"ASSESSMENT AND EVALUATION OF PERIPHERAL NEUROPATHY, VASCULAR CHANGES AND PLANTAR FOOT PRESSURES IN PATIENTS DIAGNOSED WITH TYPE II DIABETES MELLITUS WITHIN THE LAST 1 YEAR – A CROSS SECTIONAL STUDY"

BY

REG NO: BH0120012

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ABSTRACT

Introduction:

Diabetes Mellitus is a progressive, chronic condition, and continuous hyperglycemia may result in complications that cause severe morbidity to the patient. Diabetes-related peripheral neuropathy, elevated plantar pressures, and peripheral artery disease cause diabetic foot problems such as non-healing ulcers, gangrene, and amputation.

Aims & Objectives:

To assess peripheral neuropathy, vascular changes and plantar pressures in patients diagnosed with type 2 diabetes mellitus within the past 1 year.

The goal is to identify these changes at their onset, so that appropriate treatment can be initiated at the earliest in order to reduce long term morbidity caused to the patients.

Methods:

All patients diagnosed with type 2 diabetes within the past 1 year of their OPD visit underwent assessment of Peripheral Neuropathy, Plantar foot pressures and Vascular changes using the Diabetik Minilab machine as a part of routine examination. Neuropathy was assessed with the 10g monofilament and vibration perception test. Vascular changes were determined by calculating the Ankle Brachial Index and plantar pressures were evaluated using a Harris beath mat. The categorical data was tabulated in terms of frequencies and percentages. 'P' value less than 0.05 considered significant statistically.

Results:

A total of 150 patients were included in this study out of which 97 were males and 53 were females. Peripheral neuropathy was detected in 44% and 65% by the monofilament and vibration perception test respectively. As per the ankle brachial index 22% had peripheral

vascular disease and plantar pressure was increased in 38%. Out of 150, a total of 25 patients (16%) showed all 3 changes and when their HbA1C values were compared with the remaining 125 patients, it was not only found to be higher but was also statistically significant (p = <0.0001)

Conclusion:

Structural and pathological foot changes in diabetics are thought to occur as a function of the duration of the disease. Patients are unaware of the need and type of foot care which is required. Moreover, they too are under the impression that these changes take place after suffering from the disease for several years. However, with the above results we can provide objective evidence to these patients at the time of their diagnosis itself regarding the need for appropriate foot care, foot wear and follow up. With the help of simple tests, this study hopes to spread awareness about early diabetic foot changes so as to reduce its morbid sequelae.

Keywords: neuropathy, plantar pressure, peripheral arterial disease, diabetes, HbA1c

LIST OF ABBREVIATIONS

ABI - Ankle Brachial Index

ACC - American College of Cardiology

AHA - American Heart Association

DFP - Diabetic Foot Problems

DM - Diabetes Mellitus

DPN - Diabetic Peripheral Neuropathy

DSPN - Distal Sensory Polyneuropathy

FBS - Fasting Blood Glucose

H/O - History of

HbA1c - Glycosylated Haemoglobin

KLE - Karnataka Lingayat Education

LOPS - Loss of protective sensation

MRC - Medical research centre

OPD - Outpatient Department

PAD - Peripheral Arterial Disease

PLBS - Post prandial blood sugar

PN - Peripheral Neuropathy

SWME - Semmes Weinstein Monofilament Examination

VPT - Vibration Perception Test

WHO - World Health Organisation

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INTRODUCTION

Diabetes Mellitus is a chronic, metabolic disease characterised by raised levels of blood glucose. In 2019, the International Diabetes Federation estimated that 463 million adults (20-79 years) were living with diabetes and this number would rise to 700 million by 2045.(1)

Type II Diabetes Mellitus, is usually asymptomatic and complications of the disease such as peripheral neuropathy or vascular changes may be the presenting complaints for which patients seek professional medical advice. Less is known as to when the onset of these changes begin, but they are known to be associated with an increase in the risk for foot ulcers, gangrene and possibly lower limb amputations. (2)

Earlier it was thought that foot changes occur late in the course of Diabetics and not in every patient. However, over the years and after extensive research, specialists have concluded that amongst patients with diabetes, every foot is a "diabetic foot" from the day of diagnosis of Diabetes Mellitus.

Loss of peripheral sensation (LOPS) is the leading risk factor for increased plantar pressure. It is also noted that ulcers in diabetics usually occur on the plantar aspect and that the location of the ulcer has a higher peak plantar pressure compared to the rest of the foot. This indicates that patients with LOPS have a higher chance of plantar ulceration.

Another aspect of diabetic foot changes includes Peripheral Arterial Disease (PAD). Its severity depends of the duration of the disease and these patients are more prone to ulceration on the tip of their toes and earlier onset of gangrenous changes.

Peripheral Neuropathy, increased plantar pressures and peripheral arterial disease are all known to develop as a function of the duration of Diabetes mellitus, however it is often noticed that patients get diagnosed with Diabetes while evaluating symptoms of the aforementioned conditions.

With simple assessment tools such as a 10g monofilament, a digital biothesiometer, a and held doppler probe and a harris mat, a Diabetic Foot Evaluation can be performed on an out patient basis and patients can be stratified into risk categories for possible future ulceration. Performing this evaluation within the first few months of being diagnosed with diabetes provides dual advantage – to the surgeon as well as the patient.

From the surgeon's point of view, we are able to detect areas at high risk for ulceration can provide appropriate treatment at the earliest to prevent further sequalae. As for the patients, they become aware of the need for diabetic foot care, its consequences and understand the importance of follow up.

With this in mind, we have designed this study to assess the neuropathic, vascular and plantar pressure changes in newly diagnosed diabetics. The goal is to identify these changes at the earliest, so that appropriate treatment can be initiated at the earliest in order to reduce long term morbidity caused to the patients.

OBJECTIVES

1. To assess peripheral neuropathy, plantar foot pressure and vascular changes in patients diagnosed with type 2 diabetes mellitus within the past 1 year.

REVIEW OF LITERATURE

Diabetes Mellitus

Diabetes Mellitus is a complex, progressive and multi-factorial chronic disease which requires continuous medical care. Type 2 diabetes is the most common type of diabetes, accounting for around 90% of all diabetes worldwide.

The American Diabetes Association defines diabetes mellitus as "a group of metabolic diseases characterized by hyperglycemia resulting from defects in insulin secretion, insulin action, or both." (3)

Overtime, uncontrolled hyperglycemia leads to serious damage to the heart, blood vessels, eyes, kidneys and nerves.

Epidemiology

Diabetes is now thought to be as one of the fastest growing global health emergencies of the 21st century.

In the year 2019, the global prevalence of diabetes amounted to 463 million people which is estimated to rise to 763 million by 2045. Every 1 in 5 of the people who are above 65 years old have diabetes. It has been estimated that 374 million people are predisposed to develop type 2 diabetes as of 2020. (4)

A cause for alarm is the consistently high percentage of people with undiagnosed type II diabetes, which is currently over 45%. This reveals the urgent need to diagnose the undiagnosed people with diabetes and provide appropriate and timely care for them as early as possible.(4)

As per the latest International Diabetes Federation Atlas of 2021, there are 74.2 million Indians suffering from diabetes. Out of these, an alarming 53.1% (39.4 million) of the people were undiagnosed with the disease. (4)

Classification

Diabetes can be classified into the following general categories:(5)

1. Type 1 diabetes (β-cell damage, because of autoimmune mechanisms, leads to absolute insulin deficiency)

2. Type 2 diabetes (β-cell insulin secretion hampered along with insulin resistance)

3. Gestational diabetes mellitus (development of diabetes in the second or third trimester of pregnancy which was not overt diabetes before gestation)

4. Specific types of diabetes: For example,

   - monogenic diabetes syndromes (example- neonatal diabetes and maturity-onset diabetes of the young)

   - drug- or chemical-induced diabetes (example- with glucocorticoid use, in the treatment of HIV/AIDS, or after organ transplantation)

Diagnosis of Diabetes Mellitus[5] (Table 1)

As per the World Health Organisation (WHO) (6) Diabetes can be diagnosed based on any of the following four tests :

HbA1C	>= 6.5%
Fasting Blood Sugar	>= 126 mg/dL
2 hour plasma glucose during 'Oral Glucose Tolerance Test'	>= 200 mg/dL
Random plasma glucose	>= 200 mg/dL with classic symptoms of hyperglycemia and hyperglycemic crisis

Glycated Haemoglobin (HbA1c)

The measurement of glycated haemoglobin, primarily HbA1c, is essential for the treatment of diabetic patients. HbA1c is used to diagnose diabetes, monitor long-term glucose control, change medication, evaluate the quality of diabetic care, and anticipate the risk of complications.(7) Glycated haemoglobin is produced by the non-enzymatic addition of glucose to haemoglobin's amino groups. HbA1c is a type of glycated haemoglobin that is made when glucose binds to the valine at the N-terminus of the -chain of haemoglobin.

HbA1c concentration is dependent on the concentration of glucose in the blood as well as the erythrocyte life span. Due to the fact that erythrocytes circulate for about 120 days, HbA1c indicates the glucose concentration averaged over the last 3 months. Therefore, it is not subject to the daily changes in blood glucose concentrations and is a good indicator of a patient's glycemic control.

Pathophysiology of diabetes mellitus

In Type 2 Diabetes, at the cellular level, inflammatory, autoimmune, and metabolic stress states are influenced by a combination of genetic factors associated with the impaired insulin secretion, environmental factors and insulin resistance.(8)

These result in the bulk of beta cells being destroyed, which affects the production and/or action of insulin. Reduced tissue responsiveness to insulin and insufficient insulin secretion impacts the intricate hormonal action pathways at various levels.

Diabetes-related impairment in the metabolism of protein, fat, and carbohydrate is caused by inadequate insulin action on target tissues. Halim et al(8)

said that classically, the harmful effects of hyperglycemia are categorized into two parts namely macrovascular complications and microvascular complications.

The microvascular complications include retinopathy, diabetic nephropathy, and neuropathy, whereas, the macrovascular complications include stroke, coronary artery disease, peripheral arterial disease and changes in the anatomy of the feet resulting in alteration of plantar pressure.

Complications of Type 2 Diabetes Mellitus (9)

The complications of uncontrolled diabetes mellitus can be broadly classified as acute, such as diabetic ketoacidosis or non ketotic hyperosmolar syndrome or chronic.

The chronic complications may be divided into microvascular and macrovascular. The microvascular consequences include neuropathy, nephropathy - which is a major cause of renal failure, diabetic retinopathy- which results in progressive visual loss.

After ruling out other potential causes of neuropathy, diabetic neuropathy is defined as the presence of clinical signs of peripheral nerve damage in diabetics. Diabetics who have peripheral neuropathy may experience sensory, multifocal, or autonomic symptoms.

The most prevalent form of diabetic neuropathy, distal sensorimotor symmetric polyneuropathy causes an alteration in the complex anatomy of the foot leading to changes in their plantar pressure, ulceration, charcot joints, and amputations.

The root cause of macrovascular problems in diabetes individuals is the increased risk of atherosclerosis, platelet adhesion, and hypercoagulability.

Cardiovascular, cerebrovascular, and peripheral artery disease are more likely to affect diabetics. Chronic hyperglycemia leads to impairment of growth and susceptibility to peripheral vascular changes such as ischemic ulcers and gangrene.

Lower limb complications in Diabetes Mellitus(9)

Due to the microvascular and macrovascular effects of diabetes, lower extremities are subject to a wide range of problems. A diabetic patient's foot is vulnerable due to a combination of distal sensory peripheral and autonomic neuropathy, vasculopathy, and infection. As a result, individuals experience diminished pain, temperature, and proprioception feelings as well as minor muscle atrophy that causes foot deformities, alteration of pressure points which promotes callus formation. All of them increase the likelihood that an ulcer will grow on the patient's foot. The risk of developing an ulcer is increased when the foot is repeatedly traumatised because of diminished sensation, poor blood supply and changes in its anatomy.

Surgical Anatomy of foot

An elaborate neurovascular infrastructure supports the foot's integrated complex of tendons, ligaments, muscles, and bones that are arranged in arches.
It serves as a support for the body's full weight and serves as the primary means of locomotion.

The skeletal framework of the foot comprises of 7 tarsals, 5 metatarsals and 14 phalanges. The hindfoot comprises of talus and calcaneum. The midfoot includes

cuboid, navicular and cuneiforms and the forefoot consists of phalanges and metatarsals.(10)

Skin and Subcutaneous Tissue(11)

Compared to the dorsal aspect, the skin on the plantar aspect, or sole of the foot, is thicker and more sensitive. In comparison to the loose tissue deep to the dorsal skin, the subcutaneous tissue deep to the plantar skin is more fibrous and compact.

As you walk, the skin is held in place by fibrous septa that connect to the plantar aponeurosis. This enhances plantar grip while walking. Additionally, these "modified skin ligaments" concentrate subcutaneous fat on the weight-bearing parts of the heel, lateral heel border, and plantar aspect of the metatarsal heads. This makes those areas best equipped to serve as shock absorbers.

The skin over the sole is characterised by the absence of hair follicles and sebaceous glands, as well as the conspicuous presence of numerous sweat glands.(10)(11)

Deep fascia

The deep fascia is a distant extension of the inferior extensor retinaculum that extends over the dorsum of the foot.

Over the foot's sole, it has a more complicated structure, and is known as the "plantar fascia", where the distribution is weaker in the medial and lateral portions and thicker in the centre.

The maintenance of the longitudinal arches of the foot depends heavily on the plantar fascia. Additionally, it maintains the integrity of the foot's components and safeguards the plantar aspect from harm.

The "plantar aponeurosis" is the name given to the thicker centre section. It reaches out from the "fibrous digital sheaths," which are its own modifications, and terminates in the calcaneum distally, covering the metatarsal head with the "superficial transverse metatarsal ligament" and the five flexor tendons heads.(10)(11)

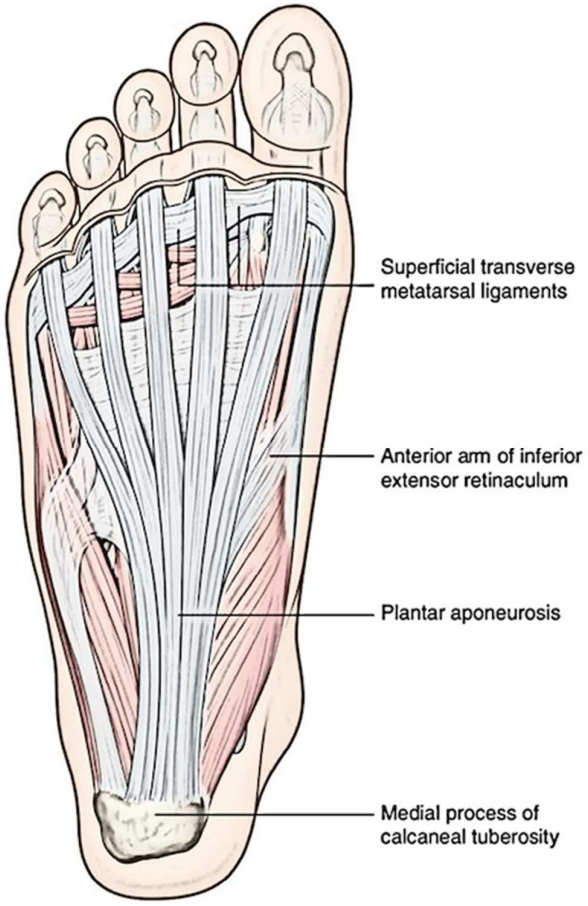


Figure 1: Plantar aponeurosis

Muscles of the foot

Twenty muscles make up each foot. There are two on the dorsal side, four in the middle and 14 muscles on the sole of the foot. The muscles on the sole of the foot are separated into four compartments, each with four layers.

By preserving the integrity of the foot arches, all of the muscles in the sole stabilise the foot during the support phase of stance. (10)(11)

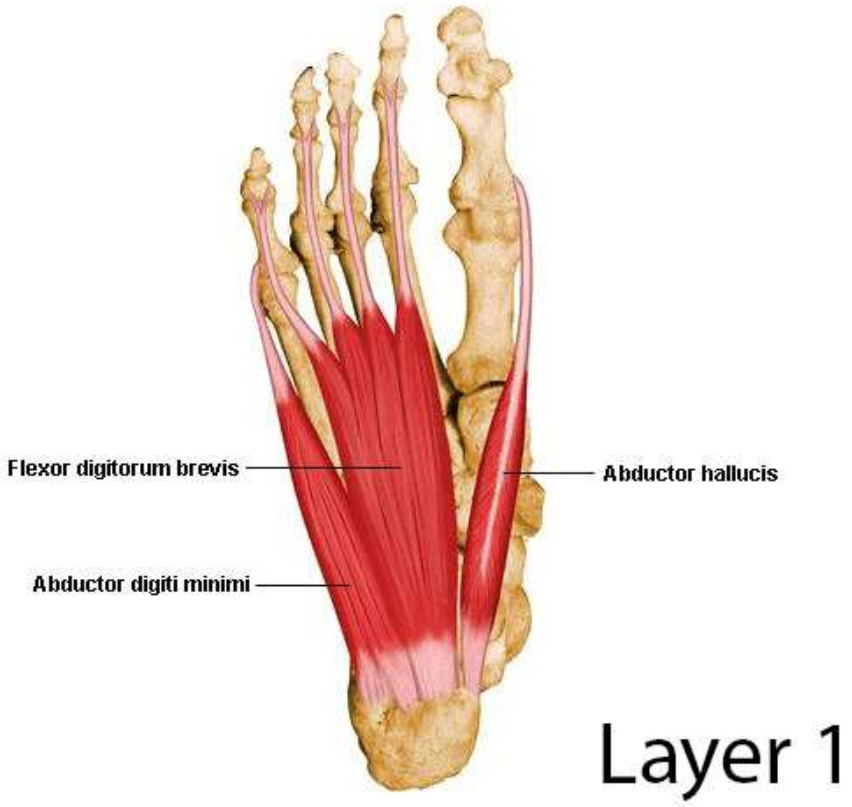


Figure 2a: Muscles of the sole of the foot- 1st layer

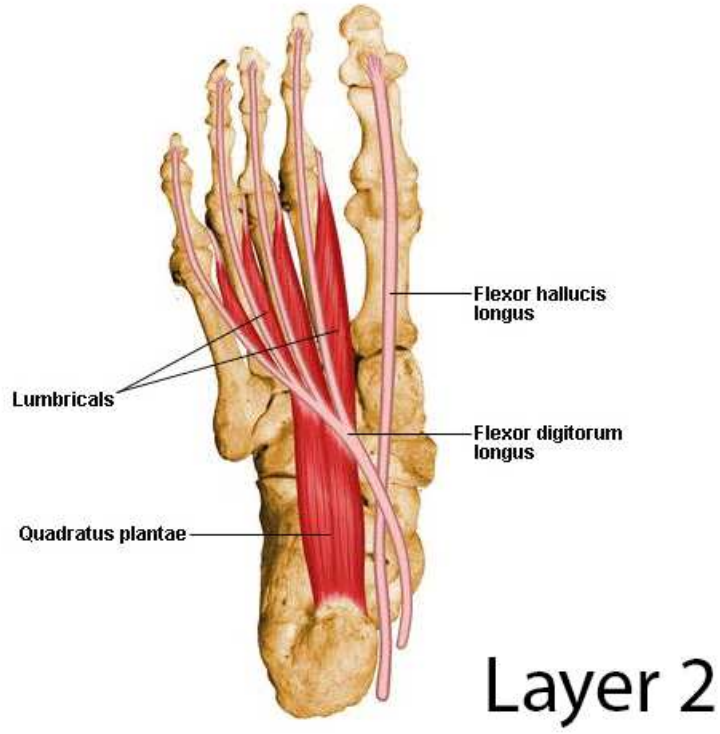


Figure 2b: Muscles of the sole of the foot- 2nd layer

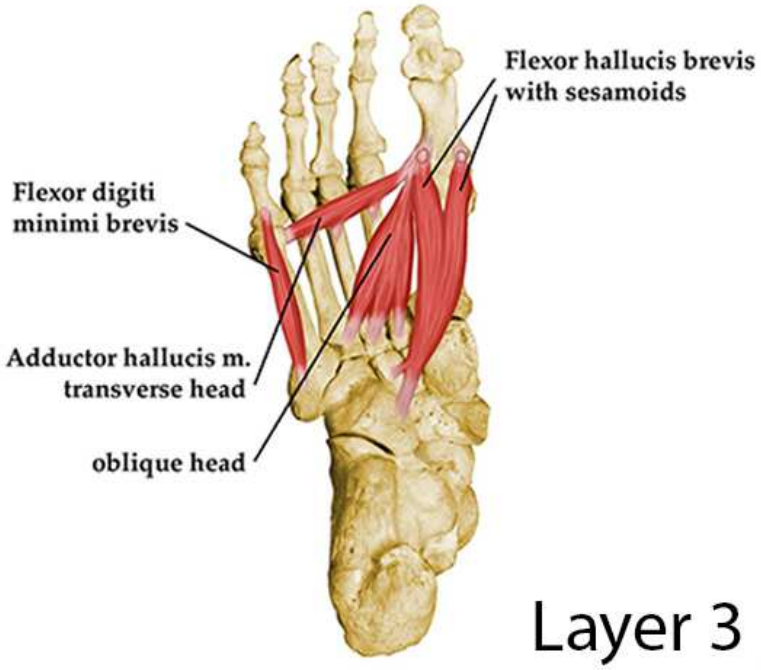


Figure 2c: Muscles of the sole of the foot- 3rd layer

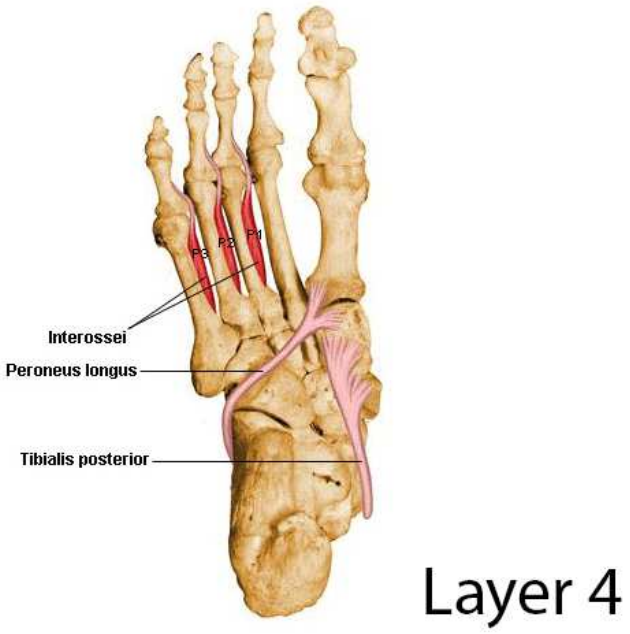


Figure 2d: Muscles of the sole of the foot- 4thlayer

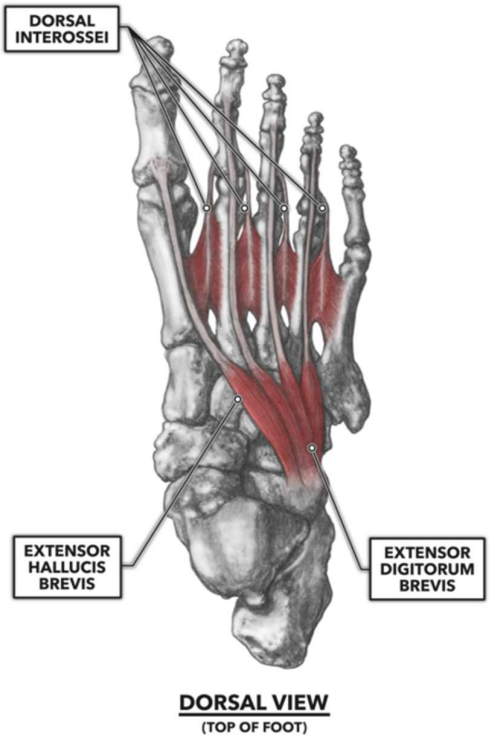


Figure 3: Muscles of the dorsum of the foot

Nerve supply of foot(10)(11)

The medial and lateral plantar nerves innervate the muscles of sole of foot. They are end branches of the tibial nerve.

Only the Extensor digitorum brevis muscle on the dorsum of foot is innervated by lateral branch of deep fibular nerve.

The cutaneous nerve supply of foot is by the following nerves:

1) Superficial fibular (peroneal) nerve – medial and lateral cutaneous branches
2) Deep fibular (peroneal) nerve
3) Sural nerve
4) Saphenous nerve
5) Medial and lateral plantar nerves

Figure 4: Cutaneous nerve supply of the foot

Arterial supply of foot(10)(11)

Dorsum of Foot-

The Dorsalis pedis artery, that is the continuation of the Anterior tibial artery supplies the dorsum of the foot.

Dorsalis Pedis artery branches : -Lateral tarsal artery

-Arcuate artery

-First dorsal metatarsal artery

-Deep plantar artery (terminal branch)

PLANTAR ARCH

Sole of Foot

Supplied by end branches of Posterior tibial artery:

-Medial plantar artery

-Lateral plantar artery

The Plantar arch is created by an anastomosis between the deep plantar artery and lateral plantar artery, and gives plantar metatarsal arteries to the toes.

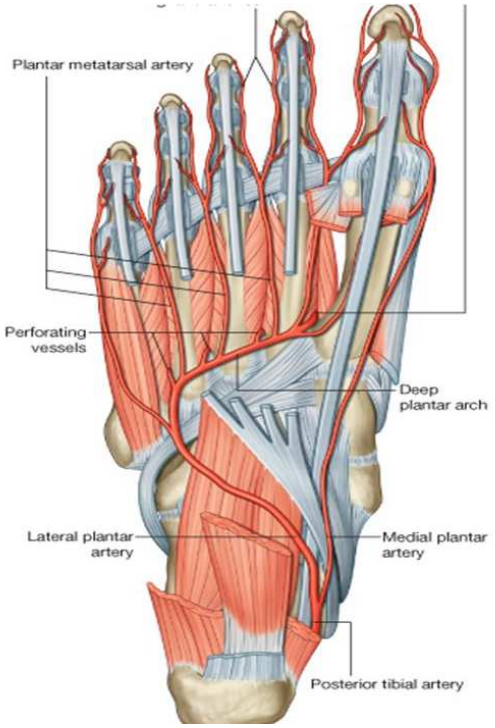


Figure 5a: Arterial supply of the foot

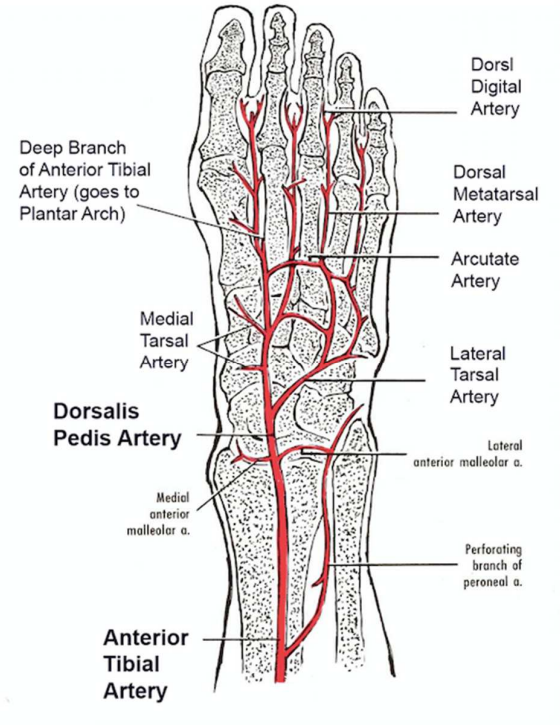


Figure 5b: Arterial supply of the foot

Venous drainage of foot(10)(11)

The majority of the blood in the foot is drained from the sole into the great saphenous vein medially and the short saphenous vein laterally, forming the dorsal venous arch.

The posterior tibial venae comitantes are formed by the union of the medial and lateral plantar veins on the bottom of the foot, which travel with their corresponding arteries.

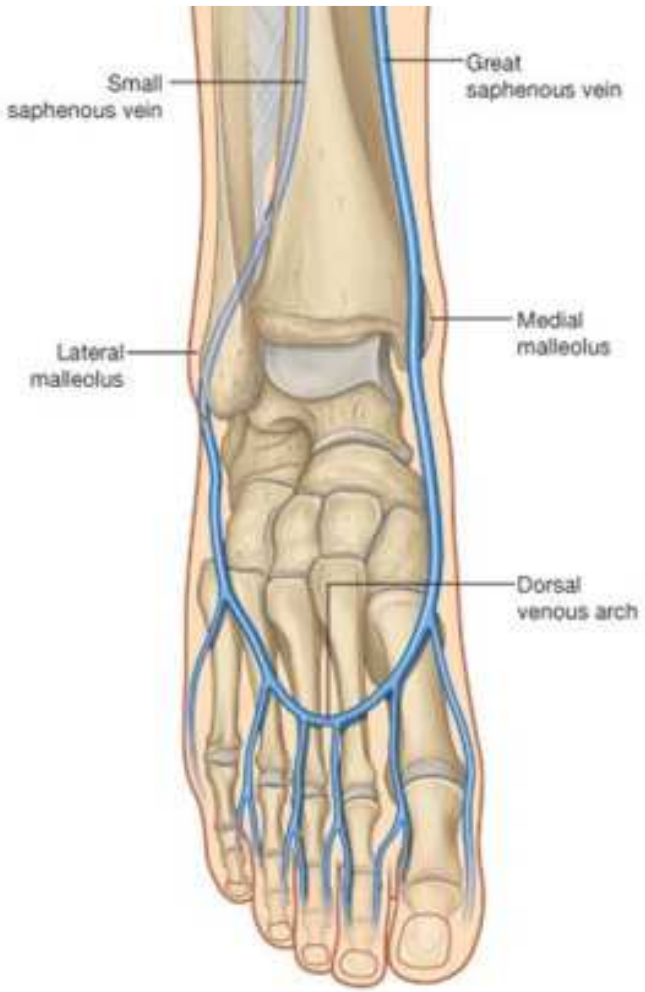


Figure 6: Venous drainage of the foot

Arches of the Foot (6)(7)

The bones that make up the arch of the foot are connected by ligaments, tendons, and muscles to form a complex elastic framework. These arches allow the foot to support the entire weight of body.

The various arches of the foot are as follows:

-Longitudinal arch: 1) Medial longitudinal arch

2) Lateral longitudinal arch

- Transverse arch

Functions of the arches of the foot are as follows:

1) Provide uniform distribution of the weight of the body over the entire foot
2) Act as a propellant for locomotion such as during walking, running and jumping
3) Enable the foot to adapt to changes in surface contour

Supports for the foot arches:

A variety of variables in the foot help to maintain the health of the arches.

PASSIVE SUPPORTS:

1) Ligaments of the foot –

-The most important factor in maintainence of the arches

-Includes the Spring ligament or Plantar Calcaneonavicular ligament, Short and Long Plantar ligaments

2) The plantar aponeurosis

3) The structural integrity of the bones themselves.

DYNAMIC SUPPORTS:

1) The action of intrinsic and extrinsic muscles

2) The action of the long tendons extending into the foot

- Flexor digitorum longus, Flexor hallucis longus, Fibularis longus, Tibialis posterior

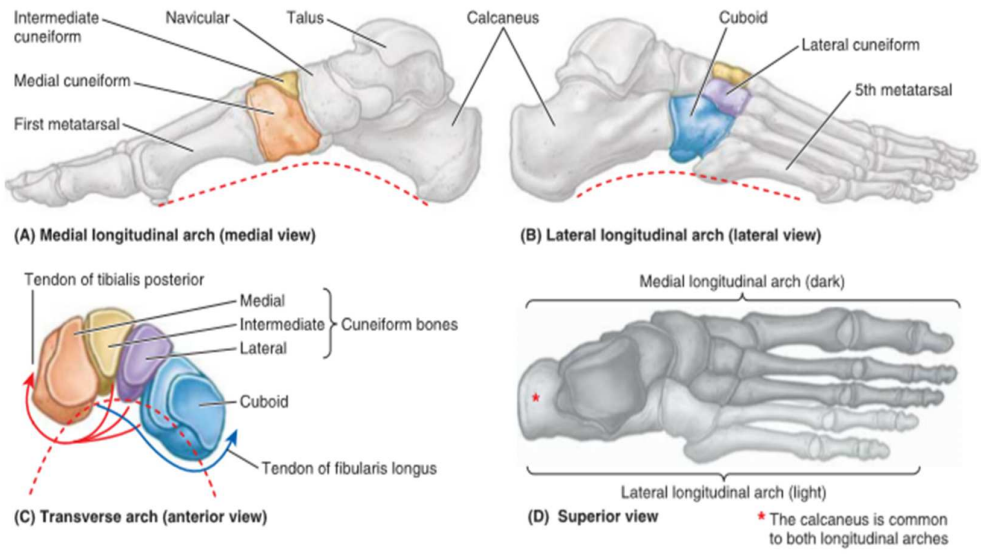


Figure 7: Arches of the foot

BIOMECHANICS OF THE DIABETIC FOOT

The foot is subjected to a variety of forces acting simultaneously on various structures. This serves as the foundation for foot biomechanics. It is common for aberrant biomechanics to be the etiological cause of excessive callus, foot deformities, and foot ulcers in a diabetic foot that appears normal from the outside. These ulcers heal slowly and frequently recur.

Peripheral neuropathy and increased plantar pressure, particularly over regions of bony prominences, provide the basis of abnormal biomechanics in a diabetic foot. The underlying tissue is exposed to harm and forms calluses as a result of the loss of sensation over places where subcutaneous tissue and fat are lacking. Once haemorrhage has established itself in the callus, the area serves as a precursor to an ulcer. Additionally, noteworthy contributors to tissue disintegration include peripheral vascular disease and trauma.(12)

Ulcers grow on the heads of metatarsals, tips of toes, and midfoot in conditions such clawing of the toes, hammer toes, and Charcot's arthropathy. When the foot is malformed, the lack of subcutaneous fat in certain areas emphasises tissue disintegration even more.(12)

Elevated plantar pressure results from restricted joint movement at the metatarso-phalangeal, especially that of the first toe, as well as sub-talar joints. This is brought on by non-enzymatic glycosylation, which causes collagen at joint capsules to stiffen.

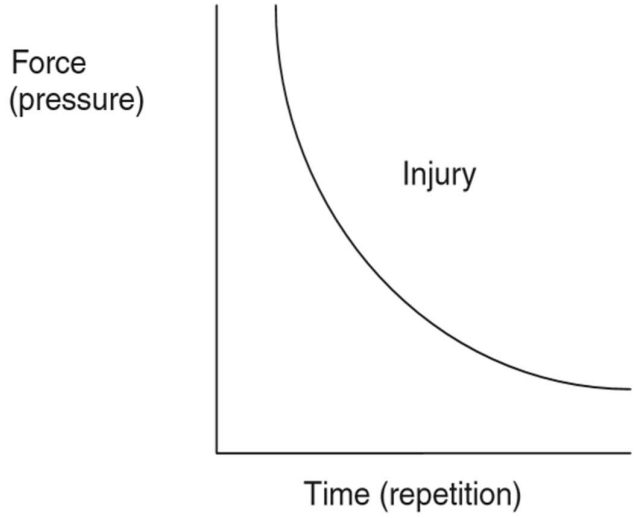
The extrinsic factors that have the most observable effects on increasing plantar pressure are poorly fitted, incorrect shoes, and barefoot walking.(13)

Three mechanisms which can lead to elevated pressure in a diabetic foot predisposing it to ulcer formation are as follows :

1. Increased magnitude of pressures- When a diabetic patient with peripheral neuropathy experiences trauma from a sharp nail or shard of glass, a significant amount of force is delivered across a very small region of skin. Similar injuries can result from a "foot slap." In this instance, weak dorsiflexor muscles result in a reduced forefoot slowing down once the foot contacts the ground.

2. Increased number of pressures- The tissue is unable to preserve integrity as a result of repeated loading, as in a neuropathic diabetic foot. Due to the absence of sensation in the foot, injuries occur repeatedly. This is additionally known as "mechanical weariness." In his research on ischemic ulcers, Kosiak found that force and time have a reversible link. The time or frequency of force(s) necessary to harm tissue must decrease as force increases.(13)

3. Increased duration of pressures- a prolonged period of low pressure that causes ischemia and subsequent tissue damage. Most often because of improper footwear or prolonged heel positioning over a flat surface.



GRAPH 1: Relationship between Force and Time

PATHOPHYSIOLOGY OF THE DIABETIC FOOT

The hyperglycemic state of diabetes is the core of diabetic foot disease. Diabetes-related foot changes are caused by a combination of neuropathy, vasculopathy, increased plantar pressure and infection.

DIABETIC NEUROPATHY

The chronic state of hyperglycemia in diabetics causes oxidative stress to the neurons. Metabolic derangements like nonenzymatic glycation and polyol pathway hyperactivity leads to the production of free radicals.

This has a direct toxic effect on neurons along with reduction in nitric oxide production. The decrease in nitric oxide then leads to endothelial dysfunction and thus blood flow to the nerves is depleted.

Type II Diabetes is commonly an asymptomatic disease and so an extended duration of impaired glucose metabolism may precede its onset. Therefore, diabetic neuropathy may already be present at the time of diagnosis.(14) However, little is known as to when exactly diabetic patients without any diabetic foot problems develop sensory neuropathy.(2)

Involvement of peripheral nerves is the commonest complication of diabetes, with about 30% of diabetics suffering from neuropathy during their lifetime(15). The severity of neuropathy is a function of the duration of diabetes(16). A study by *Nather et al* concluded that while they found the incidence of neuropathy to be higher in patients with diabetes for >10 years, they also found a significant number of people suffering from neuropathy who had a duration of diabetes for less that 5 years. (2)

Thus, suggesting that evaluation of the neuropathic status of the limb should be done at the time of diagnosis of diabetes itself.

Patients with neuropathy are said to have “insensate feet”. They ignore trivial trauma to their feet which can lead to recurrent foot infections, ulcerations, gangrene and eventually may lead to an amputation.

The neuropathy manifests in the sensory, motor, and autonomic nervous systems.

Diabetes-related neuropathy is divided into several clinical disorders. Depending on the component of the peripheral nervous system that is impaired, each syndrome has its own unique set of symptoms and manifestations.

The most prevalent neuropathies include the following:(16)

1. Distal symmetric polyneuropathy (DSPN)
2. Autonomic neuropathy
3. Thoracic and lumbar nerve root disease, causing polyradiculopathies
4. Individual cranial and peripheral nerve involvement causing focal mononeuropathies
5. Asymmetric involvement of multiple peripheral nerves, resulting in a mononeuropathy multiplex.

CLASSIFICATION OF DIABETIC NEUROPATHIES

(TABLE 2)([16])

Diabetic neuropathies
A. Diffuse neuropathy
▪ DSPN
• Primarily small-fiber neuropathy
• Primarily large-fiber neuropathy
• Mixed small- and large-fiber neuropathy (most common)
▪ Autonomic
• Cardiovascular
◦ Reduced HRV
◦ Resting tachycardia
◦ Orthostatic hypotension
◦ Sudden death (malignant arrhythmia)
• Gastrointestinal
◦ Diabetic gastroparesis (gastropathy)
◦ Diabetic enteropathy (diarrhea)
◦ Colonic hypomotility (constipation)
• Urogenital
◦ Diabetic cystopathy (neurogenic bladder)
◦ Erectile dysfunction
◦ Female sexual dysfunction
• Sudomotor dysfunction
◦ Distal hypohydrosis/anhidrosis
◦ Gustatory sweating
• Hypoglycemia unawareness
• Abnormal pupillary function
B. Mononeuropathy (mononeuritis multiplex) (atypical forms)
▪ Isolated cranial or peripheral nerve (eg, CN III, ulnar, median, femoral, peroneal)
▪ Mononeuritis multiplex (if confluent may resemble polyneuropathy)
C. Radiculopathy or polyradiculopathy (atypical forms)
▪ Radiculoplexus neuropathy (ie, lumbosacral polyradiculopathy, proximal motor amyotrophy)
▪ Thoracic radiculopathy

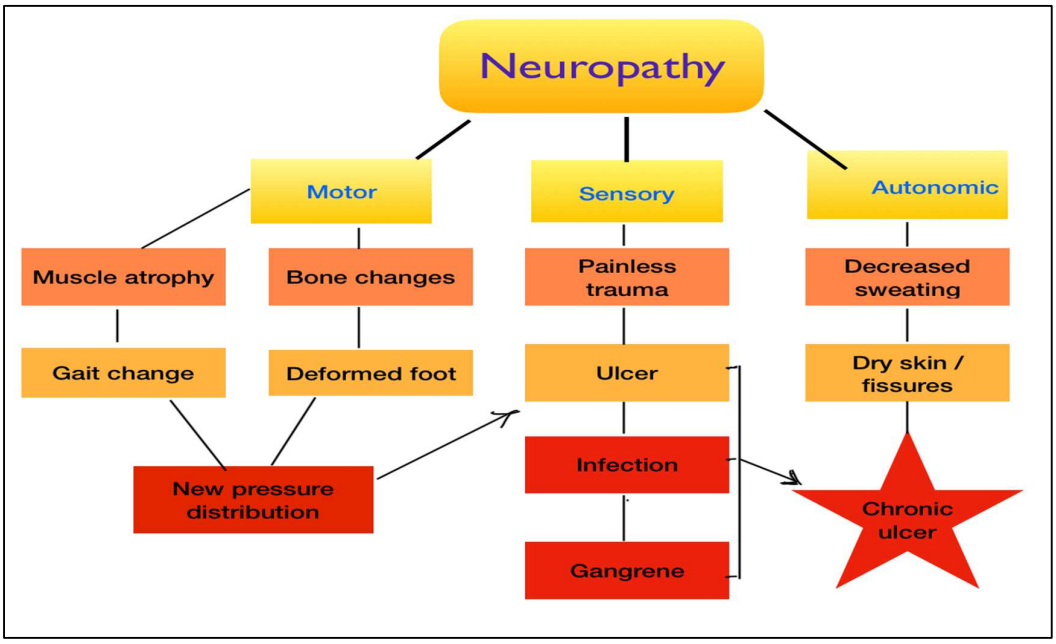
Sequalae of neuropathy

Sensory- The loss of protective sensation in diabetic feet increases their susceptibility to trauma and ulcer formation.

Motor- Due to the loss of the motor fibres, the nerve supply to intrinsic foot muscles is compromised. Due to the atrophy of the intrinsic muscles, the flexion and extension of the foot become unequal. Claw toe and hammer toe abnormalities result from hyperextension of the metatarso-phalangeal joint with flexion of the proximal or distal interphalangeal joints, respectively.

The resulting abnormalities of the foot expose bony prominences that function as pressure points. This makes the foot susceptible to skin erosion and ulcer development.

Autonomic - Loss of sympathetic tone results in increased arteriovenous shunting and ineffective nutrition flow. The functional impairment of sweat and sebaceous glands causes the foot to become dry and prone to skin rupture. This increases the risk for secondary infection and predisposes the foot to ulcer formation.



Sequalae of diabetic neuropathy (TABLE 3)

Quantitative sensory testing (QST) is a non invasive way of measuring and quantifying sensory nerve function. (17) It can be carried out using simple tests such as the Semmes Weinstein monofilament examination (SWME) and Vibration perception. (18)

The large myelinated A-alpha, A-beta and C sensory fibers are evaluated with light touch and vibration testing. (17) The Semmes Weinstein monofilament examination (SWME) is a non-invasive, inexpensive, quick, and easy test used in clinical examination. The monofilaments are applied to each test site perpendicularly until they bend for about one second. Blindfolded patients are told to say “yes” each time they sense the monofilament on their foot. If patients fail to sense the monofilament after it bends, the test site is considered to be insensate. The buckling force for the standard 5.07 monofilament is 10 grams, which is also the force felt by the patient when the monofilament bends. (19)

Using a biothesiometer, in similar fashion as for the SWME, a vibration probe is placed at chosen test sites. A signal is initiated starting from 0V and the patient is asked to say “yes” as soon as they feel a sense of vibration at each site, while being blindfolded. An average value of >25V in at least one foot signifies an increased cumulative risk of ulceration on that foot, as high as seven-fold(20).

The optimal screening tool for diabetic neuropathy is one that offers precise and objective results in a short amount of time, is quick to administer, is readily accessible, and has high sensitivity, specificity, and positive predictive value.

With simple tools such as the SWME and Vibration perception testing, we could determine the status of neuropathy when patients are diagnosed with type 2

diabetes mellitus. This information can in turn be used as objective evidence to educate patients regarding the importance of foot care and glycemic control which could before sensory neuropathy sets in. With early intervention, the sequalae of diabetic neuropathy can be avoided.

PLANTAR PRESSURE

Walking, also known as ambulation, is the primary mode of human mobility and the most prevalent action in our daily lives. While standing, walking or running, the ground exerts a pressure on our feet which is experienced by our sole known as plantar pressure. The foot is the most caudally located weight-bearing body part. The arrangement of bones, muscles, and joints facilitates mobility through the absorption and support of intense pressure during standing and walking.

Taha et al. in 2016 concluded in their study that physiologically, there are three significant points of the highest pressure on the sole i.e central part of the heel and the 1st and 4th-5th metatarsal heads.(21) This physiological distribution pattern is almost symmetrical and hence provides the feet optimal stabilization and balance.

Pressure distribution and quantity below the plantar surface depends on: body weight, age, and foot abnormalities secondary to disease. (22). It is known that in poorly controlled diabetics, the plantar pressure distribution is altered and that areas with increased pressure are more prone to ulceration than the rest of the foot – this area mainly being at the head of the 1st metatarsal.This is seen particularly when combined with foot deformity and peripheral neuropathy

As discussed above, peripheral motor neuropathy leads to structural changes in the foot such as hammer toes, charcots foot, bunions and autonomic changes cause

thickened skin and callus formation. These changes are responsible for the altered areas of high pressure zones in diabetic feet.

Plantar pressure distributions are widely recognised as a dependable biomechanical measure for studying and diagnosing a variety of foot problems in the modern day.

To assist in clinical diagnosis, decision-making, and follow-up, the Harris and Beath footprint is a low-cost, non-invasive way of capturing foot pressure patterns. Once the footprint is obtained over the mat, a pedobarograph can be used to determine the visual pattern and value of the plantar pressure. The pedobarograph is a device that uses a pressure measurement to turn the applied pressure into a visual light pattern.

Figure 8a: Harris Beath mat equipment

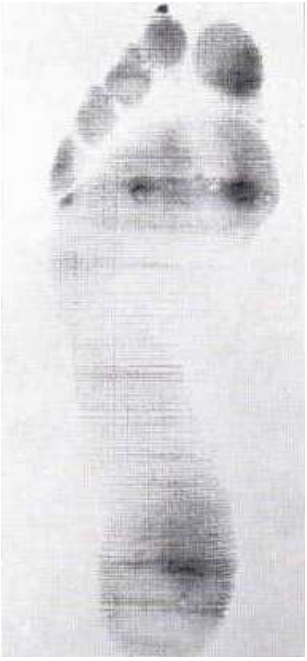


Figure 8b: Harris &Beath mat print

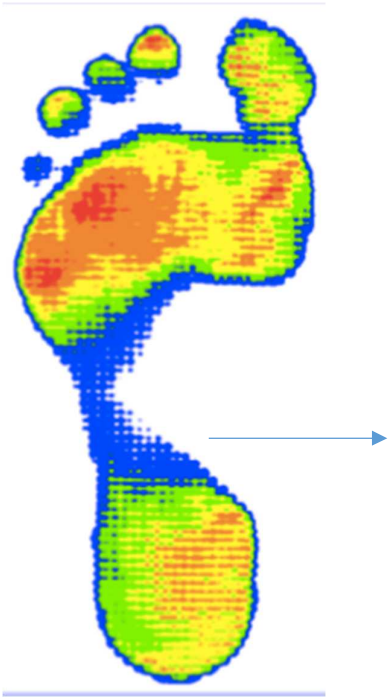


Figure 8c: Pedobarograph representation

The Diabetik foot software used in this study, categorizes the pressure levels using the following colour codes:

- Red – Very high pressure
- Orange – High pressure
- Yellow – Mild Pressure
- Green – Normal pressure
- Blue – Less pressure
- White - No contact area

Since the average, disease free human has 3 high pressure points, any diabetic subject having 4 or more pressure points which are represented with Yellow, orange or red colours were considered as those having an increased plantar pressure.

The optimum way to use foot pressure would be to predict possible ulceration areas with measurements before an ulcer develops and to efficiently provide off-loading of ulcerated regions to speed up wound healing.

However, even with the advent of advanced and digitalized methods to determine plantar pressure, a paper by Fernando et al in 2018 stated that there is no standardized protocol for this assessment up to now.(23)

Using one of the oldest methods, i.e the Harris beath mat along with newer technology, we can successfully provide a pictorial view of the pressure directly to the patient to help them understand the problem at hand. This pictorial representation can be used to educate patients regarding the importance of foot care at the time of diagnosis.

VASCULAR CHANGES

The prolonged hyperglycemic state eventually has a lasting effect on the vasculature, leading to the development of peripheral arterial disease.

Peripheral artery disease (PAD) is one of the most prevalent complications of diabetes, carrying the risk of developing critical limb ischemia and necessitating amputation of the affected limb.

As with neuropathy, the severity of PAD depends on the duration of diabetes. However, a study by *Faglia et al* sowed that it was also prevalent amongst recently diagnosed diabetics(24). As per his study, 21% of newly diagnosed diabetics had an Ankle Brachial Index of <0.9, suggestive of peripheral vascular disease.

As per the American Heart Association, the Ankle Brachial Index (ABI) is the initial diagnostic test for PAD and might be the only test required to establish the diagnosis and initiate treatment.(25)

The method of choice for ABI measurement is the use of Doppler handheld probe with a regular sphygmamometer. The ankle brachial index is calculated by dividing the higher of the two ankle pressures (Dorsalis pedis artery or posterior tibial artery) divided by the higher arm pressure.

$$\text{Ankle Brachial Index} = \frac{\text{Higher of the two Ankle pressures}}{\text{Arm Pressure}}$$

The normal ABI is 0.9-1.2

An ABI < 0.9 suggests peripheral arterial disease

An ABI > 1.3 suggests incompressible arteries

As per the American Heart Association, those with ABI more than 1.3 due to non-compressible vessels can be subjected to other tests such measurement of systolic pressures in toes, pulse volume assessment, duplex ultrasound or transcutaneous oxygen quantification. Derangement in these tests prove the presence of peripheral arterial disease.(25)

This technique is limited by user dependence, equipment accessibility, and lack of training. Because diabetics have calcified arteries with poor compressibility, there is a chance that the value will be falsely high.

TABLE 4: Interpretation of Ankle Brachial index results

ABI[a]	Interpretation
>1.30	Poorly compressible vessels, arterial calcification
0.90–1.30	Normal
0.60–0.89	Mild arterial obstruction
0.40–0.59	Moderate obstruction
<0.40	Severe obstruction

Pathophysiology of vascular changes

Diabetes mellitus (DM) is characterized by hyperglycemia, dyslipidemia, and insulin resistance. The underlying metabolic abnormalities in DM exacerbate vascular inflammation, endothelial dysfunction, vasoconstriction, platelet activation, and thrombotic risk, which are key processes in the pathogenesis of PAD in diabetic patients.(26)

The diabetic vasculopathy has two distinct manifestations: a nonocclusive microcirculatory impairment in the blood vessels of the retina, peripheral nerves, and kidney, and a macrocirculatory impairment characterised by atherosclerosis of the cardiovascular and peripheral vascular blood vessels.

Microvascular - Thickening of the capillary basement membrane impedes leukocyte movement and causes functional microcirculatory ischemia in injured tissues. This is exacerbated by the absence of a neurogenic vasodilatory response to injury, which renders the foot more susceptible to infection. This is exacerbated by the absence of a neurogenic vasodilatory response to injury, which renders the foot more susceptible to infection.

Macrovascular - This condition is distinguished by the presence of peripheral arterial disease. The vascular disease of the lower extremities is predominantly caused by accelerated atherosclerosis of the tibial arteries. This ultimately leads to critical limb ischaemia and the possibility of limb loss. (27)(28)

Using the ankle brachial index as an indicator for PAD, we can detect changes in the vasculature at its initial stage and intervene appropriately to prevent further complications like ulcers and gangrene from setting in.

NEED FOR THE STUDY

The diabetic pandemic is a continuous, ever growing problem which lacks in patient education and awareness regarding the severity of the disease and its sequalae. The older concept of diabetics developing foot complications after suffering from the disease for years, needs to change. Diabetics need to be aware of what symptoms and signs to look for which may lead to foot changes causing ulceration. With the means of simple tests which can be carried out in the out- patient department, this study wishes to assess the incidence of neuropathy, changes in plantar pressure and vasculopathy in patients who have been diagnosed with type 2 diabetes mellitus within the last 1 year. This study is carried out with the aim to provide objective visual evidence to diabetics which will make it easier for them to comprehend the need, urgency and importance of foot care.

MATERIALS AND METHODS

The source of data were patients who came to the outpatient department at KLES PRABHAKAR KORE HOSPITAL AND MRC, BELAGAVI between January 2021 and December 2021.

Study Design: CROSS – SECTIONAL STUDY

Duration of data collection: 1 YEAR

Study Period: January 2021 - December 2021

Study Population: Patients who came to the outpatient department and were diagnosed with Type II Diabetes mellitus within the last one year of their visit.

Selection Criteria:

INCLUSION CRITERIA	EXCLUSION CRITERIA
1. Patients diagnosed with type 2 diabetes mellitus within the past 1 yearof their visit to the OPD. 2. Above or equal to 35 years of age. 3. Patients who give consent to participate in this study.	1. Patients with known history of diabetes for a duration of more than 1 year 2. Patients with known history of Peripheral Vascular Diseases or Varicose Veins 3. Patients with history of foot ulcers. 4. Patients with known history of Peripheral neuropathy due to other cause

Table 5: Selection criteria

Sample Size: Sample size was calculated using Cochran's Sample Size formula:

$$N_0 = \frac{Z^2 pq}{e^2}$$

Where, N_0 = Sample size

Z = Z value is found using a Z table (related to Confidence Interval)

e = desired level of precision (i.e. the margin of error)

p = the (estimated) proportion of the population which has the attribute in question

q = 1 – p

Using a 94% Confidence Interval (with corresponding Z value of 1.55) and allowing a 6% error, the sample size was calculated.

For the Objective:

p = 0.29

q = 0.71

Substituting values in the above equation, the Sample size (N0) = **137**

137 was the minimum amount of sample size required to fulfill the desired level of precision.

In this study, 150 was the final total sample size.

Method:

- Patients who have been diagnosed with diabetes mellitus within the last 1 year of their OPD visit based on the WHO criteria, were asked to participate in the study.

As per the World Health Organisation (WHO) (6) Diabetes can be diagnosed based on any of the following four tests :

HbA1C	>= 6.5%
Fasting Blood Sugar	>= 126 mg/dL
2 hour plasma glucose during 'Oral Glucose Tolerance Test'	>= 200 mg/dL
Random plasma glucose	>= 200 mg/dL with classic symptoms of hyperglycemia and hyperglycemic crisis

Table 6: WHO criteria of diagnosing diabetes mellitus

- Informed consent was taken.
- A detailed history was taken and general examination was performed.
- The Diabetik Minilab machine available in our surgery OPD was used to examine all patients for Peripheral Neuropathy, Vascular changes and Plantar foot pressures during their OPD visit.

Figure 9: Diabetik Minilab machine

- The following components from the Diabetik Minilab were used to assess the patient:

    1. For Peripheral Neuropathy:

        - 10g monofilament test
        - Vibrotest Digital Biothesiometer

    2. For Vasculopathy ABI was calculated using:

        - Unidirectional 8MHz Doppler probe

- Manual Blood Pressure Apparatus

3. For Plantar Pressure:

    - Foot Imprinter Harris Mat FM1111
    - Software to scan greyscale image into Multi colour image

PROCEDURE:

1. All patients diagnosed with type 2 diabetes within the past 1 year of their visit underwent assessment of Peripheral Neuropathy, Plantar foot pressures and Vascular changes and using the Diabetik Minilab during their OPD visit as a part of routine examination.

2. To assess neuropathy, 2 tests were conducted on the following 6 sites of the plantar surface of the foot –

    1. The great toe
    2. 1st metatarsal Head
    3. 3rd metatarsal head
    4. 5th Metatarsal head
    5. Arch of foot
    6. Heel

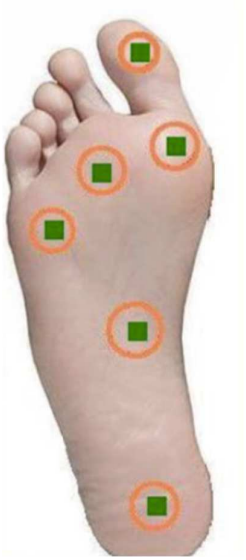


Figure 10: Sites checked over the plantar aspect of the foot

- **10g Monofilament Test:** With the patients unable to see their feet, the monofilament is placed on the plantar surface of the foot at right angles to the skin on the 6 sites mentioned above. The pressure is increased only until the filament buckles (Fig). The patients were asked to say 'yes' when they felt the tip of the monofilament at the respective sites. The foot was said to have loss of protective sensation when 4 or less than 4 sites were felt by the patient. If four or more sites are felt, then the foot is normal. The results are displayed on the screen as a diagrammatic representation of the foot and whether sensation at a particular area are present or not.

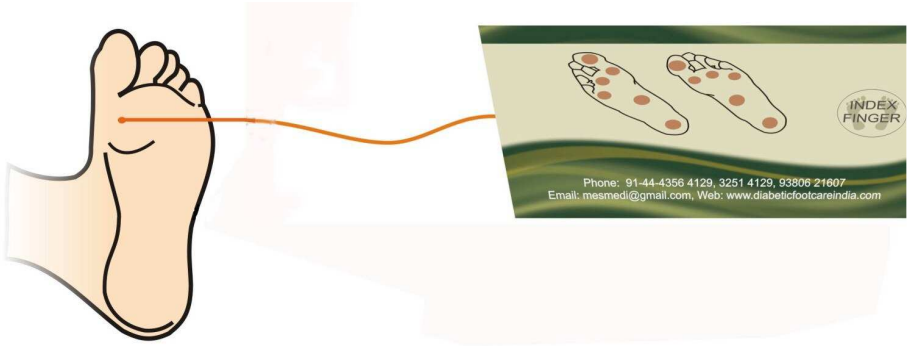


Figure 11: Procedure for the 10g monofilament test

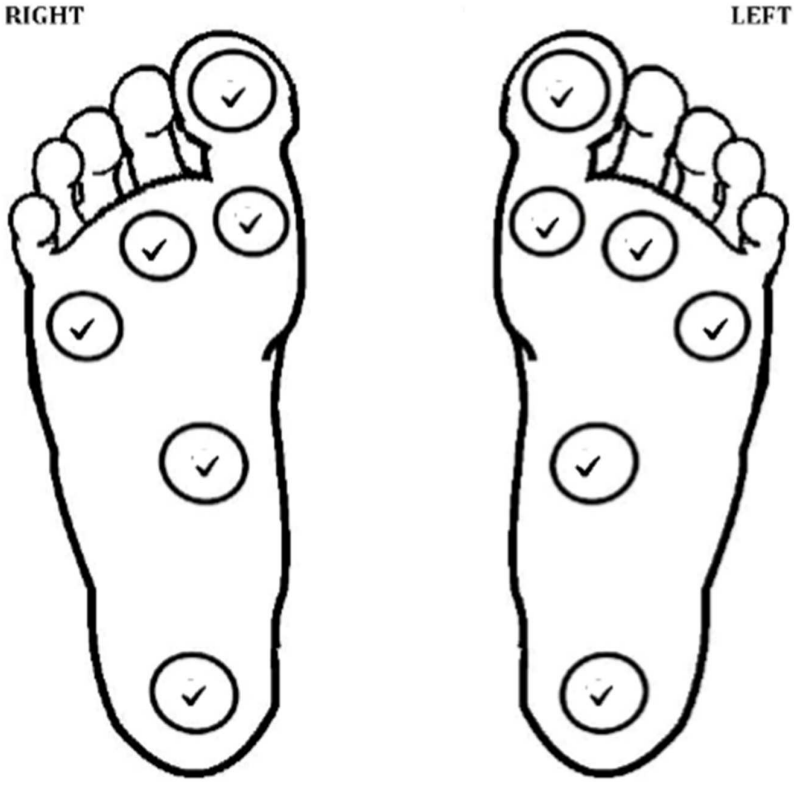


Figure 12: 6 points over the sole where the monofilament was tested

- **Vibration Perception Test (VPT):** With the patients unable to see their feet, the Vibrotest Digital Biothesiometer is probed at the aforementioned mentioned six sites over the sole. A digital volt indicator provides voltage from 0-50V at each site and the voltage at which the patient feels the vibration first is recorded. The average voltage of the six sites is calculated. An average VPT value of >25 V in at least one foot has been associated with a high cumulative risk of neuropathic ulceration.

Figure 13: Vibrotest Digital Biothesiometer probe

Results are displayed on the screen as a diagrammatic representation of the foot with the voltage reading at each site and the average voltage of each foot.

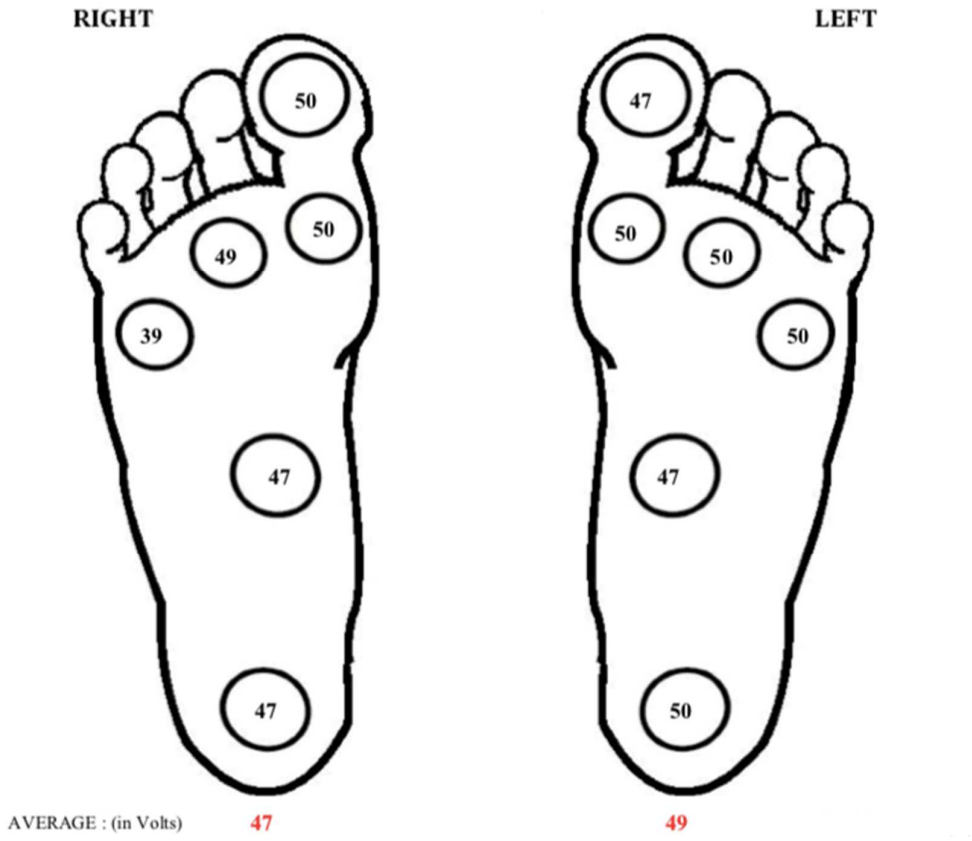


Figure 14: Diagrammatic representation of the voltage recorded at each site over the sole and the average voltage of each foot highlighted in red.

2. To assess **Plantar Pressure**, the Foot Imprinter Harris Mat FM1111 is used.

- Dynamic foot pressure is recorded by asking the patients to casually walk over the mat with each foot. The footprint is then scanned to a software in the Diabetik Minilab which converts the greyscale image into a coloured pictorial representation of different pressures found at different sites on the foot.

- Any Foot having more than 4 or more areas of High / Very high pressure were considered as those having increased plantar pressure.

- The software categorizes the pressure levels using the following colour codes:

    - Red – Very high pressure
    - Orange – High pressure
    - Yellow – Mild Pressure
    - Green – Normal pressure
    - Blue – Less pressure
    - White - No contact area

Figure 15: Procedure to get a black and white foot imprint using the Harris Mat

This imprint is then scanned into the Diabetik minilab software, where it is converted to a coloured pictorial representation of the plantar pressure.

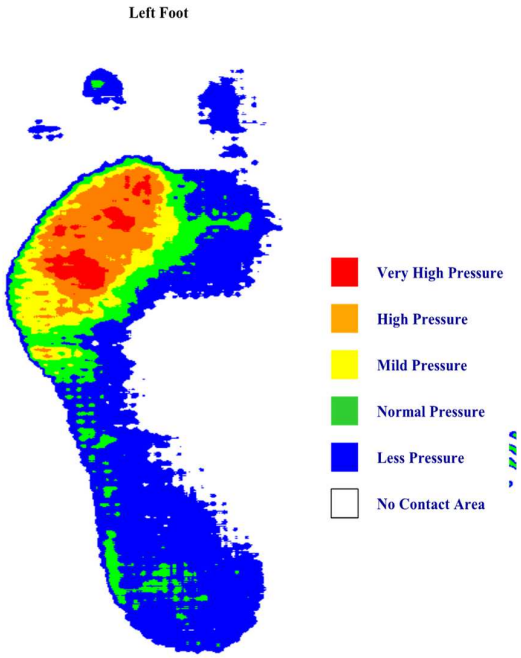


Figure 16: Coloured representation of the foot print stratified into zones of pressure by colour

3. To assess **Peripheral Arterial Disease**, the ABI index is calculated.

- A sphygmomanometer was placed at the level of the ankle. A doppler probe was placed at a 45 degrees angle over the point at which the Dorsalis Pedis pulsations were palpated i.e lateral to the tendon of extensor hallucis longer against the navicular bone. Once a wave was seen, the sphygmomanometer cuff was inflated till the signal disappeared and slowly deflated till it returned. At this point, the pressure was recorded. The doppler probe was then placed over the anatomical site of the Posterior tibial artery (i.e between the medial malleolus and tendoachilles) and the procedure was repeated with the sphygmomanometer. The same was repeated for the other lower limb and with the brachial artery for both the upper limbs.

- The ABI was then calculated for each foot by dividing the higher of the two ankle pressures (i.e Dorsalis pedis and posterior tibial artery) divided by the brachial pressure.

$$\text{ABI} = \frac{\text{Higher of the two Ankle pressures (Dorsalis Pedis Artery OR Posterior Tibial Artery)}}{\text{Brachial Pressure}}$$

Normal ABI is 1.10 ± 0.10

ABI of **< 0.9** is used as a cut-off value as a marker for PAD.

Results were displayed on the screen as follows:

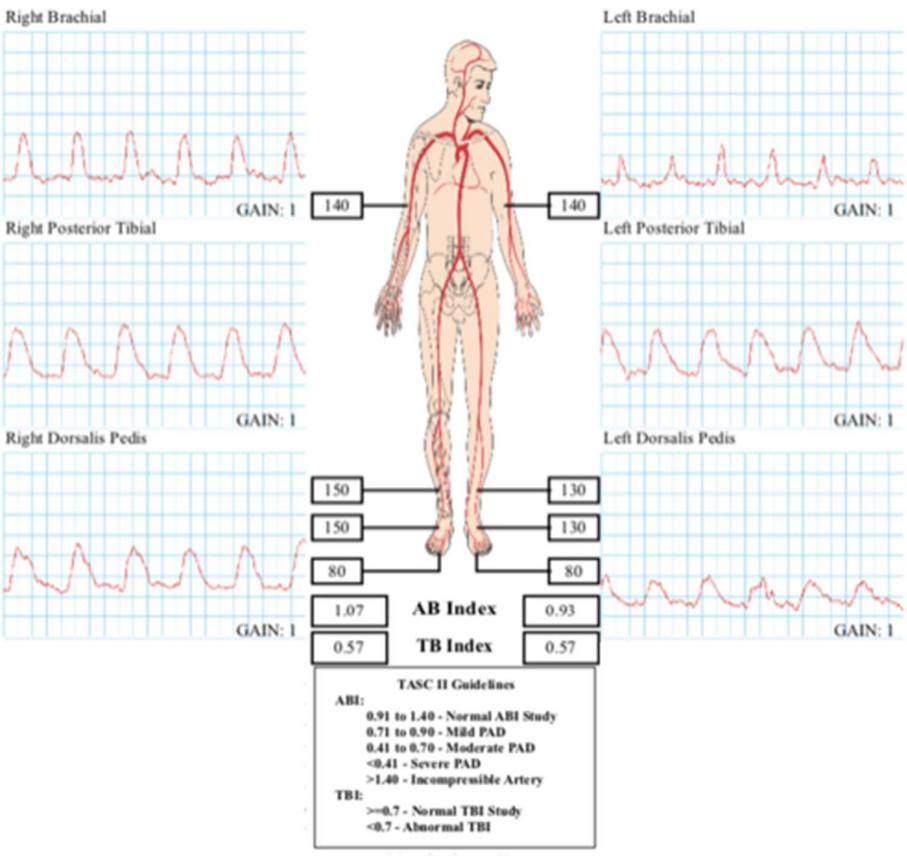


Figure 17: Representation of doppler wave forms at the Posterior Tibial, Dorsalis Pedis and Brachial Artery and the calculated ABI

STATISTICAL TOOLS USED IN THE ANALYSIS OF DATA:

All the data collected from the patients was coded and tabulated in a Microsoft excel spreadsheet. The data was statistically analyzed.

The categorical data was tabulated in terms of frequencies (Numbers) and percentages.

For quantitative variables mean, standard deviation, minimum value and maximum value were calculated.

For comparison of data, student's unpaired t test was used and a value of p less than 5% (0.05) was considered as significant.

Kappa statistics were calculated to check the agreement between the two methods of assessment of neuropathy by Vibration Perception Test and by monofilament method.

RESULTS

This study was conducted in KLE'S Dr. Prabhakar Kore Hospital and Medical Research Centre, Belagavi, from January 2021 to December 2021.

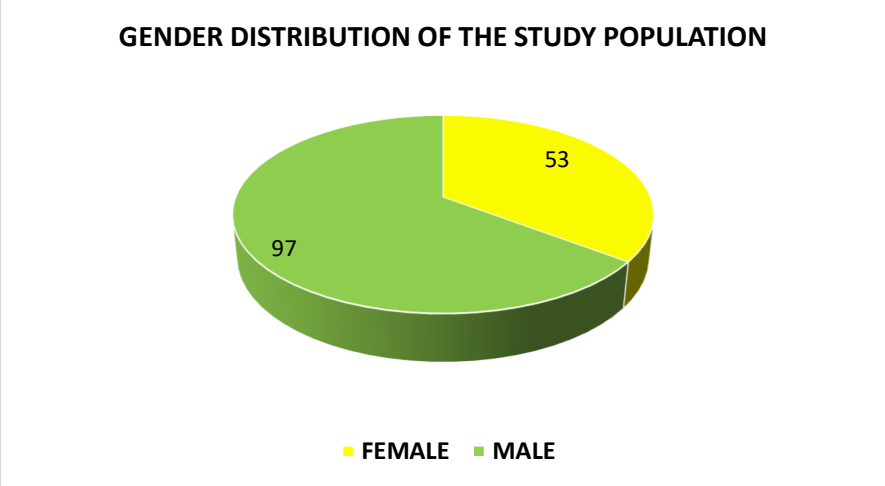
A total of 150 patients who were diagnosed with Type 2 Diabetes Mellitus in the last 1 year from the day they presented to the Out Patient Department were included in this study.

Data collected was typed into spreadsheets of Microsoft excel. The data was analysed and the results obtained were tabulated as represented below.

'P' value less than 0.05 considered significant statistically.

1. **Gender distribution in the study**

GENDER	NUMBER	%
FEMALE	53	35.33
MALE	97	64.67
TOTAL	150	100.00

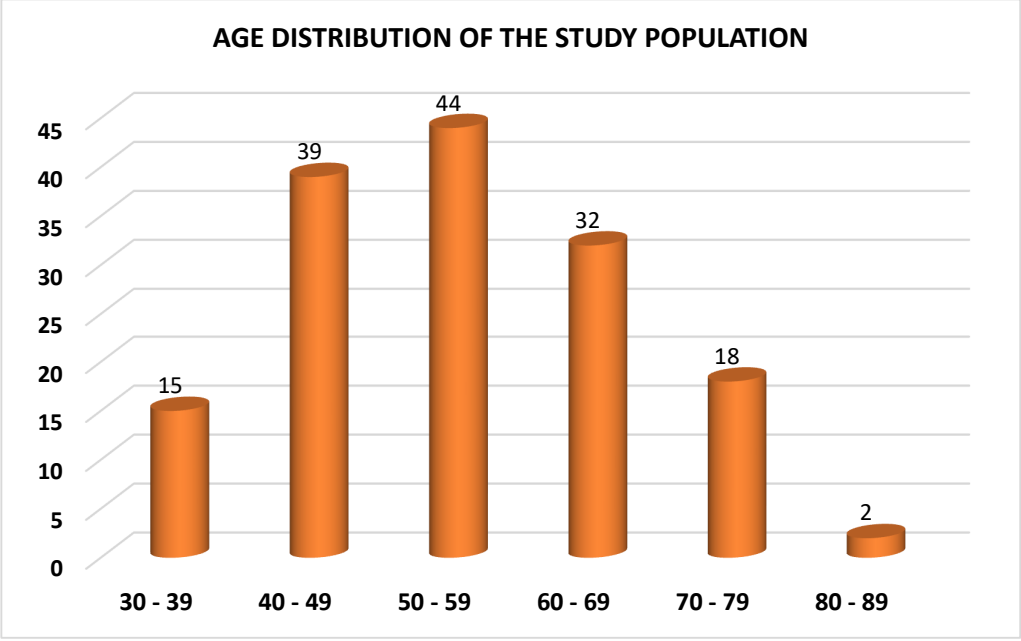


Graph 2: Gender distribution of the study population

Out of 150 study participants, 97 were males and 53 were females.

2. Age Distribution of the study population

AGE	NUMBER	%
30 - 39	15	10.00
40 - 49	39	26.00
50 - 59	44	29.33
60 - 69	32	21.33
70 - 79	18	12.00
80 - 89	2	1.33
TOTAL	150	100.00



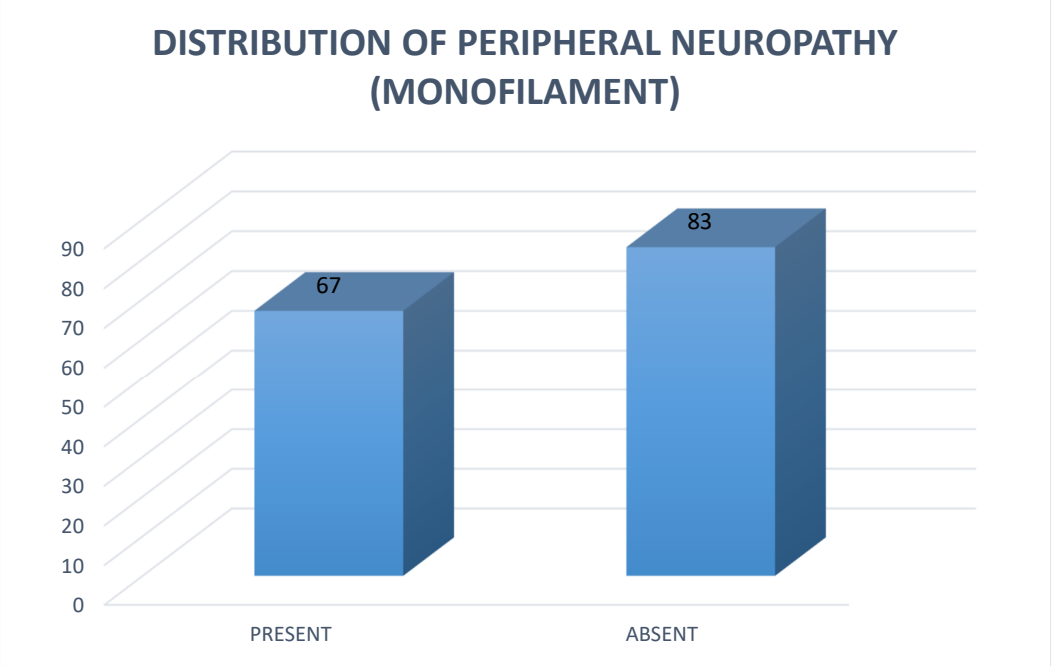
Graph 3: Age distribution

	MEAN	S.D.	MIN	MAX
AGE (yrs)	54.74	11.88	35	82

The maximum number of study participants belonged to the age bracket of 50-59yrs.

3. Distribution of peripheral neuropathy as per 10g Monofilament test amongst the study population

NEUROPATHY	NUMBER	%
PRESENT	67	44.67
ABSENT	83	55.33
TOTAL	150	100.00

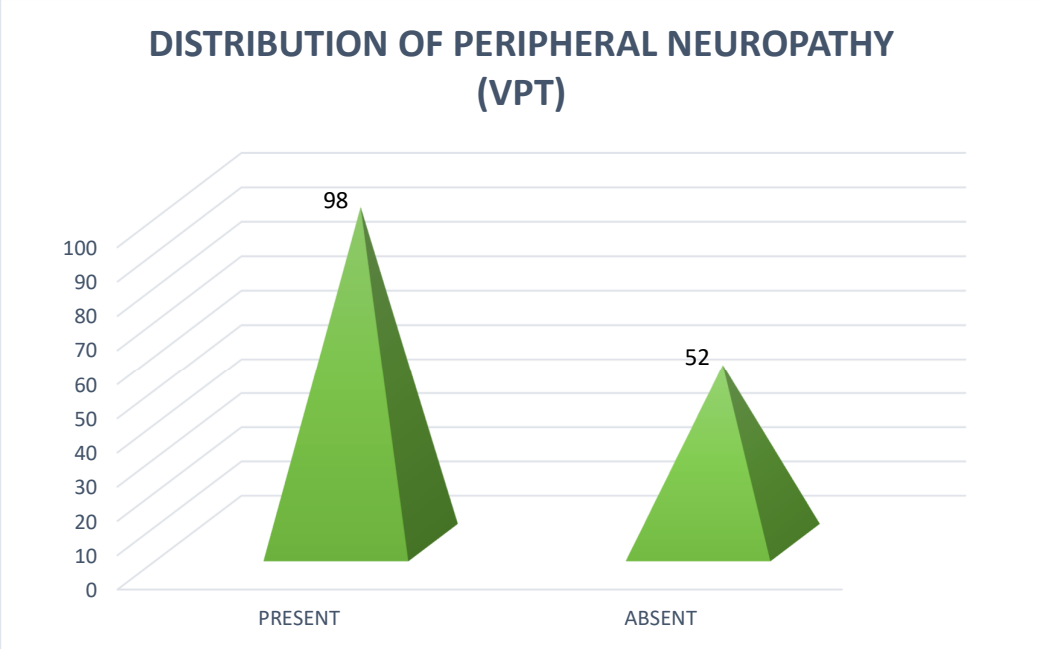


Graph 4: Distribution of peripheral neuropathy as per 10g Monofilament test

ACCORDING TO THE 10G MONOFILAMENT TEST, THE RATE OF PREVALENCE OF PERIPHERAL NEUROPATHY IN THIS STUDY IS 44.67%

4. Distribution of peripheral neuropathy as per Vibration Perception test (VPT) amongst the study population

NEUROPATHY	NUMBER	%
PRESENT	98	65.33
ABSENT	52	34.67
TOTAL	150	100.00



Graph 5: Distribution of peripheral neuropathy as per Vibration Perception test (VPT)

ACCORDING TO THE VIBRATION PERCEPTION TEST, THE RATE OF PREVALENCE OF PERIPHERAL NEUROPATHY IN THIS STUDY IS 65.33%

5. Degree of agreement between the 10g monofilament test and the vibration perception test used for evaluating neuropathy

	NEUROPATHY	BY MONOFILAMENT		
		PRESENT	**ABSENT**	**TOTAL**
BY VPT	**PRESENT**	63	35	98
	ABSENT	4	48	52
	TOTAL	67	83	150

From the above table we can infer that out of 98 people who showed signs of neuropathy via the vibration perception test, only 63 were also positive for neuropathy via the monofilament test. Also, out of the 67 people who tested positive for neuropathy by the monofilament test, 63 gave a positive vibration perception test as well.

To determine the degree of agreement between the two tests, the kappa value is calculated.

For the above table the value of kappa is 0.1465 with $p < 0.0001$ (highly significant). Hence, it can be said that there is a moderate agreement between the outcomes of the vibration perception test and monofilament test.

While the results from the vibration perception test alone showed a higher prevalence of neuropathy as compared to the 10g monofilament test, the combination of both these tests is proven to be highly sensitive towards the diagnosis of neuropathy.

6. Distribution of Plantar pressure amongst the study population

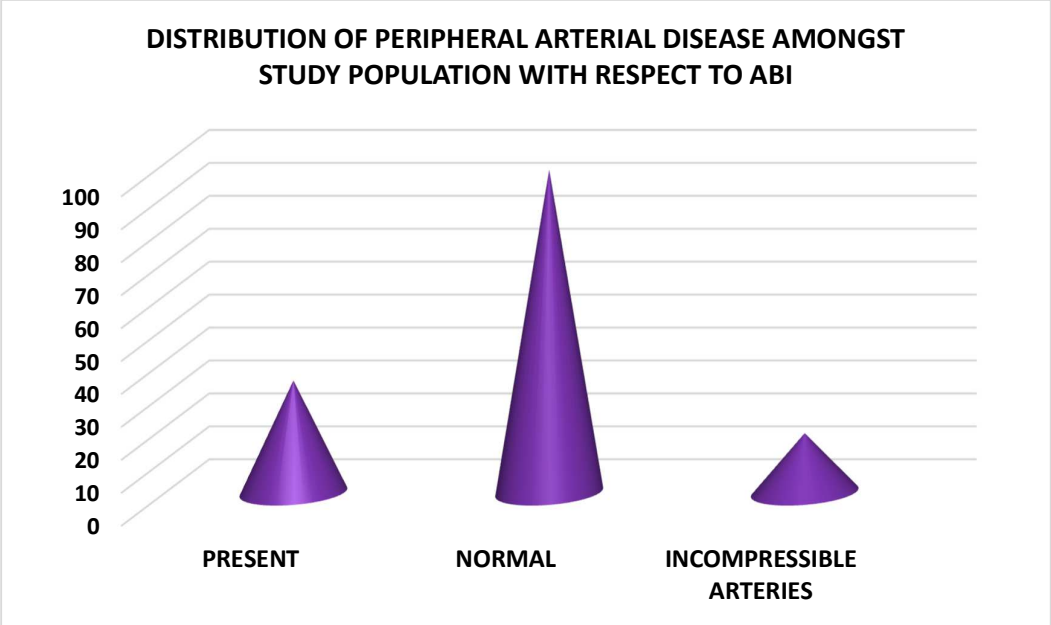
TYPE	NUMBER	%
NORMAL	93	62.00
INCREASED	57	38.00
TOTAL	150	100.00

Graph 6: Distribution of Plantar pressure

THE RATE OF PREVALENCE OF INCREASED PLANTAR PRESSURE IS 38.00% AMONGST THE STUDY POPULATION.

7. Distribution of Peripheral Arterial Disease as calculated by the Ankle Brachial Index (ABI) amongst the study population:

TYPE	NUMBER	%
PERIPHERAL ARTERIAL DISEASE	34	22.67
NORMAL	98	65.33
INCOMPRESSIBLE ARTERIES	18	12.00
TOTAL	150	100.00



Graph 7: Distribution of Peripheral Arterial Disease

According to the Ankle Brachial Index, 22.67% of the study population had peripheral arterial disease while 65.33 % had a normal value. 12% of the population had evidence of incompressible arteries as per the ABI.

8. A) Distribution of study population with respect to HbA1C

In the above study out of a total of 150 participants, the evidence of Peripheral Neuropathy, Peripheral Arterial Disease and Increased Plantar Pressure was present in **25** patients. The remaining 125 patients had a combination of any of the 2 parameters present.

Out of 150 participants, 25 participants account for **16.67%** of the study population.

The mean HbA1C in the study was 8.53%

HbA1c FOR ALL THE 150 CASES			
MEAN	**S.D.**	**MIN**	**MAX**
8.53	1.18	6.5	11.4

B) The following table shows the mean HbA1C value for the aforementioned 25 patients as compared to the remaining 125 study population:

	MEAN	**S.D.**	**MIN**	**MAX**	**p VALUE**	**INFERENCE**
HbA1c FOR THE 25 CASES	9.41	1.22	7	11.4	< 0.0001	HS
HbA1c FOR THE REMAINING 125 CASES	8.36	1.09	6.5	11.3		

The table above shows that out of the 150 participants, the HbA1C for the 25 patients with Peripheral Neuropathy, Increased Plantar Pressure and Peripheral Arterial Disease was significantly higher as compared to the HbA1c of the remaining 125 participants.

With the p value being < 0.0001, the association of HbA1c with the development of all 3 parameters i.e Peripheral Neuropathy, Increased Plantar Pressure and Peripheral Arterial Disease is highly significant amongst the study population.

DISCUSSION

The prevalence of diabetes has reached an alarming level. As per the 10th edition of the International Diabetes Federation, currently approximately half a billion people worldwide are suffering from diabetes (4)

While the general population is aware of the disease, the knowledge about the spectrum of its consequences is limited. Poorly controlled diabetes can lead to dramatic side effects of hyperglycemia, namely peripheral neuropathy, changes in distal vasculature and ultimately a change in the complete architecture of the foot. Progression of the disease eventually leads to non healing ulcers requiring regular dressings with frequent visits to the out patient department. In addition, with compromise to peripheral blood supply patients can end up with amputations of varied levels. These consequences alter the lifestyle of these patients and not only cause significant morbidity to them, but also act as an economic burden on their families and care givers.

In India, 77 million people were estimated to have diabetes in 2019, and by 2045, that number is projected to reach over 134 million.(29) Even though the sequalae of diabetes mellitus occur as the disease progresses over a period of time, in India, there is a delay in the diagnosis of the disease itself (30). In a study conducted in Bangalore by *Rayappa et al.*, there was a ten-year gap in the age of diagnosis between actively working and non-working respondents, a seven-year gap between the most educated and the least educated, and a four-year gap between the highest and lowest socioeconomic groups.(31) The latter half of the people who have a delay in their diagnosis usually present with a variety of symptoms such as lower limb cramps at night, non healing ulcers or recurrent trivial wounds. This already implies that the onset of the Diabetes had begun a long time ago.

Research in the field of diabetes is dynamic, vast and ever growing. However, it is still not known when the onset of neuropathic or vascular changes really begin after a patient has been diagnosed with the disease. Even though the onset is thought to be after a few years of the diagnoses, in most instances by the time the patient gets diagnosed, they suffer from symptoms of neuropathy or vasculopathy already. Hence, foot care and education needs to be at the forefront in the management of diabetes.

This study is an attempt to create awareness amongst medical health care professionals as well as the general population suffering from diabetes about the urgency that is The Diabetic Foot. With prompt diagnosis, we can initiate foot care and education early and potentially reduce the burden of the diabetic foot complications.

This case series was executed under the department of general surgery at KLES Dr. Prabhakar Kore Charitable Hospital and Medical Research Centre, Nehru nagar. Belagavi, between January 2021 to December 2021. A total of 150 patients who satisfied the selection criteria and were willing to participate comprised the study population.

All of them were subjected to a thorough diabetic foot scan using the Diabetik Minilab as a part of their routine physical examination.

The incidence of complications is anticipated to rise in tandem with the prevalence of diabetes. One of the most significant complications of Diabetes is Diabetic Peripheral Neuropathy, which is brought on by consistently elevated glucose levels. For diabetic patients, this issue is a significant contributor to their impairment and poor quality of life. Additionally, it has been linked to a rise in foot ulcer incidence. According to reports, those with DPN have amputations 10–20 times more frequently than those without the disease.(15)

Peripheral neuropathy behaves as a silent form of the progression of diabetes mellitus. Its symptoms may often go unnoticed to the common man and the presenting complaint to the clinic is usually that of repeated trivial trauma to the sole of the foot which the patent only notices once it doesn't heal for prolonged periods of time. Little is known about when diabetic people who do not have diabetic foot problems (DFP) acquire sensory neuropathy. A study by *Nather e*t al showed that 15.9% of patients with a duration of diabetes of < 5 years had sensory neuropathy(2). In another study by *Ratzmann et al*, peripheral neuropathy was evaluated in newly diagnosed diabetics within only 10 days of their diagnosis. In their study, there was a reduction in vibration perception in 80% of their participants.(14)

In this present study, out of the 150 population, according to the vibration perception test and 10g monofilament test the rate of prevalence of peripheral neuropathy was 65.33% and 44.67% respectively. This result is in concordance with the aforementioned studies. As per text by *Anandhanarayanan et al*, the most sensitive assessment for neuropathy is vibration perception while the 10g monofilament is sensitive upto 86-100% in screening for neuropathy. They further mention that a combination of both tests can increase diagnostic sensitivity upto 87%. This agrees with the present study as even in our results, the prevalence of neuropathy was higher with vibration perception (65.33%) versus the 10g monofilament (44.67%). In this study the degree of agreement between the 10g monofilament test and the vibration perception test was used for evaluating neuropathy. With a kappa value of 0.1465 and p value being <0.0001, it was concluded that there was moderate degree of outcomes between the two tests, meaning that the combination of the two were proven to be highly sensitive towards the diagnosis of neuropathy. This too is in agreement with the text by *Anandhanarayanan et al.* (32)

With simple tools such as the SWME and Vibration perception testing, we could determine the status of neuropathy when patients are diagnosed with type 2 diabetes mellitus. This information can in turn be used as objective evidence to educate patients regarding the importance of foot care and glycemic control which could before sensory neuropathy sets in. With early intervention, the sequalae of diabetic neuropathy can be avoided.

The processes that are responsible for the rise in plantar pressure caused by peripheral diabetic neuropathy have been well described. One of the causes is an abnormality in proprioception, which manifests itself as an imbalance between the long flexors and extensors of the toes (33). This condition, in its most severe form, results in claw toes and large metatarsal heads. Clawing of the toes is accompanied by an anterior shift of the fat pads located on top of the submetatarsal head (34). Because of these structural alterations, neuropathic feet experience greater supinatory moments, which is accompanied by an increase in pressure under the 4th and 5th metatarsal heads. These sorts of shifts in plantar pressure could happen before the clinical manifestation of peripheral neuropathy.

Stokes et al. reported one of the earliest investigations on plantar pressure in diabetic patients in 1975. Not only did he discover that patients who had an active ulcer had the highest possible load, but he also saw, in comparison to normal subjects, a lateral shift in pressure, which manifested itself as an increased amount of pressure under the 4th and 5th metatarsal heads.(35)

In our study, the rate of prevalence of increased plantar pressure is 38% amongst the target population *Lei et al,* noted that peak pressures at the hallux were

at an upward trend within 5 years of diagnosis of diabetes (36), although *Anjos et al* reported that it takes atleast 10 years for plantar pressures to increase in diabetics.

In another study Pataky et al discuss how plantar pressure abnormalities were found even in patients without signs of neuropathy, indicating that raised plantar pressure in itself is an indicator of future loss of protective sensation.(37)

When it comes to arterial disease, extensive research has been done in the relationship of diabetes with cardiovascular and cerebrovascular events. However its relation with peripheral arterial disease is comparatively limited. (24) Peripheral arterial disease (PAD) is a condition that frequently affects younger people and arises in 10% of diabetics that have just been diagnosed.(38) Because of this, it is essential to have an easy and speedy method for determining whether or not someone has vascular disease.

As per the 2016 AHA/ACC Guideline on the Management of Patients With Lower Extremity Peripheral Artery Disease(25) the initial diagnostic test for PAD is called the Ankle Brachial Index and it is possible that it is the only test that is required to establish the diagnosis.

In our study amongst the 150-study population, 22.67% had an ABI of <0.9 suggestive of peripheral arterial disease while 65.33 % had a normal value. 12% of the population had evidence of incompressible arteries as per the ABI of >1.3. While the percentage of patients with PAD is underwhelming, it is in agreement with a study by Chevtchouk et al which suggests that PAD occurs in 10% of diabetics that have just been diagnosed(38). Medial arterial wall calcification limits the use of ABI and an ABI > 1.30 in 12% of our patients was a strong indicator of calcification.

Many people who have diabetes don't have many symptoms because, despite the massive loss of vascularized tissue, distal sensory polyneuropathy often leads to a loss of sensation. This is the most prevalent complication of diabetes. Mortality and morbidity due to artery disease is a common reason for hospitalization, and patients admitted with this condition have a 15–25% chance of developing ulcers.

Due to the fact that vascular disease, which is a risk factor regardless of ulceration and amputation, is present in fifty percent of patients who have ulcers, early diagnosis is of utmost concern. (38)

Another study by *Khalil et al* showed that 9.8% of their study population showed evidence of Peripheral arterial disease and 10.4% had diabetic kidney disease at the time of diagnosis itself indicating that microvascular complications and peripheral arterial disease are present at the time of or even years before diagnosis of diabetes mellitus. (39)

Plantar pressure distribution has been the subject of a significant amount of research in diabetic neuropathy (DN) patients over the past several decades due to its connection to an increased risk of tissue breakdown and plantar ulcer formation. (40) An increase in plantar pressure is a pre-requisite for ulcer formation in diabetics. More often than not it is found that ulcers over the plantar aspect are found over the heel, the ball of the great toe or head of the first meta tarsal. This is due to increased weight bearing over these areas.

The present study was conducted amongst 150 patients out of which 25 had signs of peripheral neuropathy, increased plantar pressure as well as peripheral arterial disease. Moreover, the mean HbA1c of these 25 patients was 9.41, as compared to the

remaining 125 patients whose mean HbA1c was 8.36. This was statistically significant, proving that patients with uncontrolled glycemic control are more likely to have all three sequalae of diabetes.

A study by *Su et al* also discusses that short term variability in HbA1c is an independent contributor to diabetic peripheral neuropathy and that long term poor glycemic control is a contributing factor to loss of protective sensation.(41)

The relationship between peripheral arterial disease and HbA1c has been well established and agrees with our study. Selvan et al (42) and Wilbert et al (43) successfully conclude in their studies that higher the glycosylated haemoglobin, more is the degree of peripheral arterial disease amongst the patients.

As per literature, there is no direct relationship between HbA1c and plantar pressure which has been established. The HbA1c, which is used as a surrogate for glycemic control, did not have any direct impact on peak planter pressure; however, it did have an indirect impact on the progression of neuropathy during the length of the disease, which ultimately led to significant alterations in planter pressure and gait biomechanics.(44) Hence, it can be inferred that due to increased HbA1c values there is significant neuropathy in patients which ultimately leads to increased plantar pressure.

This study was conducted to prove that diabetic foot sequalae are not always a function of the duration of the disease, but are present even at the time of diagnosis itself. In a country like India, where diabetes is considered a major non communicable disease with significant impact on a patients social and economic life, this study hopes to enlighten not only the patients and care givers but also medical professionals about the utmost importance about foot care and glycemic control. Moreover, with this

study we demonstrated that with simple tests which can be done in the out patient department, it is possible to screen for neuropathy, PAD and plantar pressures.

Diabetic foot is a common cause of hospitilzation in India and the lifetime risk of a diabetic patient developing an ulcer is almost 15-20%.

Ulceration of the foot caused by diabetes causes physical and emotional anguish, as well as a loss of productivity and financial loss, which all contribute to a decrease in overall quality of life. Ulcers are complicated by impaired healing and wound infection, which puts patients at a higher risk for osteomyelitis, gangrene, and ultimately limb amputation.

This study is the first of its kind wherein attempts to evaluate neuropathy, arterial disease and plantar pressure in patients diagnosed with diabetes within 1 year was done. There was a prevalence of 65.3%, 22.6% and 38% of neuropathy, peripheral arterial disease and raised plantar pressures respectively amongst the 150 study population. With this study we hope to provide objective evidence to patients and medical professionals regarding the early onset of the same and that with prompt screening and early diagnosis timely treatment can be initiated to prevent these sequalae.

LIMITATIONS OF THE STUDY

This was a case series which is a limitation and a comparison group of patients with different durations of diabetes would improve the statistical value of this study. Considering the number of patients being diagnosed with diabetes, the sample size and duration of this study is relatively less. Also, the lack of follow up in this study prevents us from assessing whether the patients are adhering to advice following the foot examination. A follow up of every 2 months initially and then 6 monthly could help us note whether the application of this study of diabetic foot care and patient education is successful amongst patients.

CONCLUSION

From this study, we conclude that peripheral neuropathy, raised plantar pressure and peripheral arterial disease are found even in patients diagnosed with type 2 diabetes mellitus within the last one year. More over, the prevalence of these parameters is higher in patients with raised Glycosylated haemoglobin values.

With this study, we also highlight the ease and importance of timely screening of patients on an out patient basis. With prompt diagnosis of neuropathy and signs of peripheral arterial disease, we can begin intervention at the earliest and prevent morbid sequalae like non healing ulcers, gangrene and amputation.

In the future, a larger study cohort and a comparison of these parameters with different duration of diabetes could be more helpful in clinical practice.

SUMMARY

Diabetes Mellitus is a chronic progressive disease and persistent hyperglycemia can lead to sequalae which cause significant morbidity to the patient. Diabetes associates peripheral neuropathy, raised plantar pressures and peripheral arterial disease lead to complications of the diabetic foot such as non healing ulcers, gangrene and amputation.

The goal of this study is to identify these changes at their onset, so that appropriate treatment can be initiated at the earliest in order to reduce long term morbidity caused to the patients.

This study was conducted on 150 selected patients who visited the surgery outpatient department in KLE Dr.Prabhakar Kore charitable hospital and MRC, Belagavi.

All patients diagnosed with type 2 diabetes within the past 1 year of their OPD visit underwent assessment of Peripheral Neuropathy, Plantar foot pressures and Vascular changes using the Diabetik Minilab machine as a part of routine examination.

Neuropathy was assessed with the 10g monofilament test and the vibration perception test. Vascular changes were determined by calculating the Ankle Brachial Index and plantar pressures were evaluated using a Harris beath mat. The categorical data was tabulated in terms of frequencies and percentages. 'P' value less than 0.05 considered significant statistically.

A total of 150 patients were included in this study out of which 97 were males and 53 were females. Peripheral neuropathy was detected in 44% and 65% by the monofilament and vibration perception test respectively. Plantar pressure was increased in 38% and as per the ankle brachial index 22% had peripheral vascular disease.

Out of the 150 participants, 25 patients (16%) showed all 3 changes and when their mean HbA1C values (9.41%) were compared with the remaining 125 patients (8.36%), it was not only found to be higher but was also statistically significant.

Structural and pathological foot changes in diabetics are thought to occur as a function of the duration of the disease. Patients are unaware of the need and type of foot care which is required. Moreover they too are under the impression that these changes take place after suffering from the disease for several years. However, with the above results we can provide objective evidence to these patients at the time of their diagnosis itself regarding the need for appropriate foot care, foot wear and follow up.

Type 2 Diabetes is a leading cause of foot ulceration, gangrene and amputation when not managed properly. With the help of simple tests, this study hopes to spread awareness about early diabetic foot changes so as to reduce its morbid sequelae.

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ANNEXURE I

CONSENT FOR PARTICIPATION IN RESEARCH STUDY

Mr/Mrs/Miss. _____, we are requesting you to participate in study titled **"ASSESSMENT AND EVALUATION OF PERIPHERAL NEUROPATHY, VASCULAR CHANGES AND PLANTAR FOOT PRESSURES IN PATIENTS DIAGNOSED WITH TYPE II DIABETES WITHIN THE LAST 1 YEAR – A CROSS SECTIONAL STUDY**", conducted by **REG NO: BH0120012**, Post Graduate in M.S. General Surgery under the guidance of Dr. _____, Professor & Head, Department of General Surgery, J.N. Medical College, Belagavi under KAHER, Belagavi.

Respected Sir/Madam,

We request you to participate in our study. Your participation in the research is voluntary. Your decision to participate in the study or otherwise will not affect the relationship with KLES Prabhakar Kore Hospital. If you decide not to participate, you are free to withdraw at any time. During the study you will be asked some questions to aid with the evaluation.

Purpose of the study:

The purpose of this study is to-

1. To assess and evaluate peripheral neuropathy, plantar foot pressure and vascular changes in patients diagnosed with Type II diabetes within the last one year.

The principal investigator of the study is REG NO: BH0120012, under the guidance of Dr._____.

Procedure Involved:

If you agree to enroll yourself in this study, your detailed history will be taken and you will be clinically examined in detail. You will undergo a complete analysis of peripheral neuropathy, vascular changes and plantar pressure using the Diabetik Minilab device. To assess neuropathy, the Monofilament Test (SWMT) and Vibration Perception Test (VPT) will be performed. For vascular changes, the Ankle-Brachial Index is calculated using a sphygmomanometer and a continuous wave form doppler. For plantar pressure, the Foot Imprinter Harris Mat FM1111 and the diabetik minilab software is use. All the data will be collated and printed in a systematic manner which will be handed over to you.

Risks and Benefits:

There is no increased risk involved in being a part of this study. This study will help to assess and evaluate if you have any neuropathic, vascular or plantar pressure changes due to diabetes. It will guide us with further therapy and to initiate appropriate intervention. This study may help similar patients elsewhere.

Withdrawing/removal from the study:

The participant has freedom to withdraw from the study whenever he/she wishes and without any prior notice. Even if you decline to participate, there will not be any change in the line of your management or the relationship with your doctor. You will be told about all the information that affects your decision to participate in the study. The investigator may also exclude a participant from the study at any point of time.

Privacy and Confidentiality:

The only people to know that you are a research subject are members of the research team. No information about you or information provided by you during the research will be disclosed to other without your written permission except:

1. In emergency to protect your rights and welfare.
2. If required by law.

Institutional/sponsors policy:

If any unforeseen complications or injury occurs during the period of study, the participant will be given treatment within the limitations of KLES Prabhakar Kore Hospital.

Financial Incentives for participation:

The participant neither gets any financial incentives during the period of study nor will be asked to pay for this study.

Authorization to Publish Results:

When the results of the research are published, or discussed in a conference, no information will be displayed that would disclose your identity. Any information that is obtained in this study that can be associated with your identity will remain confidential.

In case the participants have any queries related to the study, they can contact REG NO: BH0120012, Dept of General Surgery, KLES Hospital and MRC, Belagavi, or Dr. _____., Professor & Head, Department of General Surgery, KLES Hospital and MRC, Belagavi. In case they have any questions regarding their rights as a study subject, they may contact Dr. Harsha Hegde, Chairperson, JNMC, IEC & Scientist D, ICMR, National Institute of Traditional Medicine.

CONSENT STATEMENT

I, Mr/Ms/Mrs. _____ voluntarily agree for the participation as a subject of study. By signing this consent form, I am not giving up any of my legal rights. I may withdraw from the study anytime. I am signing the consent form after having read or been read for me in my vernacular language, including the risks and the benefits and having all my questions answered.

Subject Name : _____

Signature or Left Thumb Print of Subject : _____

Witness Name: _____ Signature: _____

Investigators Name: _____ Signature: _____

Date: _____

Place: _____

ANNEXURE II

PROFORMA

DATA COLLECTION INSTRUMENT

ASSESSMENT AND EVALUATION OF PERIPHERAL NEUROPATHY, VASCULAR CHANGES AND PLANTAR FOOT PRESSURES IN PATIENTS DIAGNOSED WITH TYPE II DIABETES MELLITUS WITHIN THE LAST 1 YEAR – A CROSS SECTIONAL STUDY

Screening number: ☐☐☐☐

Date of Data Collection(dd-mm-yyyy): ☐☐ ☐☐ ☐☐☐☐

First name : ☐☐☐☐☐☐☐☐☐

Last name : ☐☐☐☐☐☐☐☐☐☐

Age (years). : ☐☐

History of Symptoms before diagnosis of Diabetes:

(Y= yes, N= no)

Polydipsia ☐

Polyphagia ☐

Polyuria ☐

Poor wound healing ☐

Family History of Diabetes – Yes / No

If yes, relation to family member-

General Examination-

Height (in cms) – ☐☐☐

Weight (in kgs)- ☐☐☐

Body Mass Index - ☐☐.☐

Blood pressure (mmHg) ☐☐☐ / ☐☐

Pulse rate (beats per minute) ☐☐☐

Peripheral Pulses :

(Present : + ; Absent : -)

Peripheral Pulse Location	Right Limb	Left Limb
Posterior Tibial		
Dorsalis Pedis		

INVESTIGATIONS:

Diabetes was diagnosed using – FBS / PLBS / Glycosylated Hemoglobin

FBS (mg/dl)- ☐☐☐

PLBS (mg/dl)– ☐☐☐

HbA1c (%) - ☐☐.☐

FOOT EVALUATION

NAME OF TEST	ASSESMENT TOOL	RIGHT FOOT	LEFT FOOT
10g Monofilament test	Number of sites where monofilament was felt	/10	/10
Vibration Perception Test	Average volts of vibration felt (0-50V)		
Ankle – Brachial Index for Peripheral Arterial Disease	Ankle – Brachial Index		
Plantar Pressure	Number of high risk clusters on foot		

ANNEXURE III

MASTERCHART

Sr No.	Name	Age	Gender	Onset of DBM	HBA1C	Peripheral Neuropathy (Monofilament)		Peripheral Neuropathy (VPT)		ABI		Plantar Pressure	
						Right	Left	Right	Left	Right	Left	Right	Left
1	Abdul	69	M	0 days	8.4	5	5	40	25	1.07	0.93	3	3
2	Abdul Rehman	45	M	3 months	6.8	6	6	23	26	1.07	1.07	3	3
3	Ankit Mishra	36	M	1 year	7.8	6	6	17	27	1.36	1.40	3	3
4	Bajirao	67	M	1year	9	2	3	50	44	1.17	1.17	4	4
5	Ballappa Appagol	63	M	1year	8	3	4	16	14	1.18	1.18	4	4
6	Bhanudas Khatavkar	69	M	1 year	6.5	6	6	30	26	1.00	1.00	3	3
7	Chandrappa Deernnavar	42	M	1 year	7.1	6	6	6	8	1.08	1.00	3	3
8	Deepika Darwi	48	M	1 year	7.8	6	6	14	10	0.97	0.97	3	3
9	Edwin Singoraj	47	M	1 week	8.2	6	6	27	24	1.07	1.09	3	3
10	Gangangouda Patil	38	M	0 days	8.3	6	6	5	8	1.36	1.00	3	3
11	Ganpati Chavan	58	M	1year	7.8	3	3	30	21	0.82	1.01	4	4
12	Girish Murgad	48	M	1 year	8.2	2	3	30	28	0.91	1.00	4	4
13	Hansa Shah	74	F	1year	7.3	5	5	19	15	1.40	1.40	3	3
14	Iswar Darvi	48	M	1 year	8.4	6	6	20	21	0.92	1.00	3	3
15	Jyothi	37	F	1 year	7.4	6	6	6	4	1.40	1.30	3	3
16	Lalitha	67	F	1 year	9.1	5	5	23	26	0.93	0.93	3	3
17	Madhukar Honageka	54	M	1 year	9.4	1	2	44	31	0.75	1.33	5	5
18	Manuprit Das	30	M	1 year	7.6	6	6	8	8	1.00	1.00	3	3
19	Mubarak Abdulraza	66	M	1 year	7	4	4	25	22	1.40	1.40	4	4
20	Nagendra Gujala	35	M	1year	7	6	6	21	23	0.86	0.86	3	3
21	Prakash Malage	41	M	1 year	7.9	6	6	12	33	1.15	1.20	3	3
22	Praveen Kumar	42	M	1 year	8	6	6	10	13	1.40	1.40	3	3
23	Praveen Kumar C	42	M	1 year	8.3	5	6	33	44	1.40	1.40	5	5
24	Rubina Kotwal	42	F	1 week	9.2	6	6	6	5	1.13	1.13	3	3
25	Rukmini Narayan Hundre	61	F	0 days	10	4	4	32	30	0.80	0.82	4	4
26	Samradhini	36	F	1week	8.3	6	6	6	6	1.27	1.00	3	3
27	Sanjay Mannurkar	35	M	6 months	8.3	5	5	35	32	0.90	0.91	4	4

28	Shaila Hanchinamani	52	F	1 year	10.7	5	6	10	6	0.97	1.00	3	3
29	Shivagouda B Patil	58	M	1 year	9	1	0	50	50	0.73	0.53	5	5
30	Shivanand Shankareppa	47	M	8 months	9.3	2	6	19	23	1.23	1.23	4	4
31	Shivanand Solapure	50	M	1 month	9.8	1	1	17	15	1.14	1.14	3	3
32	Shrimanda Annapa	58	F	1 year	9.1	4	4	28	30	0.82	0.90	3	3
33	Shrimandhar	58	M	1 year	6.5	0	0	46	50	1.40	1.40	3	3
34	Sudhar Poojari	42	M	8 months	9.3	5	6	29	29	0.82	0.84	4	4
35	Sunitha Bhandarkar	51	F	0 days	8.7	6	6	8	6	1.30	1.20	3	3
36	Supriya Abhujeet	40	F	2 years	8.3	3	5	32	30	1.00	1.20	5	5
37	Surash Kadgaoudar	58	M	0 days	9.7	6	6	6	5	0.90	0.90	3	3
38	Surekha Metri	52	F	1 month	10	6	6	17	12	1.17	1.17	3	3
39	Suresh Pandit	53	M	1 year	8.4	6	6	16	11	1.30	1.20	3	3
40	Suvarna Hiremath	46	F	1 year	7.9	5	6	22	25	1.30	1.08	3	3
41	Suvarna Kattimani	50	F	0 days	8.9	6	6	16	16	0.87	0.93	3	3
42	Uday Bogar	56	M	1 month	9.2	5	6	9	10	0.92	0.92	3	3
43	Venkartramanna Katti	44	M	1 year	10	2	4	29	14	1.08	0.92	4	4
44	Vinosha	35	F	2 years	8.2	6	6	4	2	1.09	1.09	3	3
45	Woleppa Gharte	73	M	1 month	9.2	3	3	30	33	0.92	0.95	4	4
46	Bhimraj Hadpadi	62	M	10 months	11	2	2	44	42	0.74	0.72	5	5
47	Fareerappa Kure	41	M	7 months	9.1	2	2	41	42	0.78	0.80	4	4
48	Avinash Patil	57	M	6 months	7.2	3	3	30	31	0.90	0.96	4	4
49	Radhava Naik	42	F	11 months	6.9	5	5	20	21	1.00	1.04	3	3
50	Ravindra Kalkar	52	F	6 months	8.2	3	3	26	27	0.92	0.98	3	3
51	Yallawa Ankale	71	F	10 months	7	2	2	31	33	0.81	0.83	4	4
52	Ashok Hanji	52	M	11 months	7.8	3	3	28	29	0.90	0.93	3	3
53	Saurav	42	M	3 months	7.6	4	4	22	25	92.00	1.00	3	3
54	Rajesh Porli	56	M	9 months	6.8	3	4	27	29	0.90	0.90	3	3
55	Mahadev	59	M	11 months	9.2	3	3	41	43	0.76	0.79	4	4
56	Basamma	64	F	10 months	8.1	1	1	42	44	0.78	0.70	4	4
57	Reshma Desai	46	F	11 months	7.3	5	5	18	17	1.01	0.97	3	3
58	Prakash Yedawad	52	M	5 months	6.5	4	4	20	21	0.85	0.90	3	3
59	Madhsudnan Reddy	47	M	6 months	8	4	4	26	27	0.82	0.91	3	3

60	Ravikiran	39	M	11 months	7.3	5	5	22	21	0.92	1.02	3	3
61	Yallava Kumbar	64	F	11 months	6.9	5	5	23	20	0.90	1.00	3	3
62	Bhamadev P	60	M	4 months	9.1	0	0	49	50	0.90	0.85	5	5
63	Rangappa Dyanu	58	M	3 months	8.2	3	4	29	30	1.10	1.10	4	4
64	Babu G.	55	M	10 months	8	0	0	48	48	1.60	1.70	5	5
65	Kallappa Mahadev	75	M	11 months	8.3	1	1	36	40	1.50	1.50	5	5
66	Fatema Shaikh	36	F	2 months	7.5	4	4	27	28	1.08	1.00	3	3
67	Yashwanth Kyari	42	M	9 months	8.3	4	4	29	31	1.08	1.20	3	3
68	Urmila Yardi	76	F	4 months	7.1	0	0	43	44	0.82	1.09	5	5
69	Manda Kumbar	65	M	4 days	7.5	4	4	14	22	1.11	1.00	3	3
70	Anita K	50	F	2 days	7.1	0	0	45	50	1.08	1.33	3	4
71	Mohd Khan	45	M	5months	8.2	5	5	22	25	1.00	1.00	3	3
72	Farzana	50	F	11 months	7.6	3	3	30	32	1.20	1.20	4	4
73	rajshekhar	53	M	6 months	8	5	5	20	20	0.90	0.93	3	3
74	Dilshad	60	F	1 year	10.1	2	2	37	38	1.20	1.22	4	4
75	Shweta S	39	F	10 months	7.8	5	6	10	12	1.00	1.00	3	3
76	Pallavi	42	F	7 months	9.2	3	3	32	33	0.92	0.97	3	3
77	Shaad Ali	66	M	4 months	9.8	0	0	48	50	0.60	0.60	5	5
78	Prakash F	47	M	8 months	8.3	3	3	29	32	0.90	0.80	4	4
79	Tirakappa	38	M	4 months	7.5	6	6	7	8	0.92	0.93	3	3
80	maganlal	63	M	11 months	9.2	1	1	47	41	0.70	0.72	5	5
81	Shantavva	56	F	5months	8	5	5	13	15	0.99	0.98	3	3
82	Basamma	63	F	9 months	8.4	3	3	22	20	0.97	0.98	3	3
83	Shivalingappa	55	M	10 months	8.3	4	4	16	18	0.97	0.99	3	3
84	Krishnappa	70	M	8 months	10.3	1	1	46	44	1.50	1.40	5	5
85	Rakesh Sutra	64	M	4 months	9	3	3	30	32	1.10	1.00	4	4
86	Prashant K	40	M	4 months	8.8	4	4	33	31	0.90	0.91	4	4
87	Rashmi	52	F	1 year	10	2	2	34	30	0.97	0.96	4	4
88	Basallingappa	74	M	8 months	8.6	2	2	40	37	1.12	1.20	4	4
89	Mahesh	55	M	4 months	7.8	4	4	25	25	0.99	0.99	3	3
90	Kareppa	62	M	7 months	11.3	2	2	36	37	1.20	1.20	3	3
91	Shivalingappa	72	M	1 year	9	1	1	40	42	0.79	0.80	4	4

92	Kalleshi	82	M	1 year	11	0	0	50	50	0.70	0.70	5	5
93	Radha	42	F	10 months	8.4	4	4	18	19	0.93	0.97	3	3
94	Basavva	53	F	3 months	9.2	3	3	32	34	0.89	0.90	3	3
95	Fakeerappa	76	M	1 year	9.8	2	2	40	42	0.67	0.70	4	4
96	Shrishail	54	M	8 months	7.3	4	4	30	30	0.90	0.90	3	3
97	Ramchandra	62	M	9 months	7.5	5	5	20	20	0.90	0.90	3	3
98	Roopa	50	F	3 months	10	3	3	25	27	0.90	0.90	3	3
99	Ramedios	77	M	9 months	11	2	2	36	36	0.80	0.80	4	4
100	Shankar	60	M	10 months	9	3	3	40	38	0.90	0.99	4	4
101	Ramesh	65	M	5months	8	4	4	30	30	1.00	1.00	3	3
102	Kuruppa	73	M	7 months	9.4	3	3	32	33	1.00	1.20	3	3
103	Prajakta	80	F	10 months	10.1	2	2	40	41	0.60	0.60	4	4
104	Kalleshwari	58	F	9 months	8.1	4	4	26	28	0.80	0.80	3	3
105	Mahadev	60	M	11 months	9.2	3	3	29	30	0.97	0.97	3	3
106	Sankesh	77	M	11 months	9.7	3	3	32	33	1.30	1.30	3	3
107	Prakash	39	M	10 months	7.4	4	4	26	25	0.90	0.90	3	3
108	Savitri	53	F	2 months	7.8	4	4	28	27	0.99	0.99	3	3
109	Pawan	67	M	9 months	10.6	3	3	34	35	0.87	0.88	3	3
110	Ridham	39	M	4 months	7.2	6	6	10	12	0.99	0.99	3	3
111	Priyanka	42	F	5months	8.6	4	4	26	27	0.90	0.90	3	3
112	Prashant L	57	M	8 months	8.5	4	4	30	31	0.80	0.80	3	3
113	Sabiha	65	F	7 months	7.5	4	4	32	32	0.80	0.80	3	3
114	Shaabri	57	M	5months	7	5	5	27	26	0.93	0.94	3	3
115	Basallingappa	59	M	10 months	8.8	3	3	31	36	0.87	0.87	4	4
116	Sagar	47	M	8 months	7.3	5	5	16	18	0.93	0.93	3	3
117	Mallawwa	49	F	9 months	8.3	4	4	27	28	0.90	0.90	3	3
118	Jacinta	40	F	1 year	10.8	2	2	35	36	0.87	0.88	4	4
119	Shivaling	67	M	3 months	9.5	4	4	27	26	0.93	0.94	3	3
120	Nurjan	58	F	7 months	9.9	3	3	31	35	1.03	1.02	3	3
121	Channappa	75	M	9 months	11.3	2	2	42	46	1.30	1.30	4	4
122	Rama	50	M	2 months	7.1	4	4	25	26	0.90	0.93	3	3
123	Suresh	68	M	11 months	9.1	3	3	28	29	0.95	0.97	4	4

124	Mahadevi	43	F	5months	7	6	6	17	15	1.00	1.00	3	3
125	Hussainsab	42	M	7 months	7.4	5	5	17	16	1.00	1.00	3	3
126	Kallappa	66	M	10 months	7.1	6	6	10	14	1.00	1.00	3	3
127	Sarasvati	72	F	11 months	8.6	3	3	34	33	0.89	0.90	4	4
128	Rajashri	54	F	2 months	8.9	4	4	26	27	1.00	1.00	3	3
129	mohan	73	M	6 months	8.1	5	5	16	17	1.00	1.00	3	3
130	Shankar	47	M	7 months	10.6	2	2	30	32	1.10	1.10	4	4
131	Padmashri	55	F	5 months	8.5	4	4	27	28	1.11	1.09	3	3
132	Sunita	41	F	10 months	8	5	5	17	16	1.00	1.00	3	3
133	Sadev	57	M	5 months	11.2	2	2	35	36	0.90	0.90	4	4
134	Muttappa	45	M	8 months	9.7	3	3	34	33	1.30	1.30	4	4
135	Bhimavva	72	M	5 months	7.5	4	4	20	21	1.00	1.00	3	3
136	Shivappa	64	M	11 months	10	2	2	37	36	0.90	0.90	5	5
137	Rajendra	45	M	9 months	9.8	5	5	16	15	0.91	0.93	3	3
138	Sarvamangala	53	F	2 months	9.3	5	5	12	14	0.91	0.91	3	3
139	Lokamma	71	M	9 months	10.8	2	2	42	44	0.87	0.87	4	4
140	Riyaz	63	M	10 months	9.9	3	3	40	40	0.93	0.93	4	4
141	Pragati	48	F	4 months	7.4	5	5	21	22	0.98	0.98	3	3
142	Anil	69	M	6 months	9.6	3	3	29	28	1.00	1.00	4	4
143	Anusuya	43	F	5 months	7.5	5	5	14	16	1.10	1.10	3	3
144	Shilpa	62	F	11 months	8	5	5	16	14	0.98	0.98	3	3
145	Salini	39	F	1 year	7.2	6	6	8	9	0.98	0.98	3	3
146	Subhash	60	M	1 year	11.4	2	2	46	47	0.78	0.78	5	5
147	Deepak	75	M	8 months	8.4	3	3	30	31	0.93	0.93	4	4
148	Shruthi	47	F	7 months	8.7	3	3	32	35	1.00	1.00	3	3
149	Amith	54	M	6 months	7.4	5	5	15	16	1.10	1.10	3	3
150	Sikavva	58	F	4 months	7.1	6	6	10	12	1.00	1.00	3	3