stage D: Infection and ischaemia present.

Grading

–Grade 0: Epithelialized wound

–Grade 1: Superficial wound

–Grade 2: Wound penetrates to tendon or capsule

–Grade 3: Wound penetrates to bone or joint

The other classification is **SAD system**, which adds to the Texas system the cross-sectional area and the presence of neuropathy or not.

The 'PEDIS' system includes

• Perfusion (ischaemia)

• Extent

• Depth

• Infection

• Sensation (neuropathy)

More recently, International Working Group on Diabetic Foot (IWGDF) has created a classification that grades wound size ,perfusion of foot, the presence of infection, and the presence of sensation.The flaw in all classifications is that none of these systems considers the duration of the foot ulcer, which has great bearing on healing.

ASSESSMENT OF DIABETIC FOOT ULCER

Proper assessment of diabetic foot involves adequate history, clinical examination and investigations.

HISTORY

• MEDICAL HISTORY

1. Diabetes – Duration
2. Treatment history
3. Co-morbidities
4. Nutrition
5. Addictions
6. Current drug intake
7. Hypersensitivities
8. Past Medical or surgical history

• GENERAL HISTORY

1. Everyday activities
2. Foot Protection
3. Callus formation
4. Bony deformities of the foot
5. Neuropathic symptoms
6. Claudication or rest pain

• WOUND/ ULCER HISTORY

1. Area involved
2. Number of months involved
3. Precipitating cause
4. Previous involvement in the same site
5. Infection
6. Hospitalization
7. Wound care
8. Wound healing
9. Patient adherence to advice
10. Social problems hampering adequate wound care
11. Previous foot trauma or surgery
12. Presence of pedal edema
13. Charcot arthroneuropathy

CLINICAL EXAMINATION

All diabetic patients who present to any physician will require a detailed foot inspection and examination at least annually. Patients who are having diabetic foot related complaints need to be evaluated in detail. Systematic examination is mandatory so that we should not overlook any important aspects. All the key components required for a detailed foot examination are summarized in a bulletin format. In addition to that patient's vital signs and assessment of his/her general condition is also mandatory even though it is not mentioned here specifically.

Examination of vascular system

1. Palpation of peripheral pulses (dorsalis pedis, anterior tibial, posterior tibial, popliteal, femoral)
2. Sub papillary venous plexus filling time (normal <3 seconds)
3. Venous filling time (normal <20 seconds)
4. Color changes: Cyanosis, Dependent rubor erythema
5. Edema
6. Temperature gradient
7. Dermal thermometry
8. Integumentary changes consistent with ischemia:

- Atrophy of skin
- Atrophy of nail
- Abnormal wrinkling
- Loss of hair

Examination of nervous system

1. Vibration perception

   Tuning fork 128Hz

   Measurement of vibration perception threshold (Biothesiometer)
2. Light pressure : Semmes-Weinstein 10 gram monofilament
3. Light touch : Cotton wool
4. Two-point discrimination
5. Pain : Pin-prick
6. Temperature perception : Hot and cold
7. Deep tendon reflexes : Ankle, knee
8. Clonus testing
9. Babinski test
10. Rhomberg's test

Examination of musculoskeletal system

1. Evaluation of Biomechanical abnormalities

    Orthopedic deformities

    Hammertoes

    Bunion(s) or Tailor's bunion(s)

    Flat or high arched feet

    Charcot deformities

    Iatrogenic deformities (e.g, amputations)

    Restricted joint mobility

    Tendo-Achilles contractures/equines

2. Evaluation of gait
3. Testing of muscle group strength

    Passive and active, non-weight bearing and weight bearing Foot drop

    Atrophy – intrinsic muscle atrophy

4. Assessment of plantar pressure

    Computerised devices

    Harris ink mat

Examination of skin and appendages

1. Appearance of skin : Color, texture, turgor, quality dry skin.

2. Calluses : Discoloration/subcallus hemorrhage
3. Fissures (especially posterior heels)
4. Appearance of Nail : Onychomycosis, dystrophic, atrophy, hypertrophy, paronychia
5. Presence/absence of hair
6. Ulceration, gangrene, infection (Note location, size, depth, infection, etc)
7. Interdigital lesions
8. Fungal infections like Tinea pedis
9. Markers of diabetes

    Shin spots – diabetic dermopathy

    Necrobiosi lipoidica diabeticorum

    Bullosum diabeticorum

    Granuloma annulare

Examination of footwear

1. Type of shoe
2. Fit
3. Shoe wear, patterns of wear
4. Lining wear
5. Foreign bodies
6. Insoles

Communication and Classification of Cumulative Risk

Following a detailed diabetic foot examination, the patient is classified based on cumulative risk category. This helps the doctor to design a treatment plan which will reduce lower extremity amputations and reduce the patient from a high - risk category to the lowest risk level. Several risk stratification schemes have been proposed assigning different weights to various risk factors such as peripheral neuropathy, arterial insufficiency, deformity, high plantar pressures and previous history of ulceration or amputation. International working group has accepted the following simplified risk categorization system.

Table 2

Risk categorization system

Category	Risk Profile	Evaluation Frequency
0	No neuropathy	Annual
1	Neuropathy	Semi - annual
2	Neuropathy, PVD and/or deformity	Quarterly
3	Previous ulcer or amputation	Monthly to quarterly

LABORATORY STUDIES:

Blood sugar levels

HB%

Renal function test

TC/DC

ESR

CRP

Complete lipid profile

Urine sugar &deposits

Urine acetone

Imaging studies

The complex nature of diabetic foot disease along with its complications predispose it to various infections and non – infectious process. Hence imaging presentations are more likely to vary because of lack of specificity in complex clinical situations. Obviously it will be a great challenge to interpret the imaging studies in diabetic foot disease. Hence these imaging studies should be restricted to confirm a diagnosis and to treat the patients.

Plain X – rays are always the first imaging study in diabetic patients who are presenting with signs and symptoms of foot ulcer. X-ray finding in diabetic foot infection for example osteomyelitis will not be able to demonstrate in an obvious osseous changes for upto two weeks.

Plain X-rays are mainly indicated to detect osteomyelitis, osseolysis, fractures/dislocations seen in neuropathic arthropathy, arterial calcification and soft tissue gas shadows.

The role of CT scans come in to play only when X-rays do not show any suspected bone or joint pathology. CT scan provides high anatomical details and resolution of bone with osseous fragmentation and also subluxation of joints are visualized better. Though 99Technitium scan lacks specificity in neuropathic patients, they are also used in evaluation of diabetic foot infections. For early detection of osteomyelitis, fracture, charcot's arthropathy the three phase bone scans are beneficial. All these imaging modalities when combined with other scintigraphic procedures like WBC scans have a higher specificity. Gallium 67 citrate is used along with 99Tc scan to help in the diagnosis of osteomyelitis and acute osteo arthropathy.

Indium 111 leukocyte scans, TcGG-labelled white cell scan have high sensitivity and specificity to distinguish between osteomyelitis and neuropathic arthropathy. Even though these investigations are costly and time consuming, they are available in most of the hospitals which aim at an early identification of bone infections. MRI scan is also used in evaluating soft tissue, bone, pathologies. Indications for MRI are osteomyelitis, deep seated abscesses, septic arthropathy and tendon rupture. MRI has high sensitivity for bone infection and it is readily available. It can be used for planning surgical interventions. Even though it is costlier, MRI has been accepted widely in the treatment of diabetic foot ulcers.

Vascular Procedures

Whenever there is ischemia and/or a non healing ulcer with absent peripheral pulses, further non-invasive arterial studies are warranted. They should be performed to determine perfusion in lower limbs. They include arterial Doppler study, ankle brachial pressure index, toe pressure, transcutaneous oxygen tension. Sometimes ankle pressures may be falsely elevated due to calcification of tunia media of the arteries and also the affected arteries may be non-compressible. In such cases the ankle brachial pressure index may be misleading. Toe blood pressures have a role in predicting the diabetic patients who are at risk for foot ulceration and also in the prediction of successful wound healing. Trans-cutaneous oxygen tension measurement has also received similar kind of supports in the previous publications. When it is less than 25mmHg, it predicts that there will be failure of proper tissue oxygenation and there are more chances of poor wound healing. These two tests can be performed distally on the foot regardless of calcification of pedal arteries. The favourable range of pressure is around 40mmHg.

Laser Doppler velocimetry is useful in assessment of skin perfusion pressure and blood flow velocity in superficial arterioles and capillaries of skin. At levels less than 30mmHg it is highly predictive of critical limb ischemia and poor wound healing. Digital subtraction angiography/magnetic resonance angiography are the other modalities used to evaluate distal arterial perfusion.

Neurologic Procedures

Biothesiometer is used to assess the vibration perception threshold. This is also useful to predict the patients who are having high risk for

ulceration. Nerve conduction studies are also useful to diagnose peripheral sensory neuropathy. Patients who are having neuropathic ulceration always have a profound sensory neuropathy.

Plantar Foot Pressure Assessment

High plantar foot pressure is an important risk factor for foot ulcerations. Various modalities are used to measure the plantar foot pressures. There are many computerized systems which quantitatively measure the plantar foot pressures. They identify the foot which are at risk of ulceration and also evaluate the orthotic adjustments. Harris mat, it is not only useful in qualitative measurement of plantar pressure but also useful in identifying vulnerable areas of ulceration.

Assessment of diabetic wound infections

During evaluation , a proper detailed history taking and physical examination should be done in all cases. Complete assessment of these patients can be achieved through a systematic approach.

The past history of patients about any pre –existing comorbidities should be obtained. Treatment/drug history is also important. Since most of the diabetics have associated peripheral neuropathy, pain is not a reliable symptom in these patients. History of previous ulcertaions, infections, injury , surgery at the present site should be obtained. Patients with the infected diabetic foot can have constitutional symptoms such as-

i. Fever
ii. Chills

iii. Nausea
iv. Vomiting
v. Malaise
vi. Fatigue

In about 50 to 60% of diabetic patients, who have severe infections the systemic signs such as fever and leukocytosis are absent. Recalcitrant hyperglycemia is the only indicator of infection in certain diabetic foot patients where systemic signs are absent

Laboratory investigations

i. Complete blood count
ii. Total and differential counts
iii. Blood culture and sensitivity
iv. Estimation of glycated haemoglobin
v. Fasting/post prandial blood glucose
vi. Erythrocyte sedimentation rate
vii. Urine ketone bodies
viii. Renal function tests
ix. Routine urine analysis

The patients should be asked about the mode of onset, duration of the ulcer and appearance of area before infection.

Examination of the ulcers include

i. Size of ulcer
ii. Shape

iii. Margins
iv. Depth
v. Floor
vi. Base
vii. Discharge if any
viii. Swelling
ix. Color of the surrounding skin
x. Odour
xi. Extent of the ulcer
xii. Presence or absence of slough

Probing of the ulcer should be done to determine

i. Bone and joint involvement
ii. Sinus tract
iii. Extension into tendon sheets

The extension into tendon sheets is the most common route of spread of infection proximally and distally. If the bone gets exposed it is presumed that there is underlying osteomyelitis unless proved otherwise . Swab should be taken from the ulcer base and should be sent for culture. This will help to minimise the false negative results and to identify the true pathogen involved in ulcer causation. Nowadays tissue culture holds good results compared to conventional pus culture.

Bone culture should be taken in case of osteomyelitis in order to arrive at a definite diagnosis and to plan appropriate treatment. There is a possibility of contamination of non-infected bone when there is active soft

tissue infection. As already mentioned imaging studies plays a vital role in overall assessment of diabetic foot infection.

Plain X-rays shows presence of bony erosion and /or soft tissue gas shadows. The demonstration of osteomyelitis by plain X-rays lags the onset of bony involvement by 10 to 14 days. Tc99 bone scans may demonstrate abnormal uptake of radionuclides before obvious changes are visible on plain radiographs.

But Tc99 scans lags specificity in patients having peripheral neuropathy or having any pre-existing osseous conditions which causes increased bone turnover (surgery, fracture, neuropathic arthropathy). Tc99 scan and indium labelled leukocyte scan and Tc99 HMPAO labelled scans are the combination of scans which are helpful in distinguishing between osteomyelitis and charcot's arthropathy.

MRI scan has higher tissue contrast and so it is very useful to detect both soft tissue and bone marrow inflammation. MRI scan can also be used in the follow up of resolution of infection and also used in planning surgery. All the imaging modalities which are mentioned above do not have 100% sensitivity and specificity to diagnose bone infection. In addition to that these imaging modalities are not readily available and also very costly. Hence proper clinical assessment is the mainstay of planning management.

MANAGEMENT OF DIABETIC FOOT ULCERS

General principles and goals

The main aim in the treatment of diabetic foot ulcer is to obtain wound closure as early as possible. In diabetic patients the rate of lower limb amputation can be reduced by treatment of foot ulcer and reducing the rate of occurrence. The treatment objectives are summarized as follows

i. Appropriate wound management
ii. Debridement
iii. Management of ischemia and infection
iv. Pressure relief (off-loading)
v. Surgical management
vi. Management of comorbidities

Treatment of diabetic foot infections are divided into non-limb threatening and threatening infections.

Non -limb threatening infections

To define, these are medically stable patients having no signs of systemic sepsis. They are ideal candidates for outpatient management under close supervision.

Clinically, these group of patients have superficial ulcers with minimal ischaemia and lack bone or joint involvement. Cellulitis is confined to within

2cm of the ulcer margins. Many of these cases are monomicrobial with staphylococcus aureus, staphylococcus epidermiditis and beta hemolytic streptococci being the most common ones to be isolated. Samples for cultures are acquired by curettage of the infected ulcer. Oral antibiotic therapy is initially given. MRSA should be covered under the spectrum. The ulcer should be debrided as often as necessary.

Limb-threatening infections

This group includes

• Cellulitis beyond 2cm of ulcer margin

• lymphangitis

• Ischemic Tissue necrosis

• Odour

• Gangrene

• Bone involvement

These patients require emergent hospital admission and appropriate intervention. The patient's co-morbidities should be evaluated. Extent of infection should be thoroughly assessed. A team approach in treatment improves outcomes.

Initial management by a surgeon includes debridement, drainage of toxic fluid or pus, decompression by fasciotomy or limited amputations to curtail

progress of infection. During this, wide range of culture specimens should be obtained to identify bacteria and fungal growth. Sub-Arachnoid Blocks should be avoided in these patients.

The presence of hemodynamic imbalance or profound systemic compromise should not deter the surgeon from taking up debridement. Only clearance of infected tissue will improve the internal milieu of the patient. Most of such cases have poly-microbial infections requiring intravenous antibiotics initially rather than oral medication. Osteomyelitis should be evaluated radiologically and microbiologically. Debridement of the infected bone and even minor amputations may be needed. Bone cement containing antibiotics are available for use after adequate debridement, provided adequate soft tissue cover is available.

DRESSINGS

Dressings are done to keep the wound clean and free. It removes excess bacteria from the wound. It removes excess fluid. Materials used should not cause toxicity to the wound. It should be non-adhesive. Various types are

1. Honey dressing- provides moisture, decreases odour, reduce inflammation, reduce oedema and exudates.
2. Silver-containing dressings- Silver is having broad spectrum of activity. It kills yeast, fungi, viruses and methicillin and vancomycin strains.
3. Iodine-Dressings.

4. Hydrocolloid Dressings- Create moist healing environment, should not be used in heavily infected wound

5. Alginate dressings- Derived from sea weed, have the capacity to absorb large amounts of exudates.

6. Hydrogels- Promote autolytic wound debridement in dry wounds, absorb exudates in moist wounds.

7. Hyalofill- Derivative of hyaluronic acid, should be used on clean wounds.

8. Living skin equivalents- Dermagraft or graftskin.

LARVAL THERAPY

Maggots are living chemical factories. Worms remove dead tissues by the production of a mixture of proteolytic enzymes that breakdown dead tissue to a semi-liquid form ingested by creatures, stimulate fibroblast growth in vitro, eliminate odour and reduce wound related pain.

GROWTH FACTORS

Growth factors play a key role, regulating all aspects of wound healing. Epidermal growth factor(EGF), fibroblast growth factor 2 (FGF-2) and platelet derived growth factor(PDGF) all have been approved in the treatment of diabetic foot ulcers.

OFFLOADING THERAPY

The choice of technique is based on the physical form and compliance with the treatment, and also site and severity of the ulcer. The aim is to reduce dynamic foot pressure.

Total contact cast (TCC) is gold standard in management. Total contact cast should not be used in patients with local sepsis and vascular compromise. Other options are Removable cast walkers, Charcot restraint orthotic walkers, Healing sandals and half shoes, felted foam padded dressings, therapeutic shoes and insoles.

This line of management should be continued till there is complete healing of ulcers.

SURGICAL INTERVENTION

It is often indicated for deep infection or for the treatment of recurrent or recalcitrant ulceration. They include debridement of recalcitrant or infected ulcers, drainage and debridement of soft tissue infection,bone resection for relief of pressure and or for osteomyelitis; closer techniques; Achilles lengthening surgery; and reconstruction of forefoot deformities. Primary or delayed closure is advocated as more effective, much more cost-efficient and successful. Low pressure or vacuum assisted closure is particularly valuable when there is insufficient soft tissue surplus to allow for primary closure of the wound edges.

DEBRIDEMENT

Debridement serves several purposes:

• Excision of dead tissues and callus.

• Pressure alleviation

• Ulcer bed assessment

• Probing of tracks and tunnels

• Decreasing microbiological load

There are Five types of debridement

• Surgical

• Enzymatic

• Autolytic

• Mechanical

• Biological

Early surgical intervention by debridement has been demonstrated to be the best.

SURGICAL DEBRIDEMENT

Aggressive removal of the devitalized tissues is done by sharp scissors and scalpels. Extent of debridement should be sufficient enough to reach all margins of infection both in the deeper plane and also in the horizontal plane. The aim of this approach is to convert a chronic non-healing wound into an actively bleeding acute wound that will have the capacity to granulate and

HEALTHY GRANULATED TISSUE

SPLIT SKIN GRAFT

regenerate. Deep seated abscess mandates hospitalisation and immediate incision and drainage. Removal of bones either locally or by limited amputations may be necessary. Repeated removal of necrotic tissue expedites rate of healing. This is known as as “maintenance debridement”.

Hydro surgery

Its properties are

• Precise and

• Limited Excision

• Negligible thermal damage to the tissues.

An extremely painful wound may benefit from enzymatic debridement.

Vascular wounds are candidates for enzymatic debridement.

MOISTURE BALANCE

Moisture accelerates re-epithelization in a wound. Tissue moisture is a term used to convey the importance of keeping wounds moist and free of excess fluids. A moist wound environment promotes granulation and autolytic processes. Effective management of chronic wound fluids is an essential part of wound bed preparation. It also helps in addressing the issues of cellular dysfunction and biochemical imbalance.

ADVANCES IN WOUND CARE

Preparation of ulcer bed enables removing barriers which hinder healing and to start the healing process. Advanced care sometimes becomes the only means of attaining wound closure. The discovery of recombinant growth factors, genetic manipulation, artificial tissues, stem cell therapy have empowered the surgeon and wound-care provider to aid in angiogenesis to accelerate healing.

VAC THERAPY

Delivery of intermittent or continuous sub-atmospheric pressure through a specialized pump connected to open-celled foam surface dressing covered with an adhesive drape to maintain a closed environment. It increses blood flow, decreases local tissue edema, removes excessive fluid and proinflammatory exudates from the wound bed.

AMPUTATIONS

Amputations, an unpleasant but often final end result of the diabetic foot. It is performed for multiple reasons and can be either curative or emergent. Amputation level selection aims at achieving balance between preservation of limb length and function with the ability of the wound to heal properly. Currently available vascular surgical advances have made ‘limb sparing’ more and more feasible. Endovascular restoration of vascularity have made it

possible to do more distal amputations. Pre amputation vascular intervention must be done to limit level of amputation and also to facilitate proper stump healing.

AIM OF DECIDING LEVEL OF AMPUTATION

• To leave behind a stump that can readily accept prosthetic shoe, orthotic device or complete limb prosthesis

• To create a stump that is less likely to breakdown from external pressures

• To prevent dynamic imbalances that may occur due to migration of digits such as in migration of the other digits after 1st MTP joint disarticulation, varus deformity that occurs due to lateral loading of the foot following 5th Toe Ray amputation.

• To facilitate primary wound healing so as to enable rapid healing of the stump and early rehabilitation of the amputee.

If infections are not controlled or due to the advancement in the disease process, diabetics usually succumb to lower extremity amputation. The incidence of amputations for non-traumatic etiologies is ten times higher in Diabetes. Costs of amputation and its descendent managements are very high. This is due to length of hospitalization that is required and due to multiple investigations and repeated surgical and vascular interventions that may be required.

Various amputations of lower extremity are

• Ray's amputation

• transmetatarsal (Gillies)

• tarsometatarsal (lisfranc's)

• midtarsal(chopart's)

• syme's

• Below-knee(Burgess)

• Transcondylar

• above-knee

The three most common indications for major lower extremity amputations are

• acute limb ischaemia

• chronic critical limb ischaemia

• major infection due to malperforans ulcers in diabetics with normal arterial circulation.

GOALS

The goals of major lower extremity amputations are :

• to eliminate the nonviable tissue

• to provide a stump with best chance to heal

• to provide a stump with best chance of long term function-ambulation with prosthesis.

IDEAL STUMP

• The ideal stump should heal adequately

• should have rounded ,gentle contour with adequate muscle padding

• should have adequate length to bear prosthesis

• should have thin scar which does not interfere with prosthetic function

• should have adequate joint movement

• should have adequate blood supply.

GENERAL PRINCIPLES APPLICABLE TO AMPUTATION SURGERY

SKIN

Flaps should be sutured in a tension free manner and the scar should be well healed and non-adherent to the bone.

MUSCLE

Myodesis should be performed to facilitate balanced action of opposing muscle groups.

NERVE

Neuroma formation should be prevented by dividing the nerve at a higher level by applying adequate traction and allowing it to retract into the stump under cover of muscles. Nerves should never be ligated.

BLOOD VESSELS

Visible bleeding alone does not indicate optimum level of amputation. Wound healing in reality is dependent on micro-circulation. Vessels must be suture ligated, arteries and veins in separate group to avoid iatrogenic AV fistula formation.

BONES

Bone should be cut at a higher level and ends beveled so as to avoid protruding bone that will interfere with healing of stump and also result in a painful end bearing stump.

STUMP DRESSINGS

A cotton wool followed by crepe bandage is commonly used dressing for the amputation stump. A rigid cast support enables wound protection, contracture prevention and oedema reduction.

TYPES OF AMPUTATION

RAY AMPUTATION

Amputation of the toe with the head of metatarsal or metacarpals.

TRANSMETATARSAL AMPUTATION (GILLIES')

Amputation is done proximal to the neck of the metatarsals, distal to the base.

LISFRANC'S AMPUTATION (TARSOMETATARSAL)

Here tarsometatarsal joint is disarticulated with a long volar flap.

CHOPART'S AMPUTATION (MIDTARSAL)

Here talonavicular and calcaneocuboid joints are disarticulated. Tibialis anterior is sutured to the drilled talus bone. A long volar flap is used and immobilized for six weeks after surgery.

SYME'S AMPUTATION

It is removal of the foot with calcaneum and cutting tibia and fibula just above the ankle joint with retaining heel flap (dividing both malleoli).Heel flap is supplied by medial and lateral calcaneal vessels . Elephant boot is used for the limb after syme's amputation. Many patients walk well with syme's stump without difficulty. It is presently mainly used in trauma(crush injuries) and malignancies of the distal part of the foot.

PIROGOFF'S AMPUTATION

It is like syme's amputation except the posterior part of the calcaneum is retained along with heel flap. It provides longer stump than syme's amputation.

TRANSTIBIAL(BELOW-KNEE) AMPUTATION

Knee joint is spared. The ideal stump is 15cms long

The advantages of preserving the knee joint are

• lower kinetic energy requirement

• near normal gait

• Ease of using prosthesis

• Self Sufficiency and reduced dependancy

• Quicker rehabilitation

• Less expensive prosthesis

KNEE DISARTICULATION(THROUGH-KNEE) AMPUTATION

It is through the joint and does not disturb the bone. It is used in patients with poor general condition and those who are not amenable to prosthetic mobilization

TRANSFEMORAL(ABOVE-KNEE) AMPUTATION

About 12-15cm of lower end of femur should be removed. Usually equal anterior and posterior flaps are used. If femur length less than 10cms this procedure is not possible. If femur length is less than 10 cms, then should proceed with hip disarticulation. The marked reduction in limb length drastically reduces propulsive power and manipulation of the prosthesis. Efficient ambulation depends solely on the user's ability to mobilize the artificial knee joint in the prosthesis.

CURATIVE VERSUS EMERGENT SURGERY

Performance of amputation in the elective setting may not always be a possibility. When serious infections such as gas gangrene are starting to set in, it becomes mandatory to perform an emergency amputation. Before surgical intervention, pre-existing infection should be dealt with. Elective amputations are usually curative ie, primary wound healing is facilitated by raising flaps and

closing the wound primarily. Emergency amputations aim at removal of necrotic tissue only and not at healing the stump primarily. Subsequent surgery may be required to close the wound once the infection has been controlled.

COMPLICATIONS OF AMPUTATION SURGERY

Early complications:

• Hemorrhage

• Infection

• Haematoma

Late complications:

• Pain

• Flap necrosis

• Ring sequestrum formation

• Ulceration of the stump

• Painful scar

• Phantom limb

POSTOPERATIVE PERIOD AFTER AMPUTATION

• Regular physiotherapy

• Regular dressing

• Crutch is used initially

• After 3 months prosthesis is used

• Rehabilitation

Prevention of recurrence of ulcer

Always prevention is better than cure. A multidisciplinary team approach is essential for the prevention of recurrence of ulcer. The team compromises of

1. Podiatric surgeon
2. Internist
3. Endocrinologist
4. General physician
5. Cardiologist
6. Vascular surgeon
7. Orthopaedic surgeon
8. Nephrologist
9. Neurologist
10. Physiotherapist
11. Pedortist
12. Attending nurse

HEALTH EDUCATION

Health education of the patients regarding

1. Foot hygiene
2. Daily self inspection of wounds
3. Wear proper fitting footwear
4. Prompt treatment of new ulcers
5. Regular hospital visits

OBSERVATION AND RESULTS

TOTAL NUMBER OF PATIENTS – 120

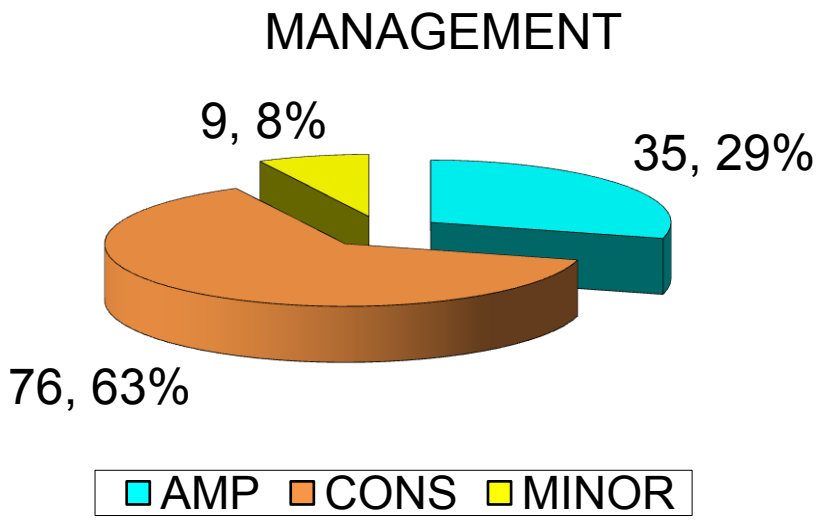
CONSERVATIVELY MANAGED – 76

MAJOR AMPUTATIONS – 35

- Amputation at trans tibial level (below knee amputation) or higher.

MINOR AMPUTATIONS – 9

- Toe disarticulation
- Ray amputation
- Mid-tarsal amputation
- Tarso-metatarso amputation

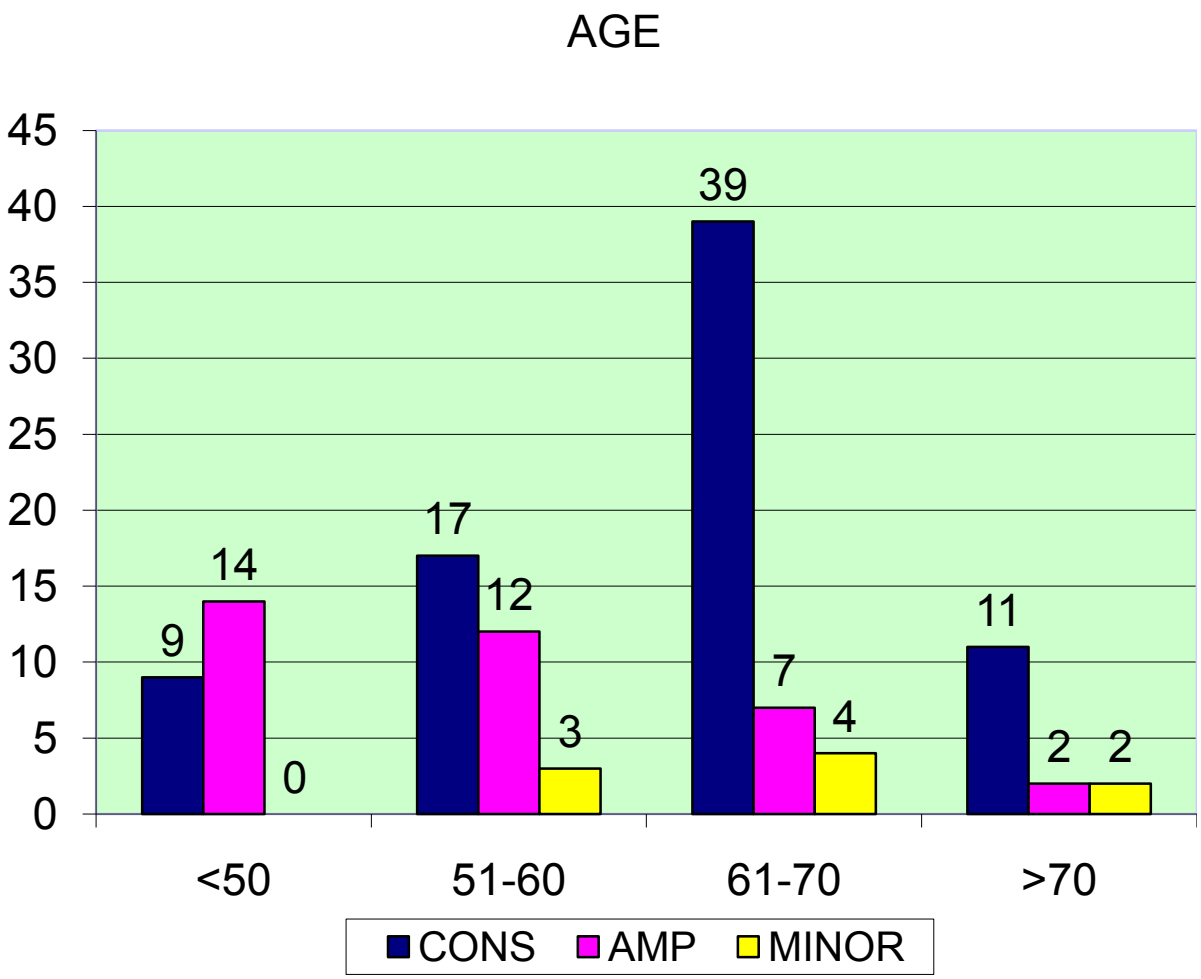


1. AGE DISTRIBUTION

Age distribution of 120 cases studied at Govt. Rajaji hospital, youngest patient was 35 yrs old, eldest patient was 75 yrs old. Highest number of cases were found in the age group of 61-70.

AGE	CONSERVATIVE	AMPUTATION	MINOR
<50	9	14	0
51-60	17	12	3
61-70	39	7	4
>70	11	2	2

By age distribution, patients with younger age tend to have aggressive disease.

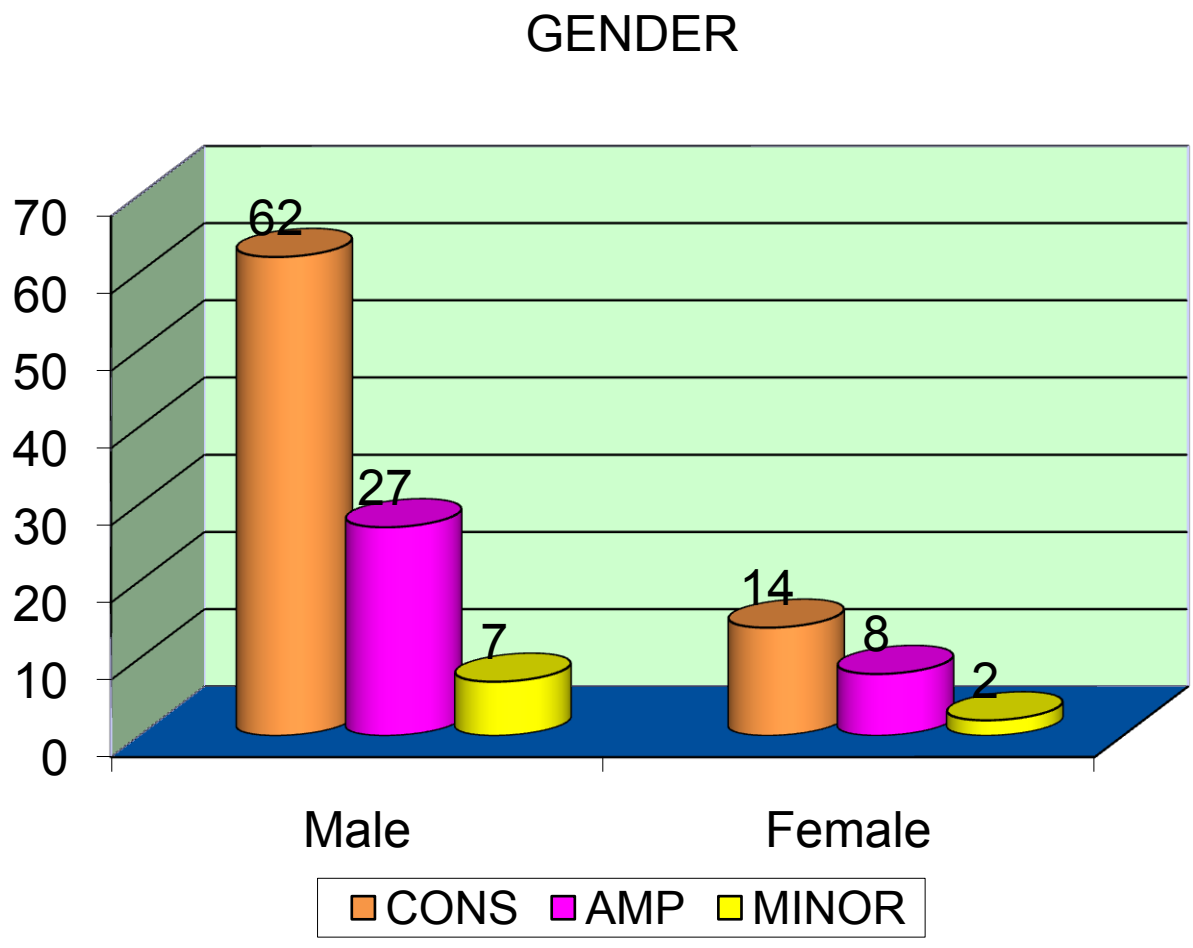

AGE
45
40
35
30
25
20
15
10
5
0
9
14
0
17
12
3
39
7
4
11
2
2
<50
51-60
61-70
>70
CONS
AMP
MINOR

2. SEX DISTRIBUTION

Out of 120 patients 96 were male and 24 were female.

SEX	CONSERVATIVE	AMPUTATION	MINOR
Male	62	27	7
Female	14	8	2

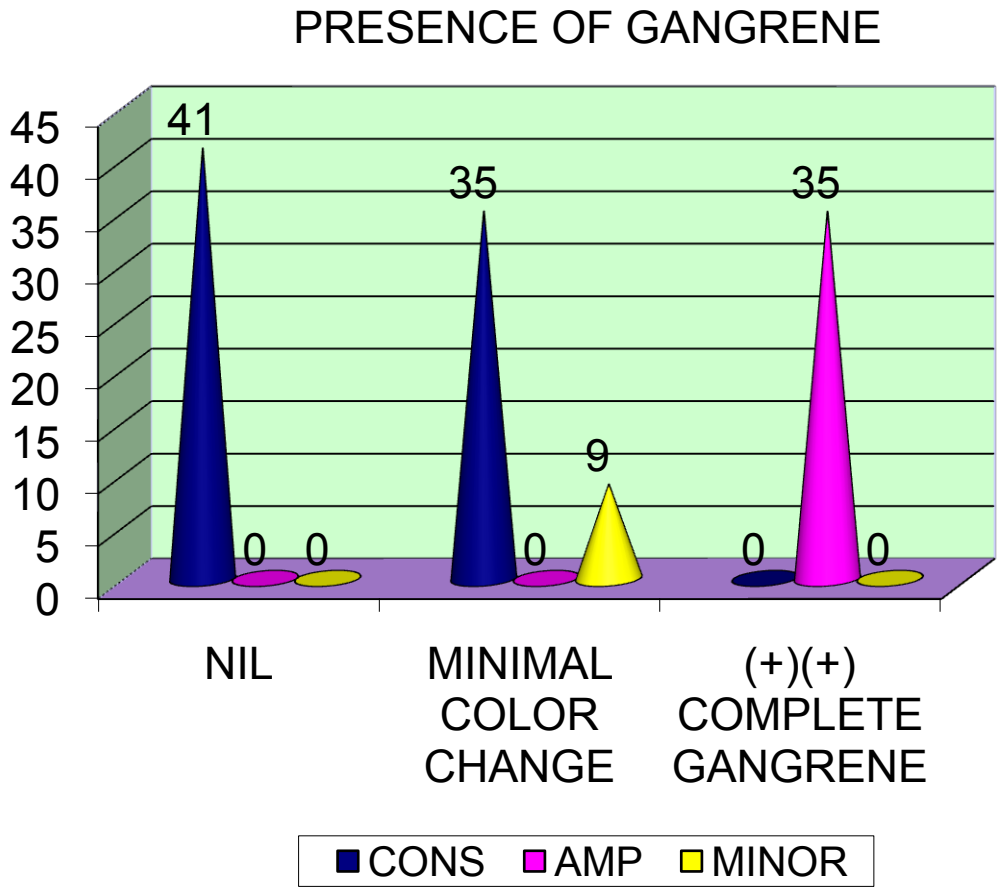
Males are more commonly affected in diabetic foot infections and amputation rates are also higher compared to females.



3. PRESENCE OF GANGRENE

PRESENCE OF GANGRENE	CONSERVATIVE	AMPUTATION	MINOR
NIL	41	0	0
MINIMAL COLOR CHANGE	35	0	9
(+)(+) COMPLETE GANGRENE	0	35	0

100% of patients with complete gangrene are going for amputation.

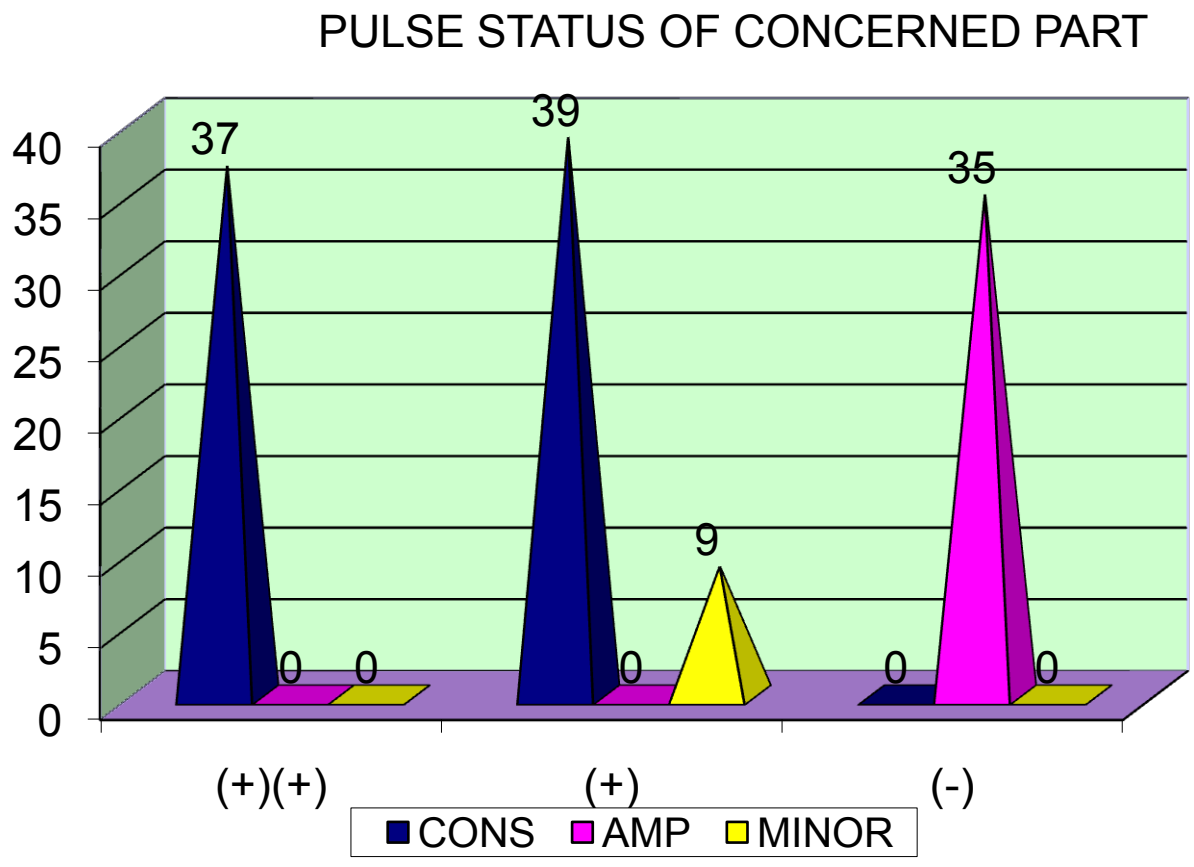


4. PULSE STATUS OF THE CONCERNED PART

PULSE STATUS OF CONCERNED PART	CONSERVATIVE	AMPUTATION	MINOR
(+)(+)	37	0	0
(+)	39	0	9
(-)	0	35	0

100% of patients with absent pulse are going for amputations.

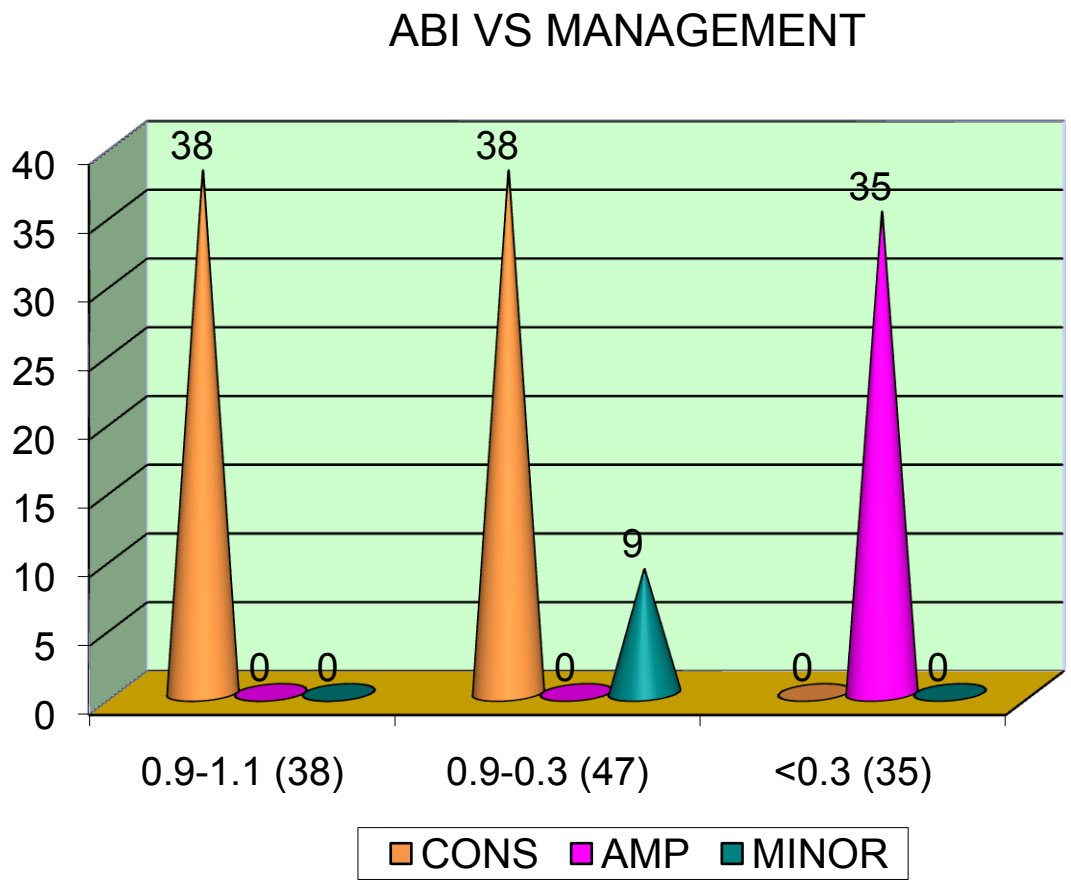
In case of weak pulse, out of 48 patients, 9 are going for minor amputation



5. ANKLE BRACHIAL INDEX

ABI	CONSERVATIVE	AMPUTATION	MINOR
0.9-1.1 (38)	38	0	0
0.9-0.3 (47)	38	0	9
<0.3 (35)	0	35	0

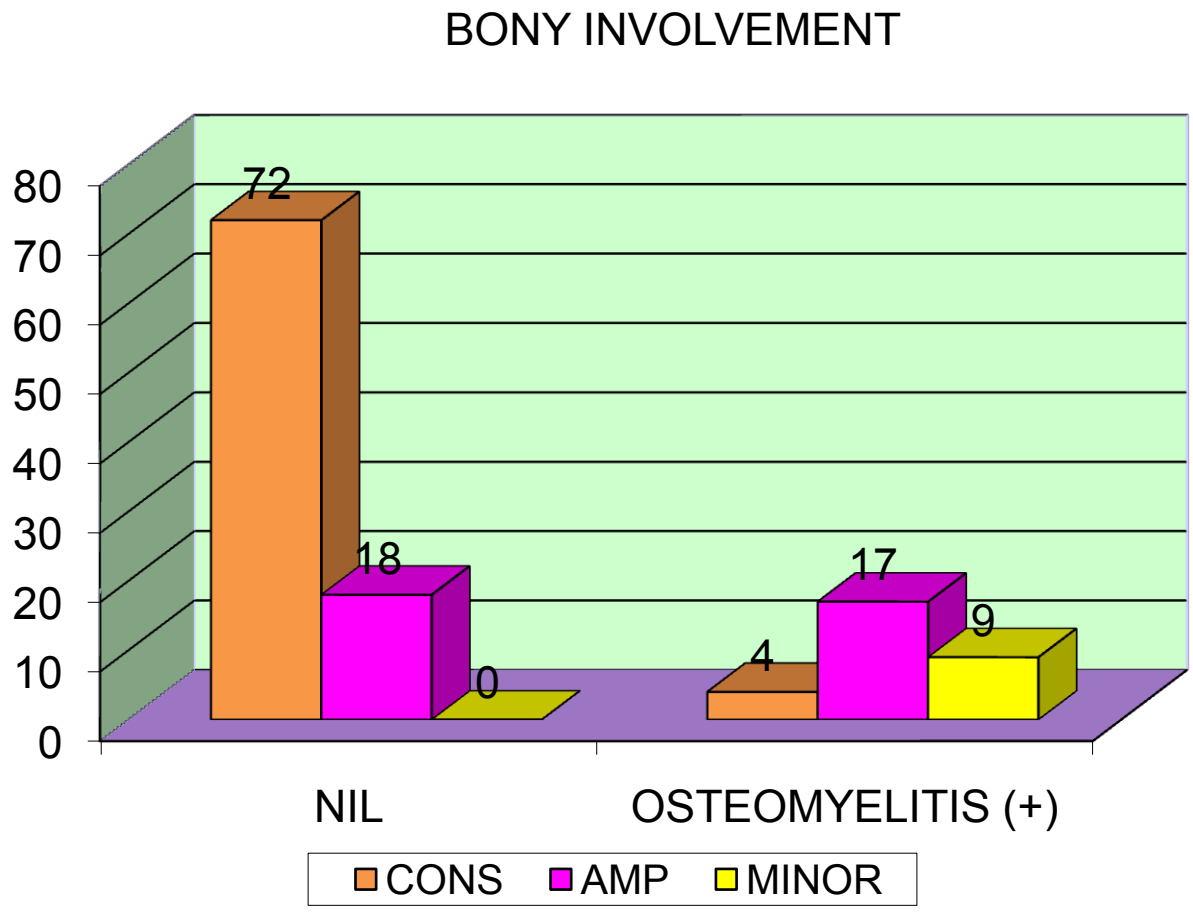
In case of ABI, critical limb i.e. less than 0.3 - 100% unsalvageable



6. BONY INVOLVEMENT

BONY INVOLVEMENT	CONSERVATIVE	AMPUTATION	MINOR
NIL	72	18	0
OSTEOMYELITIS (+)	4	17	9

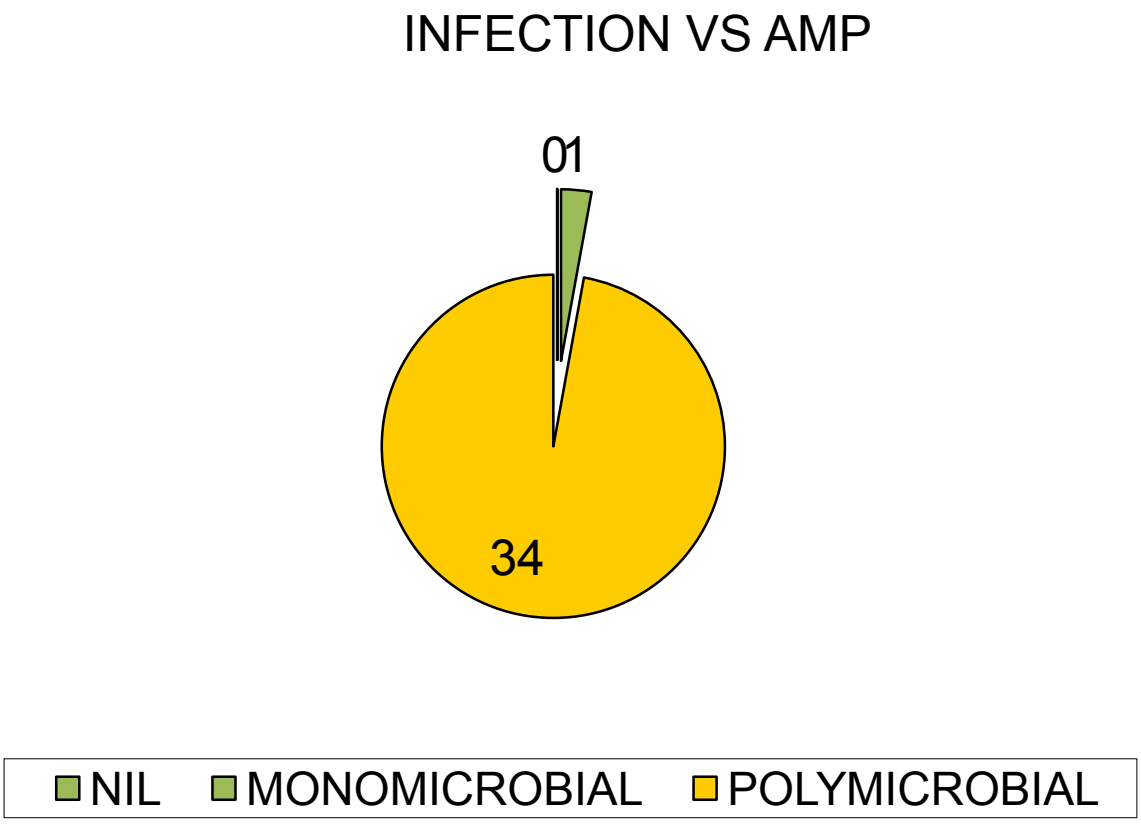
In case of bony involvement i.e. Osteomyelitis, out of 30 patients more than 50% are going for amputation and more than 25% are going for minor amputation.



7. INFECTIONS

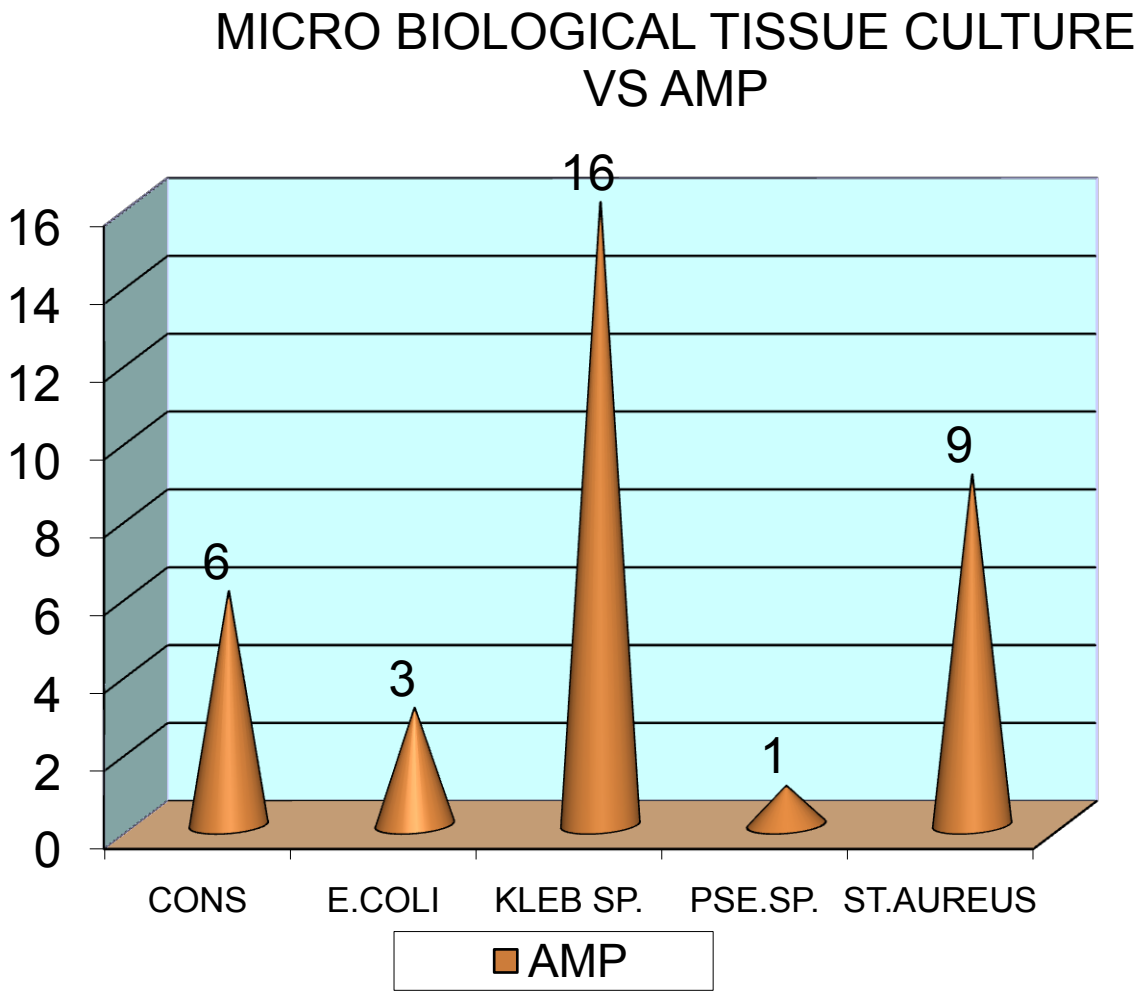
INFECTIONS	AMPUTATION
NIL	0
MONOMICROBIAL	1
POLYMICROBIAL	34

Infections tend to be more severe in case of polymicrobes. Out of 35 amputations, 34 are infected with polymicrobes. Klebsiella species is the predominant infection in our locality.



8. MICROBIOLOGICAL TISSUE CULTURE

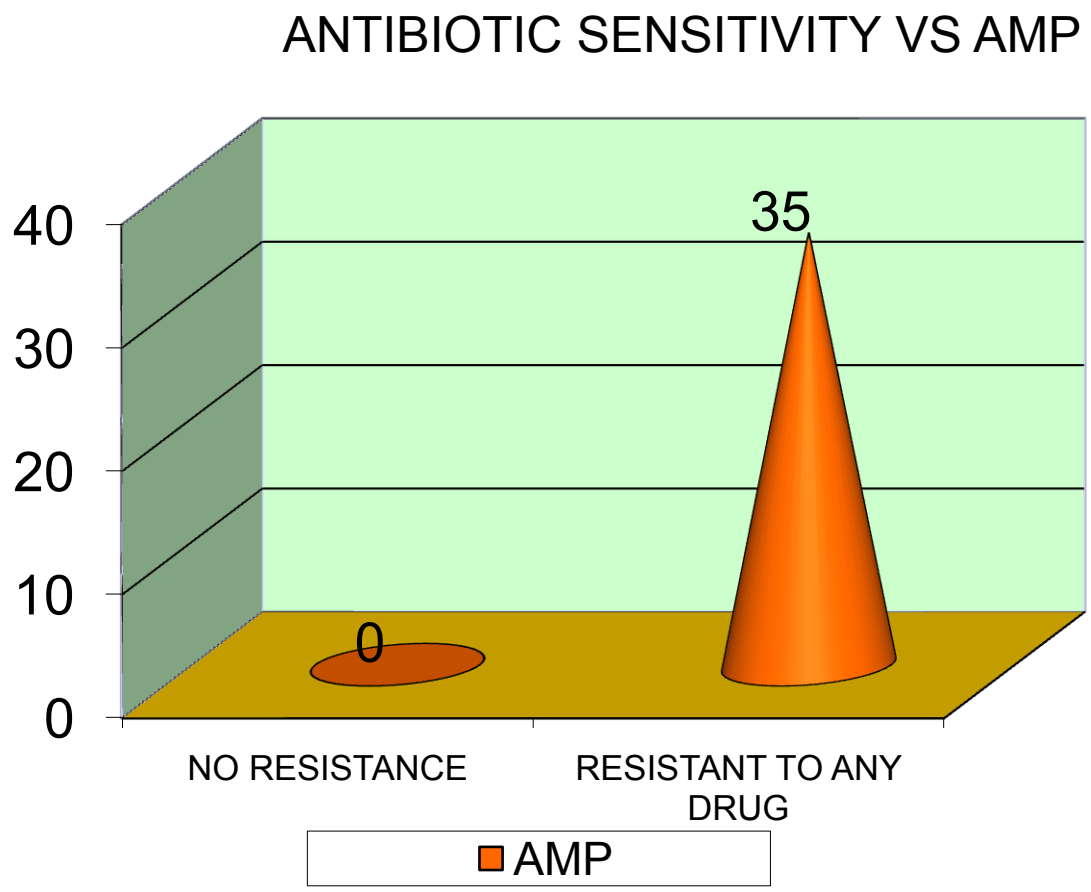
MICROBIOLOGICAL TISSUE CULTURE	AMPUTATION
COAGULASE NEGATIVE STAPH. AUREUS	6
E.COLI	3
KLEBSIELLA SP.	16
PSEUDOMONAS SP.	1
STAPH. AUREUS	9



9. ANTIBIOTIC SENSITIVITY/ RESISTANT PATTERN

ANTIBIOTIC SENSITIVITY	AMPUTATION
NO RESISTANCE	0
RESISTANT TO ANY DRUG	35

In all those 35 amputations, the organisms were resistant to atleast one drug.



Antibiotics	**No of resistant strains**								
	MRS spp (n=10)	**MSS spp (n=30)**	***Streptococcus* spp (n = 8)**	***P. aeruginosa* (n = 5)**	***K. pneumoniae* (n=35)**	***E.coli* (n =8)**	***Enterococci* spp (n=4)**	***Actinomycetes* spp (n=3)**	***Proteus* spp (n=4)**
Amikacin (30mcg)	5	4	1	2	**12**	2			
Ampicillin (10mcg)	**7**	5	2	**4**	**22**	**6**			
Ampicillin/ Sulbactam (10 by 10)	4	1	3	**3**	**17**	2			
Azithromycin (15mcg)	4	8	2	2	**21**	1			

Carbenicillin (100mcg)	4	7	1	**3**	9	4			
Cefazolin (30mcg)	5	3	2	0	13	4			
Cefepime (30mcg)	4	7	2	2	10	3			
Cefaperazone (75mcg)	5	2	2	1	10	3			
Ceftizoxime (30mcg)	**8**	7	1	2	9	4			
Cefuroxime (30mcg)	**7**	5	1	0	9	4			
Chloramphenicol (30mcg)	2	4	1	**3**	8	2			

Gatifloxacin (5mcg)	2	2	2	2	4	3			
Lomefloxacin (10mcg)	**6**	**19**	3	**3**	10	**5**			
Meropenem (10mcg)	5	2	1	1	3	1			
Nitrofurantoin (300mcg)	0	1	2	2	8	2			
Penicillin (10mcg)	**9**	3	2	**3**	**18**	2			
Ticacillin/Clavulanic acid	5	2	3	2	11	**5**			

From the table it is clear that there are some organisms showing high resistance to the antibiotics. Those organisms showing more than 50% resistant strains are highlighted in bold.

More than 50% of the MRS spp showed resistance to Ampicillin (10mcg), ceftizoxime (30 µg), cefuroxime (30 µg), lomefloxacin (10 µg) and penicillin (10 µg).

While more than 50% of the MSS spp showed resistance to lomefloxacin (10 µg). Gram – positive *Streptococcus* spp showed reasonably good sensitivity to all the tested antibiotics with very little resistance seen.

In the Gram – negative aerobes more than 50% of *the K.pneumoniae* strains showed resistance to ampicillin (10 µg), Ampicillin/ Sulbactam (10 by 10), azithromycin (15 µg) and Penicillin (10mcg). More than 50% of the *E. coli* strains showed resistance to ampicillin (10 µg), lomefloxacin (10 µg) and ticarcillin/clavulanic acid (75 by 10).

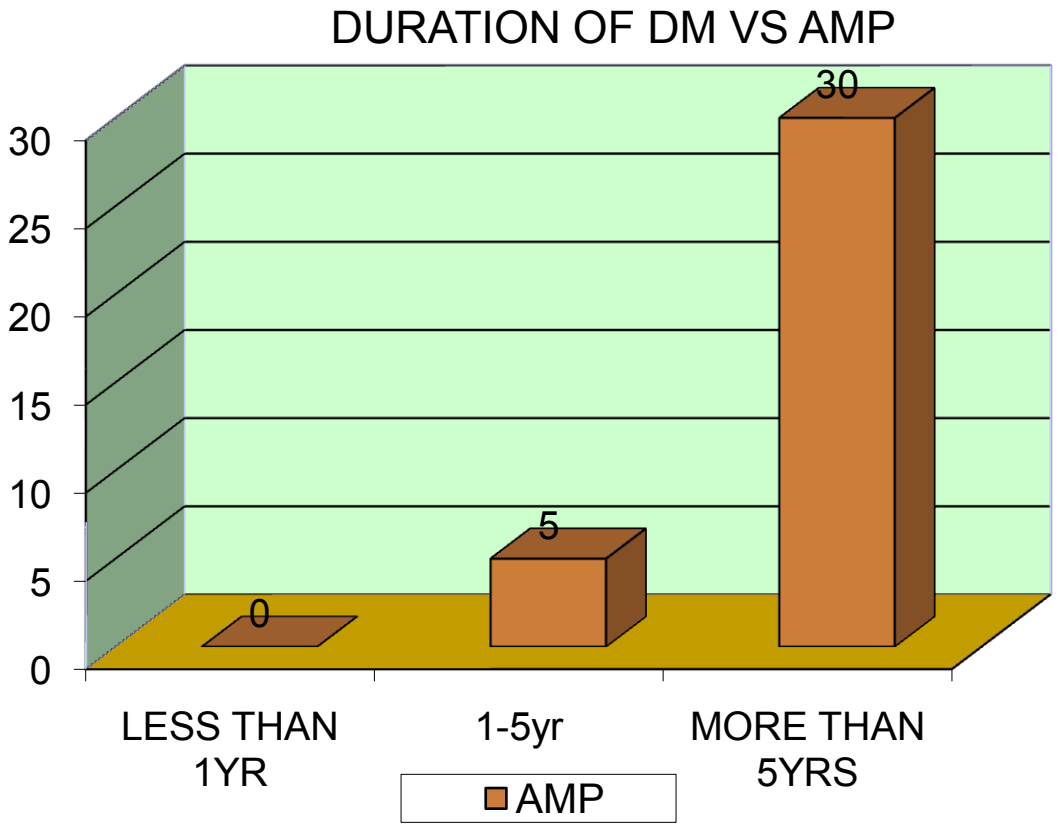
Common organism and their antibiotic sensitivity pattern in our locality

- Klebsiella species- Cefuroxime/ Ceftizoxime/ Meropenem
- Staph. aureus- Cefaperazone Sulbactam/ Amoxyclav. Acid/ Meropenem
- Coagulase negative Staph. aureus- Cefepime
- E.coli- Cefaperazone Sulbactam/ Azithromycin/ Meropenem
- Pseudomonas species- Cefuroxime/ Cefaperazone Sulbactam/ Amikacin

10. DURATION OF DIABESTES

DURATION OF DIABETES	AMPUTATION
LESS THAN 1YR	0
1-5yr	5
MORE THAN 5YRS	30

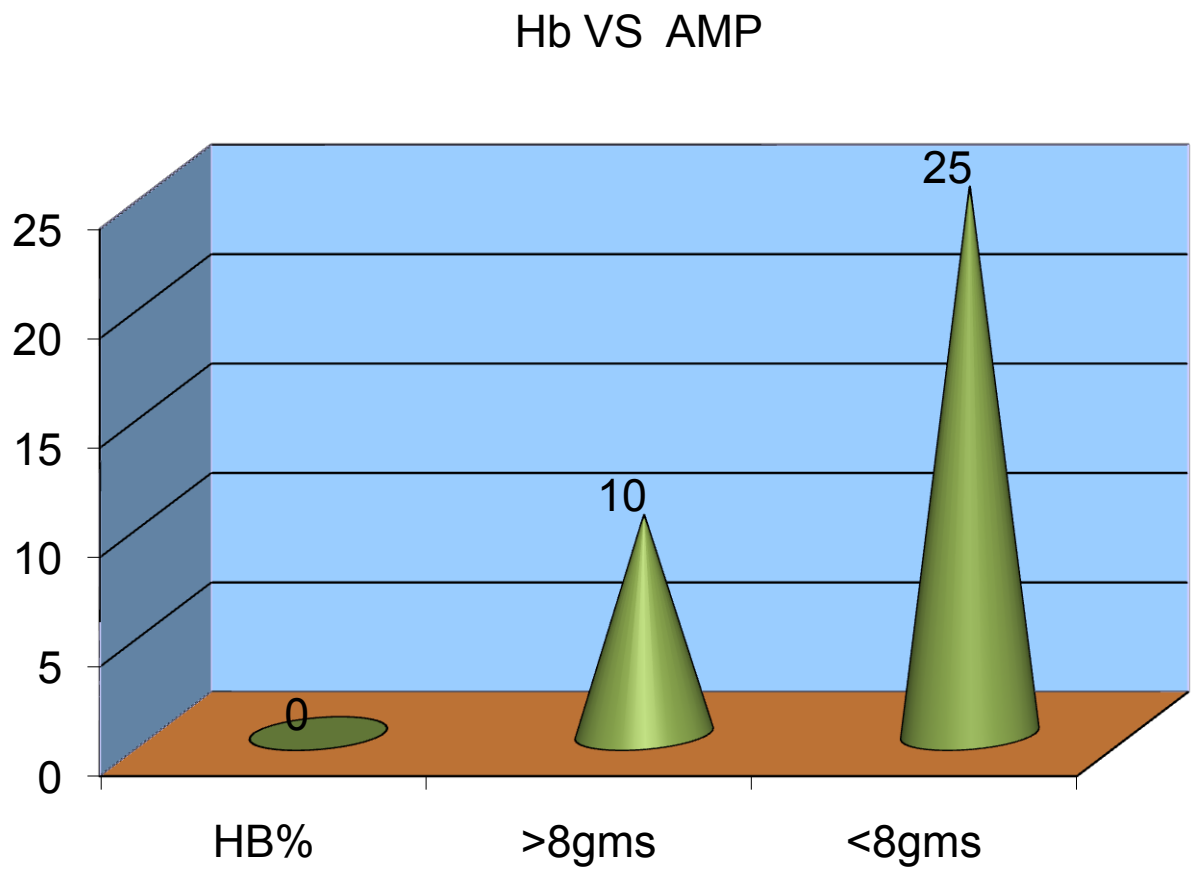
Risk of amputation increases with increase in duration of Diabetes.



11. HAEMOGLOBIN

HB%	AMPUTATION
>8gms	10
<8gms	25

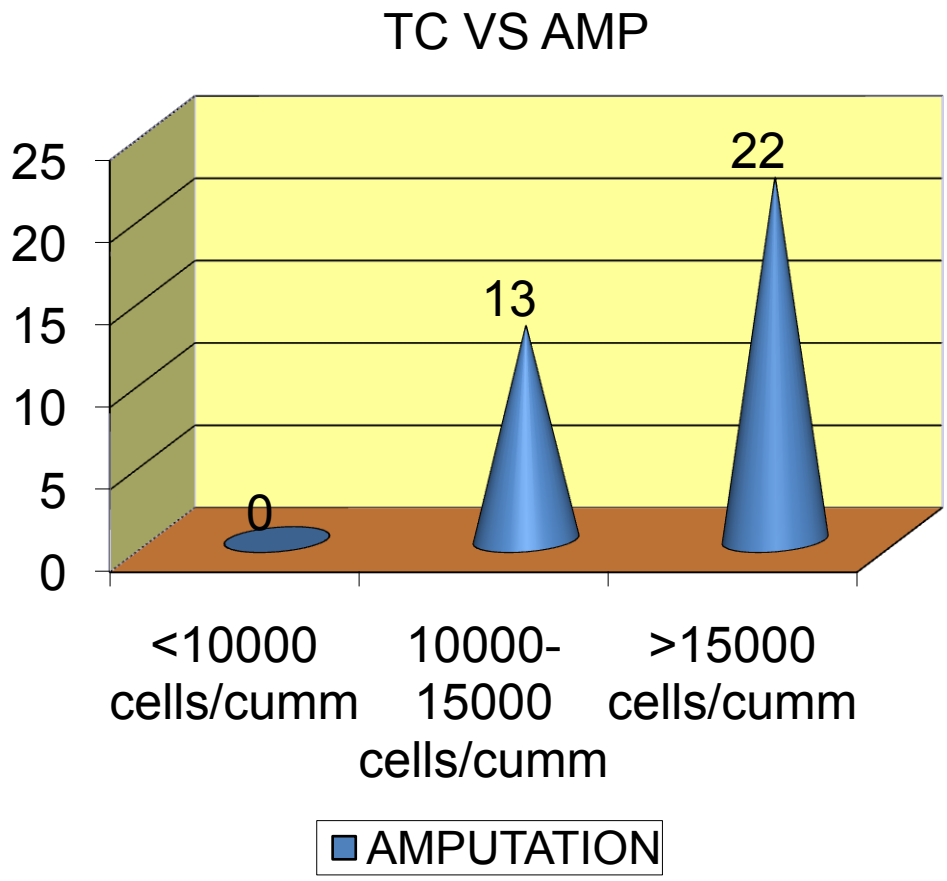
Out of 35 amputations, amputation tendency increases with fall in Hb%.



12.TOTAL WBC COUNT

TC	AMPUTATION
<10000 cells/cumm	0
10000-15000 cells/cumm	13
>15000 cells/cumm	22

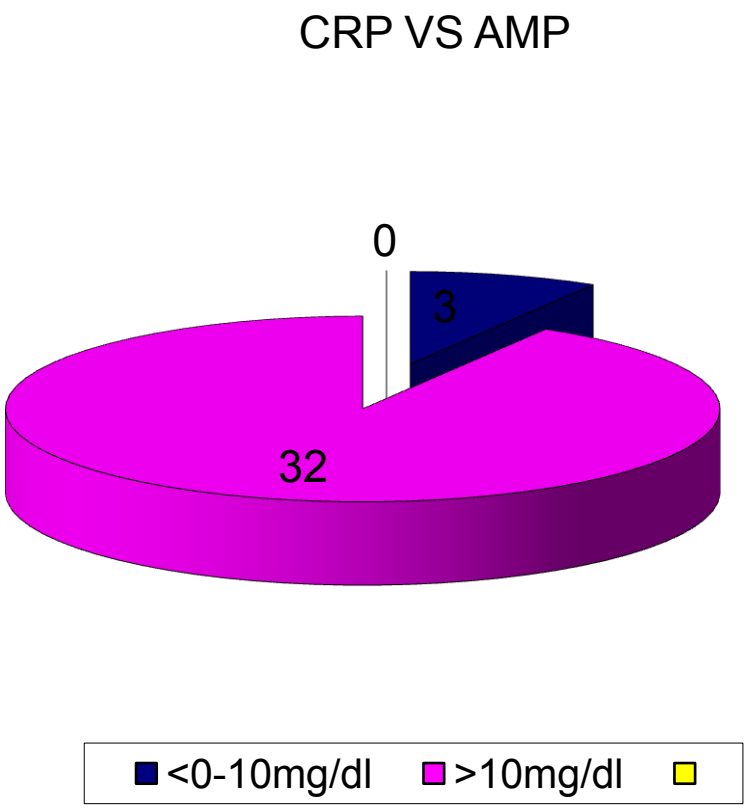
Total count reflects the rate of wound infection and hence amputation rate increases with increase in TC.



13. C-REACTIVE PROTEIN

CRP	AMPUTATION
<0-10mg/dl	3
>10mg/dl	32

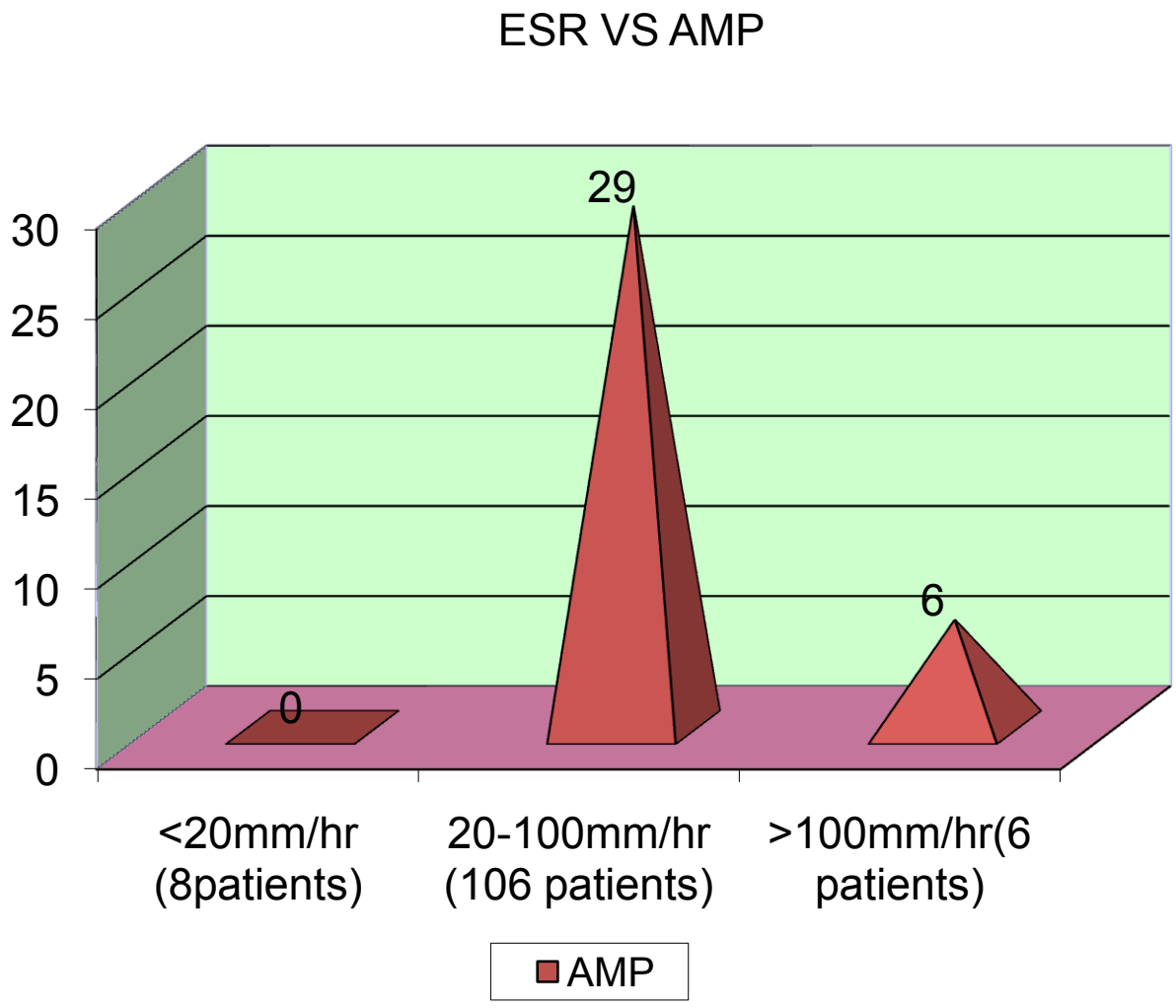
CRP denotes the acute severity of infection. Out of 35 amputations, 32 patients had elevated CRP.



14. ERYTHROCYTE SEDIMENTATION RATE

ESR	AMPUTATION
<20mm/hr (8 patients)	0
20-100mm/hr (106 patients)	29
>100mm/hr (6 patients)	6

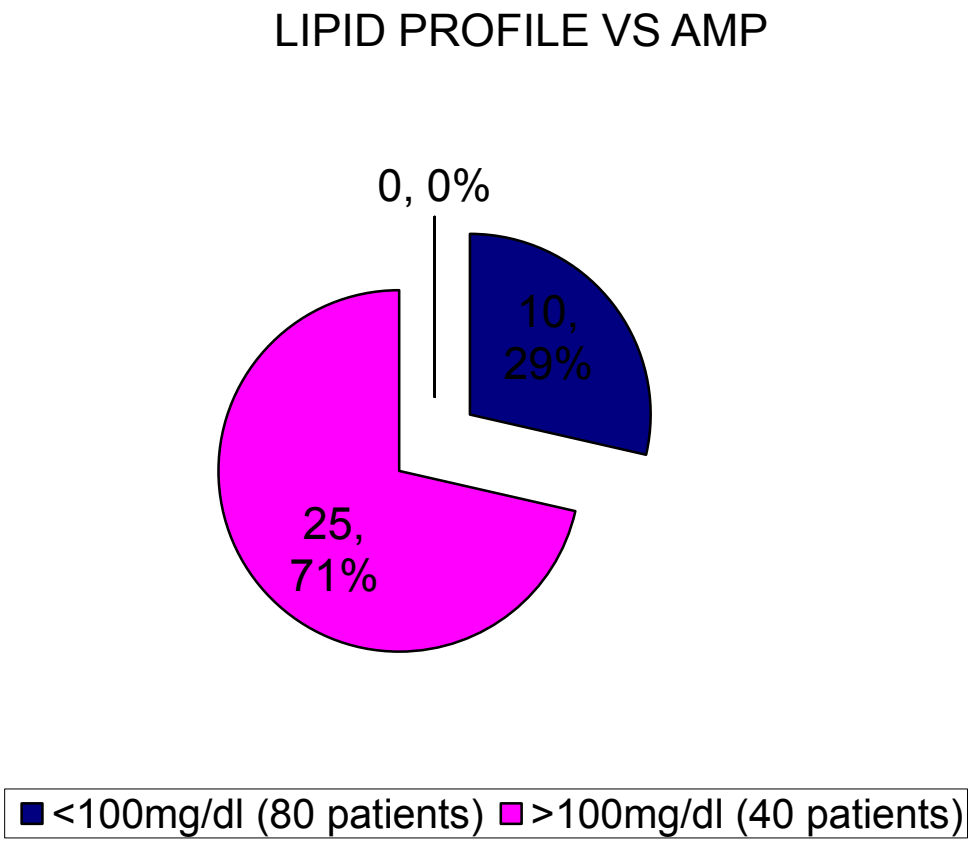
In case of ESR<20mm/hr, all patients were managed conservatively while patients with ESR>100mm/hr went for amputation.



15. LIPID PROFILE (LDL)

LIPID PROFILE (LDL)	AMPUTATION
<100mg/dl (80 patients)	10 (29%)
>100mg/dl (40 patients)	25 (71%)

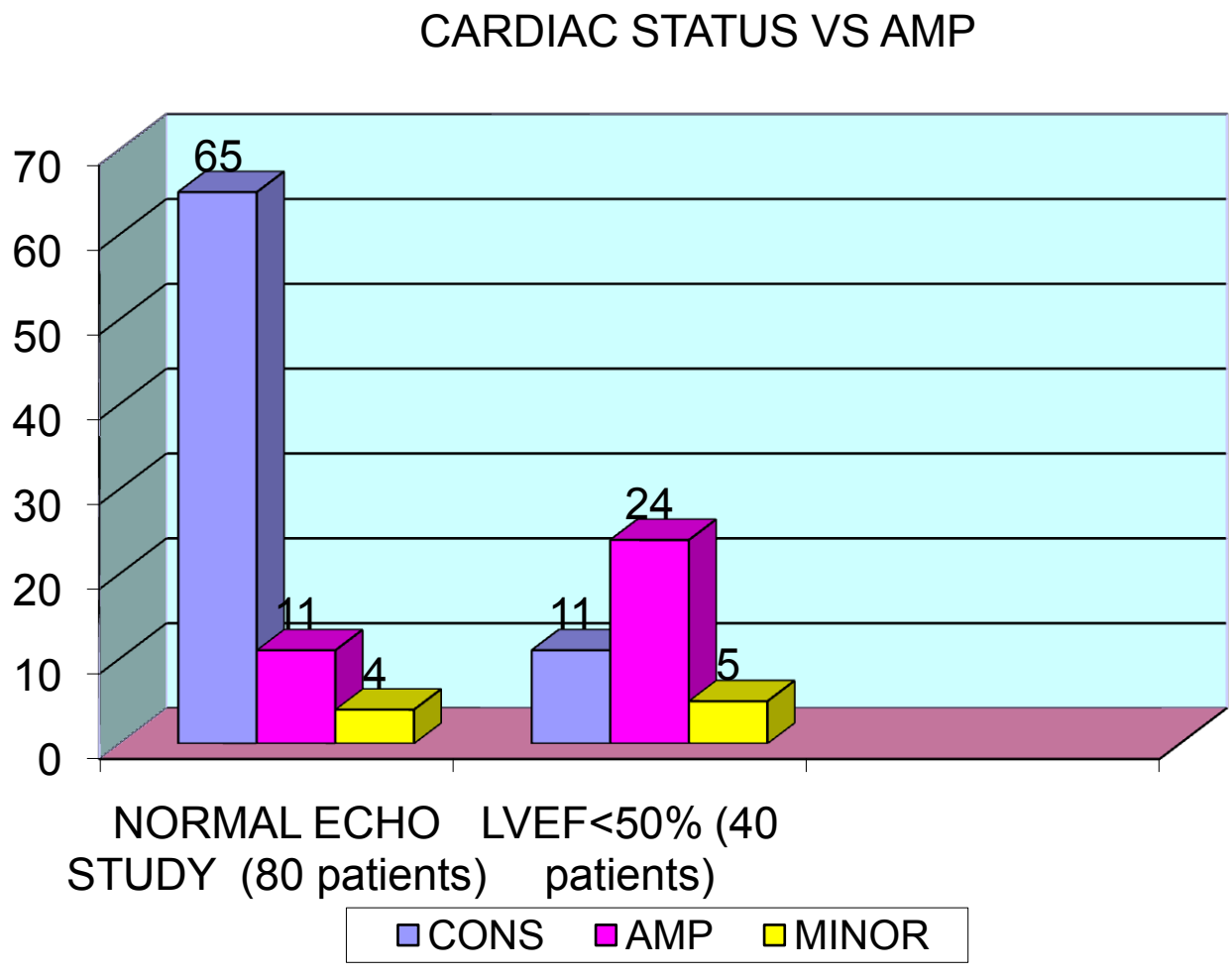
Lipid profile plays an important role in influencing diabetic foot amputation. Out of the total 35 amputees, 10 had <100mg/dl of LDL level whereas 25 had >100mg/dl.



16. CARDIAC STATUS

CARDIAC STATUS	CONSERVATIVE	AMPUTATION	MINOR
NORMAL ECHO STUDY	65	11	4
LVEF<50%	11	24	5

A decrease in Ejection fraction compromises peripheral blood supply which in turn leads to poor oxygenation to the tissues and aggrevates infections. In almost 60% of amputed patients LVEF is less than 50%.



MADURAI MEDICAL (MM) SCORING SYSTEM FOR ANALYSING MAJOR CONTRIBUTING FACTORS FOR LOWER LIMB AMPUTATIONS IN DIABETIC FOOT INFECTIONS

	0	1	2
PRESENCE OF GANGRENE	NIL	MINIMAL COLOR CHANGE	(+)(+) COMPLETE GANGRENE
PULSE STATUS OF CONCERNED PART	(+)(+)	(+)	(-)
ABI	0.9-1.1	0.9-0.3	<0.3
BONY INVOLVEMENT	NIL		OSTEOMYELITIS (+)
INFECTIONS	NIL	MONOMICROBIAL	POLYMICROBIAL
DURATION OF DIABETES	LESS THAN 1YR	1-5yr	MORE THAN 5YRS
ANTIBIOTIC SENSITIVITY	NO RESISTANCE		RESISTANT TO ANY DRUG
HB%	>12gms	8-12gms	<8gms
TC	<10000 cells/cumm	10000-15000 cells/cumm	>15000 cells/cumm
ESR	<20mm/hr	20-100mm/hr	>100mm/hr
CRP	<0-10mg/dl	10-15mg/dl	>15mg/dl
LIPID PROFILE (LDL)	<100mg/dl	100-189mg/dl	>190mg/dl
CARDIAC STATUS	NORMAL ECHO STUDY	LVEF<50%	LV THROMBUS; EF<40%

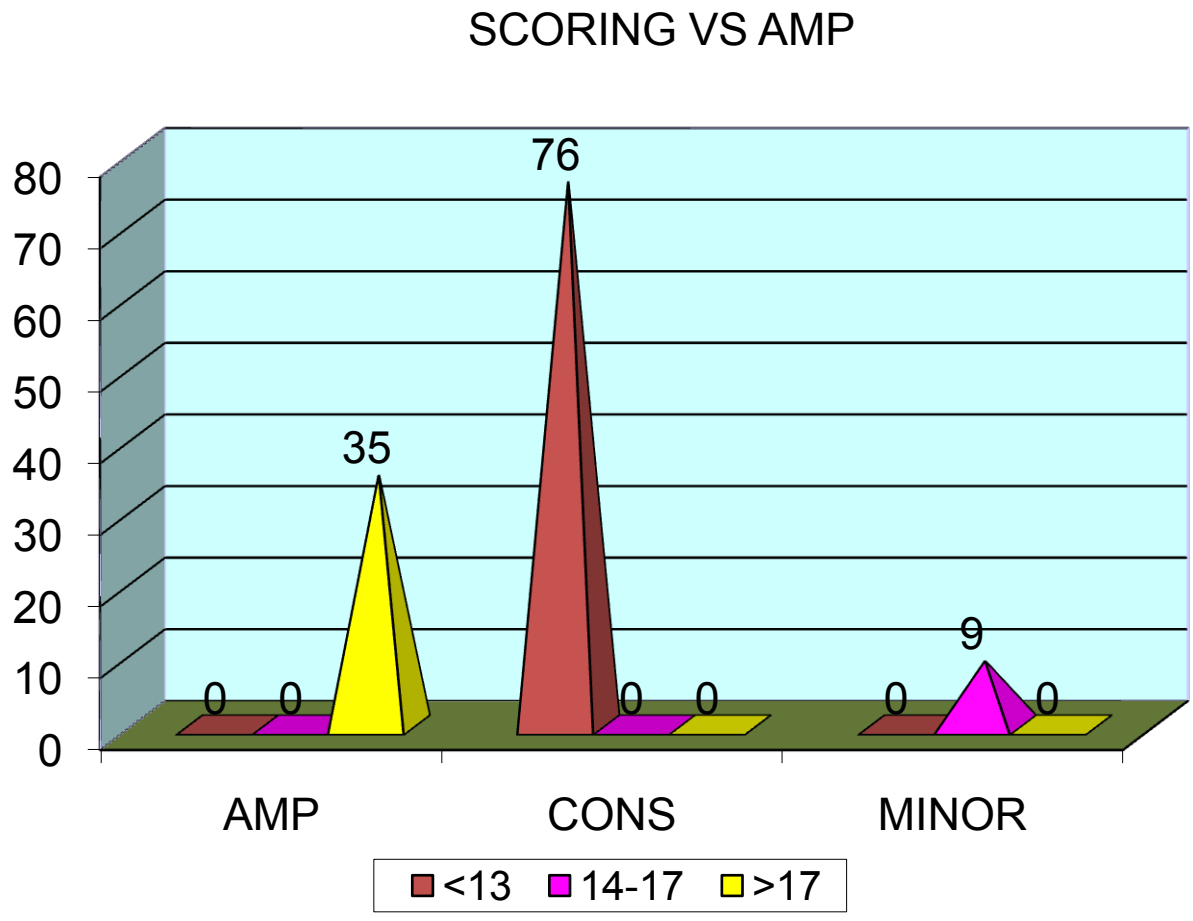
CONCLUSION

The conclusion derived from the above observation are:

1. The following factors are the major contributory/predictive factors for amputation in Diabetic foot patients -
    - Presence of gangrene
    - Pulse status of concerned part
    - Ankle brachial index
    - Presence of osteomyelitis
    - Soft tissue Infections
2. The minor contributory/predictive factors for amputation in Diabetic foot patients -
    - Haemoglobin status
    - Total white blood cell count
    - Erythrocyte sedimentation rate
    - C-reactive protein
    - Lipid profile (LDL)
    - Cardiac status

Conclusion of Madurai Medical (MM) Scoring system -

SCORE	SURGICAL MANAGEMENT
13 & BELOW	DEBRIDEMENT ALONE
14-17	MINOR AMPUTATION
18 & ABOVE	NON-SALVAGEABLE



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CASE PROFORMA

- NAME :
- AGE/SEX :
- ADDRESS :
- PHONE NO :
- COMPLAINTS :
- SITE OF INVOLVEMENT :
- HISTORY OF CLAUDICATION PAIN :
- HISTORY OF NUMBNESS/LOSS OF SENSATION :
- HISTORY OF TRAUMA :
- HISTORY OF FOUL SMELLING DISCHARGE :
- HISTORY OF PREVIOUS SURGERY :
- DRUG HISTORY :
- FAMILY HISTORY :
- DIET HISTORY :
- GENERAL EXAMINATION :
- LOCAL EXAMINATION :
  - INSPECTION :
  - PALPATION :
  - PULSE STATUS :
  - ABI :

❖ DIAGNOSIS :

❖ INVESTIGATIONS :

- Hb%:
- Tc/Dc:
- Creatinine:
- ESR:
- CRP:
- Lipid profile:
- X-Ray:
- Echocardigram:
- Bacteriological tissue culture:
- Monomicrobial:
- Polymicrobial:
- Antibiotic Sensitivity Patterns:

MASTERCHART

S.No.	NAME	AGE/SEX	IP No.	GANGRENE	PULSE STATUS	ABI	BONY INVOLVEMENT	INFECTIONS	DURATION OF DIABETES	ANTIBIOTIC SENSITIVITY	HB%	TC	ESR	CRP	(LDL)	CARDIAC STATUS	MICROBIOLOGICAL TISSUE CULTURE	SCORING	MANAGEMENT
1	SUBRAMANI	47/M	11177	1	1	1	0	2	1	2	0	1	1	1	0	0	CO.NS	11	CONS
2	VELUSAMY	49/M	40456	1	0	0	0	2	1	2	1	1	1	1	0	0	CO.NS	10	CONS
3	AJMERKHAN	35/M	1080885	0	0	0	0	2	1	2	0	1	1	1	1	1	E.COLI	10	CONS
4	VELUKONAR	60/M	17773	2	2	2	0	2	2	2	2	1	1	1	2	0	CO.NS	19	AMP
5	MUNIYASAMY	50/M	1053663	2	2	2	0	2	2	2	1	2	1	1	1	1	KLEB SP.	19	AMP
6	MEENAKSHI	55/F	76771	0	0	0	0	1	1	0	2	1	0	0	0	0	CO.NS	5	CONS
7	KOTHAIAMMAL	80/F	79696	1	1	1	2	2	1	2	1	1	1	1	1	1	KLEB SP.	16	MINOR
8	SHANMUGAVEL	45/M	1082152	0	0	0	0	2	1	2	1	1	1	1	1	0	CO.NS	10	CONS
9	AYYAPAN	50/M	1057491	2	2	2	0	2	2	2	2	2	2	1	1	0	S.AUREUS	20	AMP
10	THIRUMALAI	48/M	1056439	0	0	0	0	1	1	1	1	1	1	0	0	0	CO.NS	6	CONS
11	MOOKAMAL	60/F	78231	2	2	2	2	2	2	2	0	1	1	1	0	1	S.AUREUS	18	AMP
12	DHANALAKSMI	35/F	79760	1	1	1	0	2	1	2	0	1	1	1	0	2	S.AUREUS	13	CONS
13	SELVARAJ	55/M	1088271	1	0	0	0	2	0	2	1	1	1	1	1	1	CO.NS	11	CONS
14	SITHUPANDI	48/M	1120943	2	2	2	0	2	2	2	2	2	1	1	1	1	S.AUREUS	20	AMP
15	JANAKI	44/M	75731	1	1	1	0	2	1	2	2	1	1	1	0	0	E.COLI	13	CONS
16	JAYAMANI	60/F	16781	1	0	0	0	2	1	2	1	1	1	1	1	0	CO.NS	11	CONS
17	NAGENDRAN	55/M	1092231	0	0	0	0	1	0	0	1	1	0	0	0	0	KLEB SP.	3	CONS
18	BALASUBBU	56/M	1125429	1	1	1	2	2	1	2	1	1	1	1	0	0	CO.NS	14	MINOR
19	RAGHU	42/M	50293	0	0	0	0	2	1	2	0	1	1	1	1	0	S.AUREUS	9	CONS
20	KANNAN	50/M	1135441	2	2	2	0	2	2	2	2	2	2	1	1	1	CO.NS	21	AMP

21	ANANDHAM	45/M	287	1	1	1	0	2	2	2	1	1	1	1	0	0	PSE.SP.	13	CONS
22	MANIMARAN	42/M	1140096	2	2	2	2	2	2	2	1	2	1	1	0	1	CO.NS	20	AMP
23	MEENAKSHI	70/F	58821	1	1	1	0	2	1	2	1	1	1	1	0	0	KLEB SP.	12	CONS
24	PALANIAMMAL	50/F	1096694	2	2	2	2	2	2	2	2	1	1	1	1	0	S.AUREUS	20	AMP
25	SEENIAMMAAL	65/F	37793	1	0	0	0	2	1	2	2	2	1	1	0	0	PSE.SP.	12	CONS
26	SUBBAIYA	65/M	1135123	0	0	0	0	1	0	2	1	1	1	1	1	0	E.COLI	8	CONS
27	VEERAPAN	68/M	58710	1	1	1	0	2	1	2	1	1	1	1	1	0	CO.NS	13	CONS
28	SUBRAMANI	54/M	1142021	2	2	2	0	2	2	2	1	2	1	1	1	1	S.AUREUS	19	AMP
29	KARUPPAIYA	65/M	1110742	2	2	2	0	2	2	2	2	1	1	1	1	2	E.COLI	20	AMP
30	VARADHARAJ	64/M	1101145	2	2	2	2	2	2	2	1	1	1	1	1	1	S.AUREUS	20	AMP
31	MARIAMMAL	55/F	1115674	0	1	1	2	2	1	2	1	1	1	0	1	0	E.COLI	13	CONS
32	LAKSHMANAN	61/M	53456	1	1	1	0	2	1	2	1	1	1	0	0	0	KLEB SP.	11	CONS
33	KALIAPPA	58/M	1111245	1	1	1	0	2	1	2	1	1	1	0	0	0	S.AUREUS	11	CONS
34	VASANTHAM	64/F	15634	1	1	1	0	2	1	2	1	1	1	0	1	0	CO.NS	12	CONS
35	JAYAMANI	60/F	31566	2	2	2	0	2	2	2	2	2	2	1	1	1	CO.NS	21	AMP
36	PERIYASAMY	69/M	1118765	0	0	0	0	0	1	2	1	1	1	0	0	0	E.COLI	6	CONS
37	KULANDHAIVEL	72/M	1118976	0	0	0	0	2	1	2	0	1	1	0	0	0	S.AUREUS	7	CONS
38	NATARAJA	75/M	1115321	2	2	2	0	2	1	2	2	1	1	1	1	2	KLEB SP.	19	AMP
39	NADARAJAH	59/M	56453	0	0	0	0	2	1	0	0	1	0	0	0	0	KLEB SP.	4	CONS
40	RAVINDRAN	74/M	1119864	0	1	0	0	2	1	2	1	1	1	0	0	0	CO.NS	9	CONS
41	JOHN PETER	43/M	11304	2	2	2	0	2	2	2	2	2	1	1	0	1	KLEB SP.	19	AMP
42	RAMAKRISNAN	66/M	52678	0	0	0	0	2	1	2	1	1	1	0	0	0	PSE.SP.	8	CONS
43	VADIVELU	60/M	1115674	1	1	1	2	2	1	2	1	1	1	0	0	0	KLEB SP.	13	CONS
44	SAVITHRI	63/F	1117635	1	1	1	0	2	1	2	1	1	1	0	1	1	CO.NS	13	CONS
45	CHINNASAMY	55/M	145221	2	2	2	0	2	2	2	2	2	1	1	1	1	KLEB SP.	20	AMP
46	GOUNDER	50/M	48281	2	2	2	2	2	2	2	2	2	1	1	0	0	S.AUREUS	20	AMP

47	DHARMAN	57/M	1118078	2	2	2	2	2	1	2	2	1	1	1	1	0	E.COLI	19	AMP
48	KATHIR	65/M	14534	1	1	1	0	2	1	2	1	1	1	0	0	1	KLEB SP.	12	CONS
49	CHITRA	65/F	1111546	0	1	1	0	1	1	2	1	1	1	0	1	0	CO.NS	10	CONS
50	SOLAIRAJA	64/M	34562	1	1	1	0	2	1	2	1	1	1	0	0	0	S.AUREUS	11	CONS
51	RAMALINGAM	59/M	1115632	1	1	0	0	2	1	2	1	1	1	0	0	0	KLEB SP.	10	CONS
52	ANNAMAYIL	35/F	147623	2	2	2	0	2	2	2	2	2	1	1	1	1	KLEB SP.	20	AMP
53	ANANDHASAMY	61/M	1114367	1	1	1	0	2	1	2	1	1	1	0	0	0	CO.NS	11	CONS
54	XAVIER	60/M	53423	1	1	1	2	2	1	2	1	1	1	0	0	0	KLEB SP.	13	CONS
55	SAKTHIVEL	58/M	1113456	0	0	0	0	0	0	1	0	1	0	0	0	0	S.AUREUS	2	CONS
56	SIVAGAMI	60/F	1198231	0	1	1	0	2	1	2	2	1	1	0	1	0	KLEB SP.	12	CONS
57	PANDIRAJ	62/M	1117634	0	0	0	0	2	2	2	2	1	0	0	0	0	CO.NS	9	CONS
58	KRISHNASAMY	63/M	147623	2	2	2	0	2	2	2	1	2	2	1	1	0	KLEB SP.	19	AMP
59	BHARATHI	59/F	143871	1	1	1	2	2	2	2	2	1	1	0	0	0	PSE.SP.	15	MINOR
60	ANANDHI	62/M	1114567	2	2	2	0	2	2	2	2	2	1	1	1	1	KLEB SP.	20	AMP
61	ANNADURAI	65/M	1114598	0	1	1	0	2	2	2	2	1	1	0	0	0	CO.NS	12	CONS
62	KARPAGAM	66/F	34287	0	0	0	0	2	1	2	1	1	1	0	0	0	KLEB SP.	8	CONS
63	KALAMARAN	72/M	1134982	0	0	0	0	2	1	2	2	1	1	0	0	0	S.AUREUS	9	CONS
64	SARAVANAN	70/M	1123467	1	1	1	2	2	1	2	1	1	1	0	0	1	KLEB SP.	14	MINOR
65	SIVANESAN	69/M	1128761	1	1	1	0	2	1	2	1	1	1	0	0	0	CO.NS	11	CONS
66	VANITHA	65/F	1118124	2	2	2	2	2	2	2	2	1	1	2	1	1	S.AUREUS	22	AMP
67	MUTHUSAMY	73/M	1123657	1	1	1	0	2	1	2	2	1	1	0	0	0	E.COLI	12	CONS
68	DURAIRAJ	70/M	1125673	0	1	1	0	2	1	2	1	1	1	0	0	0	CO.NS	10	CONS
69	KANNAIYA	60/M	1126745	1	0	1	0	2	1	2	1	1	1	0	0	2	KLEB SP.	12	CONS
70	MUTHUPANDI	46/F	1126943	2	2	2	2	2	2	2	2	1	1	0	1	1	KLEB SP.	20	AMP
71	SYED ALI	71/M	1124578	0	0	0	0	1	0	2	1	0	1	0	0	0	CO.NS	5	CONS
72	RAMASAMY	68/M	1122342	0	0	0	0	2	1	2	0	1	1	0	0	0	KLEB SP.	7	CONS

73	MOORTHI	52/M	1127551	2	2	2	0	2	2	2	2	2	1	1	0	1	KLEB SP.	19	AMP
74	KRISHNAMURTHY	64/M	1124576	1	1	1	0	2	1	2	2	1	1	0	0	0	CO.NS	12	CONS
75	PAULRAJ	65/M	1124567	0	0	0	0	2	1	2	1	1	1	0	0	0	KLEB SP.	8	CONS
76	ABDUL HAMEED	70/M	1125567	1	1	1	0	2	1	2	1	2	1	0	0	0	CO.NS	12	CONS
77	TAMILVANAN	40/M	1128895	2	2	2	0	2	2	2	2	2	2	1	1	1	S.AUREUS	21	AMP
78	CHELLAM	60/F	1127902	2	2	2	2	2	2	2	1	1	1	1	0	1	E.COLI	19	AMP
79	KANNAGAVEL	72/M	1122348	1	1	1	0	2	1	2	2	2	1	0	0	0	E.COLI	13	CONS
80	KUMARASAMY	69/M	1123367	0	1	1	0	2	1	2	1	1	1	0	0	0	CO.NS	10	CONS
81	VENKATACHALAM	68/M	112387	1	1	1	0	2	1	2	1	1	1	0	0	2	KLEB SP.	13	CONS
82	AIAGAR	64/M	1124702	2	2	2	2	2	1	2	2	2	1	1	0	1	KLEB SP.	20	AMP
83	KRISHNAN	56/M	1127836	2	2	2	0	2	1	2	2	2	1	1	1	0	KLEB SP.	18	AMP
84	MAILUSAMY	69/M	1125675	1	1	1	0	2	2	2	1	1	1	0	0	0	CO.NS	12	CONS
85	ALAMELU	59/F	1125648	0	0	0	0	1	2	2	1	0	0	0	1	0	KLEB SP.	7	CONS
86	VETRIVEL	69/M	1126756	1	1	1	2	2	2	2	2	1	1	0	0	0	PSE.SP.	15	MINOR
87	PANDI	63/M	1126788	0	0	0	0	2	2	2	0	1	1	0	0	0	KLEB SP.	8	CONS
88	THAGAPANSAMY	70/M	1127891	1	1	1	0	2	2	2	0	1	1	0	0	0	CO.NS	11	CONS
89	AYYER	65/M	1120384	2	2	2	2	1	1	2	1	2	2	1	0	0	KLEB SP.	18	AMP
90	VAITHISWERAN	66/M	1128976	0	1	1	0	2	2	2	2	1	1	0	0	0	KLEB SP.	12	CONS
91	MURUGESAN	60/M	58070	2	2	2	0	2	2	2	2	2	1	1	1	1	KLEB SP.	20	AMP
92	THANGARAJ	69/M	1132345	1	1	1	2	2	1	2	2	1	1	0	0	1	CO.NS	15	MINOR
93	KARIMUTHU	66/M	1135674	1	1	1	0	2	1	2	1	1	1	0	0	0	E.COLI	11	CONS
94	MARIAM	64/M	1135674	0	0	0	0	1	1	0	1	0	0	0	0	0	KLEB SP.	3	CONS
95	VELUSAMI	73/M	1135645	0	0	0	0	2	1	2	1	1	1	0	0	0	PSE.SP.	8	CONS
96	JOHNSON	70/M	1135678	0	0	0	0	2	1	2	2	1	1	0	0	1	KLEB SP.	10	CONS
97	PATHIRAKALI	51/M	1137683	2	2	2	2	2	2	2	2	1	1	1	0	1	CO.NS	20	AMP
98	ANBARASU	70/M	1136578	0	0	0	0	2	2	2	2	1	1	0	0	0	KLEB SP.	10	CONS

99	ASAIMUTHU	71/M	1136713	1	1	1	0	2	1	2	2	1	1	0	0	0	E.COLI	12	CONS
100	PETCHIAMAL	45/F	1137866	2	2	2	2	2	2	2	2	2	1	1	1	0	KLEB SP.	21	AMP
101	JAYARAJ	73/M	1136578	0	0	0	0	2	1	2	1	1	1	0	0	0	CO.NS	8	CONS
102	RATHNASAMY	71/M	1136798	1	1	1	2	2	2	2	1	1	1	0	0	0	E.COLI	14	MINOR
103	KUPPANAN	68/M	1137789	0	0	0	0	2	1	2	0	1	1	0	0	0	KLEB SP.	7	CONS
104	MADASAMY	65/M	1137823	1	1	1	2	2	2	2	0	1	1	0	0	2	KLEB SP.	15	MINOR
105	IRUDHAYARAJ	69/M	1137589	0	0	0	0	2	1	2	1	1	1	0	0	0	CO.NS	8	CONS
106	KARUPUSAMY	75/M	1136354	0	0	0	0	2	1	2	2	1	1	0	0	0	KLEB SP.	9	CONS
107	PERIYAKARUPU	74/M	1138764	0	1	1	0	2	1	2	2	1	1	0	0	0	E.COLI	11	CONS
108	SHEIK ALI	56/M	1137705	2	2	2	2	2	2	2	2	2	1	1	1	0	KLEB SP.	21	AMP
109	KARMEGAM	59/M	1136798	1	1	1	2	2	2	2	2	1	1	0	0	1	CO.NS	16	MINOR
110	SATHYAMOORTHY	50/M	1139994	2	2	2	2	2	2	2	2	1	1	0	1	1	PSE.SP.	20	AMP
111	VEERAGURU	66/M	1134897	1	1	1	0	2	1	2	1	1	1	0	0	0	KLEB SP.	11	CONS
112	CHINNAPONNU	69/F	1132467	0	0	0	0	2	1	2	1	1	1	0	1	0	CO.NS	9	CONS
113	MUTHUKUMAR	45/M	11721	2	2	2	2	2	2	2	1	1	1	1	0	0	KLEB SP.	18	AMP
114	RAMESH	62/M	1136789	1	1	1	0	2	1	2	1	1	1	0	0	1	CO.NS	12	CONS
115	ELAMARAN	59/M	1139878	0	1	1	0	2	1	2	1	1	1	0	0	0	CO.NS	10	CONS
116	GANESAN	60/M	1082341	0	0	0	0	2	0	2	1	1	1	0	0	1	KLEB SP.	8	CONS
117	MANJULA	60/F	35784	0	0	0	0	2	0	2	1	1	1	0	0	0	KLEB SP.	7	CONS
118	MANAVALAGAN	65/M	1139087	1	1	1	2	2	1	2	1	1	1	0	0	0	CO.NS	13	CONS
119	NATARAJAN	72/M	1133542	2	2	2	2	2	2	2	1	2	1	0	1	1	CO.NS	20	AMP
120	ALAGAMMAL	70/F	37871	1	0	0	0	1	2	0	0	1	0	0	0	1	E.COLI	6	CONS

LIST OF ABBREVIATIONS:

1. KLEB SP : KLEBSIELLA SPECIES
2. CO.NS : COAGULASE NEGATIVE STAPHYLOCOCCI
3. E.COLI : ESCHERICHIA COLI
4. S.AUREUS : STAPHYLOCOCCI AUREUS
5. PSE SP : PSEUDOMONAS SPECIES
6. CONS : CONSERVATIVE
7. MINOR : MINOR AMPUTATION
8. AMP : MAJOR AMPUTATION

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Name of the Candidate	:	Dr.B.Sathiyaseelan
Course	:	PG in MS, General Surgery
Period of Study	:	2014-2017
College	:	MADURAI MEDICAL COLLEGE
Research Topic	:	Prospective study of "Analysing predictive factors for major lower extremity amputations in Diabetic foot infections
Ethical Committee as on	:	16.03.2016

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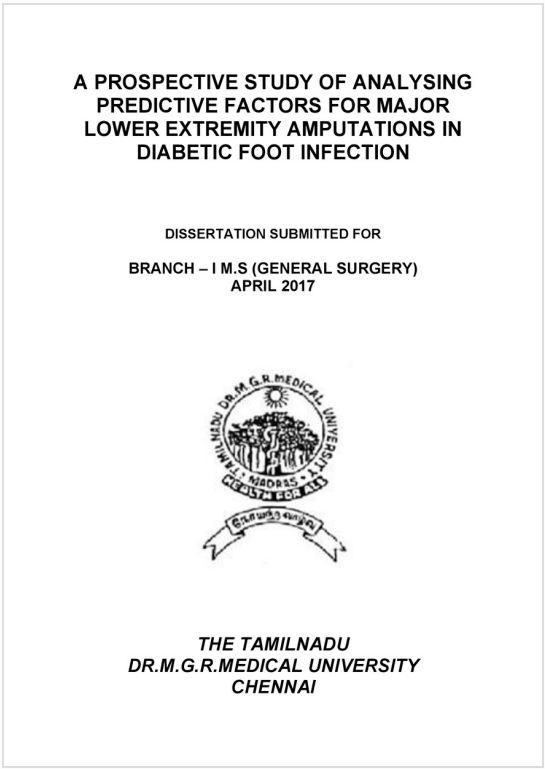

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DISSERTATION SUBMITTED FOR

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