# "A STUDY OF ANKLE – BRACHIAL INDEX AS A PROGNOSTIC INDICATOR IN DIABETIC FOOT ULCERS".

**By**

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## Dissertation

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**Under the guidance of**

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## LIST OF ABBREVATIONS USED.

| | | |
|---|---|---|
| CBC | - | Complete blood count. |
| CVD | - | Cardio vascular diasease. |
| DM | - | Diabetes mellitus. |
| MMP | - | Matrix metalloproteases. |
| IL-6 | - | Interleukin 6. |
| TNF α | - | Tumour necrosis factor α. |
| NF κ B | - | Nuclear factor κ B. |
| AGE | - | Advanced glycosylation end products. |
| FFA | - | Free Fatty Acids. |
| LEAD | - | Lower extremity arterial disease. |
| UKB | - | Urine ketone bodies. |
| Hba1c | - | Glycosylated haemoglobin. |
| PAD | - | Peripheral arterial disease. |
| IP | - | In patient. |

# ABSTRACT

**Background and objectives.**

Foot ulcerations are a common complication of diabetes accounting to 15% of all diabetics. 15% of diabetics undergo lower limb amputation. Ankle-Brachial index has a sensitivity of 70.6% & specificity of 88.5% in diagnosis of peripheral vascular diseases. But no literature is available where ABI is studied as a prognostic indicator in diabetic foot ulcers. Can ABI predict the outcome of diabetic foot ulcers ? The objective of this study was to assess ABI as a prognostic indicator in diabetic foot ulcers.

**Methodology.**

The present study was conducted in the Department of Surgery K.L.E.S Dr. Prabhakar Kore Hospital and Medical Research Centre , Belgaum on 74 patients with diabetic foot ulcers between Nov 2008 to Nov 2009. The ABI was measured by Colour Doppler on the day of admission. The patients were treated as per hospital protocols. They were followed up for 6 months. The outcome of the ulcers were categorized into 2 groups: limb salvaged & major amputation. Retrospectively the ABI were reviewed & analysis was done.

**Results.**

There were 66 males & 8 females in the study. 64.9% of patients were in the age group of 71 –90 years. 43.2 % of patients had diabetes since 6 – 9 years. Infection was present in 59.5% & absent in 40.5%. Neuropathy was present in 56.8% of patients & absent in 43.2%. ABI for patients who underwent major

amputation was < 0.84 ( 0.74 +/- 0.1). ABI for patients in whom limb salvage was possible was 0.84 – 1.0 (0.92 +/- 0.08).

**Conclusion.**

ABI is falsely raised in diabetics due to atherosclerotic calcification of vessels. ABI is an excellent prognostic indicator in diabetic foot ulcers. If ABI is < 0. 84, one should consider angiography & revascularization procedure. If angiography shows diffuse disease unsuitable for revascularization or if the angiography is not affordable to the patient then, surgeon should consider major amputation without wasting time. ABI between 0.84 – 1.0 is more in favour of healing of the ulcer & salvage of limb. However larger trials are required for better assessment of ABI as a prognostic indicator in diabetic foot ulcers.

**Key words:** diabetic foot ulcers; diabetes mellitus ; ankle – brachial index.

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# INTRODUCTION

Diabetes mellitus, a chronic disease once thought to be uncommon in the developing world, has now emerged as a serious public health problem in Asia.At least 177 million people worldwide suffer from diabetes; This figure is likely to be more than double by 2030.Around 4million deaths every year are attributable to complications of diabetes[1].Top five countries with the most diabetes sufferers in 2003 were: India : 35.5 million, China : 23.8 million, USA : 16 million, Russia : 9.7 million and Japan : 6.7million[2]

Foot ulcerations are a leading cause of hospitalisation in diabetics. "Rule of 15" in diabetes mellitus is 15% of diabetics develop lower limb ulcers, 15% of ulcers have osteomyelitis & 15% of ulcers result in amputation.Half of patients undergoing amputation of one limb will undergo amputation of contralateral limb within two years[3].Diabetes is the most common cause of nontraumatic amputation of lower limb[1].

Peripheral neuropathy, peripheral vascular disease & susceptibility to infection are the three most important causes for development of diabetic foot ulcers.

Diabetes mellitus should also be considered a vascular disease because diabetic patients have a strong predilection for atherosclerosis. Macroangiopathy & microangiopathy are both features of diabetes mellitus[4].

Major causes of amputation in diabetics include profound infection, gangrene, lower limb ischaemia & foot deformities. Vascularity of lower limb is the most important factor for prognosis of diabetic foot ulcers.

Vascularity of lower limb is assessed by clinical exam, by a hand held Doppler, Colour Doppler or angiographic studies. Ankle – brachial index is measured by dividing the ankle systolic pressure to brachial systolic pressure using a hand held Doppler or colour Doppler . **Normal value:** 1.11 +/- 0.10.

In limbs with intermittent claudication ABI ranges from 0.2- 1.0 with mean of 0.59 +/- 0.15. ABI in limbs with ischaemic rest pain 0 – 0.65. ABI in limbs with impending gangrene is 0.05 +/- 0.08.Many limbs with impending gangrene or ischaemic ulceration ABI is 0 due to absent flow signals. ABI ( ankle – brachial index) <0.3 denotes critical limb ischaemia[4].Amputation in diabetic foot ulcers (in the absence of gangrene & profound infection ) generally gets delayed as such patients are subjected to multiple debridements before decision for amputation is taken.

In the present day the need for amputation in diabetic foot ulcers is taken based on two observations, 1) that the ulcer is showing no signs of healing inspite of repeated debridement & daily dressings &2) the various vascularity studies like colour Doppler or angiography showing reduced/ absent flow in the lower limbs. Angiography, considered gold standard, may not be possible in Acute/Chronic renal failure patients or may not be affordable to the patients.

Ankle-Brachial Index is cheap, bedside & easy to perform investigation.ABI has been previously studied in peripheral vascular diseases but it has not been adequately studied in diabetic foot ulcers till date.The role of ABI in aiding the early decision making in diabetic foot ulcers is still not clearly answered, thus encouraging us to take up the study.

# OBJECTIVES OF THE STUDY

The objective of the present study was to know whether ankle brachial index can act as a prognostic indicator in diabetic foot ulcers.

# REVIEW OF LITERATURE.

**Introduction.** *"The era of coma has given way to the era of complications"* – Elliot P.Joslin. Professor Joslin's quote from first half of last century is as fitting today as it was some 70 years ago. Diabetics no longer die from acute complications but rather it is the chronic complications that predominate[3].

**Definition of diabetes[5-11].**

"Diabetes mellitus (DM) is characterized by chronic hyperglycemia with disturbances of carbohydrates, fat, and protein metabolism resulting from defects in insulin secretion, insulin action, or both".

**Classification[5-11]**

***Type I***

IA : Autoimmune beta cell destruction which leads to insulin deficiency.

IB : Lack of immunologic markers indicative of an autoimmune destructive process of the beta cells..

***Type II***

It is a heterogeneous group of disorders characterized by:-

- Impaired insulin secretion.
- Variable degree of insulin resistance.

- Increased glucose production

One million amputations each year are caused by diabetes. A diabetic is up to 40 times more likely to need a lower-limb amputation when compared to a person who does not have diabetes[2].Using a component model, Pecoraro, Reiber, & Burgess delineated the "causal pathway" to amputation with a landmark analysis of individual clinical factors in patients with diabetes. No factor alone was sufficient to result in amputation, but several together could. Nearly three – quarters of amputation had the following component pathway.

1. Peripheral neuropathy : an essential aspect.
2. Trauma : usually from just daily ambulation.
3. Ulceration : typically of the plantar skin.
4. *Faulty healing*.

   With time infection & ischaemia may worsen & patients succumb to amputation.

**Healing in acute wounds[12].**

An acute wound heals through 4 phases. 1) coagulation. 2) inflammation. 3)proliferation. 4) remodelling.

**Coagulation:** Occurs immediately at the time of injury. Platelet gets activated, binds thrombin & forms a plug. Platelet derived growth factors & fibroblast growth factors are released. They act as chemoattractants for neutrophils & lymphocytes.

**Inflammation:** Vasodilatation occurs. Neutrophils & macrophages enter the wound & produce more cytokines & growth factors. Cytokines attract fibroblasts which infilterate the provisional matrix & begin to produce fibronectin & collagen. Inflammatory cells produce MMPs ( matrix metalloproteases ). MMps clear the way for angiogenesis.

**Proliferation:** Fibroblasts & endothelial cells are recruited locally. The newly formed matrix with rich arcade of new vessels appears to the naked eye as "granulation tissue". Migration of the keratinocytes across the wound surface occurs to create a new epidermis. Wound is closed completely by 10 – 14 days.

**Remodeling:** Continues for 6 – 12 months. Collagen fibrils in the scar tissue are remodeled in a turnover process that results from the interplay of degradation from MMPs & the production of matrix components from the fibroblasts.

Good vascularity is necessary for the stage of inflammation & proliferation to occur successfully.

**Chronic wounds**[13] differ from acute wounds in the lack of orderly progression to healing. Diabetic foot ulcers are unique chronic wounds in their pathogenesis & pathophysiology. The chronic wound is stuck in a disorderly mix of inflammation & failed bursts of proliferation. The chronic wound is characterized by 4 cardinal defects.

1. ***Inflammatory excess:*** excessive inflammation occurs, with over production of inflammatory cytokines , such as IL -6, TNF ( tumour necrosis factor ) alpha,

& MMPs particularly MMP-1, MMP – 8, & MMP-13. This allows altered matrix substance to accumulate, such as fibronectin, which have been rendered ineffective by protein degradation.

2. ***Deficiency of essential growth factors:*** Essential growth factors are lacking, in part because of excessive degradation by this hostile wound environment. As a result, cell recruitment, matrix formation, & angiogenesis become impaired.

3. ***Bacterial overgrowth & colonization.*** Bacteria often congregate in colonies under protective structures called biofilms, which protect them from host defences. Moreover, the bacteria in their sequestered spaces communicate with small molecules in a process called quorum sensing, & they activate virulence factors. The bacterial cell wall & lipopolysaccharide are capable of activating inflammatory pathways through a toll-like receptor mechanism, which activates NF κ B.

4. ***Senescence of fibroblasts:*** abnormal aging of fibroblasts makes them less responsive to the stimulatory signals of the healing wound.

Providing good wound care addresses these individual defects, treats the underlying cause ( pressure, edema, vasculitis), & reverses the abnormalities, allowing the wound to progress to proliferation with granulation, contraction, re-epithelialization, & scarring—that is, to healing.

**Causes of impaired wound healing in diabetics:**

1. Peripheral neuropathy:
   a) loss of protective sensation
   b) autonomic dysfunction
   c) impaired neuroinflammatory reflex.
2. Tissue hypoxia : Macrovascular disease & microvascular disease:
   a ) capillary loss
   b) microvascular endothelial dysfunction.
3. Abnormal cellular pathways :
   a) chemotaxis
   b) fibroblast responsiveness.
4. Excess inflammation.
5. Deficient precursor cells.

**Neuropathy[14]:**

Clearly, the most important factor contributing to the development of foot ulcers & faulty healing is peripheral neuropathy, especially the loss of protective sensation. Neuropathy permits the recurrent injury sustained in daily walking to build into a crescendo of inflammatory activity that leads to tissue strain & injury, all without detection by the host. The ulceration's initial lesion is a "hot spot" of inflammation over an area of high pressures, thus creating the unfortunate combination of high foot pressures & inability to feel them. This results in callus formation, more pressure & injury, & ultimately, ulceration.

Neuropathy is present in over 80 percent of patients with foot ulcers.

*Peripheral sensory neuropathy*

Interferes with normal protective mechanisms and allows the patient to sustain major or repeated minor trauma to the foot, often without knowledge of the injury.

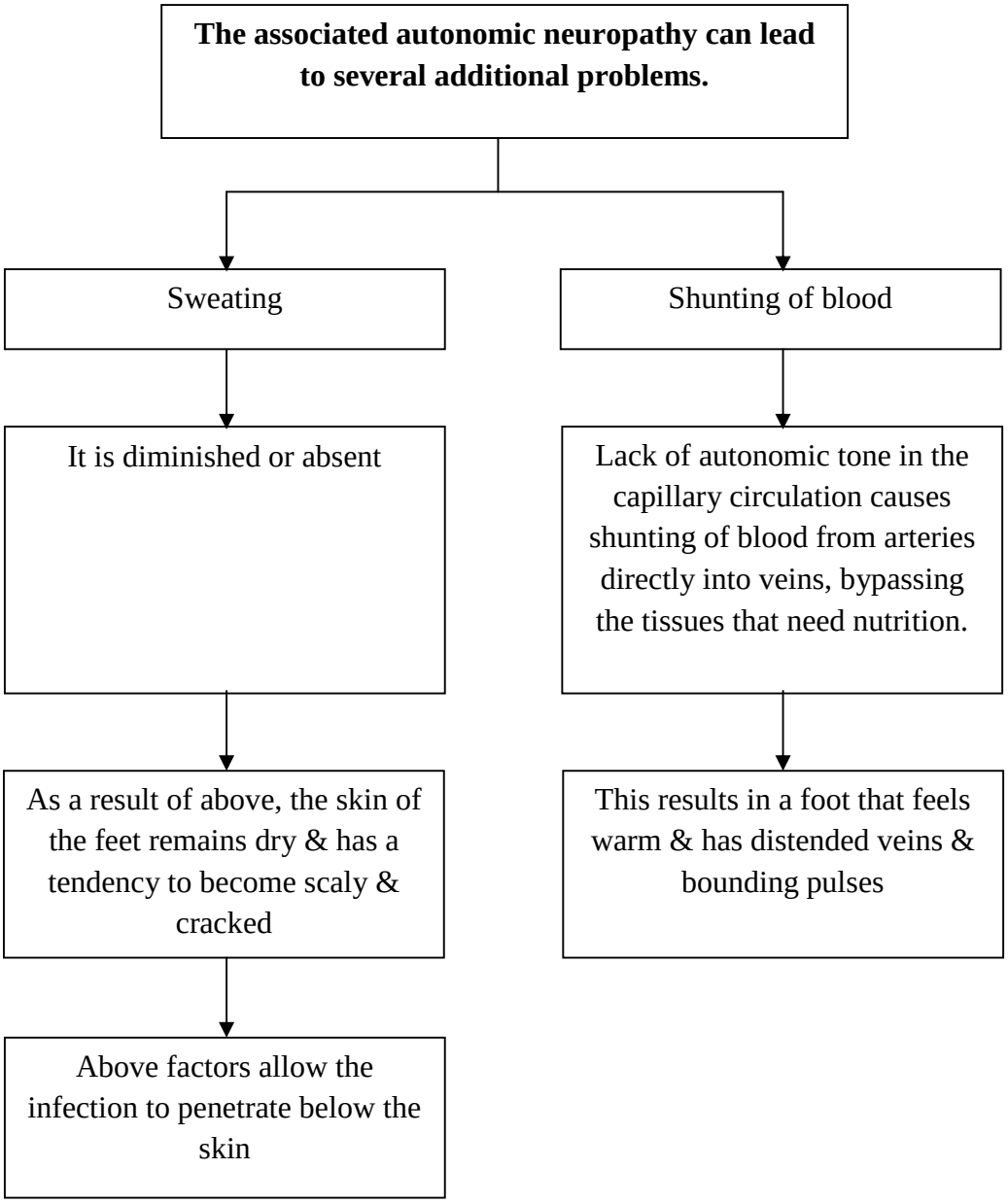
*Motor neuropathy*

Lead to abnormal foot muscle mechanics and to structural changes in the foot [hammer toe, claw toe deformity, prominent metatarsal heads, Charoct arthropathy].

*Autonomic neuropathy*

Results in anhidrosis and altered superficial blood flow in the foot, which promotes drying of the skin and fissure formation.

Figure: 1 Pathophysiology of Autonomic Neuropathy in Diabetes Mellitus

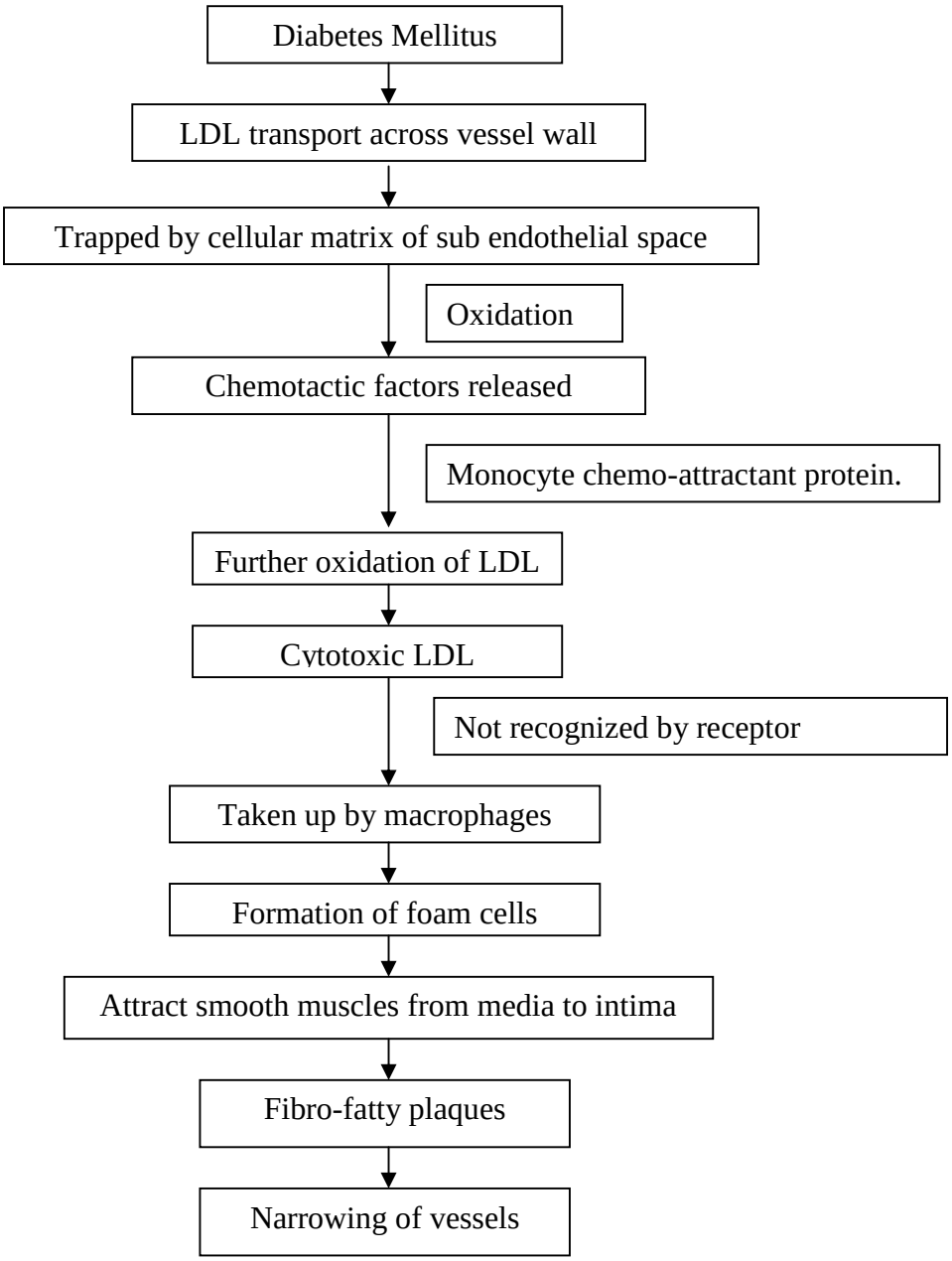


**Macrovascular disease[15]:**

Faulty healing seen in diabetes can be attributed to wound hypoxia from both microvascular & macrovacular diseases. In the extremities, people with diabetes are 4 to 5 times more likely to develop peripheral vascular disease than are people without diabetes. PAD risk increases even before the onset of hyperglycaemia in type 2 diabetes, implicating the prediabetic state in its pathogenesis. This state is characterized by insulin resistence,oxidative stress, & altered free fatty acid metabolism. This milieu leads to endothelial dysfunction, with impaired nitric oxide signalling & vasoreactivity, which through complex mechanisms, result in vasoconstriction, inflammation & hyper coagulabilty. These factors may account for the high risk of vascular disease seen in diabetes.

Diabetes is unique as a risk factor not only for its power but also for its predilection to involve the small arteries below the knee, the tibial vessels. Here the diseases is typically diffuse & distal, but it spares the arteries of the foot. In addition, PAD is strongly associated with neuropathy, which allows the vascular disease to worsen slowly without sensory feedback. Thus a patient with diabetes may have severe PAD & ischaemia with few or no symptoms & may present late in the course of the disease with an ischaemic ulceration.

Figure : 2 Pathophysiology of Diabetic Vasculopathy




**Microvascular disease:**

Patients with diabetes & neuropathy also have significant microvascular defects. Hyperglycaemia is associated with ubiquitous involvement of the

microvasculature that is manifested by capillary sclerosis & drop out, particularly in people with type 1 DM. This can be seen with capillaroscopy & measurement of elevated capillary pressures. In type 2 patients with neuropathy, functional microvascular abnormalities can be seen, specifically endothelial dysfunction & impaired vasoreactivity. These abnormalities are best demonstrated by laser Doppler imaging of the microcirculation with defective vasodilatory response to heat, acetyl choline, & sodium nitroprusside. In addition neuropathy affects the neuroinflammatory microvascular vasodilatation in response to injury or noxious stimuli

**Cellular / inflammatory pathways:**

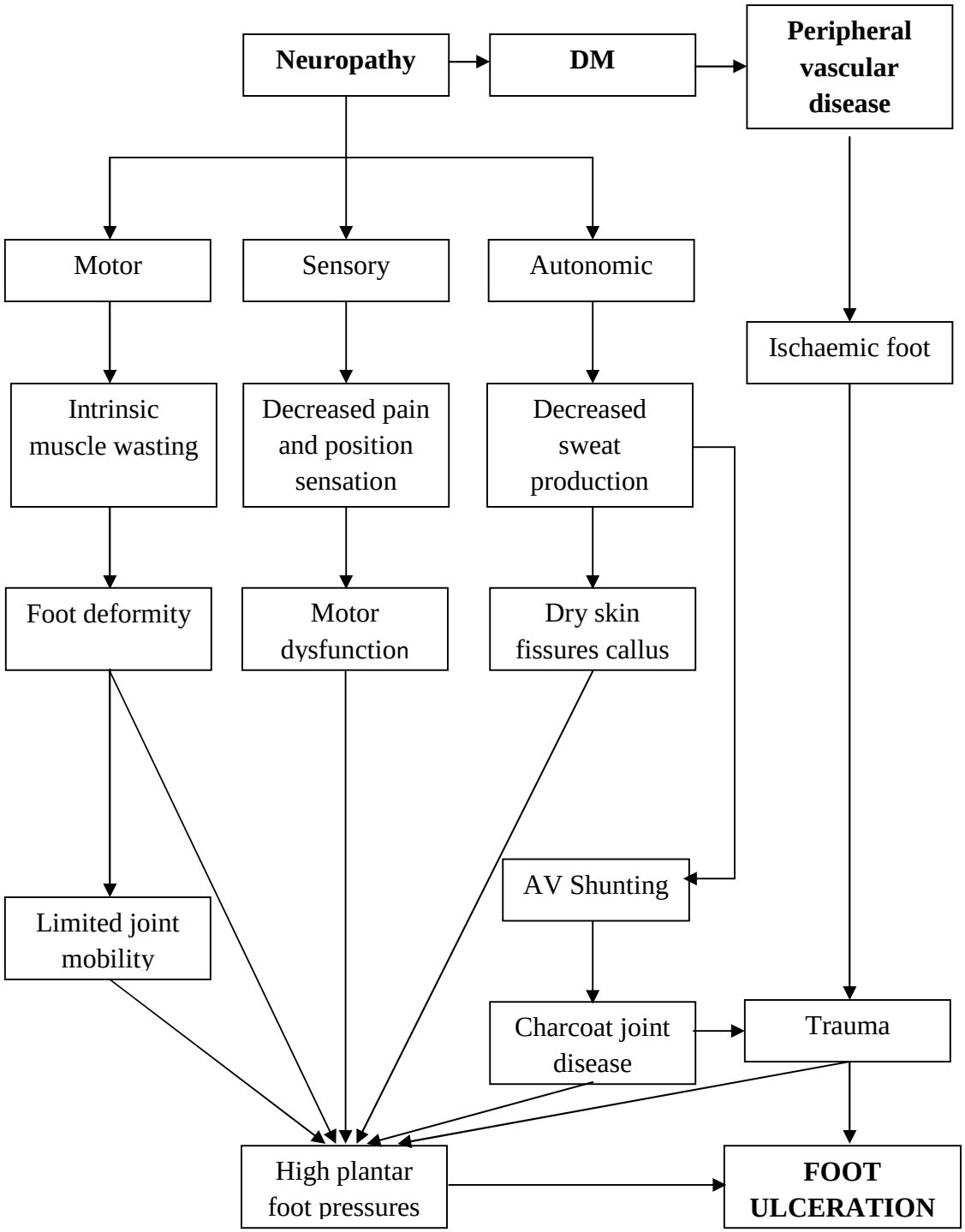
Diabetes is known to affect the cellular & inflammatory pathways that are involved in wound healing. Hyperglycaemia , primarily through a hyperosmotic effect, may slow neutrophil chemotaxis.Fibroblast taken from subjects with diabetes show altered function & response to stimulatory challenges.

Diabetes is a state of chronic vascular inflammation. A vast body of literature suggests that altered glucose & FFA metabolism results in oxidative stress, endothelial dysfunction & activation of inflammatory cytokines, in particular those regulated by NF κ B.

Systemic interventions in addition to good foot ulcer care, may affect inflammation. Controlling hyperglycaemia, oxidative stress, endothelial dysfunction, AGE( advanced glycosylation end products) formation, or other metabolic consequences of diabetes may help reduce inflammatory activity generally &

specifically, its contribution to the excess inflammation seen in diabetic foot ulcers.

Figure : 3 Clinical pathways leading to foot ulceration[16-17]



**Complications of Diabetes.**

Diabetes has both acute and long term complications. They are:

***Acute***

- Diabetic ketoacidosis
- Hyperglycemic Hyperosmolar state
- Hypoglycemia

***Long term:***

- Retinopathy
- Neuropathy
- Nephropathy
- Ischemic heart disease
- Cerebrovascular disease
- Peripheral vascular disease
- Hypertension.

Others

- ***Infections***
  - UTI
  - Tuberculosis
  - Candidiasis – oral / volvovaginal
  - Mucor mycosis

- Necrotising fasciitis
- Periodontitis

o Duputrens contracture

Pseudogout

## Wagner's classification:

Wagner ulcer classification system was developed for the "dysvascular" foot. It was skewed toward severe disease and contains all infections within a single grade.

**Grade 0 -** Preulcer. No open lesions, skin intact ,deformities, erythematous areas of pressure or hyperkeratosis.

**Grade 1** – Superficial ulcer. Disruption of skin without penetration of the subcutaneous fat layer.

**Grade 2** – Full – thickness ulcer. Penetrates through fat to tendon ,joint capsule wihtout deep abscess or osteomyelitis

**Grade 3 –** Deep ulcer not bone deep, with abscess, osteomyelitis, or joint sepsis and tendon sheath infections.

**Grade 4---** Denotes gangrene of a geographical portion of the foot such as toe forefoot or heel.

**Grade 5**--- Gangrene or necrosis foot is beyond salvage require a major limb or life saving amputation.

## Peripheral vascular diasease in Diabetes mellitus[18-22].

Introduction .

Lower extremity arterial disease (LEAD) is clinically identified by intermittent claudication and/or absence of peripheral pulses in the lower legs and feet. These clinical manifestations reflect decreased arterial perfusion of the extremity. With the use of Doppler technology and blood pressure measurements of the extremity, LEAD can be identified noninvasively before clinical manifestation.X-ray of the extremities can detect arterial calcification that is indicative of arterial disease with or without an occlusive component. Ultrasound with duplex scanning can also detect occlusive LEAD noninvasively, while angiography remains the gold standard for identification and diagnosis of LEAD.

The incidence and prevalence of LEAD increase with age in both diabetic and nondiabetic subjects and, in those with diabetes, increase with duration of diabetes.Many elderly diabetic persons have LEAD at the time of diabetes diagnosis. Diabetes is an important risk factor for LEAD. Hypertension, smoking, and hyperlipidemia, which are frequently present in patients with diabetes, contribute additional risk for vascular disease. LEAD in diabetes is compounded by the presence of peripheral neuropathy and by susceptibility to infection. These confounding factors in diabetic patients contribute to progression of LEAD to foot

ulcerations, gangrene, and ultimately to amputation of part of the affected extremity.

Assessment of LEAD.

Palpation of peripheral pulses has been used as a clinical tool to assess occlusive LEAD in diabetic and non diabetic patients, particularly when intermittent claudication is present. However, it is sometimes difficult to interpret the significance of diminished peripheral pulses when symptoms are not present. Ambient temperature, anatomic variation, and expertise in palpating peripheral pulses may contribute to variation in the clinical examination. Absence of pulses remains a significant clinical finding. Absent posterior tibial, popliteal, or femoral pulses with or without bruits that persist on repeated examination are clinically significant and indicate significant occlusive LEAD whether intermittent claudication is present or not. However, clinical findings such as diminution or absence of peripheral pulses and presence of bruits are more meaningful of occlusive disease in the context of clinical symptoms such as intermittent claudication.Because of anatomic variation, absence of the dorsalis pedis pulse alone may not indicate LEAD.

Measurement of the ankle-brachial index (ABI),which represents the systolic blood pressure at the posterior tibial or dorsalis pedal level compared with brachial blood pressure, can be used to define clinically significant occlusive LEAD. An index of <0.9 is suggestive of occlusive LEAD, particularly if symptoms or clinical

findings such as bruits or absent pulses are present. ABI levels <0.8 indicate LEAD regardless of symptoms. The lower the ABI, the more significant the occlusion whether symptoms are present or not. It is highly unlikely that symptoms would not be present in patients whose ABI is <0.5. The ABI may be more sensitive with exercise, and a 5-minute exercise period with measurement of the ABI post-exercise may indicate significant occlusive LEAD before the resting ABI becomes abnormal . The post-exercise ABI helps differentiate the etiology of exercise-induced leg pain.

X-ray of the extremities will identify calcified arteries that may be associated with high ABI levels, indicating non compressible arteries. It is more difficult to identify occlusive LEAD in these patients because of the high ABI levels and the continued presence of peripheral pulses. Velocimetry with continuous-wave Doppler technique may be able to identify occlusive LEAD in the presence of non compressible vessels.

Also, the toe systolic blood pressure index (TSPI) may be helpful in identifying occlusive LEAD in this circumstance. Otherwise, the first symptoms of occlusive LEAD in these patients may be related to gangrene or ulceration.Measurement of toe pressures has received a great deal of attention because of their predictability in defining individuals at high risk of gangrene, ulceration, and infection associated with occlusive arterial disease, even in patients with non compressible vessels. Reduced toe pressures are highly associated with progression of LEAD to gangrene, ulceration, and the need for amputation.

Screening and diagnosis of LEAD are best accomplished with measurement of ABI and TSPI.

Doppler ultrasound alluded to above provides an excellent measure of occlusive LEAD, but the ABI and TSPI are the definitive quantitative diagnostic indices for occlusive disease in compressible arteries.

Transcutaneous oxygen measurement(Po2) may help assess the healing of ischemic skin lesions. The measurement is not useful for screening or diagnosis of LEAD.Angiography remains the gold standard for identifying occlusive LEAD and the areas of occlusion in the arterial system. Patients being considered for amputation because of occlusive LEAD should have angiography performed to determine whether revascularization may be effective in salvaging the limb or in lowering the level of amputation.

Morbidity.

The morbidity of LEAD includes intermittent claudication,foot ulcers, gangrene, and amputation.Foot ulcers and gangrene are frequent comorbid conditions with LEAD. Concurrent peripheral neuropathy with impaired sensation make the foot susceptible to trauma, ulceration, and infection.The progression of LEAD in diabetes is compounded by such comorbidity as peripheral neuropathy and insensitivity of the feet and lower extremities to pain and trauma. With impaired circulation and impaired sensation, ulceration and infection occur. Progression to osteomyelitis and gangrene may necessitate amputation.Revascularization

procedures have assisted with improving perfusion and flow to the lower extremities but have apparently not decreased the frequency of amputation.

**Summary of vascular problems in diabetics[24].**

Peripheral vascular disease is usually only one manifestation of severe vascular disease throughout the body in diabetics. Peripheral vascular disease in diabetics is usually bilateral, multisegmental & more distal vessels are involved. In the non diabetics the iliac & femoral vessels are commonly involved with stenosis & blockages but in diabetics it is the trifurcation of the tibial vessels at popliteal level which is most commonly involved. Intermittent claudication is not a feature in PVD in diabetics due to neuropathy. For many diabetics the first awareness about ischaemia comes with development of an **ulcer** or **gangrene.**

**Ischaemic foot do not heal`[24].**

Not all ischaemic feet have ulcers but once the ischaemic foot develops ulcerations, these ulcers do not heal. The reason being that the level of blood flow necessary to maintain an intact skin is much less than that required to heal an injured foot. Many ischaemic feet do well as long as the skin is intact. But when an injury occurs the foot is no longer in a position to mount an inflammatory response & fight infection, thus the ulcers do not heal.

**Diabetic foot ulcers are deceptive[24].**

Unlike an ischaemic foot in a non diabetic patient, the diabetic feet are deceptive. A nondiabetic ischaemic foot shows classical signs of ischaemia. This is not always true in a diabetic foot.

**Cold feet:** An ischaemic foot should be cold but diabetic ischaemic feet can be worm due to the 1) associated autonomic neuropathy causing lack of autonomic tone in the capillary circulation which causes shunting of blood from arteries directly into veins, bypassing the tissues that need nutrition.This results in foot that feels warm with bounding pulses & dilated veins & 2) secondary infection.

1) **Claudication pain:** The claudication pain which occurs in the ischaemic foot is an agonizing symptom to the patient & also a warning sign of ischaemia to the doctor. This claudication pain makes the patient aware that something is wrong in his lower limb & thus he takes great care to avoid injuring his extremely pain sensitive feet. But claudication is not always present in a diabetic ischaemic limb due to the associated neuropathy. These patients keep on walking & thus develop ulcerations much earlier than non diabetic patients with lower limb ischaemia. Non diabetic patients with lower limb ischaemia develop ulcerations at ABI <0.3, where as diabetic patients develop ulcerations at a much higher ABI.
2) **Pale , bluish skin:** an ischaemic foot should have a pale or blue skin but in diabetics with critical ischaemia the foots become pink due to dilatation of capillaries in an attempt to increase blood supply. Thus due to this pink

colour the diabetic foot appears to be well vascularised even in the setting of severe ischaemia.

**Decision making in diabetic foot ulcers:**

Diabetic foot ulcers have a deceptive appearance as discussed above. In such a setting it is often difficult for the treating doctor to take decision of a limb salvage procedure (debridement, toe disarticulations, fore foot amputations) or a radical operation (above knee/ below knee amputation).Often such a patient is subjected to one or more debridements/ disarticulations & daily dressings & finally once the ulcer doesn't show any signs of healing or frank gangrene appears, then the decision for amputation is taken.This results in prolonged hospital stay& its accompanied complications.

Prolonged hospital stay is an economic burden to the family. Shobhana et all[16] in 2001 did a study on the costs spent by each patient with diabetic foot ulcers per year. She concluded that a single patient with diabetic foot ulcer spent Rs16,910 per year on his treatment.The patient will also not earn his livelihood as long as he is admitted. Other morbidities with prolonged hospital stayinclude loss of weight,malnutrition, psychological problems, anaemia, hypoproteinemia, bed sores & hospital acquired infections.

Diabetic foot ulcers are also a burden on the government hospitals as most of the resources are consumed during their management.

These issues are a result of prolonged hospital stay. Ischaemic diabetic foot ulcers are often deceptive & these patients tend to have prolonged hospital stay due to the dilemma they cause in decision making. A solution to address these issues will be early & proper decision making. This requires reliable investigations that will determine the vascular status of the patient. The vascular status is studied with various radiological investigations like Doppler, duplex sonography& angiography. Colour doppler has been used widely & is practically the first investigation desired in any patient with peripheral vascular disease. But colour Doppler has drawbacks: it's not widely available , is observer dependent & status of collaterals cannot be commented . Angiography is the best investigation to asses the vascularity of the limbs. But angiography cannot be performed in acute or chronic renal failure patients. Ankle – Brachial index is a simple bed side test which gives sufficient information about vascularity of the involved limb. The objective of this study was to know whether ABI can act as a prognostic indicator in diabetic foot ulcers ie can ABI predict accurately limb salvage or amputation in an ischaemic diabetic foot ulcer& thus aid in early decision making.

## ANKLE – BRACHIAL INDEX.

**D**efinition: The ratio of ankle systolic pressure to brachial systolic pressure is called as ankle brachial index (ABI).

### Blood pressure measurement by Doppler[24].

Measurement of the blood pressure in various arteries is very useful in the evaluation of peripheral arterial disease. A blood pressure cuff is wrapped around the affected limb, and a Doppler ultrasound probe is placed on the skin over the artery. A doctor or examiner inflates the pressure cuff to a pressure high enough that the colour signals of the artery disappear, and then slowly deflates the cuff. The pressure measured by the cuff when the colour signals reappear again is the systolic pressure under the cuff. These measurements can be taken at the ankle, toe, points on the leg (upper thigh, above the knee, upper calf), and points on the arm (elbow, forearm, wrist).

### Ankle pressure

In most people, the resting ankle pressure is greater than the pressure at the crook of the arm, known as the brachial blood pressure. The ratio of the ankle systolic pressure to the brachial systolic pressure is called the ankle – brachial index (ABI). A normal ABI is 0.9 to 1.3. A person with an ABI less than 0.9 may have peripheral arterial disease.

**Toe pressure**

Toe pressures can be measured with miniature blood pressure cuffs. In general, a toe pressure measuring less than 30 millimeters of mercury (mm Hg) means that the person has severe arterial insufficiency at the level of the toes.

**Segmental leg pressures**

Arterial pressure can be estimated in the upper thigh, above the knee, and in the upper calf by placing blood pressure cuffs at the appropriate levels. The pressures can be compared between the two legs or at different levels in the same leg.

**Arm pressures**

Blood pressures can be measured at the elbow (brachial), forearm, or wrist. Large differences between pressures at the various levels suggest arterial blockage. As with toes , finger pressures can be measured. And a finger - brachial index can be calculated. Finger - brachial ratios of less than 0.8 suggest that a person has arterial disease of the upper extremity.

**Introduction to ankle brachial index [26].**

The ankle systolic pressure varies with central aortic pressure. The brachial pressure reflects the central aortic pressure.In the arterial circulation, peak systolic pressure is amplified as the pulse wave progresses down the lower limb. This amplification is a result of reflected waves originating from the relatively high

peripheral resistance & differences in compliance between the central & peripheral arteries.Thus the systolic pressure measured at the ankle is normally higher than that in upper arm. However, the diastolic & mean pressures gradually decrease as the pulse wave moves distally. Diastolic pressure in the lower limb is reduced only in the presence of severe proximal stenosis, Whereas the peak systolic pressure decreases with lesser degrees of disease. Thus determination of systolic blood pressure is the most reliable pressure parameter for diagnosis of arterial narrowing.Because the ankle systolic pressure varies with central aortic pressure it is desirable to compare ankle systolic pressure to central aortic pressure. Brachial systolic pressure is essentially equal to central aortic pressure.

**Technique of measurement of ABI:** The systolic pressure at any level of the lower extremity can be measured by positioning a pneumatic cuff at the desired site.The cuff is tied around the ankle above the malleoli. Colour doppler probe is placed on the anterior tibial ( or posterior tibial or dorsalis pedis) for flow detection. The cuff pressure is gradually inflated until the flow signals disappear.

Now the cuff pressure is deflated & the pressure at which the flow signals appear is taken as the ankle systolic pressure.

The brachial systolic pressure is also measured in a similar manner. The cuff is tied to upper arm. The cuff is inflated until the flow signals disappear. Then slowly the pressure is released & the pressure at which the flow signals appear is taken as the brachial systolic pressure.

The ratio of ankle systolic pressure to brachial systolic pressure is called **ankle – brachial index.** Also called ankle pressure index or ankle arm index.The use of this test compensates the variation in central perfusion pressure.

**Normal value:** 1.11 +/- 0.10.

Because of variability related to pressure measurement technique, value > 0.9 are considered normal.

In limbs with intermittent claudication ABI ranges from 0.2- 1.0 with mean of 0.59 +/- 0.15. ABI in limbs with ischaemic rest pain 0 – 0.65. ABI in limbs with impending gangrene is 0.05 +/- 0.08.Many limbs with impending gangrene or ischaemic ulceration ABI is 0 due to absent flow signals.

However , ABPI has known issues:

- ABPI is known to be unreliable on patients with arterial calcification (hardening of the arteries) which results in less or incompressible arteries, as the stiff arteries produce falsely elevated ankle pressure, giving false negatives). This is often found in patients with diabetes mellitus (41% of PAD patients have diabetes[]), renal failure or heavy smokers. ABPI values < 0.9 & >1.3 should be investigated further.
- Performing ABPI is time consuming.

- Resting ABPI is insensitive to mild PAD. Treadmill tests (6 minute) are sometimes used to increase ABPI sensitivity, but this is unsuitable for patients who are obese or have co-morbidities such as Aortic aneurysm, and increases assessment duration.
- Lack of protocol standardisation, which reduces intra-observer reliability.
- Skilled Operators are required for consistent, accurate results.

In a normal subject the pressure at the ankle is slightly higher than at the elbow (there is reflection of the pulse pressure from the vascular bed of the feet, whereas at the elbow the artery continues on some distance to the wrist). The ABPI is the ratio of the highest ankle to brachial artery systolic pressure and an ABPI of greater than 0.9 is considered normal (Free from significant PAD).

However, an ABPI value greater than 1.3 is considered abnormal, and suggests calcification of the walls of the arteries and incompressible vessels, reflecting severe peripheral vascular disease.

Some studies have been performed in the past where ABI has been studied in peripheral vascular disease. Permalatha et all[27] in 2002 studied ABI as a diagnostic tool for peripheral vascular disease & compared it with colour Doppler as the gold standard investigation in diagnosing peripheral vascular disease in type 2 DM. She concluded that ABI has sensitivity of 70.6% & a high specificity of 88.5%. XiaomingGuo et all[28] in 2007 studied the sensitivity & specificity of ABI for assessing the lower extremity arteries & concluded that ABI is an accurate &

reliable non-invasive alternative to conventional DSA in the assessment of lower extremity arteries.

Flanigan D et all[29] in 2008 concluded that colour doppler of superficial femoral artery was a better screening tool for identifying lower extremity atherosclerosis as compared to ABI.

All these studies have evaluated ankle brachial index as a diagnostic tool or a screening test but there is no literature available on ABI as a prognostic indicator in the outcome of diabetic foot ulcers.This lacunae encouraged us to take up this study.

# MATERIALS & METHODS

The present study was conducted on patients with diabetic foot ulcers admitted to KLE'S Dr. Prabhakar Kore Hospital & Medical Research Centre under surgery Department , Belgaum during the period of Nov2008 to Nov 2009.

**Study design.**

One year cross sectional study.

**Study period.**

The present study was conducted during Nov 2008 to Nov 2009.

**Method of collection of data.**

**Source of Data.**

Patients with diabetic foot ulcers admitted at KLE'S,Dr.Prabhakar Kore Hospital & Medical Research Centre, Belgaum.

**Criteria for admission.**

1) Patients with ulcers over foot requiring surgical intervention.
2) Patients should be diabetic.
3) Patients requiring injectable antibiotics.
4) Patients with RBS >400.

Criteria 1,2& 3 are compulsory whereas criteria 4 is optional.

**Sample size.**

As many patients admitted to our hospital in the specified time with a minimum of 70 patients.

**Sampling procedure.**

The sample size was calculated based on patient data for the last 3 consecutive years that is 2005, 2006, 2007 of KLE'S Dr. Prabhakar Kore Hospital & Medical Research Centre, Belgaum.

**Selection criteria.**

***Inclusion criteria.***

- Type 2 diabetes with atleast 5 years duration.
- Diabetics between 30-85 years
- Wagner classification system class1, 2, 3 & 4 (only toe gangrene involved).
- Ulcers </= 6cms in the largest diameter.

***Exclusion criteria.***

- Immunocompromised patients.
- Wagner's class 5.
- Cardiac conductivity disorders.
- Hypertension.
- Serum creatinine> 2.
- Diabetic keto-acidosis.
- Skin malignancy.

**Procedure**

The study was conducted in department of Surgery at KLES Dr. Prabhakar Kore Hospital& Medical Research Centre, Belgaum during one year

duration. The study was approved by the ethical & Research Committee of Jawaharlal Nehru Medical College, Belgaum.

After finding the suitability as per the inclusion & exclusion criteria patients were selected for the study & briefed about the nature of the study, the interventions used & written informed consent was obtained ( Annexure I ).On the day of admission descriptive data of the participants like name, age, sex , detailed history, were obtained by interviewing the participants & clinical examination & necessary investigations were done 1) complete blood count 2) ECG 3) RBS 4) blood urea & serum creatinine 5) Urine ketone bodies 6) HbA1c 7)HIV,Hbsag.

Wagner's class was determined after clinical examination.

ABI of the affected limb was measured with colour doppler. The observations were recorded in a predesigned & pretested proforma .

All the patients received regular dressing with betadine & normal saline. They were skin grafted, debrided / disarticulated or amputated as per the decision of the treating doctor.

All the patients had dressing done only with betadine & hydrogen peroxide or normal saline. No topical ointments were applied.The patients were followed up monthly.At every follow up the ulcer was examined.

End point of follow up was :

1) If patient undergoes amputation of toes or forefoot amputation& the operated site heals.
2) If patient undergoes above knee or below knee amputation.

3) If patient undergoes split thickness skin grafting.

4) 6 months from the date of admission if 1,2 or 3 has not occurred.

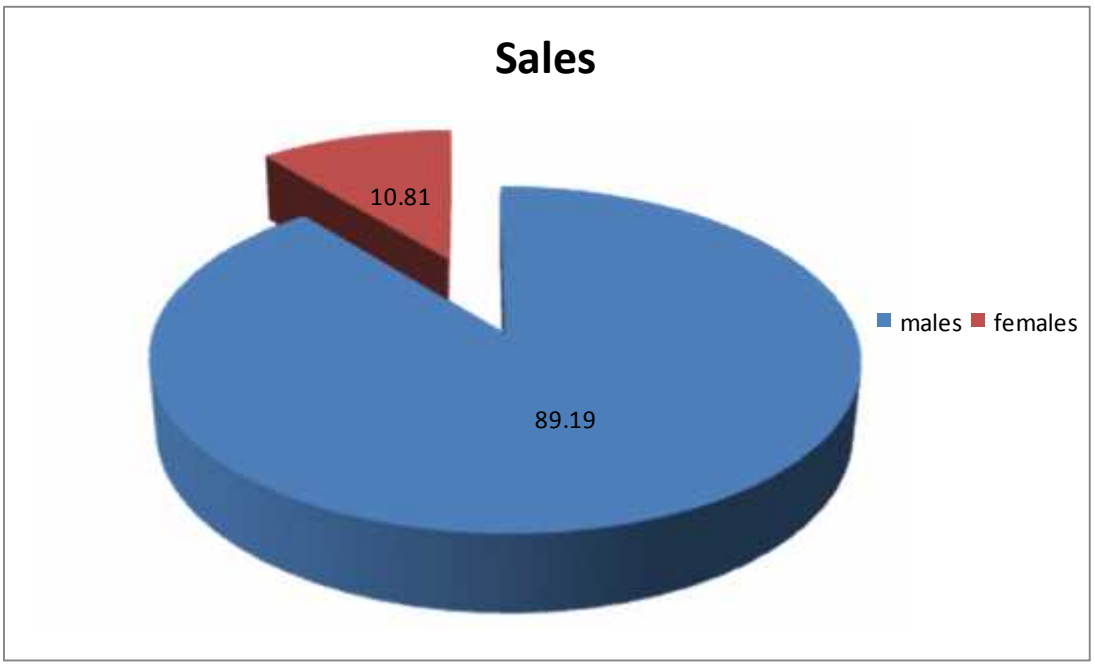
The outcome of the foot was correlated with the ABI that was measured at the time of admission & analysis was done. Data obtained was tabulated using SPSS VERSION 14 software. Means, standard deviations and percentages were used to describe the samples. Chi square test was used to identify the differences between the groups. For multiple comparisons Tukey's test was used. For comparison of Ankle – Brachial Index Student's unpaired t test was used. P value < 0.05 was considered significant.

# RESULTS

Table 1: Distribution of patients according to gender.

| Sex | Number | percentage |
|---|---|---|
| 1. Male | 66 | 89.19% |
| 2. Female | 8 | 10.81% |
| Total | 74 | 100% |

Graph 1: Distribution of patients according to gender.

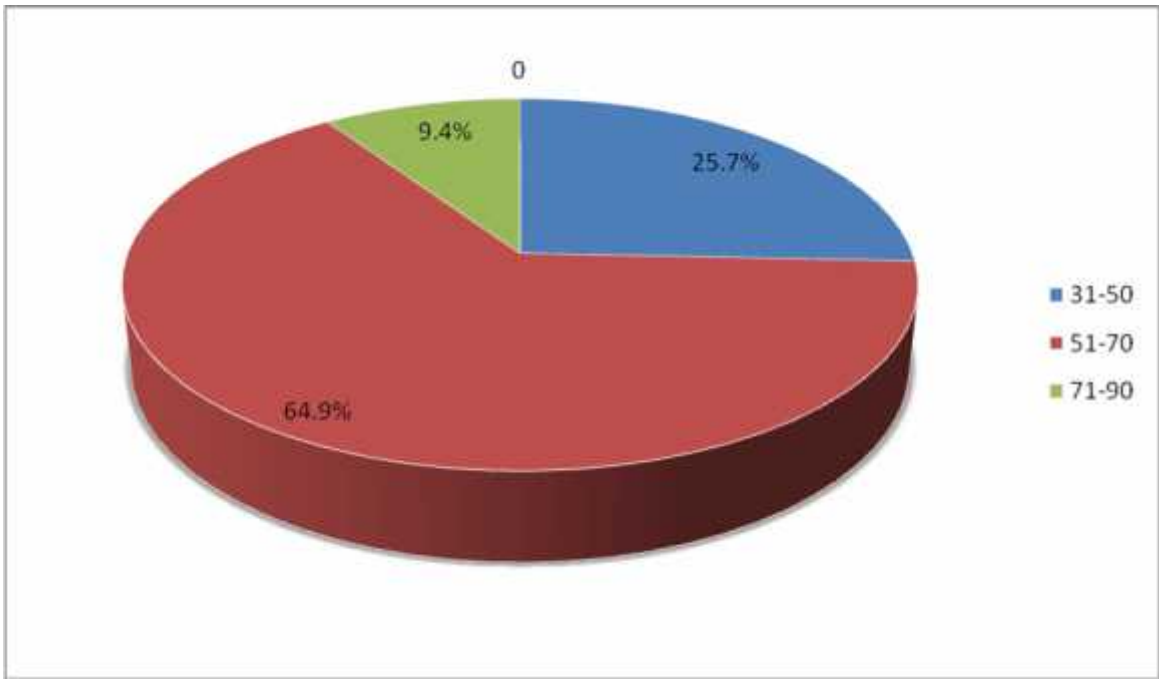



In this study 66 (89.19%) patients were males & 8 (10.81%) were females.

Table 2: Distribution of patients according to age.

| | Age | Number | Percentage |
|---|---|---|---|
| 1. | 31 – 50 | 19 | 25.7% |
| 2. | 51 – 70 | 48 | 64.9% |
| 3. | 71 - 80 | 7 | 9.4% |
| | Total | 74 | 100% |

Graph 2: Distribution of patients according to age.

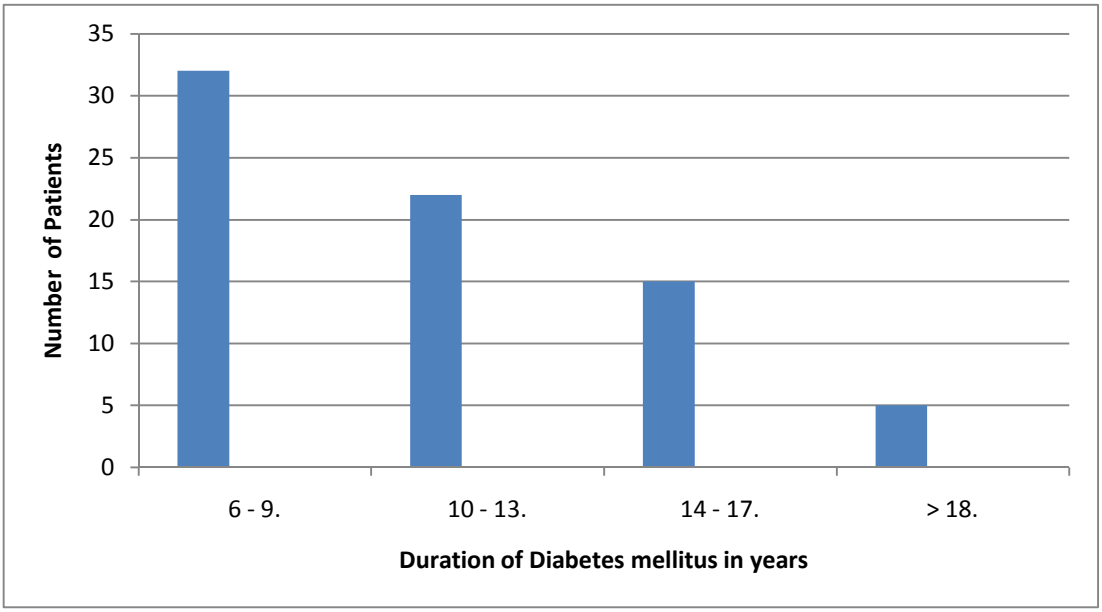



In this study majority of the patients were in the 51 to 70 years of age group. 48 ( 64.9%) patients were in the age group of 51 to 70 years, 19 ( 25.7%) were in the age group of 31 to 50 & 7 ( 9.4%) were in the age group of 71 to 80.

Table 3: Distribution of duration of diabetes.

| | Duration in years. | Number | Percentage |
|---|---|---|---|
| 1. | 6 – 9 | 32 | 43.2% |
| 2. | 10 – 13 | 22 | 29.7% |
| 3. | 14 – 17 | 15 | 20.3% |
| 4. | > 18 | 5 | 6.8% |
| Total | | 74 | 100% |

Graph 3: Distribution of duration of diabetes.




In the present study 43.2% of the patients had DM for a period of 6 to 10 years & 29.7% had DM for 10 to 13 years.

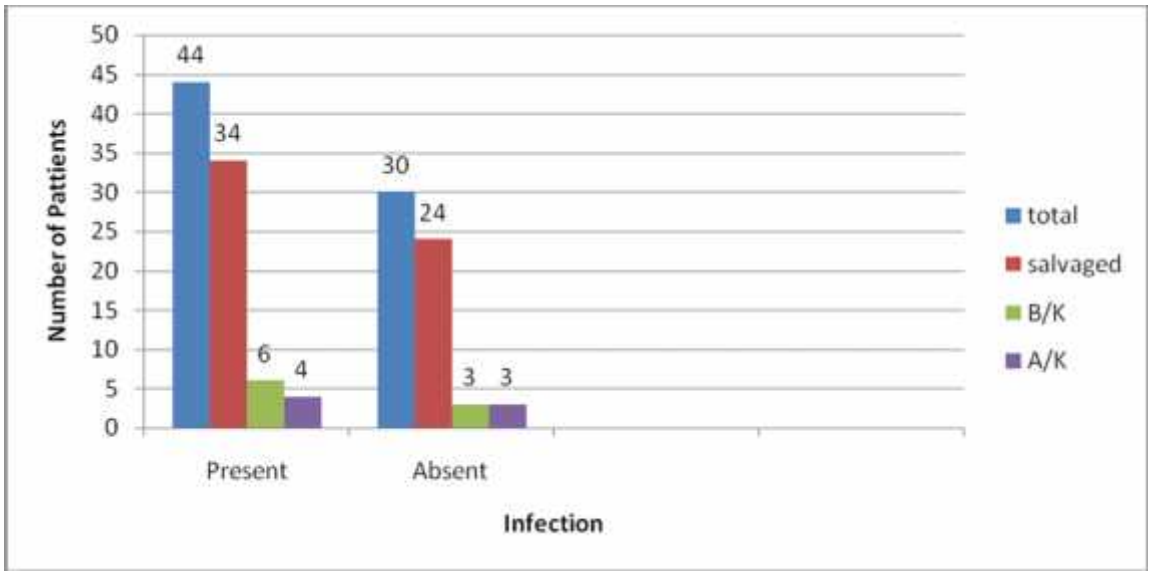
Table 4: Presence of infection & outcome of diabetic foot.

| Infection. | Above knee amputation. | Below knee amputation. | Limb salvaged. | Total |
|---|---|---|---|---|
| Present. | 4 | 6 | 34 | 44 |
| Absent. | 3 | 3 | 24 | 30 |
| | 7 | 9 | 58 | 74 |

$X^2$ = 0.2265. P value: 0.8929.

P value is not significant.

Graph 4: Presence of infection & outcome of diabetic foot.




In the present study 44 patients had infected diabetic foot ulcers & 30 patients had no infection.34 of 44 infected ulcers healed. 24 of 30 ulcers that were not infected healed. P = 0.8929, is not significant meaning the outcome of diabetic foot ulcers did not depend on the presence or absence of infection.

**Conclusion: In this study the outcome of diabetic foot ulcers was not affected with the presence or absence of infection.**

Table 5:Co-relation between palpation of pulsations & ankle – brachial index.

| Pulsations. | Number of patients. | Mean ankle – brachial index. |
|---|---|---|
| GROUP1.Absent dorsalis pedis. | 16 | 0.85. |
| GROUP 2.Absent anterior tibial& posterior tibial. | 10 | 0.78. |
| GROUP 3.Absent anterior tibial, posterior tibial& popliteal. | 5 | 0.68. |

Table 6:ONE WAY ANALYSIS OF VARIANCE

| | Sum of Squares | Df | Mean Square | F | p VALUE |
|---|---|---|---|---|---|
| Between Groups | 0.1148 | 2 | 0.0574 | 8.7436 | 0.0011 |
| Within Groups | 0.1837 | 28 | 0.0066 | | |
| Total | 0.2985 | 30 | | | |

P = 0.0011 indicates that the values of ABI in each group is not homogeneous.

Table 7: Mutilple comparisons by Tukey's test.

| GROUP | GROUP | Mean Difference | P value | Inference |
|---|---|---|---|---|
| 1 | 2 | 0.0614 | 0.1633 | NS |
| 1 | 3 | 0.1714 | 0.0008 | S |
| 2 | 3 | 0.1100 | 0.0495 | S |

P = < 0.001 indicates that difference between group 1 & 3 and group 2 & 3 is statistically significant.

The difference in ABI when only dorsalis pedis was absent on clinical palpation as compared to absent popliteal pulsations was statistically significant. Similarly the difference in ABI when anterior tibial & posterior tibial pulsations were absent as compared to absent popliteal pulsations was also significant.

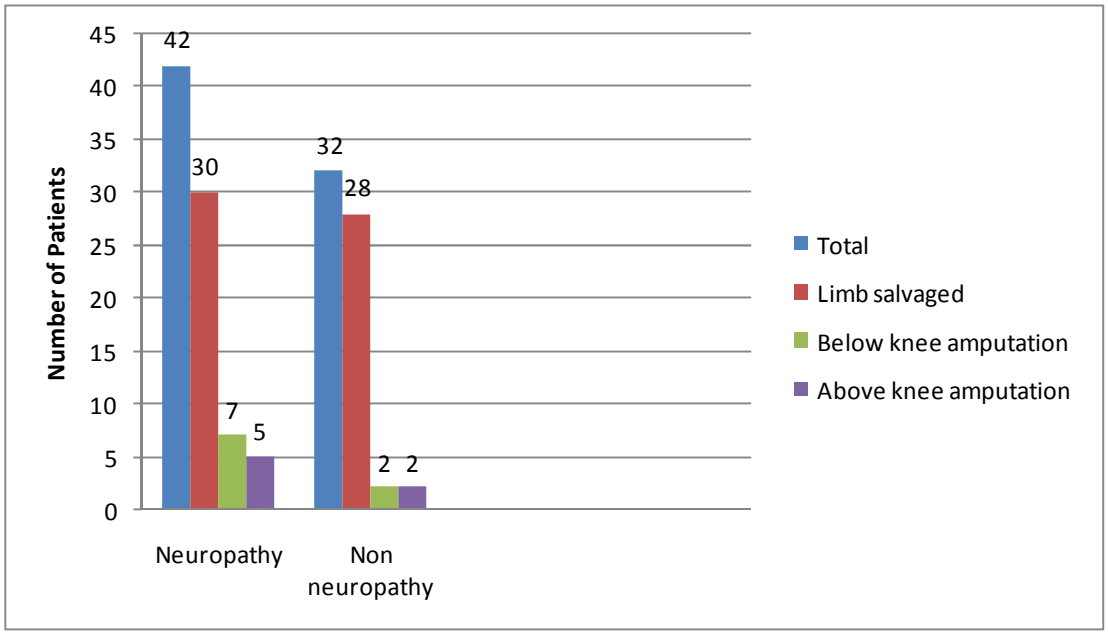
**Conclusion: The decrease in ABI correlates well with the absence of pulsations.**

Table 8: Relation between neuropathy & non neuropathy with outcome of diabetic foot.

| Neuropathy | Above/Knee amputation. | Below/Knee amputation. | Limb salvaged. | Total. |
|---|---|---|---|---|
| Present | 5 | 7 | 30 | 42 |
| Absent | 2 | 2 | 28 | 32 |

$X^2 = 0.2265$. P value= 0.8929. P value not significant.

Graph 5: Relation between neuropathy & non neuropathy with outcome of diabetic foot.



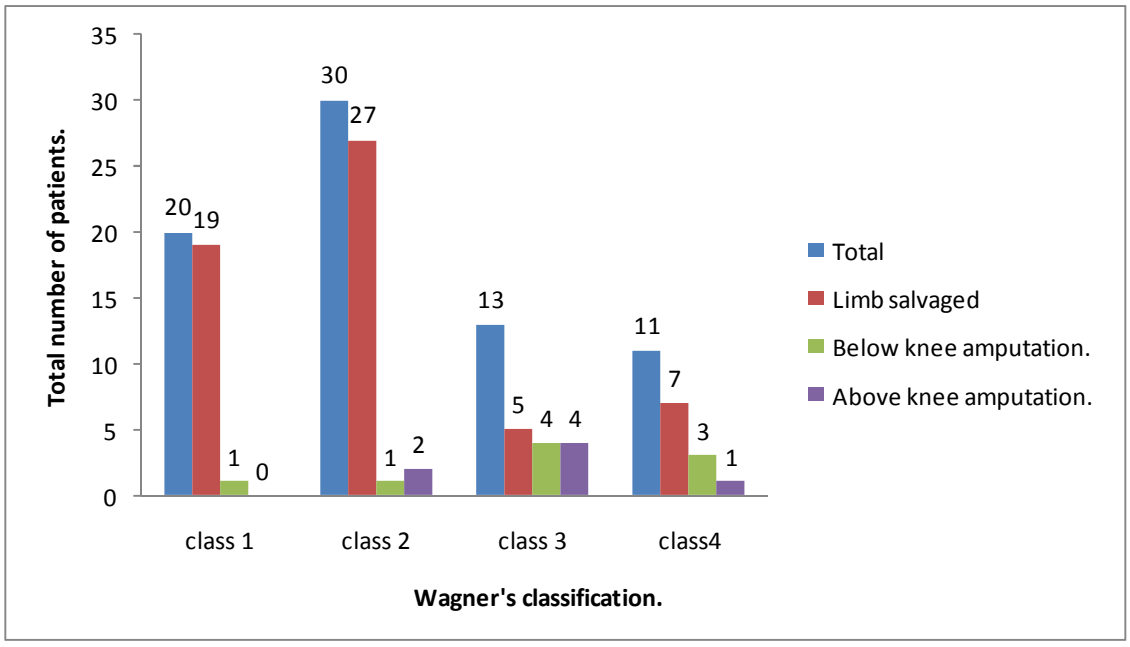

In this study 42 patients had peripheral neuropathy & 32 patients did not have neuropathy. P = 0.8929, was not significant when the outcome of these patient was analysed with the presence or absence of neuropathy.**Conclusion: In this study the outcome of diabetic foot ulcers was not affected with the presence or absence of neuropathy.**

Table 9: Relation between Wagner's class & outcome of diabetic foot.

| | A/k amputation. | B/k amputation. | Limb salvaged. | Total. |
|---|---|---|---|---|
| Class 1 | 0 | 1 | 19 | 20 |
| Class 2 | 2 | 1 | 27 | 30 |
| Class 3 | 4 | 4 | 5 | 13 |
| Class 4 | 1 | 3 | 7 | 11 |

$X^2$ = 21.08. P value = 0.0018. ( significant) . P value indicates that wagner's class is a good predictor of the outcome of diabetic foot.

Graph 6: Relation between wagner's class & outcome of diabetic foot ulcers.




In this study the maximum number of patients were in wagner's class 2 ie 30. When the outcome of diabetic foot ulcers in each wagner's class was studied P value = 0.0018 ( significant). **Conclusion: Wagner's class is a good predictor of outcome of diabetic foot ulcers.**

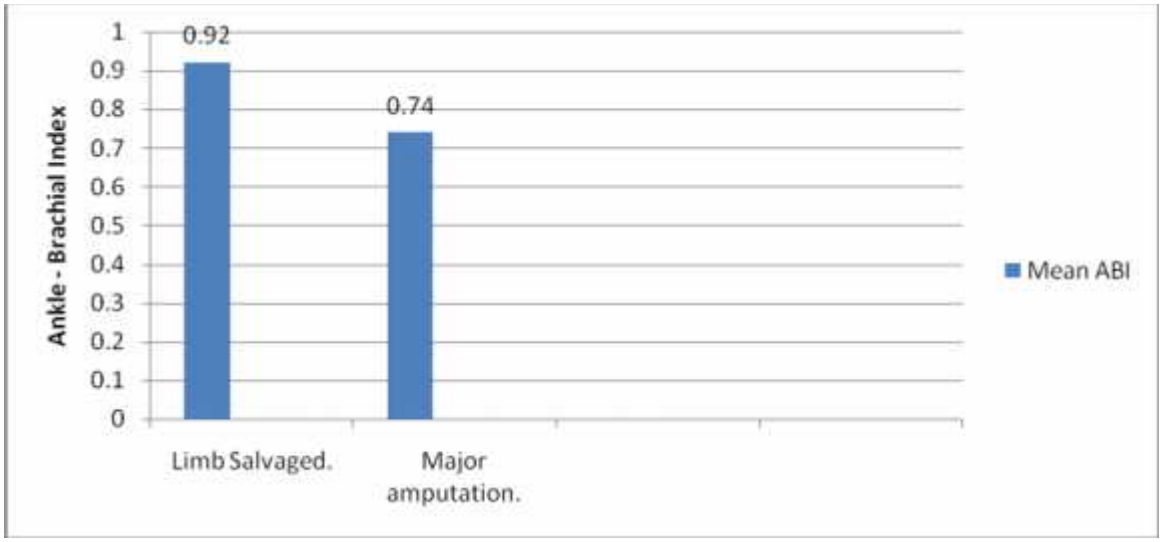
Table 10: Co- relation between Ankle – brachial index & outcome of diabetic foot.

| | Number. | Mean ABI. | +/-Standard deviation. | |
|---|---|---|---|---|
| Limb salvaged. | 58 | 0.92 | +/- 0.081 | |
| Major amputation.(above knee or below knee amputation) | 16 | 0.74 | +/- 0.10 | P < 0.0001 |
| | Total = 74. | | | |

Student's unpaired t test.

P < 0.0001 means that the differnce in ABI between limb salvagable & amputation is highly significant.

Graph 7: co-relation between Ankle - brachial index & outcome of diabetic foot.




In this study the mean ABI of patients in whom limb was salvaged was 0.92 +/- 0.08 & mean ABI of patients who underwent amputation was 0.78 +/- 0.1. P value < 0.0001 indicates that difference is statistically highly significant.

**Conclusion: ABI is a good predictor of the outcome of diabetic foot ulcers.**

# DISCUSSION

Foot ulcerations are the leading cause of hospital admission in diabetics. Peripheral neuropathy, peripheral vascular disease & susceptibilty to infection are the three most important cause for development of diabetic foot ulcers. Diabetes is the most common nontraumatic cause of amputation of lower limb. Major causes of amputation in diabetics include profound infection,gangrene, foot deformities & lower limb ischaemia. Vasularity is the most important prognostic factor for diabetic foot ulcers[30].

Selection of the proper amputation level is crucial not only to preserve the maximal length of the viable extremity, but to minimize morbidity and mortality. When overly distal amputation is selected, the blood supply may be inadequate for wound healing and additional surgery may be required. Repeated amputation, which may cause greater morbidity or mortality, should be avoided. Overly proximal amputation without prosthesis may lead to difficulty in ambulation[31].

Unlike an ischaemic foot in non diabetic patient, diabetic feet are deceptive.A non diabetic ischaemic foot shows classical signs of ischaemia, but this is not always true in diabetics. An ischaemic diabetic foot can be warm due to associated autonomic neuropathy causing lack of autonomic tone in the capillary circulation which causes shunting of blood from arteries directly into veins, bypassing the tissues that need nutrition.This results in foot that feels warm with bounding pulses & dilated veins . Claudication pain may be absent even in critical limb ischaemia in diabetics due to peripheral neuropathy.

Clinical decision making regarding conservative treatment or radical surgery in ischaemic lower limbs in diabetics is always very difficult because

diabetic foot ulcers are deceptive as mentioned above . Such a patient is subjected to multiple debridements& finally when the ulcer shows no signs of healing, the decision for amputation is taken.Prolonged hospital stays are inevitable in such patients.

Prolonged hospital stays are an economic burden to the family plus the patient will not earn his livelihood as long as he is admitted in hospital. Other problems like anaemia, hypoproteinemia, bedsores,hospital acquired infections, weight loss & psychological problems accompany prolonged hospital stay[32].Early & proper decision making is a key to avoid all these problems. An investigation that will aid in early decision making is the need of the hour. Angiography is the gold standard for accurate vascular assessment, but angiography cannot be performed in patients with chronic renal failure or acute renal failure. Affordability is also a concern.

Ankle brachial index is the ratio of ankle systolic pressure to brachial systolic pressure. ABI has been studied earlier in peripheral vascular diseases. It has a sensitivity of 70.6% & specificity of 88.5% in diagnosing peripheral vascular disease[27]. Ankle brachial index has been a reliable non invasive alternative to DSA in assessment of lower extremity arteries in peripheral vascular diseases[28]. But literature is not available on ABI as a prognostic indicator in diabetic foot ulcers.. Can Ankle brachial index in diabetic foot ulcers help in early decision making ? The present study was undertaken to answer this question.

The present study was conducted in JNMC & K.L.E.S Dr.Prabhakar.Kore Hospital & Medical Research Centre, Belgaum on Ankle brachial index as a prognostic indicator in diabetic foot ulcers.

In the present study it was seen that incidence of diabetic foot ulcers was more common in males (89.19%) as compared with females (10.81%). The national data source documented higher hospital rates in males suffering from diabetic foot ulcer.

Diabetic foot ulcers are more common between 51 to 70 years of age (64.9%). In our study 43.2% ( 32) of patients were suffering from diabetes for a duration of 6 to 9 years.

59.5% ( 44) of patients had infected diabetic foot & 40.5%(30) had no infection. Infection of the diabetic foot ulcers was documented by culture positivity. Patients with spreading infection, necrotising fasciitis & septicaemia were excluded from the study as they were candidates for emergency amputation as a life saving measure. The outcome of the diabetic foot ulcers with infection & without infection was analysed. P value was not significant, meaning the outcome of diabetic foot ulcers did not depend on the presence or absence of infection in our study.

The co-relation between clinical palpation of lower limb pulsations & ABI was analysed. Mean ABI was 0.85 in patients with absent dorsalis pedis pulsations only, 0.78 in patients with absent anterior tibial & posterior tibial & 0.68 in patients with absent polpliteal pulsations. The difference in ABI was statistically significant. Thus decrease in ABI co-relates well with absent pulsations.

In our study 56.8% (42) of patients had sensory neuropathy & 47.2% ( 32 had no neuropathy.Trophic ulcers in our study were offloaded with appropriate offloading foot wear.None of the patients had charcoat's foot.The outcome of diabetic foot ulcers with neuropathy & without neuropathy was analysed. P value was not significant, meaning the outcome of diabetic foot ulcers did not depend on the presence or absence of neuropathy.

Wagner's class 1 to 4 were included in the study. Maximum patients had class 2 ulceration (40.5%) followed by class 1, 3 & 4.The outcome of diabetic foot ulcers was studied in each wagner's class. P value was significant, meaning that wagner's clssification is a good predictor of the outcome in diabetic foot ulcers.

In our study limb was salvaged in 78.4% ( 58) of patients & major amputation was done in 21.6% ( 16). Mean ABI of patients whose limb was salvaged was 0.92 +/-0.08. The mean ABI of patients who underwent major amputation was 0.74 +/- 0.1.ABI is falsely elevated in diabetics because of the atherosclerotic calcification of the vessels[33-34]. Thus ABI of 0.74 +/- 0.1 is much higher ABI for a major amputation as compaired to 0.3 ABI in non diabetic peripheral vascular diseases[35-36].However ABI does not evaluate microangiopathy.P value < 0.0001,meaning that difference in ABI between limb salvage & major amputation is statistically significant. Thus ABI is a good predictor of the outcome of diabetic foot ulcers.

Angiography is the gold standard investigation for vascular assessment. In our study all the patients were selected from the general ward & none of them underwent angiography & revascularization procedures.ABI is a simple ,

bedside, easy to perform & economic investigation . In our study ABI has been measured with color doppler but ABI can also be measured with hand held doppler. We can conclude from this study that ABI is falsely raised in diabetic foot ulcers .If ABI is < 0.84, the chances of nonhealing ulcer following conservative amputation are high. So one should consider angiographic studies & revascularization procedures. If angiography shows diffuse disease not suitable for revascularization or the angiography is not affordable to the patient ,then surgeon should consider major amputation without wasting time. ABI is an excellent prognostic indicator in diabetics & aides in early decision making thus reducing the morbidity & mortality.

# CONCLUSION

Ankle brachial index is a good predictor of outcome of diabetic foot ulcers.

1. Diabetic foot ulcers with ABI 0.84 to 1.0 ( 0.92 +/- 0.08), in the absence of profound infection will heal & merit debridement or conservative amputation with standard wound care.

2. Diabetic foot ulcers with ABI < 0.84 ( 0.74 +/-0.1) will require further evaluation of vascularity by angiography studies & revascularization procedures if possible. If not, then major amputation in the form of above knee or below knee amputation should be considered, as there are high chances of non healing ulcer following conservative amputation.

# SUMMARY

Incidence of diabetes is on the rise globally. 177 million people world wide suffer from diabetes.15 % of all diabetics develop lower limb ulcers.Diabetes is the most common cause of nontraumatic amputation of lower limb.

Peripheral neuropathy, peripheral vascular disease & susceptibility to infection are the three most important causes of development of diabetic foot ulcer. Prognosis of diabetic foot ulcers depends on the vasularity of the lower limb.

Clinical decision making in diabetics becomes difficult as diabetic feet are deceptive.Often such a feet undergoes multiple debridements & finally the decision of amputation is taken.Prolonged hospital stays are common in diabetics due to the same. Prolonged hospital stays make the patients economically & nutritionally poor & also are a burden to the government hospitals as plenty of resources are consumed.

A solution to address these issues is early& proper decision making. Vascularity of the lower limb is the single most important predictor of healing in diabetic foot ulcers. Vacularity has been assessed by various techniques like colour doppler, angiography, etc. Ankle brachial index has been studied in peripheral vascular diseases earlier but ABI as a prognostic indicator in diabetic foot ulcers has not been studied to date.

- All patients with diabetic foot ulcers from wagner's class 1 to 4 ( toe gangrene only) were included in the study.Total 74 patients were included.
- On the day of admission all the patients underwent measurement of Ankle brachial index with the help of colour doppler. All the patients were selected from the general ward & none of the patients underwent angiography & revascularization procedures.These patients received local wound care, control of infection& offloading foot wear as per hospital protocols.Debridement, disarticulations or amputations were done as per the decision of treating surgeon.Patients were followed up monthly. End point of study was
  1. If patient underwent major amputation & flaps healed.
  2. If ulcer healed or patient underwent skin grafting or
  3. 6 months from the date of admission if 1 & 2 didn't happen.
- Males were more frequently affected as compaired with females.89.19% were males & 10.81% were females.
- 64.9% patients were in the age group of 51 -70 years.
- 59.5% of patients had infected diabetic foot ulcers & 40.5% of patients had ulcers without infection. Necrotisng fascitis & septicaemic patients were excluded from the study as they underwent emergency amputation as a life saving procedure. Outcome of diabetic foot ulcers did not depend on presence or absence of infection in our study.
- The decrease in ABI co-relates well with absent pulsations.

- Absent dorsalis pedis pulsations only, had ABI of 0.85, absent anterior & posterior tibial pulsations had ABI of 0.78 & absent popliteal pulsations had ABI of 0.68.
- 56.8% of patients had neuropathy & 43.2% had no neuropathy. No patients in our study had charcot's foot. Off loading foot wear were given to patients with trophic ulcers. In our study outcome of diabetic foot ulcers did not depend on the presence or absence of neuropathy.
- Majority of the patients belonged to wagner's class 2 ( 40.5%). Wagner's class was a good predictor of outcome of diabetic foot ulcers ie higher the class the more chances of major amputations & lower the class the more chances of healing.
- Limb salvage was possible in 78.4% of the patients & 21.6% of the patients underwent major amputation. ABI of patients with limb salvage was 0.84 to 1.0 (0.92 +/- 0.08). ABI of patients who underwent major amputation was 0.64 to 0.84 ( 0.74 +/-0.1).P vaue <0.0001 meaning the results are highly statistically significant. ABI is falsely raised in diabetic foot ulcers because of atherosclerotic calcification in arteries.
- Thus ABI is a good prognostic indicator in diabetic foot ulcers.
    1. If ABI>0.84 in diabetic foot ulcers, then the patient merits conservative debridement & conseravtive amputation.
    2. If ABI <0.84 in diabetic foot ulcers,there are high chances of non healing ulcer following debridement/conservative amputation. One should consider angiography studies & revascularization procedures . If angiography

shows diffuse athersclerosis not suitable for revascularization or if angiography is not affordable to the patient, then the surgeon should consider major amputation without wasting time.

ABI is a cheap, economic, easy to perform bedside investigation. It can also be performed with hand held doppler.ABI is falsely raised in diabetics.ABI. From this study we can conclude that ABI is an excellent prognostic indicator in diabetic foot ulcers & aids early decision making.

However larger trials are reqiured for better evaluation of ABI as a prognostic indicator in diabetic foot ulcers.

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## ANNEXURE f - CONSENT FORM

Introduction.

Mr./Miss./Mrs_____

You are invited to participate in our research study ie " A STUDY OF ANKLE – BRACHIAL INDEX AS A PROGNOSTIC INDICATOR IN DIABETIC FOOT ULCERS".

Since you are suffering from diabetes & the foot ulcer , which is not healing & will be requiring treatment for the same, you are eligible to be a part of this research & hence asked to participate. This research is about measurement of ankle brachial index in you , & following you up for a period of 6 months & then retrograde analyze the ability of ankle brachial index to predict the outcome of diabetic foot disorders.

If you agree to be a part of this research we would ask you some relevant clinical history . You are free to not to answer whichever questions you think are not relevant . A clinical examination will be done & ankle brachial index will be recorded . Empirical antibiotics will be started & regular betadine & normal saline dressings will be done as per hospital protocols. You will be followed up for a period of six months from the date of admission.

Your decision of whether or not to participate in this study will not affect the quality of treatment you will receive. Further you may withdraw from the study at anytime.

All the new information about you that is collected during the course of the study will be kept confidential to the extent permitted by law. Any information which identifies you personally will not be released without your written consent.

This study does not have any damaging aspect & there are no chances of injury during the course of the study, but if injured the investigator is not responsible. You will have to pay for all the investigations , which are a part of existing management protocol for the treatment of diabetes.

There is no commitment for any reimbursement or any compensation for the participant. The participation in this study is entirely voluntary & you may withdraw from the study at any time. At any time during or after the study , for any information you may contact the researcher.

| | |
|---|---|
| Dr.Satyajit.Godhi<br>Plot no 513, sector III,<br>Shivabasavanagar,<br>Belgaum.<br>Karnataka.<br>Ph no: 9964325835. | **Chairman,**<br>Institutional ethics Committee,<br>Dr.V.D.Patil,<br>Ph no: 0831-2471350. |

Signature of the participant or legally authorized representative:

Subject Name:_____

Signature or the left thumb print of the subject: _____

**Witness name:_____** **Signature:_____**

Investigators name:_____ Signature:_____

Date:_____ Place :_____

# ANNEXURE ff - PROFORMA

**1. PATIENT IDENTIFICATION DATA:**

Name: I.P.no:

Age: D.O.A:

Occupation: D.O.D:

Address:

**2.Chief Complaints:**

HTN:

Smoking:

**Medical history:**

Peripheral neuropathy: yes/no

Nephropathy:No/yes

Retinopathy:No/yes

C.V.D:No/yes

**Diabetic status:**

Type: Type II/I

Duration:

Medication:

**3. Ulcer Detail:**

1.Mode of onset: Spontaneous / Traumatic / Pressure / Others.

2.Duration:

3.Progress:

**4. Wound observation:**

1.size:

2.site.

3.shape.

4.edge:

5.margin:

6.floor:

7.base:

8.discharge:

9.surrounding skin:

IMP: WAGNERS CLASS :

**5.Sensations**.

**6.Vascular examination:** left right

Dorsalis pedis.

Posterior tibial.

Anterior tibial.

Popliteal artery.

**7. Any foot deformity present:**

Toe deformity

Bunion.

Charcot's foot

Foot drop.

**8. If amputation has been done earlier:**

Date:

Side:

Level:

Cause of amputation:

**9. Foot wear assessment:**

Does patient wear appropriate shoes.

Does patient require contact cast immobilisation.

**10.Investigation:**

1.CBC: hb - TLC-

2.ECG:

3.Blood urea: serum creat:

4.Ukb:

5.RBS:

6.HbA1c:

7.Xray of the affected foot:

8.ANKLE BRACHIAL INDEX OF THE AFEECTED FOOT :

9.HIV:

10.Lipid profile:

11. Culture sensitivity:

The patient was debrided on

The ulcer as observed on was

1.size:

2.site.

3.shape:

4.edge:

5.margin:

6.floor.

7.base:

8.discharge.

9.surrounding skin.

IMPRESSION:

The patient underwent _____ skin grafting / disarticutaions / major amputation.

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## ANNEXURE fff - PHOTOS

**Photo 1**: Ankle systolic pressure being measured. Probe of colour doppler placed on anterior tibial & BP cuff tied above malleoli. Pressure is raised until colour signals disappear. The pressure at which colour signals disappear is noted. Now pressure is slowly reduced.The pressure at which colour signals reappear is noted. Mean of the two pressures is taken as ankle systolic pressure.

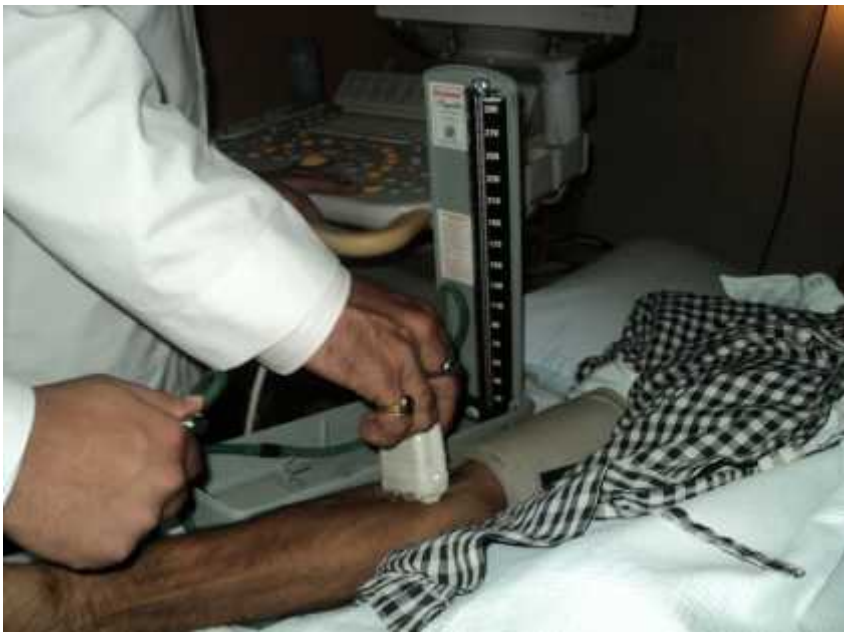

**Photo 2:** Brachial pressure being measured in a similar manner.

## ANNEXURE fff - PHOTOS

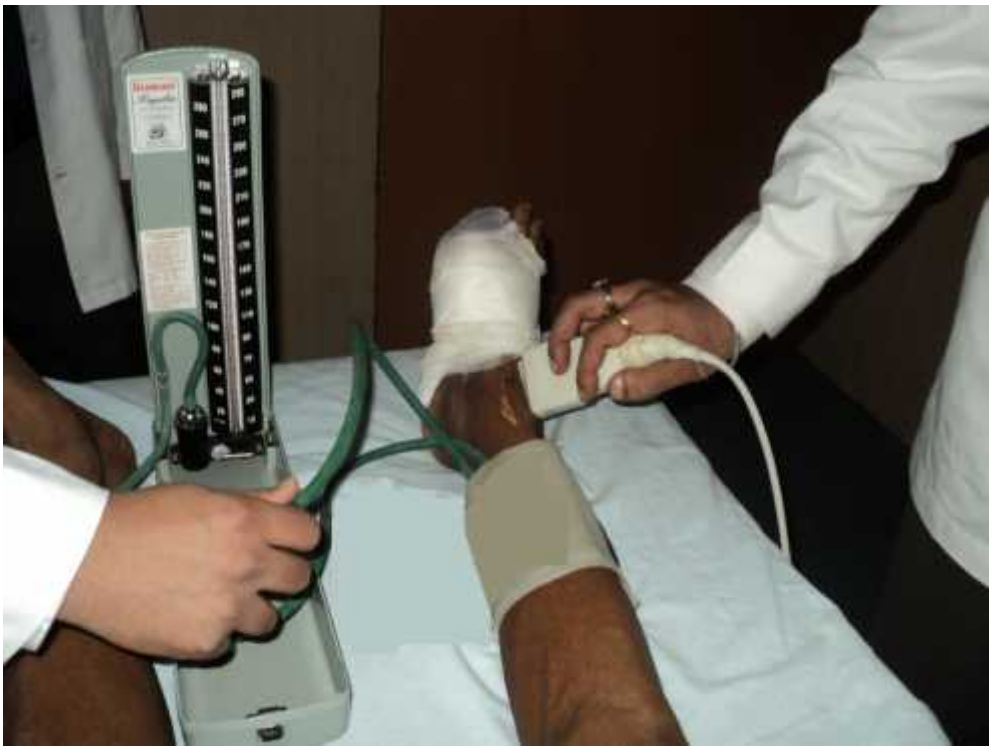

**Photo 1**: Ankle systolic pressure being measured. Probe of colour doppler placed on anterior tibial & BP cuff tied above malleoli. Pressure is raised until colour signals disappear. The pressure at which colour signals disappear is noted. Now pressure is slowly reduced.The pressure at which colour signals reappear is noted. Mean of the two pressures is taken as ankle systolic pressure.

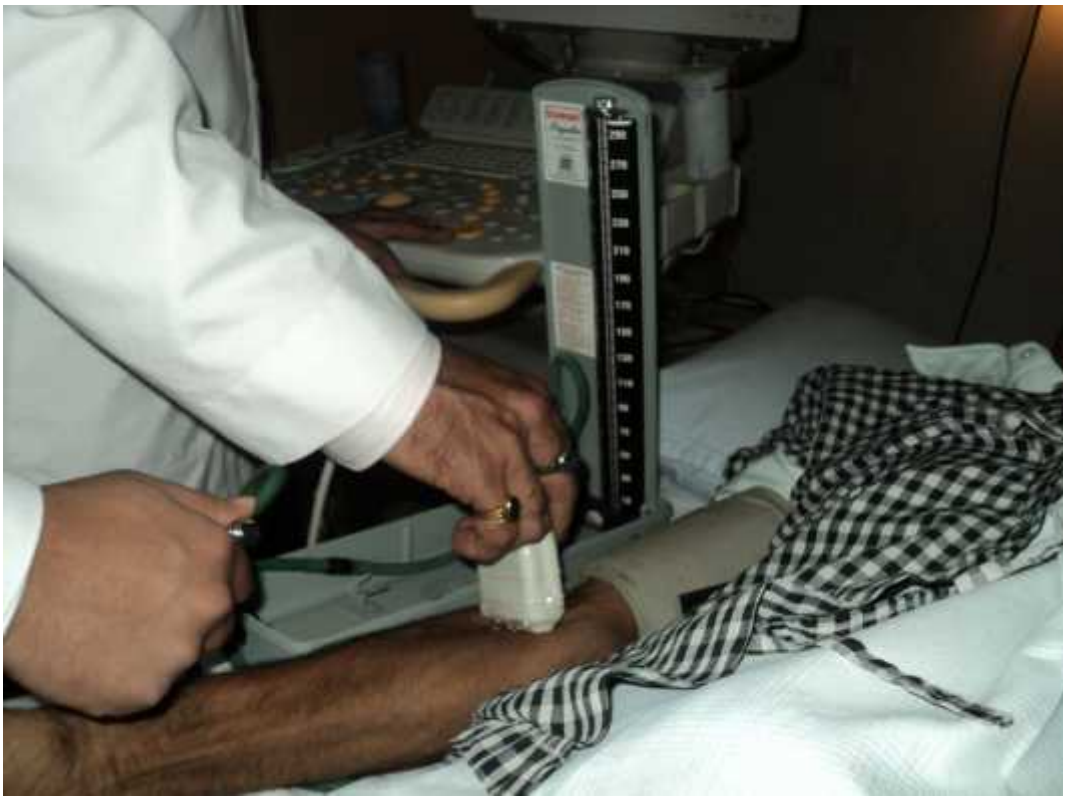

**Photo 2:** Brachial pressure being measured in a similar manner.

## ANNEXURE fV - KEY TO MASTER CHART

| | | | |
|---|---|---|---|
| 1. | M | - | Males. |
| 2. | F | - | Females. |
| 3. | R | - | Right. |
| 4. | L | - | Left. |
| 5. | A | - | Absent. |
| 6. | P | - | Present. |
| 7. | Doraslis.P | - | Dorsalis pedis artery. |
| 8. | A.T | - | Anterior tibial artery. |
| 9. | P.T | - | Posterior tibial artery |
| 10. | Pop. | - | Popliteal artery. |
| 11. | S. | - | Salvaged. |
| 12. | A/K | - | Above knee. |
| 13. | B/K | - | below knee. |
| 14. | ABI | - | Ankle – Brachial Index. |

# MASTER CHART

| SL NO | I.P Number | AGE | SEX | Duration of DM IN Yrs | Size in cms | Site | Sensations | Infection | Pulsations | Wagner's class | ABI | Outcome. |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 1 | 293999 | 42 | M | 6 | 3x3 | R | A | P | P | 2 | 1 | S |
| 2 | 285654 | 44 | M | 6 | 2x3 | L | A | P | P | 1 | 1 | S |
| 3 | 285564 | 44 | M | 9 | 2x4 | L | A | A | P | 2 | 0.94 | S |
| 4 | 300899 | 60 | M | 10 | 2x2 | L | A | P | A:Dorsalis.P | 1 | 0.9 | S |
| 5 | 300844 | 80 | M | 17 | 4x6 | R | P | P | A:AT & PT | 2 | 0.68 | S |
| 6 | 287607 | 54 | M | 8 | 4x6 | R | A | P | P | 2 | 0.99 | S |
| 7 | 245886 | 65 | F | 11 | 3x2 | R | P | P | P | 3 | 0.96 | S |
| 8 | 285995 | 60 | F | 10 | 3x3 | L | P | P | P | 1 | 0.8 | S |
| 9 | 291309 | 51 | F | 10 | 2x3 | R | P | P | P | 1 | 1 | S |
| 10 | 300388 | 66 | M | 13 | 4x4 | L | A | A | P | 1 | 1.06 | S |
| 11 | 292248 | 44 | M | 7 | 4x6 | L | A | A | P | 2 | 0.99 | S |
| 12 | 309851 | 62 | F | 13 | 6x4 | R | A | A | P | 1 | 1.06 | S |
| 13 | 288740 | 64 | M | 14 | 3x4 | L | P | A | A:Dorsalis.P | 2 | 0.82 | S |
| 14 | 295675 | 40 | M | 6 | 2x1 | R | A | P | P | 4 | 0.94 | S |
| 15 | 295961 | 64 | M | 13 | 3x3 | R | A | P | P | 1 | 0.97 | S |
| 16 | 311050 | 54 | M | 6 | 4x2 | L | A | A | P | 1 | 0.99 | S |
| 17 | 300312 | 57 | M | 16 | 6x4 | R | A | A | P | 1 | 0.97 | S |
| 18 | 312829 | 60 | M | 11 | 2x4 | L | P | P | P | 2 | 0.99 | S |
| 19 | 298423 | 46 | M | 9 | 3x4 | L | P | A | A:Dorsalis.P | 2 | 0.88 | S |
| 20 | 306413 | 70 | M | 18 | 4x6 | L | A | A | P | 2 | 1.09 | S |
| 21 | 307226 | 75 | M | 20 | 4x6 | L | A | A | P | 1 | 0.99 | S |
| 22 | 313775 | 73 | M | 17 | 4x6 | L | P | P | P | 2 | 0.89 | S |
| 23 | 307228 | 68 | M | 10 | 3x1 | L | A | p | P | 3 | 0.9 | S |
| 24 | 295362 | 59 | M | 14 | 6x6 | L | P | P | P | 2 | 0.88 | S |
| 25 | 305172 | 65 | M | 8 | 4x6 | R | A | P | A | 3 | 0.68 | A/K |
| 26 | 295594 | 64 | M | 27 | 2x3 | R | P | P | A:dorsalis.P | 1 | 0.87 | S |
| 27 | 297782 | 65 | M | 14 | 3x3 | L | P | P | A:Dorsalis.P | 2 | 0.9 | S |
| 28 | 314887 | 65 | F | 12 | 5x6 | L | A | P | A | 3 | 0.68 | A/K |
| 29 | 297796 | 40 | M | 6 | 2x3 | L | P | P | P | 1 | 0.9 | S |
| 30 | 314331 | 51 | M | 7 | 4x2 | R | P | P | P | 2 | 0.91 | S |
| 31 | 301576 | 60 | M | 9 | 4x6 | R | P | P | P | 1 | 0.92 | S |
| 32 | 298453 | 50 | M | 9 | 3x2 | R | P | A | A:A.T | 2 | 0.8 | S |
| 33 | 301171 | 71 | M | 6 | 2x3 | L | P | P | P | 1 | 0.94 | S |
| 34 | 292119 | 70 | M | 10 | 3x3 | R | A | P | P | 1 | 0.96 | S |
| 35 | 293345 | 50 | M | 7 | 3x2 | L | A | P | A:Dorsalis.P | 1 | 0.79 | B/K |
| 36 | 311246 | 70 | M | 14 | 3X1 | R | A | P | A:AT,PT,DP,Pop | 2 | 0.5 | A/K |

# MASTER CHART

| SL NO | I.P Number | AGE | SEX | Duration of DM IN Yrs | Size in cms | Site | Sensations | Infection | Pulsations | Wagner's class | ABI | Outcome. |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 37 | 299221 | 62 | M | 15 | 5x6 | R | A | A | A:Dorsalis.P | 2 | 0.89 | S |
| 38 | 302636 | 70 | M | 16 | 5x6 | R | A | A | A | 3 | 0.7 | A/K |
| 39 | 303452 | 44 | M | 8 | 4x5 | R | A | P | P:Pop | 4 | 0.8 | S |
| 40 | 300113 | 65 | M | 14 | 3x3 | L | P | P | A:Dorsalis.P | 2 | 0.9 | S |
| 41 | 300031 | 65 | M | 8 | 3x6 | R | P | P | A:Dorsalis.P | 2 | 0.75 | S |
| 42 | 298423 | 67 | M | 17 | 3x4 | R | P | P | P | 1 | 0.83 | S |
| 43 | 315549 | 55 | M | 6 | 5x2 | L | A | P | P | 2 | 0.91 | S |
| 44 | 302426 | 78 | M | 20 | 4x3 | R | P | A | A:Dorsalis.P | 1 | 0.9 | S |
| 45 | 304378 | 75 | M | 10 | 5x5 | R | A | P | P:Pop | 4 | 0.76 | B/K |
| 46 | 303953 | 46 | M | 7 | 3x4 | R | P | P | P | 2 | 0.89 | S |
| 47 | 315549 | 54 | M | 13 | 6x5 | R | P | A | A:AT,PT | 3 | 0.6 | A/K |
| 48 | 304803 | 65 | M | 8 | 1x3 | L | P | P | P | 4 | 0.94 | S |
| 49 | 303919 | 60 | M | 14 | 3x3 | L | P | P | A:Dorsalis.P | 2 | 0.9 | S |
| 50 | 316400 | 65 | M | 16 | 6x6 | R | A | A | A:Dorsalis.P | 2 | 0.87 | S |
| 51 | 305197 | 48 | M | 7 | 5x3 | L | A | P | A:Dorsalis.P | 2 | 0.79 | S |
| 52 | 313783 | 50 | F | 7 | 6x5 | L | A | A | P:Pop | 3 | 0.77 | B/K |
| 53 | 305523 | 60 | M | 11 | 4x6 | R | P | P | P | 2 | O.68 | A/K |
| 54 | 316339 | 57 | M | 8 | 4x4 | R | P | A | P | 1 | 0.96 | S |
| 55 | 306301 | 60 | M | 19 | 3x2 | L | P | A | A:AT | 2 | 0.88 | S |
| 56 | 313775 | 73 | M | 10 | 4x5 | R | A | A | P | 2 | 0.99 | S |
| 57 | 316400 | 65 | M | 8 | 5x6 | R | A | A | P | 3 | 0.84 | B/K |
| 58 | 308132 | 64 | M | 8 | 3x1 | R | A | A | P:Pop | 3 | 0.8 | S |
| 59 | 313906 | 70 | M | 9 | 2x2 | R | A | P | P | 4 | 0.98 | S |
| 60 | 291243 | 45 | M | 10 | 4x5 | R | P | P | P | 2 | 0.8 | B/K |
| 61 | 306295 | 51 | M | 8 | 3x6 | R | P | P | A:Dorsalis.P | 2 | 0.75 | S |
| 62 | 321898 | 45 | M | 10 | 4x5 | R | A | P | P:Pop | 4 | o.86 | B/K |
| 63 | 322468 | 52 | M | 9 | 4x5 | R | A | A | P | 4 | o.98 | S |
| 64 | 321886 | 60 | F | 12 | 5x4 | L | A | P | P:Pop | 4 | o.94 | S |
| 65 | 338625 | 57 | F | 9 | 5x3 | R | A | P | P | 4 | 0.98 | S |
| 66 | 334065 | 54 | M | 14 | 5x6 | L | P | A | P | 3 | 0.82 | B/K |
| 67 | 329497 | 60 | M | 10 | 6x5 | L | P | A | P | 3 | 0.9 | S |
| 68 | 329497 | 45 | M | 10 | 6x5 | R | A | A | A | 4 | 0.67 | A/K |
| 69 | 323786 | 45 | M | 8 | 3x2 | L | A | P | P:Pop | 4 | 0.83 | B/K |
| 70 | 342803 | 46 | M | 9 | 4x5 | L | A | A | P | 2 | 1 | S |
| 71 | 341503 | 65 | M | 9 | 6x6 | R | P | A | P | 1 | 0.96 | S |
| 72 | 340322 | 62 | M | 12 | 4x6 | L | A | P | P:Pop | 3 | 0.84 | B/K |

# MASTER CHART

| SL NO | I.P Number | AGE | SEX | Duration of DM IN Yrs | Size in cms | Site | Sensations | Infection | Pulsations | Wagner's class | ABI | Outcome. |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 73 | 339659 | 46 | M | 10 | 6x6 | R | A | A | P | 3 | 0.98 | S |
| 74 | 340322 | 62 | M | 14 | 3x2 | L | A | A | P | 2 | 0.97 | S |

## ANNEXURE f - CONSENT FORM

Introduction.

Mr./Miss./Mrs_____

You are invited to participate in our research study ie " A STUDY OF ANKLE – BRACHIAL INDEX AS A PROGNOSTIC INDICATOR IN DIABETIC FOOT ULCERS".

Since you are suffering from diabetes & the foot ulcer , which is not healing & will be requiring treatment for the same, you are eligible to be a part of this research & hence asked to participate. This research is about measurement of ankle brachial index in you , & following you up for a period of 6 months & then retrograde analyze the ability of ankle brachial index to predict the outcome of diabetic foot disorders.

If you agree to be a part of this research we would ask you some relevant clinical history . You are free to not to answer whichever questions you think are not relevant . A clinical examination will be done & ankle brachial index will be recorded . Empirical antibiotics will be started & regular betadine & normal saline dressings will be done as per hospital protocols. You will be followed up for a period of six months from the date of admission.

Your decision of whether or not to participate in this study will not affect the quality of treatment you will receive. Further you may withdraw from the study at anytime.

All the new information about you that is collected during the course of the study will be kept confidential to the extent permitted by law. Any information which identifies you personally will not be released without your written consent.

This study does not have any damaging aspect & there are no chances of injury during the course of the study, but if injured the investigator is not responsible. You will have to pay for all the investigations , which are a part of existing management protocol for the treatment of diabetes.

There is no commitment for any reimbursement or any compensation for the participant. The participation in this study is entirely voluntary & you may withdraw from the study at any time. At any time during or after the study , for any information you may contact the researcher.

| | |
|---|---|
| Dr.XXXXXXXX,<br>Post Graduate in department of General Surgery,<br>K.L.E.S Dr.Prabhakar.Kore Hospital.<br>Belgaum.<br>Karnataka.<br>Ph no: xxxxxxx. | **Chairman,**<br>Institutional ethics Committee,<br>Dr.XXXXXXX,<br>Ph no: xxxxxxxx. |

Signature of the participant or legally authorized representative:

Subject Name:_____

Signature or the left thumb print of the subject: _____

**Witness name:_____** **Signature:_____**

Investigators name:_____ Signature:_____

Date:_____ Place :_____