ASSESSMENT OF PERIPHERAL ARTERIAL DISEASE IN DIABETIC FOOT ULCER IN SOUTH INDIAN POPULATION:

A PROSPECTIVE STUDY

A Dissertation submitted to

THE TAMILNADU DR. M.G.R. MEDICAL UNIVERSITY

In partial fulfilment of the regulations for the award of the

M.S. DEGREE EXAMINATION

BRANCH I GENERAL SURGERY

DEPARTMENT OF GENERAL SURGERY

STANLEY MEDICAL COLLEGE AND HOSPITAL

THE TAMILNADU DR. M.G.R. MEDICAL UNIVERSITY

CHENNAI

APRIL 2017

CERTIFICATE

This is to certify that the dissertation titled "ASSESSMENT OF PERIPHERAL ARTERIAL DISEASE IN DIABETIC FOOT ULCER IN SOUTH INDIAN POPULATION: A PROSPECTIVE STUDY" is the bonafide work done by Dr. S. Vijayasarathy, Post graduate student in the Department of General Surgery, Government Stanley Medical College and Hospital, Chennai under my guidance and supervision, in partial fulfilment of the regulations of the The Tamilnadu Dr. M.G.R. Medical University, Chennai for the award of M.S. Degree Branch I General Surgery Examination to be held in April 2017.

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DECLARATION

I, Dr. S. Vijayasarathy solemnly declare that this dissertation titled "ASSESSMENT OF PERIPHERAL ARTERIAL DISEASE IN DIABETIC FOOT ULCER IN SOUTH INDIAN POPULATION: A PROSPECTIVE STUDY" is a bonafide work done by me in the Department of General Surgery, Government Stanley Medical College and Hospital, Chennai under the guidance and supervision of my unit chief

Prof. M.V. UDAYACHANDAR
Professor of Surgery

This dissertation is submitted to The Tamilnadu Dr. M.G.R. Medical University, Chennai in partial fulfilment of the university regulations for the award of M.S. Degree Branch I General Surgery Examination to be held in April 2017

Place: Chennai

Date: September 2016 **Dr. S. Vijayasarathy**

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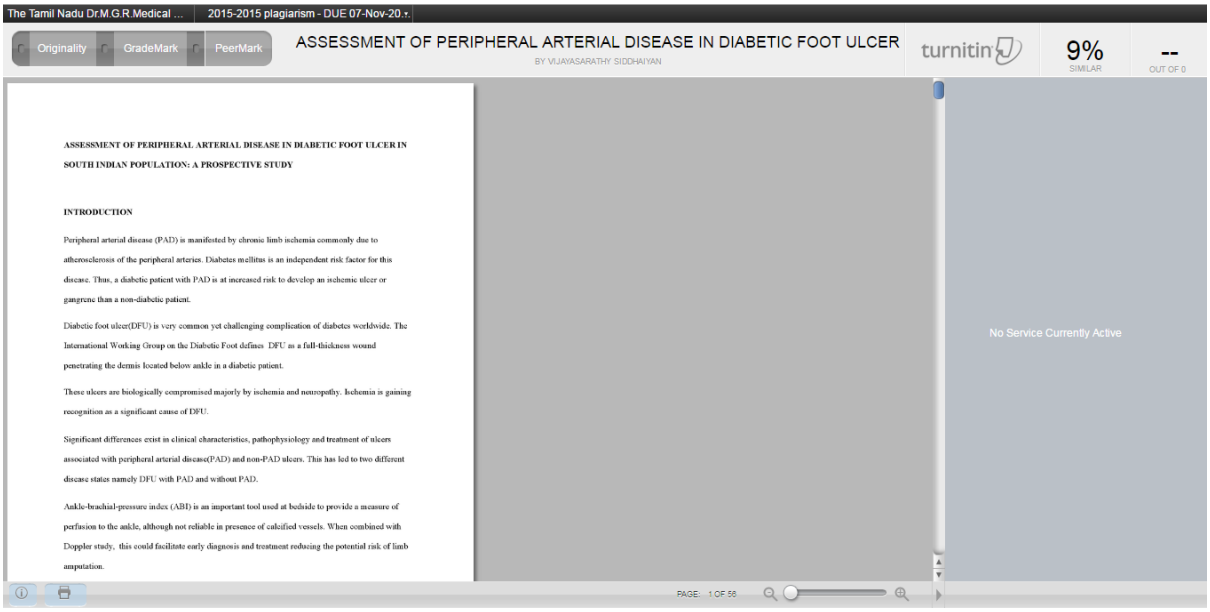
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ASSESSMENT OF PERIPHERAL ARTERIAL DISEASE IN DIABETIC FOOT ULCER IN SOUTH INDIAN POPULATION: A PROSPECTIVE STUDY
INTRODUCTION
Peripheral arterial disease (PAD) is manifested by chronic limb ischemia commonly due to atherosclerosis of the peripheral arteries. Diabetes mellitus is an independent risk factor for this disease. Thus, a diabetic patient with PAD is at increased risk to develop an ischemic ulcer or gangrene than a non-diabetic patient.
Diabetic foot ulcer(DFU) is very common yet challenging complication of diabetes worldwide. The International Working Group on the Diabetic Foot defines DFU as a full-thickness wound penetrating the dermis located below ankle in a diabetic patient.
These ulcers are biologically compromised majorly by ischemia and neuropathy. Ischemia is gaining recognition as a significant cause of DFU.
Significant differences exist in clinical characteristics, pathophysiology and treatment of ulcers associated with peripheral arterial disease(PAD) and non-PAD ulcers. This has led to two different disease states namely DFU with PAD and without PAD.
Ankle-brachial-pressure index (ABI) is an important tool used at bedside to provide a measure of perfusion to the ankle, although not reliable in presence of calcified vessels. When combined with Doppler study, this could facilitate early diagnosis and treatment reducing the potential risk of limb amputation.
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PAGE 1 OF 56

CONTENTS

ASSESSMENT OF PERIPHERAL ARTERIAL DISEASE IN DIABETIC FOOT ULCER IN SOUTH INDIAN POPULATION: A PROSPECTIVE STUDY

INTRODUCTION

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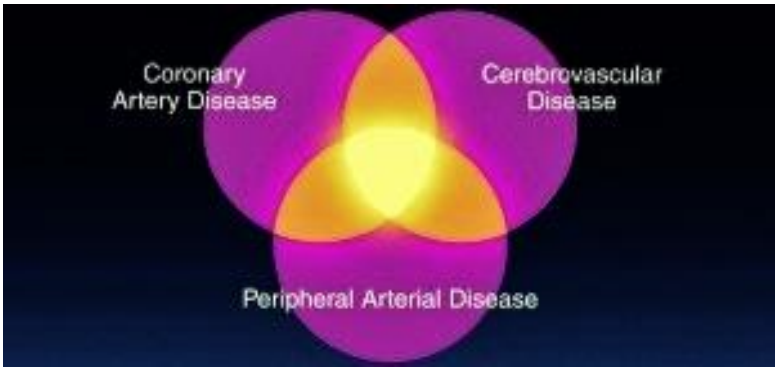
The indications for revascularization of an ischemic limb are incapacitating claudication and critical limb ischemia(CLI). Disabling claudication is a relative indication and one has to weigh the existing quality of life against the risk of procedure during selection of suitable patient and it requires significant patient counseling. Revascularization in some ischemic limbs may be deferred if no target vessel present or unavailability of an autogenous vein. Even irreversible gangrene progressing beyond midfoot may preclude revascularization. In such patients a choice must be made between prolonged medical management and primary amputation.

Major amputation in an ischemic foot is indicated only when there is life threatening sepsis or when arterial occlusion causing extensive necrosis has destroyed the foot.

Most amputations can be prevented and limbs salvaged through a multimodal treatment of infection control, wound debridement and revascularization procedures. However amputation may warrant a good quality of life, if a prolonged treatment course is anticipated with minimal likelihood of healing.

All diabetic patients should be offered full and active rehabilitation following limb amputation.

Thus the association of PAD largely impacts the treatment outcomes in terms of ulcer healing, lower limb amputations and mortality. The burden of PAD in DFU in South Indian population has not been assessed adequately in the recent years. A prompt diagnosis of ischemia and multidisciplinary approach to DFU will decrease the loss of limb and life. This study aims to throw light on the same.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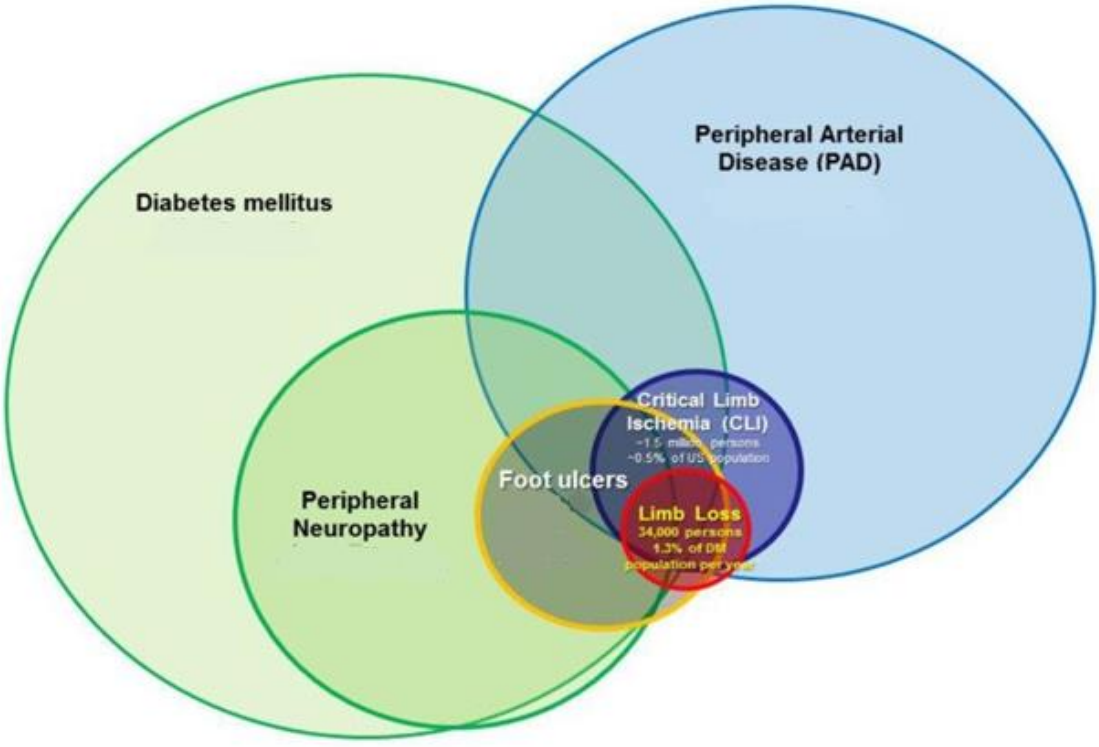
Cerebrovascular
Disease
Peripheral Arterial Disease


Diabetes mellitus
Peripheral Arterial
Disease (PAD)
Peripheral
Neuropathy
Foot ulcers
Critical Limb
Ischemia (CLI)
Limb Loss
34,000 persons

Effect of Peripheral Arterial Disease in Diabetic population

PAD is characterized by atherosclerosis of lower extremity arteries causing occlusive disease. It is a strong predictive factor for atherothrombotic disease in other vascular beds.

PAD involvement is mostly diffuse and particularly is more severe in tibial vessels. It usually involves long segment occlusions. In a non-diabetic individual, collateral vessels develop in response to occlusion of a major artery. This collateral formation is impaired in diabetes rendering the distal tissue more prone to severe ischemia.

Patient with PAD most commonly presents with a cramping pain in the calves, thighs or buttocks known as intermittent claudication. This pain relieved by rest and reappears with walking and exercise. Some patients present with extreme symptoms like rest pain, infected ulcer and gangrene. These limb-threatening symptoms are collectively termed as critical limb ischemia (CLI).

The strong risk factors for PAD are diabetes and smoking. The duration of diabetes, hypertension, hyperlipidemia and advanced age are the other established risk factors.

In diabetic patients, occurrence of PAD increases with diabetic duration, advancing age and peripheral neuropathy.

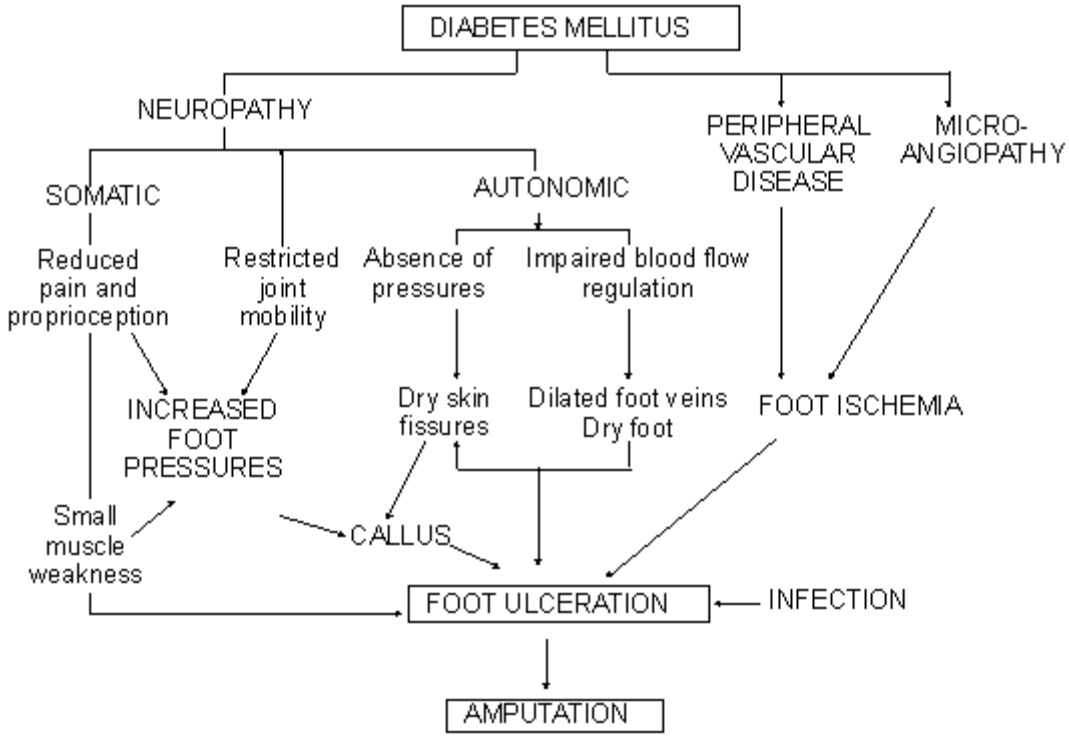
Most patients are asymptomatic or do not report symptoms due to ignorance. For some, pain is blunted by presence of neuropathy. Moreover there is no uniformly agreed consensus on screening modalities. For these reasons, the assessment of true prevalence of PAD in diabetic patients becomes difficult. Hence in presence of diabetes, PAD is more likely to present at an advanced stage.

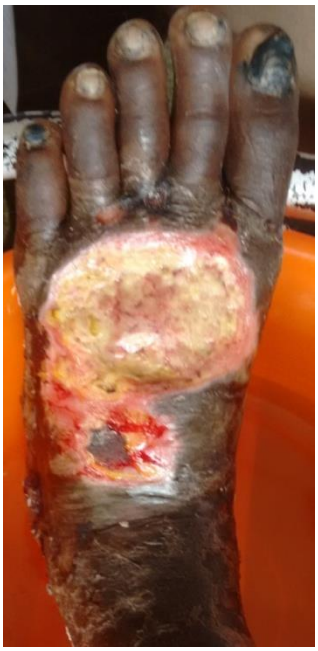
Two common assessment tools are presence of intermittent claudication and absence of distal foot pulses, both involving an element of insensitivity. For more accurate estimation of prevalence, the assessment should be based on reproducible and validated test like ankle-brachial index (ABI).

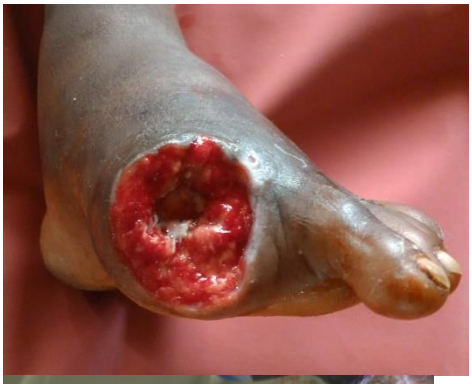
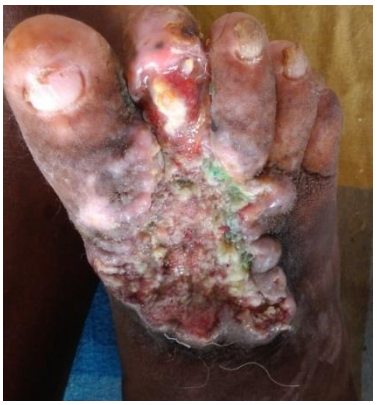
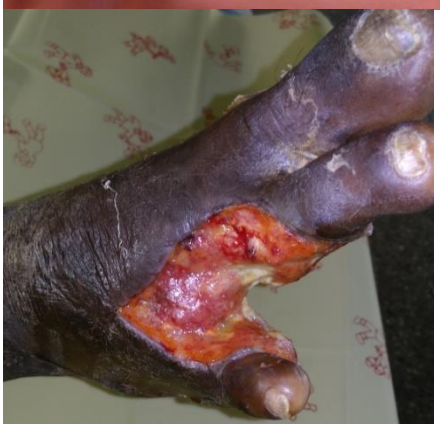
The significant differences in clinical characteristics and outcome has led to the consideration that

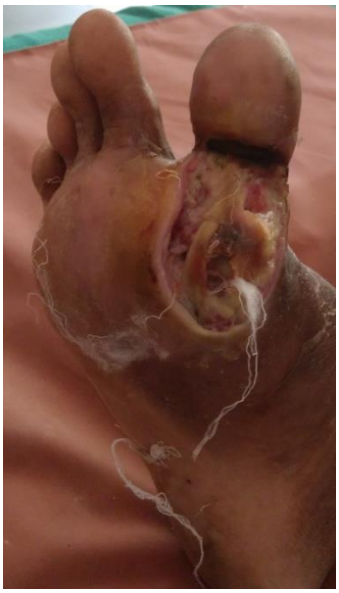
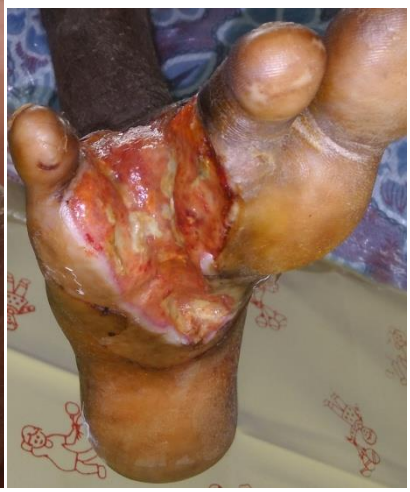
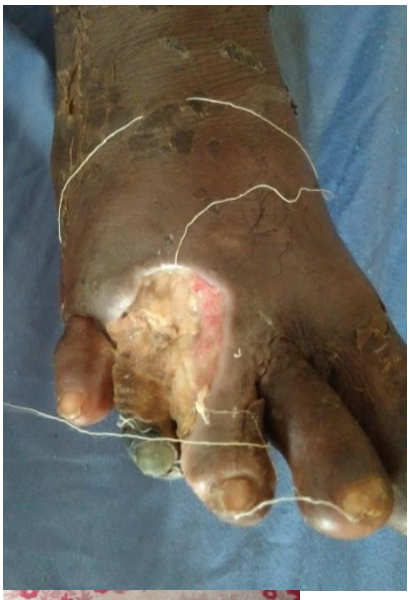
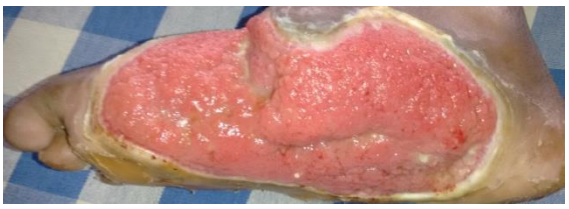
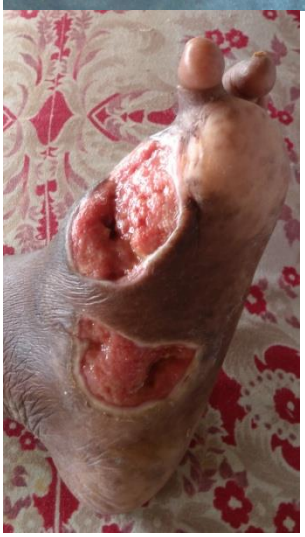
DFU with PAD and **DFU without PAD**

should potentially be defined as
two separate disease states


DIABETES MELLITUS
NEUROPATHY
PERIPHERAL VASCULAR DISEASE
MICRO-ANGIOPATHY
SOMATIC
AUTONOMIC
Reduced pain and proprioception
Restricted joint mobility
Absence of pressures
Impaired blood flow regulation
INCREASED FOOT PRESSURES
Dry skin fissures
Dilated foot veins Dry foot
FOOT ISCHEMIA
Small muscle weakness
CALLUS
FOOT ULCERATION
INFECTION
AMPUTATION



Evaluation of PAD in DFU

Clinical evaluation

The clinical evaluation should start with a detailed medical history enquiring about the onset and progression of disease. Risk factors should be enquired about and focus should be on identifying symptoms like claudication, rest pain and functional impairment. PAD patient may present in a diverse form from no symptoms to infected chronic ulcer and gangrene. Associated symptoms arising from atherothrombosis in other vascular beds like angina, stroke and abdominal ischemia should be noted. Alternative causes of claudication like spinal canal stenosis should be ruled out.

Physical examination should start with attitude of limb and presence of deformity. Detailed inspection of the affected limb involves looking for the signs of vascular insufficiency. These include muscle wasting and loss of subcutaneous fat characterized by thinning of limbs with bony prominence. The skin becomes dry and fissured with reduced temperature. Nails are dystrophic, lusterless, brittle and contain transverse ridges. There is loss of skin hair make the skin appear shiny. The interdigital spaces should be inspected for ulcers and fissures.

This should be followed by assessment of circulatory insufficiency like capillary refilling time, venous filling time(Harvey's sign) and Buerger's test.

Buerger's test involves assessing the leg elevation angle at which vascular compromise is obvious. Assessment of peripheral pulses is most important which involves charting of lower and upper limb pulses. One has to auscultate these arteries and look for bruit. This has to be followed by assessment of neighboring joints for movements and deformity. Thorough assessment of motor system, sensory system of nerves and reflexes should be done. Finally draining lymph nodes should be examined.

Non invasive test: ABI

ABI provides an easy, reasonably accurate and non invasive assessment of occurrence of PAD. It also helps assessing the severity of the disease. It is a ratio in which the numerator is the highest of the three ankle systolic blood pressures in dorsalis pedis, anterior and posterior tibial arteries in the affected lower limb. The denominator is the highest of brachial systolic pressures measured in both the upper limbs. The pressures are measured by using hand held Doppler making it a very simple and quantitative tool to assess the patency of lower limb arteries

However, in elderly, diabetic patients and in chronic kidney disease, the peripheral arteries are calcified and are poorly compressible and hence may artificially elevate the values. This complicate the evaluation of PAD.

The tools required to calculate ABI include a hand-held Doppler probe of frequency 5–10 MHz and a sphygmomanometer with blood pressure cuff. The ABI is measured by putting patient in supine position for 5 min. Systolic blood pressure in ankle is measured in posterior tibial artery and dorsalis pedis and by placing the cuff just above the ankle. Systolic blood pressure in arm is measured in brachial artery by placing the cuff just above the elbow.

ABI>1.3 indicates presence of poorly compressible arteries at the ankle level due to medial arterial calcification which occurs commonly in diabetes. This renders diagnosis of PAD by ABI alone less reliable.

Since the digital arteries are less commonly affected by calcification, toe pressure measurements involving digital arteries are more reliable in assessment of forefoot circulation in patients with diabetes. A toe-brachial index of <0.7 or toe pressure of <55 mmHg strongly indicates PAD.

Evaluation of the flow signals from arteries in foot using hand held Doppler revealing a monophasic or absent signal indicated sever ischemia.

Imaging modalities for PAD in diabetes

Doppler ultrasound:

Doppler ultrasound involves combining B-mode ultrasound and pulsed Doppler flow to assess anatomy and physiology of blood flow in specific arterial segments. The entire lower extremity arterial circulation is evaluated by sequential scanning of the abdominal aorta, iliac, femoral, popliteal and tibial arteries,.

CT angiography:

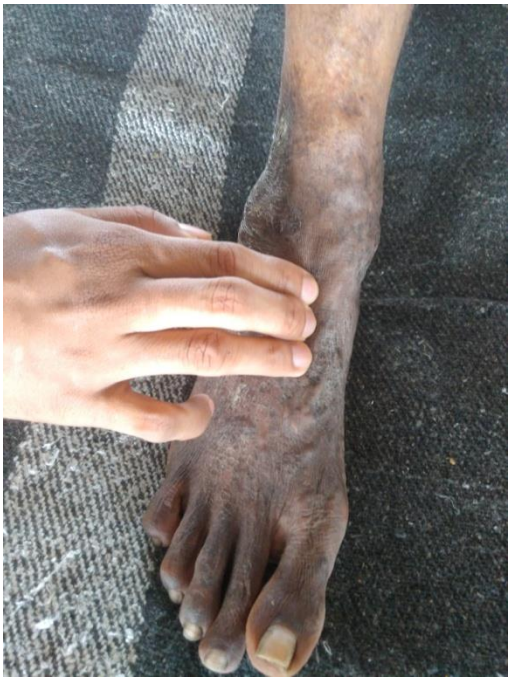
It is a minimally invasive modality to diagnose PAD. It involves use of iodinated contrast which is injected intravenously. For lower extremity, scanning is done from renal arteries to the distal foot . This is followed by 3D reconstruction of data. The advantage is that it provides high resolution images of small vessels in the calf. The disadvantage being use of radiation and potentially nephrotoxic contrast.

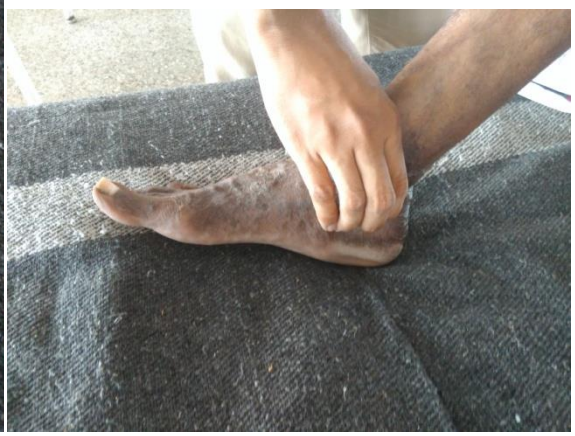
Contrast-enhanced MR angiography:

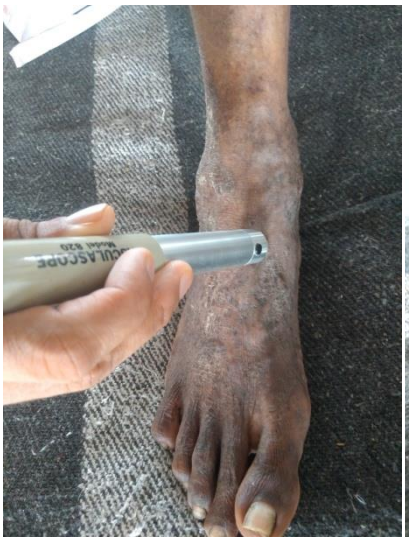
This is also a low invasive imaging technique for detecting PAD. Gadolinium is used as contrast.

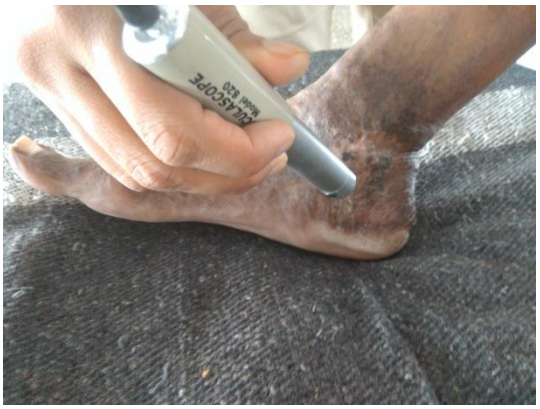
Digital subtraction angiography

Intra-arterial DSA is considered as the gold standard for imaging due to high spatial resolution of arteries. Endovascular revascularization can be performed in the same sitting. The risks involved are arterial puncture, hematoma, extravasation and contrast allergy. Preferably DSA should be performed only when revascularization is planned.









Medical management for PAD in DFU

Asymptomatic PAD

Life style modification

This involves modification of daily activities and dietary pattern.

Tobacco Smoking

Counseling of patients to abstain from all tobacco products is the first and most essential step. In PAD, tobacco is believed to increase progression of the atherosclerotic disease and hence increases amputation risk.

Blood sugar control

Adequate glycemic control to a tune of HbA1c < 7% is needed to prevent microvascular complications.

Hyperlipidemia

Many studies conclude that lipid lowering agents slow the disease progression and bring down deaths due to cardiovascular events.

Antiplatelets

Aspirin low dose exhibits antithrombotic effects and also slows down the clot propagation and reduce the cardiovascular complication. Those allergic to aspirin are put on clopidogrel.

Management of symptomatic PAD

Exercise

Moderate exercise within the limits of claudication distance is advised to improve the collateral blood supply and cardiovascular risk factor profile.

Drug therapies

Heparin is used to interfere with clotting mechanism and prevent further clot formation. However it does not act on existing clot.

Pentoxifylline is a hemorheologic modifier which is said to improve microcirculation by decreasing the blood viscosity.

Cilostazol is a phosphodiesterase III inhibitor acting as a vasodilator. It is contraindicated in heart failure.

Foot care

Patients should be counseling on foot care involving hygiene and specialized foot wear. Regular supervision reduces the risk of foot complications.

Treatment of ischemic foot

1. Debridement of nonviable tissue
2. Off-load pressure from injury site
3. Provide clean, moist environment to promote new granulation tissue and epithelialization
4. Optimize nutrition, glycemic control
5. Possible antibiotics, surgery to resolve infection
6. Possible revascularization surgery to resolve peripheral ischemia

Critical limb ischemia characterized by rest pain, tissue loss or gangrene is a limb-threatening condition which warrants emergent treatment. Peripheral neuropathy blunts the pain perception causing PAD to manifest late in a diabetic patient. Contrarily PAD accelerated nerve ischemia and worsened neuropathy.

In contrast to involvement of plantar aspect in neuropathic ulcers, ischemic ulcers commonly involve dorsum , edges of foot and toes. Conservative management includes wound debridement, ulcer offloading, appropriate dressing, and adjuvant wound healing methods.

Wound Debridement

Wound debridement should aim to remove all necrotic debris and slough to reduce infection. It should be done when there is presence of localized fluctuation, undermining of ulcer with slough, crepitus with gas in X-ray and need for drainage of pus.

Appropriate footwear

It is most important in case of a neuroischemic foot wear the aim is protection of foot from shear and pressure. Patients should be advised not to wear tight shoes. This would hinder ulcer healing. Ideal footwear should be long, deep and broad, designed to protect arch of foot and offload the pressure. This would automatically facilitate the ulcer healing.

Dressings

Dressing is applied to prevent tissue desiccation, absorb discharge, prevent external contamination. Occlusive dressing lowers risk of infection. Non adhesive dressings should cover foot ulcers at all times. Some properties like easy removability, accommodating foot pressures while walking and is

desirable. Various available dressings include foams, hydrogels, absorbent polymers, alginates, growth factor and skin replacement agents.

Treatment of infection

In a diabetic foot, the signs and symptoms of foot infection are often diminished due to impaired neuroinflammatory response.

The isolates in diabetic foot sepsis are usually polymicrobial. Commonly streptococci, methicillin resistant staphylococcus(MRSA), enterobacteriaceae and pseudomonas are encountered. Often it is mixed infection with anaerobes.

Appropriate empirical antibiotics should act against both gram positive and gram negative organisms and provide both aerobic and anaerobic coverage. Such wounds require intravenous antibiotics and these patients should be hospitalized.

Patients with mild to moderate infection, superficial ulcers without sepsis, localized cellulitis can be treated with oral antibiotics on an outpatient basis. The empirical antibiotics should be started after initial cultures are taken and changed as needed.

Incision and drainage is the core of treatment for almost all diabetic foot infections. It may be supplemented with toe or ray amputation for facilitating drainage. Aggressive wound debridement and revascularization procedure usually make limb salvage possible.

Neuroischemic foot may complicate into dry and wet gangrene. Septic arteritis following soft tissue infection and ulceration leads to wet gangrene. If gas in soft tissue is noted, immediate open drainage of all potentially infected spaces is necessary along with broad spectrum I.V. antibiotics.

In acute on chronic limb ischemia, there is severe reduction in arterial perfusion leading to dry gangrene. Surgical wound debridement should be done only after revascularization if applicable. Only then the debrided foot will be perfused adequately to heal.

Revascularization

Revascularization can be carried out by open surgical technique or endovascular procedures. These two types of procedures are not mutually exclusive. They are combined in most of the cases like iliac angioplasty with bypass grafting with saphenous vein.

Endovascular procedures are preferred in focal segmental disease.

Aortoiliac disease usually managed effectively with open aortofemoral prosthetic bypass but endovascular stenting is gaining significant recognition as an alternative.

Superficial femoral artery stenosis can be treated by open femoropopliteal bypass.

Bypass grafting with autogenous great saphenous vein is the common procedure for tibial disease. Nevertheless, endovascular procedures are increasingly becoming popular due to technical advances allowing aggressive use of tibial angioplasty.

Major limb amputation is only indicated in case of life threatening sepsis in an ischemic foot. majority of limb amputations can be prevented by timely diagnosis and intervention by a combination of revascularization, wound debridement, infection control by antibiotics, and staged closure of wound.

Revascularization
patient counseling

Critical limb ischemia
ABI<0.4

Incapacitating claudication

lack of target vessel

unavailability of autogenous vein

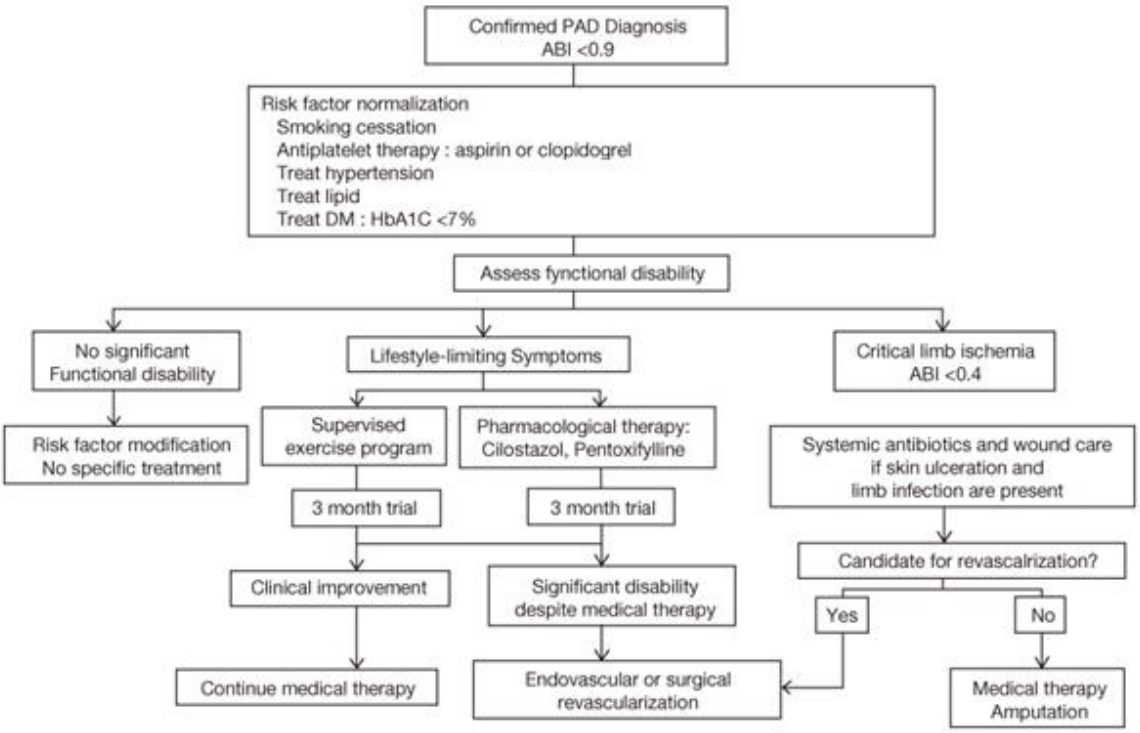
irreversible gangrene beyond the level of midfoot

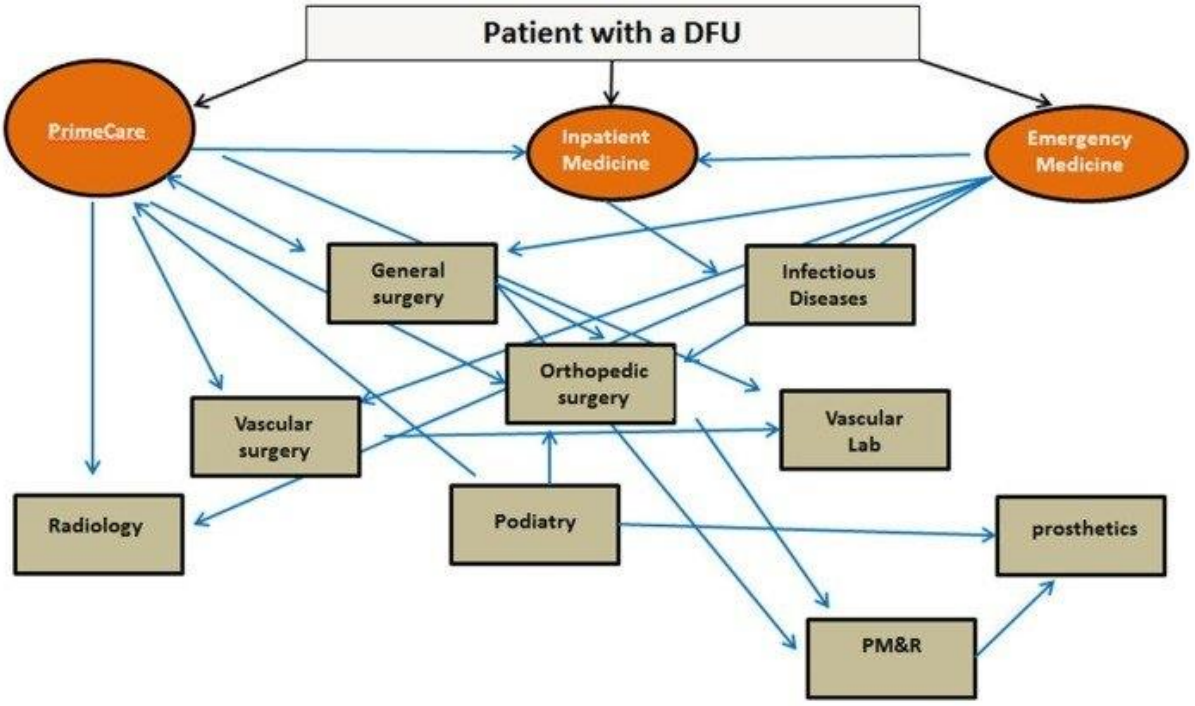
Primary amputation

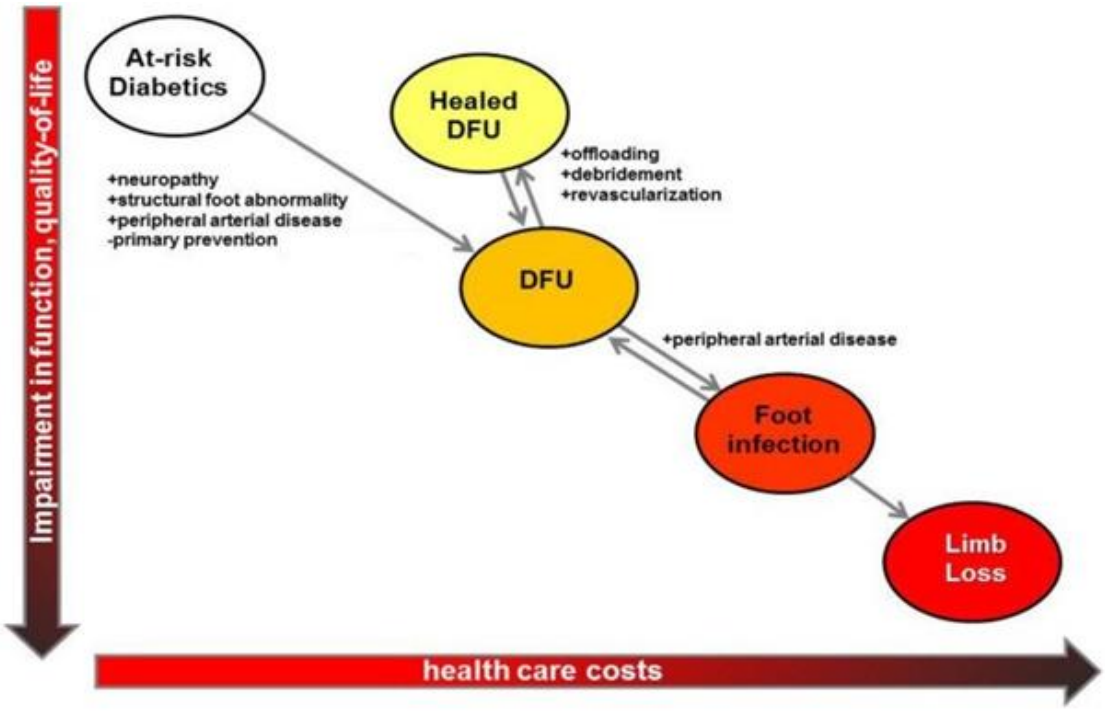
Severe ischemia leading to necrosis

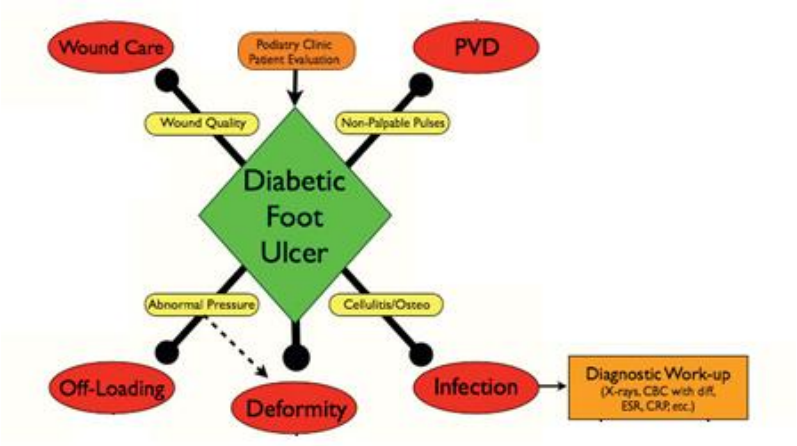
Life threatening sepsis

if a prolonged course of treatment is anticipated with little likelihood of healing, amputation may cause return to a good quality of life


Confirmed PAD Diagnosis
ABI <0.9
Risk factor normalization
Smoking cessation
Antiplatelet therapy : aspirin or clopidogrel
Treat hypertension
Treat lipid
Treat DM : HbA1C <7%
Assess fynctional disability
No significant
Functional disability
Lifestyle-limiting Symptoms
Critical limb ischemia
ABI <0.4
Risk factor modification
No specific treatment
Supervised
exercise program
Pharmacological therapy:
Cilostazol, Pentoxifylline
Systemic antibiotics and wound care
if skin ulceration and
limb infection are present
3 month trial
3 month trial
Candidate for revascalrization?
Clinical improvement
Significant disability
despite medical therapy
Yes
No
Continue medical therapy
Endovascular or surgical
revascularization
Medical therapy
Amputation


Patient with a DFU
PrimeCare
Inpatient Medicine
Emergency Medicine
General surgery
Infectious Diseases
Orthopedic surgery
Vascular surgery
Vascular Lab
Radiology
Podiatry
prosthetics
PM&R


Impairment in function, quality-of-life
At-risk Diabetics
+neuropathy
+structural foot abnormality
+peripheral arterial disease
-primary prevention
Healed DFU
+offloading
+debridement
+revascularization
DFU
+peripheral arterial disease
Foot infection
Limb Loss
health care costs


Wound Care
Podiatry Clinic Patient Evaluation
PVD
Wound Quality
Non-Palpable Pulses
Diabetic Foot Ulcer
Abnormal Pressure
Cellulitis/Osteo
Off-Loading
Deformity
Infection
Diagnostic Work-up
(X-rays, CBC with diff, ESR, CRP, etc.)

WIFI-G Index

The society of vascular surgeons formulated a new wound classification system WIFI based on wound, ischemia and foot infection. It is useful for risk stratification like other classification systems like Wagner, PEDIS, Texas. This study aims to incorporate this classification and test its correlation with PAD and limb amputation.

Table 2. Diabetic Foot classification according to Wagner

Grade	Denomination	Description
0	Foot at risk	Thick calluses, bone deformities, clawed toes, and prominent metatarsian heads
1	Superficial ulcers	Total destruction of the thickness of the skin
2	Deep ulcers	Penetrates through skin, fat and ligaments, but not affect bone. Infected
3	Abscessed deep ulcers	Limited necrosis in toes or the foot
4	Limited gangrene	Limited necrosis in toes or the foot
5	Extensive gangrene	Necrosis of the complete foot, with systemic effects

University of Texas Diabetic Wound Classification System				
Stage	**Grade**			
	0	**I**	**II**	**III**
A (no infection or ischemia)	Pre- or post-ulcerative lesion completely epithelialized	Superficial wound not involving tendon, capsule, or bone	Wound penetrating to tendon or capsule	Wound penetrating to bone or joint
B	Infection	Infection	Infection	Infection
C	Ischemia	Ischemia	Ischemia	Ischemia
D	Infection and ischemia	Infection and ischemia	Infection and ischemia	Infection and ischemia

Table 2. IWGDF and IDSA Classification of DFIs

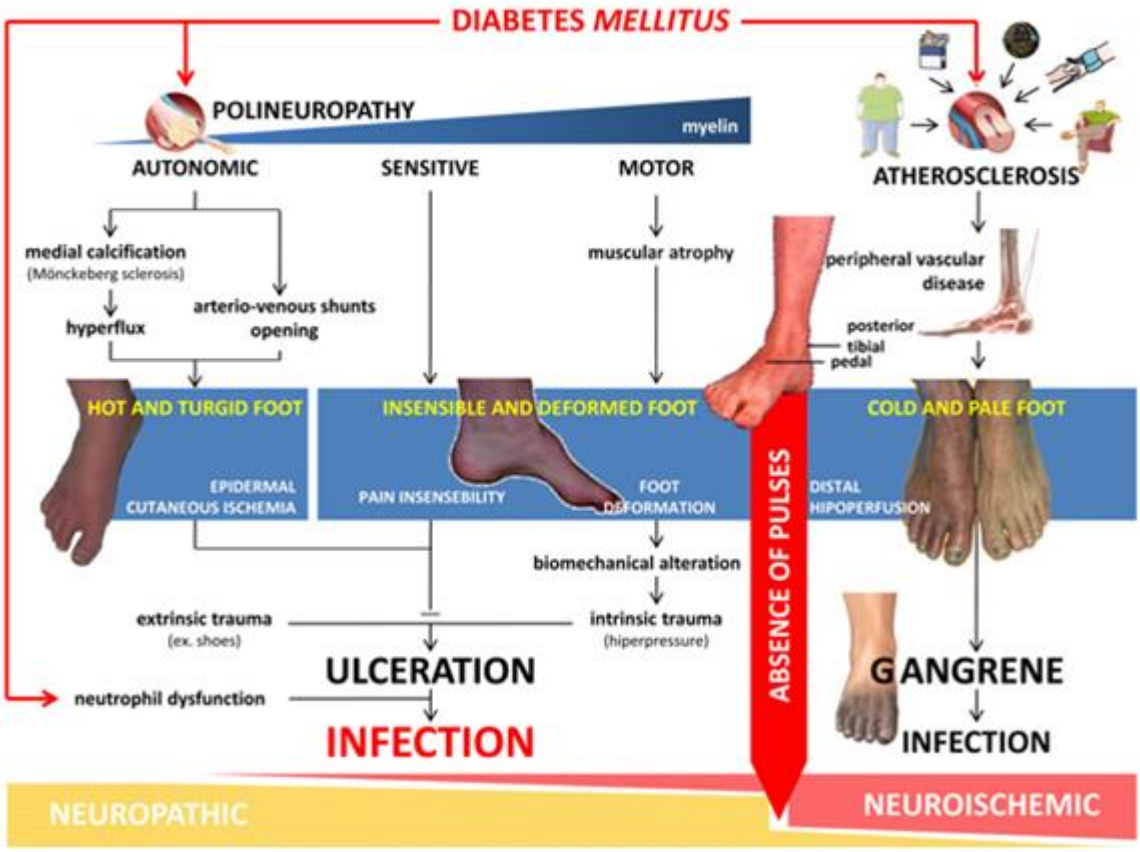
Clinical Manifestation of Infection	PEDIS	IDSA
No signs or symptoms of infection	1	Uninfected
Infection present, as defined by presence of ≥2 of the following: erythema, local swelling or induration, local tenderness or pain, local warmth, purulent discharge	N/A	N/A
Local infection involving only skin and SC tissue; if erythema present, must be >0.5-≤2 cm around ulcer; exclude other causes of inflammatory response of skin	2	Mild
Local infection as described above, with erythema >2 cm or involving structures deeper than skin and SC tissues; no SIRS	3	Moderate
Local infection as described above, with signs of SIRS as manifested by ≥2 of the following: T >38°C or <36°C, HR >90 beats/min, RR >20 breaths/min or $Paco_2$ <32 mmHg, WBC count >12,000 or <4,000 cells/mcl or ≥10% bands	4	Severe

DFI: diabetic foot infection; HR: heart rate; IDSA: Infectious Diseases Society of America; IWGDF: International Working Group on the Diabetic Foot; min: minute; N/A: not applicable; $PaCO_2$: partial pressure of carbon dioxide, arterial; PEDIS: perfusion, extent, depth, infection, sensation; RR: respiratory rate; SC: subcutaneous; SIRS: systemic inflammatory response syndrome; T: temperature.
Source: References 6, 13.

Table 3. PEDIS[a] Grades and Treatment Paradigms

Grade	Infection Severity	Clinical Manifestations	Treatment Parameters	Medications
1	Uninfected	Wound without purulence or inflammation	Outpatient	Topical antibiotics
2[b]	Mild	≥2: purulence or erythema, pain, tenderness, warmth, or induration; cellulitis ≤2 cm around ulcer; infection limited to skin/subcutaneous tissue; no other complications	Most not limb-threatening; most outpatient treatment	Cephalexin, trimethoprim-sulfamethoxazole (TMP-SMX), levofloxacin, amoxicillin-clavulanate, clindamycin
3[c]	Moderate	Infection as above plus >1: cellulitis >2 cm, streaking, deep tissue abscess, gangrene and with some life-threatening; involvement of muscle, tendon, joint, or bone	Most limb-threatening with some life-threatening; requires hospital treatment	TMP-SMX, amoxicillin-clavulanate, levofloxacin, ceftriaxone, linezolid, ertapenem, ticarcillin-clavulanate
4[d]	Severe	Infection plus systemic toxicity or metabolic instability; fever, chills, tachycardia, hypotension, confusion, vomiting, severe hyperglycemia, acidosis, or azotemia	Life-threatening; requires hospital treatment	Imipenem-cilastatin, vancomycin-ceftazidime, levofloxacin-clindamycin, piperacillin-tazobactam, ticarcillin-clavulanate

[a] PEDIS stands for perfusion, extent/size, depth/tissue loss, infection, and sensation.
[b] Medications for treatment can be oral.
[c] Medications for treatment are oral or parenteral, based on clinical situation.
[d] Medications for treatment are IV, at least initially.
Source: Reference 18.


DIABETES MELLITUS
POLINEUROPATHY
myelin
AUTONOMIC
SENSITIVE
MOTOR
ATHEROSCLEROSIS
medial calcification
(Mönckeberg sclerosis)
hyperflux
arterio-venous shunts
opening
muscular atrophy
peripheral vascular
disease
posterior
tibial
pedal
HOT AND TURGID FOOT
EPIDERMAL
CUTANEOUS ISCHEMIA
INSENSIBLE AND DEFORMED FOOT
PAIN INSENSEBILITY
FOOT
DEFORMATION
COLD AND PALE FOOT
DISTAL
HIPOPERFUSION
ABSENCE OF PULSES
biomechanical alteration
extrinsic trauma
(ex. shoes)
intrinsic trauma
(hiperpressure)
ULCERATION
neutrophil dysfunction
INFECTION
GANGRENE
INFECTION
NEUROPATHIC
NEUROISCHEMIC

OBJECTIVES

To know prevalence of peripheral arterial disease in diabetic foot ulcer

To assess the associated risk factors

To test the correlation of WIFI-G score with severity of disease

To assess the appropriateness of triple test and need for routine Doppler in DFU to evaluate PAD

To assess the limb salvage

To compare the results with previous similar studies on DFU with PAD

MATERIALS AND METHODS

PATIENT SELECTION:

In-patients admitted with isolated diabetic foot ulcer were enrolled into the study. They were randomly selected from the surgical wards on every 7th day. A total of 100 patients were sampled.

Study area: Department of General Surgery and Vascular Surgery,

Govt. Stanley Hospital

INCLUSION CRITERIA:

Age group 35- 65yrs

Both sexes

In-patient

Known diabetic

Isolated foot ulcer

EXCLUSION CRITERIA:

Out patients

Known case of peripheral vascular disease during admission

Ulcer other than in foot

Vasculitis

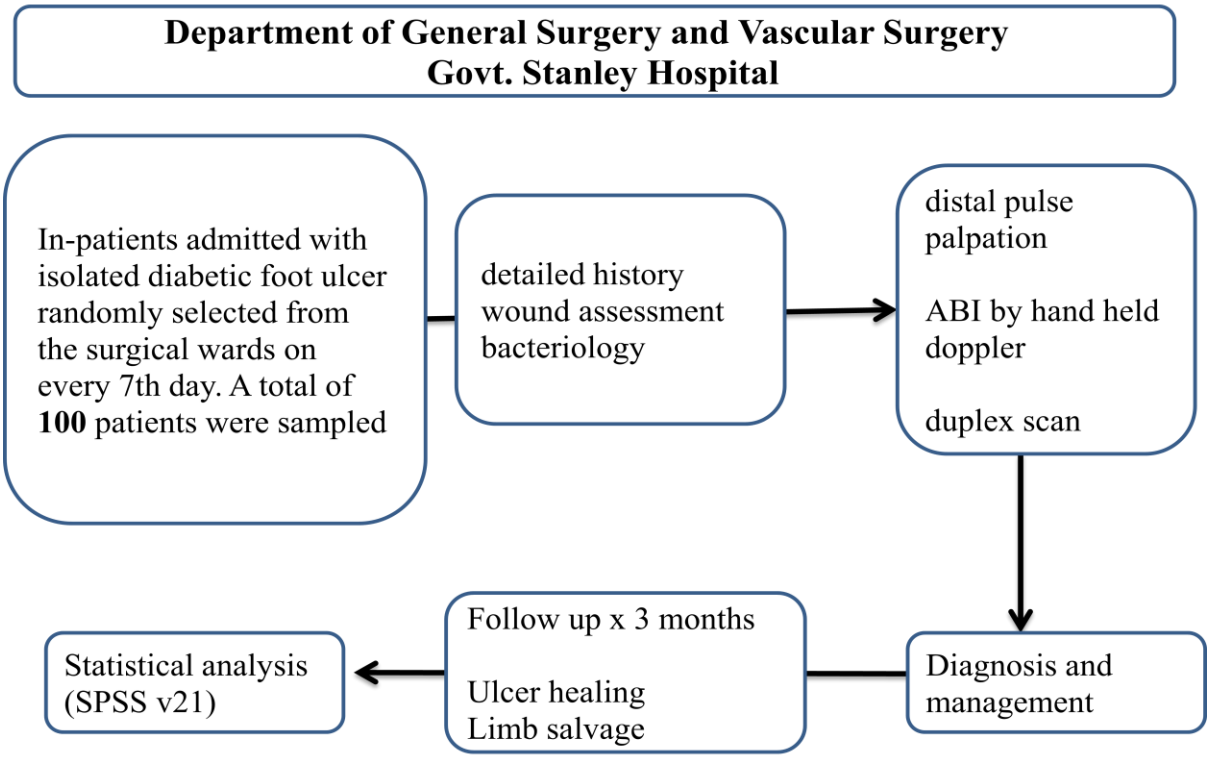
PERIOD OF STUDY: 9 months

METHODOLOGY

This is a prospective study which involved type 2 diabetic patients with isolated foot ulcer(DFU). Their history, clinical parameters and wound bacteriology were noted and documented. A total of 100 patients were evaluated in this study. The patients were subjected to detailed history by administering questionnaires to assess the diabetic foot ulcer and associated risk factors. A thorough clinical examination was carried out followed by specific examination which included distal pulse assessment, ankle-brachial index(ABI) and duplex scan to evaluate PAD. Portable hand held Doppler was used to measure ABI.

The patients were followed up for a period of 3 months from definitive procedure to assess the ulcer healing and limb salvage. The data was subjected to statistical analysis to find out association between parameters of interest.

MATERIALS AND METHODS



DIAGNOSIS CRITERIA

Peripheral arterial disease:

any **2/3** criteria of the following

- absent distal foot pulses
- ankle brachial pressure index(ABI) <0.9
- abnormal doppler flow/duplex scan

Fontaine classification:

Stage I asymptomatic
Stage IIa intermittent claudication >200m/mild
Stage IIb intermittent claudication <200m/moderate-severe/incapacitating
Stage III rest pain
Stage IV ulceration/gangrene

Proposed WIFI Index

Based upon existing validated systems or best available data with 4 point scales where

0 = none

1 = mild-moderate

2 = moderate-severe

3 = severe or advanced

W: WOUND DEPTH [based on validated UT/PEDIS Wound Classification Systems]:

Grade	Depth/Description
0	no wound, pre-ulcerative state
1	superficial/shallow; no exposed tendon, capsule or bone
2	penetrates to tendon or joint capsule
3	penetrates to bone

I: ISCHEMIA (hemodynamics, physiology): use TP or $TcpO_2$ if ABI incompressible

Grade	ABI	TP, $TcpO_2$
0	≥0.90	≥ 60 mm Hg
1	0.6-0.89	40-59 mm Hg
2	0.4-0.59	21-39 mm Hg
3	<0.39 (*or ankle pressure < 50)	< 20 mm Hg

* BASIL TP = Toe pressure

FI: FOOT INFECTION: SVS Grades 0 (none), 1 (mild), 2 (moderate), 3 (severe)

Grade	Clinical Description	IDSA	IWGDF Class
0	wound without purulence or manifestations of infection	uninfected	1
1	>2 manifestations of infection (erythema or purulence, pain tenderness, warmth or induration) any cellulitis or erythema extends < 2cm around ulcer; infection is limited to skin or subcutaneous tissues; no local complications or systemic illness	mild	2
2	Infection in patient who is systemically and metabolically stable but has ≥1 of the following: cellulitis extending 2cm, lymphangitis; spread beneath fascia; deep tissue abscess; gangrene; muscle, tendon, joint or bone involvement	moderate	3
3	Infection in patient with systemic or metabolic toxicity	severe	4

G:- GANGRENE

Grade	Clinical Description
0	None
1	Ischemic rest pain- Pre-gangrenous skin changes Minor Gangrene - limited to mid-distal digits. simple toe amputation(s) likely goal, up to a single ray amputation
2	Moderate gangrene- > 2 toes, extending onto forefoot, likely to require TMA or more than single ray amputation
3	Major gangrene- extensive gangrene > 10 cm2 or forefoot or midfoot, likely to require Chopart or Lisfranc amputation to achieve healing; full thickness heel gangrene > 5 cm2 w/o calcaneal involvement

WIFI index is intended to be analogous to the TNM staging system for cancer

A patient with diabetes, a shallow superficial foot ulcer, early cellulitis and an ABI of 0.43 with a TP of 25 mm Hg would be classified as follows:

W1 I 2 FI 1 G 0 or WIFI-G 1210, with a Total Score of 4 (1+2+1=0)

The highest possible score would be in a patient with severe ischemia (TP 15 mm Hg), dorsal foot ulcer with penetration to bone, wet gangrene of 3 toes to

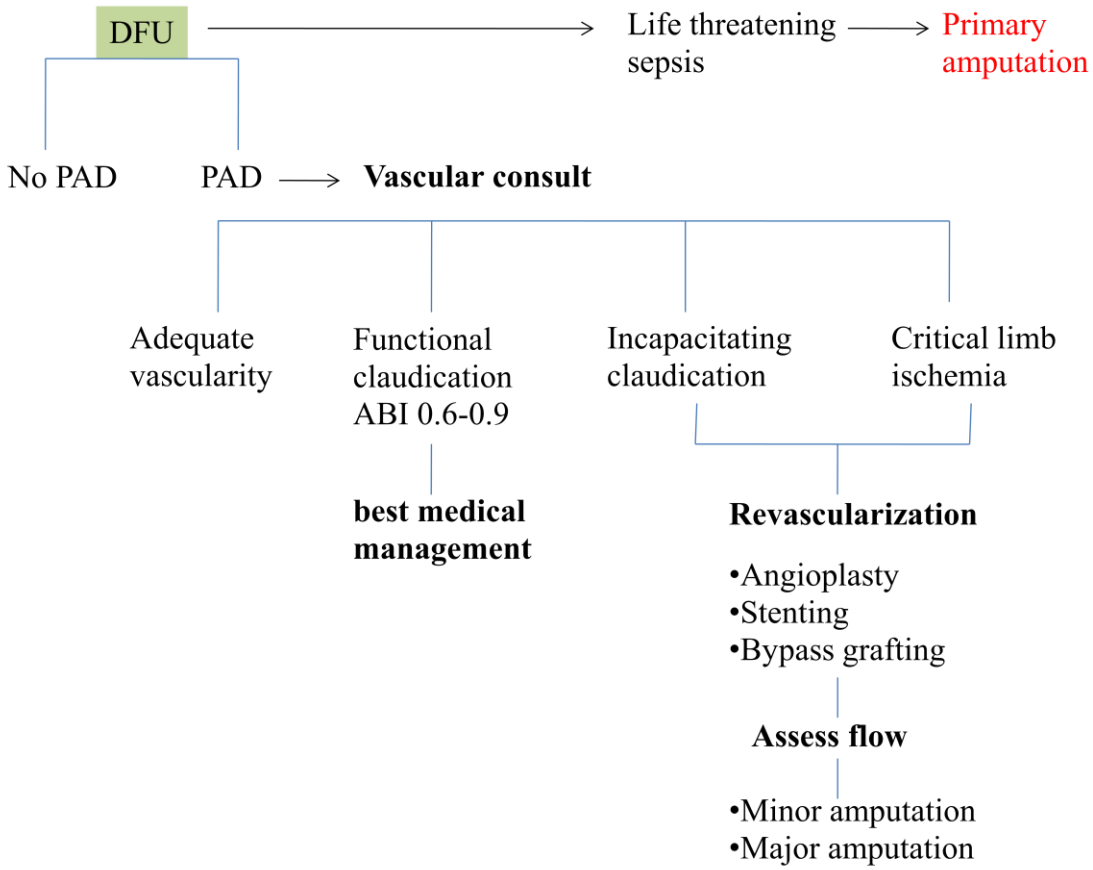
the bases and systemic sepsis: W3 I3 FI3 G3, WIFI-G 3333, generating a Total Score of 12

It might be useful to analyze outcomes based on total as well as fractionated scores

WOUND SWAB:

2 swabs were taken, one from the edge of the spreading ulcer and another from the depth of the wound. Swabs taken before empirical antibiotic administration and wound debridement.

WORKUP PROTOCOL USED IN STUDY:



RESULTS

The prevalence of PAD in DFU was found to be 36%(table 1, fig 1). It was more prevalent in males and in age>40 years and higher with increasing age(table 2). PAD was associated almost equally with plantar and dorsal ulcers, more often whole of foot was involved(p=0.008) (table 3, fig 2). There is significant association of PAD with high WIFI index(table 4,fig 3) and longer diabetic duration(p<0.0001) (table 7, fig 4) with mean disease duration of 10 years.

With only ABI as diagnostic criteria, occurrence of PAD was 28%, in another 7% cases ABI could not assessed due to non-compressibility. Combining with Doppler study, occurrence was 36%.

When >2 absent distal pulses by palpation alone was the criteria, occurrence of PAD was 24%. This could be due to human error, presence of associated pedal edema and presence of collaterals. Biphasic flow in Doppler was associated with only 74% of PAD(table 12, fig 8). This could be attributed to human error or hypodynamic circulation in septic patients.

Osteomyelitis is strongly associated with PAD(59%, p=0.003) (table 8, fig 5). PAD was associated with higher amputation rates(53.8%, p=0.003). Of minor lower limb amputations, only 47% were associated with PAD whereas of all major amputations, 87% were associated with PAD(p= 0.002) (table 14).

In a 3 month follow up, the ulcer healing was delayed in patients with PAD(~70 days) compared to non-PAD(~30 days).

36 DFU patients diagnosed with PAD were referred for vascular surgery consultation, out of which 10 patients had severe ischemia were taken up for revascularization. After a 3 month follow up, complete ulcer healing without any amputation occurred in 3 patients , 2 patients had toe amputation with complete healing of wound, one patient had a subsequent BKA with complete healing of the stump and one patient subsequently expired after angioplasty due to MI. 2 patients were lost to follow up. Therefore 60% limb salvage was possible.

Among the rest of 26 patients with PAD, 6 patients who were assessed to have adequate vascularity were put on best medical management and had complete ulcer healing.

Out of the 20 patients with PAD, 6 underwent toe amputation, one patient underwent forefoot amputation with complete healing, 9 patients underwent BKA with complete healing of stump, 3 patients underwent AKA with complete healing, and one patient was lost to follow up. Since it is a tertiary setting, the referral was late and hence primary amputation was done as a life saving measure in these patients.

Major amputations were associated more with plantar foot ulcer(16%) whereas minor amputations were associated more with dorsal foot ulcer(33%) (table 20). About 47% of ulcers involving whole of foot went in for major amputation while forefoot ulcers(33%) and midfoot ulcers(25%) commonly went in for minor amputations($p<0.0001$) (table 21).

Also higher amputation rates correlated with a high WIFI score($p<0.0001$) (table 16, fig 11) and higher Fontaine grades(table 17, fig 12). Mean diabetic duration(9.8 yrs) (table 16) was found higher in those who underwent amputation($p=0.006$).

Wound culture most commonly revealed a polymicrobial isolate(table 9, fig 6) followed by gram negative aerobes sensitive to Aminogycosides. Amikacin was sensitive to all the organisms in most of the cases(table 10, fig 7). Antibiotic resistance to Cefotaxime and Ceftriaxone was reported in few cases.

Table 1: Prevalence of PAD

		Count	Column N %
PAD	NO PAD	64	64.0%
	PAD	36	36.0%

Fig 1: Prevalence of PAD

The prevalence of PAD in DFU was found to **36%**. It was more prevalent in **males** and in **age>40** years and higher with increasing age.

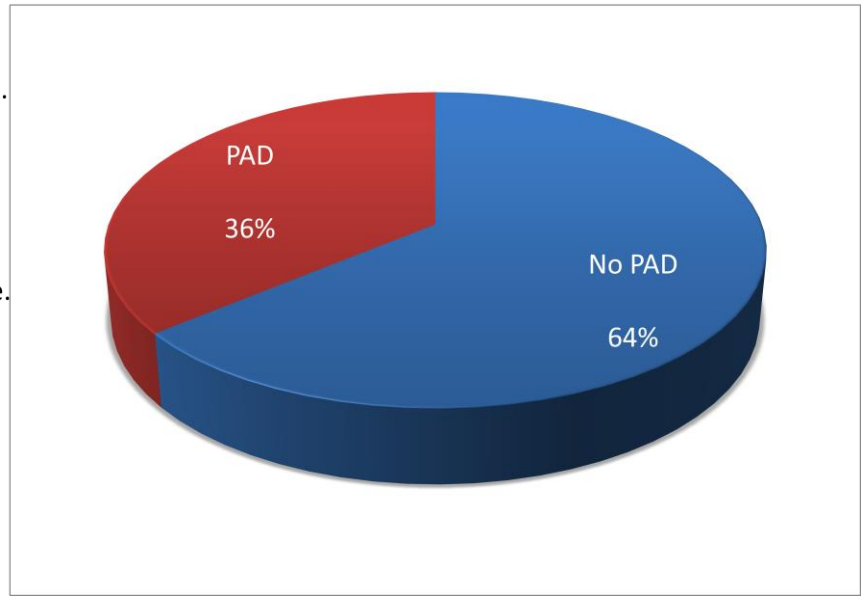


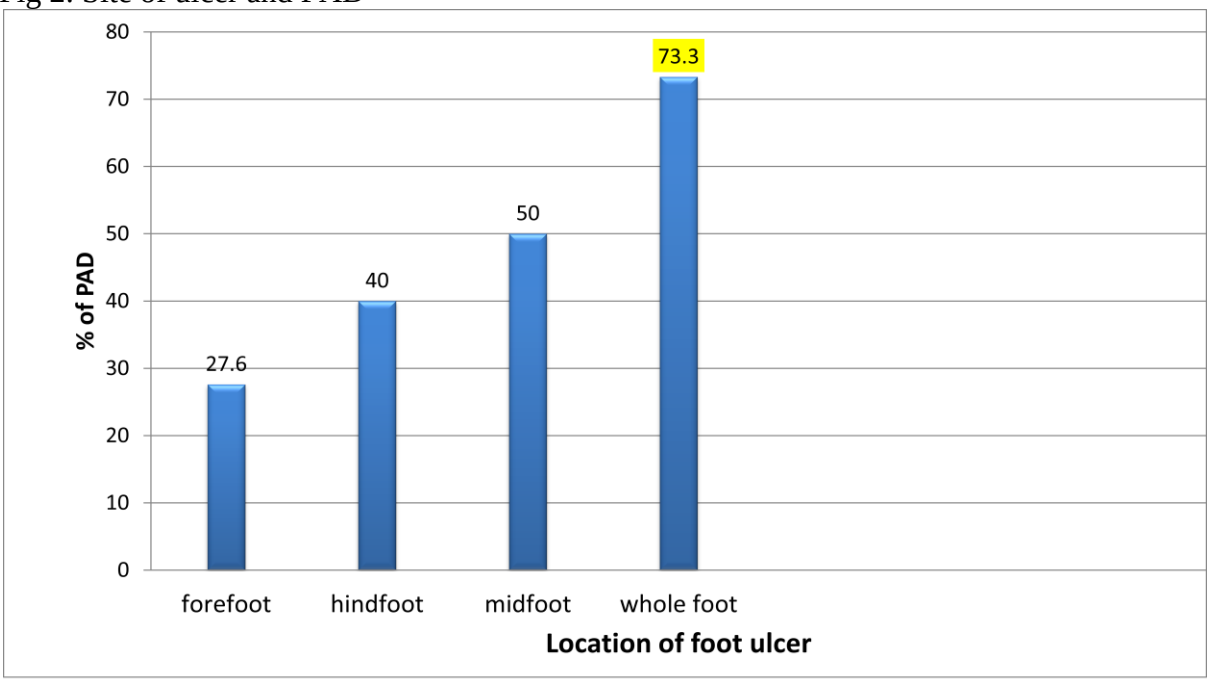
Table 2: Distribution of DFU

		PAD						
		NO PAD		PAD		Total		P
		Count	Row %	Count	Row %	Count	Column N %	
age	<40	4	80.0	1	20.0	5	5.0	0.2
	41-50	15	78.9	4	21.1	19	19.0	
	51-60	30	63.8	17	36.2	47	47.0	
	>61	15	51.7	14	48.3	29	29.0	
Sex	Male	44	65.7	23	34.3	67	67.0	0.6
	Female	20	60.6	13	39.4	33	33.0	
Education	10th	10	76.9	3	23.1	13	13.0	0.4
	5th	1	100.0	0	.0	1	1.0	
	nil	53	61.6	33	38.4	86	86.0	
Religion	christian	2	33.3	4	66.7	6	6.0	0.06
	Hindu	59	68.6	27	31.4	86	86.0	
	Muslim	3	37.5	5	62.5	8	8.0	
Socioeconomic status	low	60	63.8	34	36.2	5	5.0	0.9
	middle	4	66.7	2	33.3	19	19.0	

Table 3: Site of ulcer and PAD

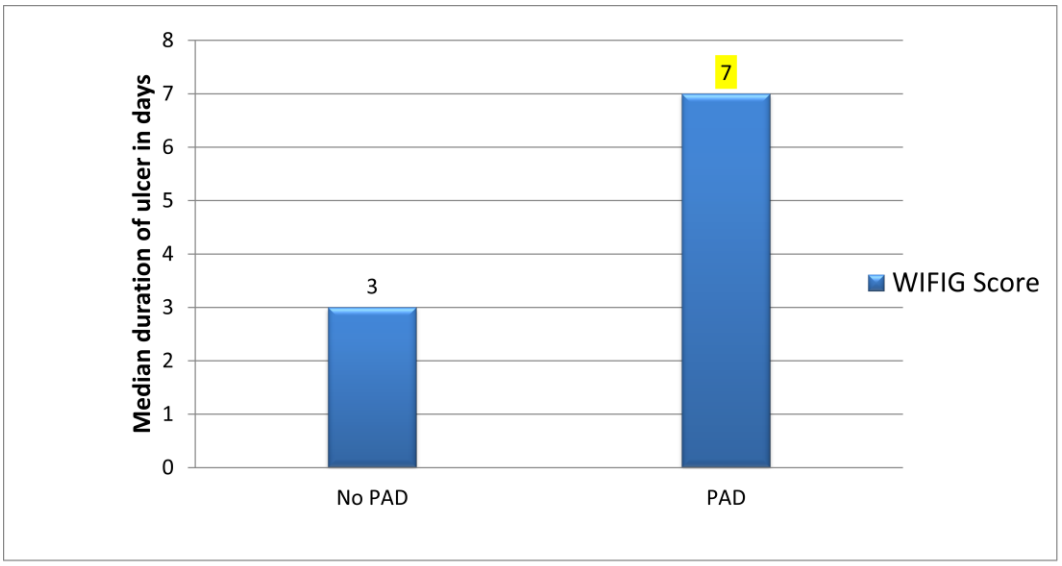
		PAD						
		NO PAD		PAD		Total		
		Count	Row N %	Count	Row N %	Count	columnN %	P
Side	left	32	65.3	17	34.7	49	49.0	0.8
	right	32	62.7	19	37.3	51	51.0	
dorsum_plantar	dorsum	53	65.4	28	34.6	81	81.0	0.5
	plantar	11	57.9	8	42.1	19	19.0	
Location of foot	forefoot	55	72.4	21	27.6	76	76	
	hindfoot	3	60.0	2	40.0	5	5	
	midfoot	2	50.0	2	50.0	4	4	
	whole foot	4	26.7	11	73.3	15	15	**0.008**

Fig 2: Site of ulcer and PAD



PAD was associated almost **equally** with plantar and dorsal ulcers, more often **whole** of foot was involved(p=0.008)

Fig 3: Ulcer duration and WIFI-G score correlation with PAD



	NO PAD (64)			PAD (36)			
	Mean	Median	SD	Mean	Median	SD	p
Ulcer durationdays	25.03	14.00	46.9	25.3	17.50	21.5	0.09
WIFIG Score	3.48	3.00	1.83	6.6	7.0	2.7	**<0.0001**

There is significant association of PAD with **high** WIFI index(p<0.0001)

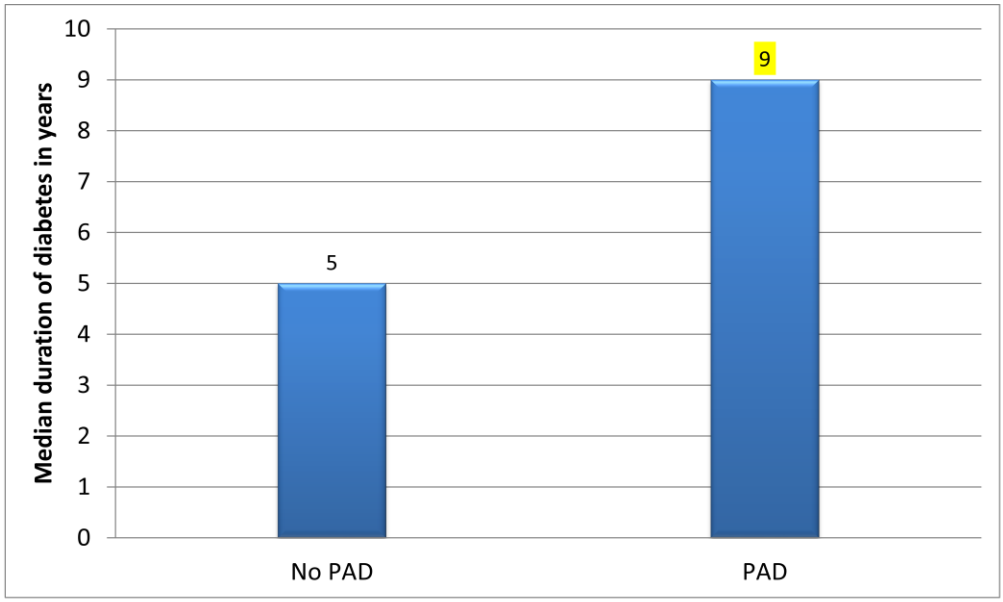
Table 5: Trauma, foot wear use and smoking in relation with PAD

		NO PAD		PAD		
		Count	Row N %	Count	Row N %	p
Trauma	no	55	64.0	31	36.0	0.9
	yes	9	64.3	5	35.7	
Footwear use	no	2	40.0	3	60.0	0.2
	yes	62	65.3	33	34.7	
Smoking	no	28	65.1	15	34.9	0.8
	yes	36	63.2	21	36.8	

Table 6: Co morbidities and PAD

		PAD				
		NO PAD		PAD		
		Count	Row N %	Count	Row N %	P
DM_Rx	no	9	60.0	6	40.0	0.8
	yes	54	64.3	30	35.7	
Hypertension	no	46	66.7	23	33.3	0.4
	yes	18	58.1	13	41.9	
CVA	no	64	64.6	35	35.4	0.2
	yes	0	.0	1	100.0	
CAD	no	60	64.5	33	35.5	0.7
	yes	4	57.1	3	42.9	
Nephropathy	no	61	64.9	33	35.1	0.5
	yes	3	50.0	3	50.0	
Retinopathy	no	64	64.0	36	36.0	
	yes	0	.0	0	.0	NA

Fig 6: Diabetic duration and PAD



	PAD						
	NO PAD			PAD			
	Mean	Median	SD	Mean	Median	SD	
Diabetes duration years	7.30	5.00	5.80	10.6	9.00	6.4	**0.007**

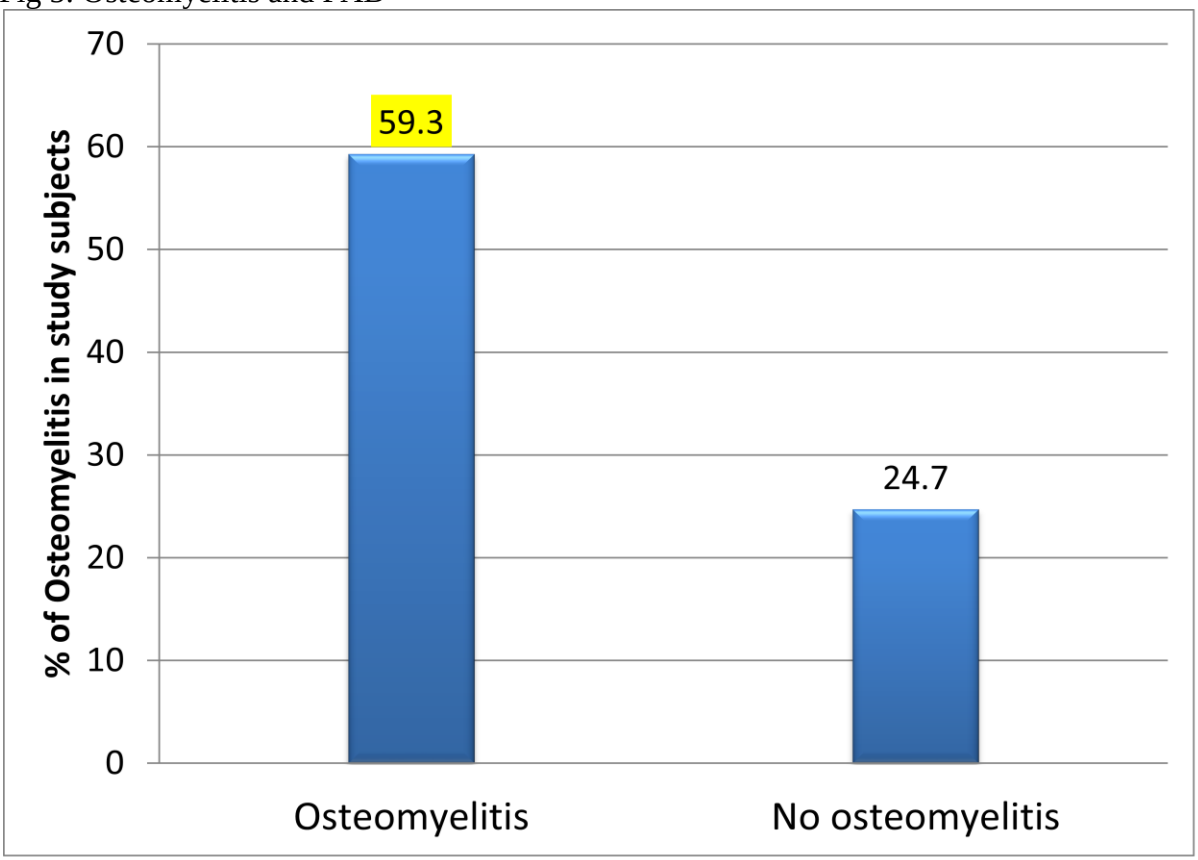
There is significant association of PAD with **longer** diabetic duration($p<0.0001$) with mean disease duration of **10 years**

Table 8: Osteomyelitis and PAD

		PAD			
		NO PAD		PAD	
		Count	Row N %	Count	Row N %
Osteomyelitis	no	53	72.6	20	27.4
	yes	11	40.7	16	59.3

P=0.003

Fig 5: Osteomyelitis and PAD



Osteomyelitis is **strongly** associated with PAD(59%, p=0.003).

Table 12: Distal pulses, Doppler flow and Fontaine grading in PAD

		PAD				
		NO PAD		PAD		
		Count	Row N %	Count	Row N %	p
Absent distal pulses	No	64	84.2	12	15.8	
	Yes	0	.0	24	100.0	**<0.0001**
Doppler flow	Normal	55	100.0	0	.0	
	No flow	0	.0	12	100.0	
	Monophasic	0	.0	4	100.0	**<0.0001**
	Biphasic	7	25.9	20	74.1	
Fontaine grade	.00	64	100.0	0	.0	
	1.00	0	.0	7	100.0	
	2.00	0	.0	10	100.0	**<0.0001**
	3.00	0	.0	8	100.0	
	4.00	0	.0	11	100.0	

Fig 8: Distal pulses, Doppler flow and Fontaine grading in PAD

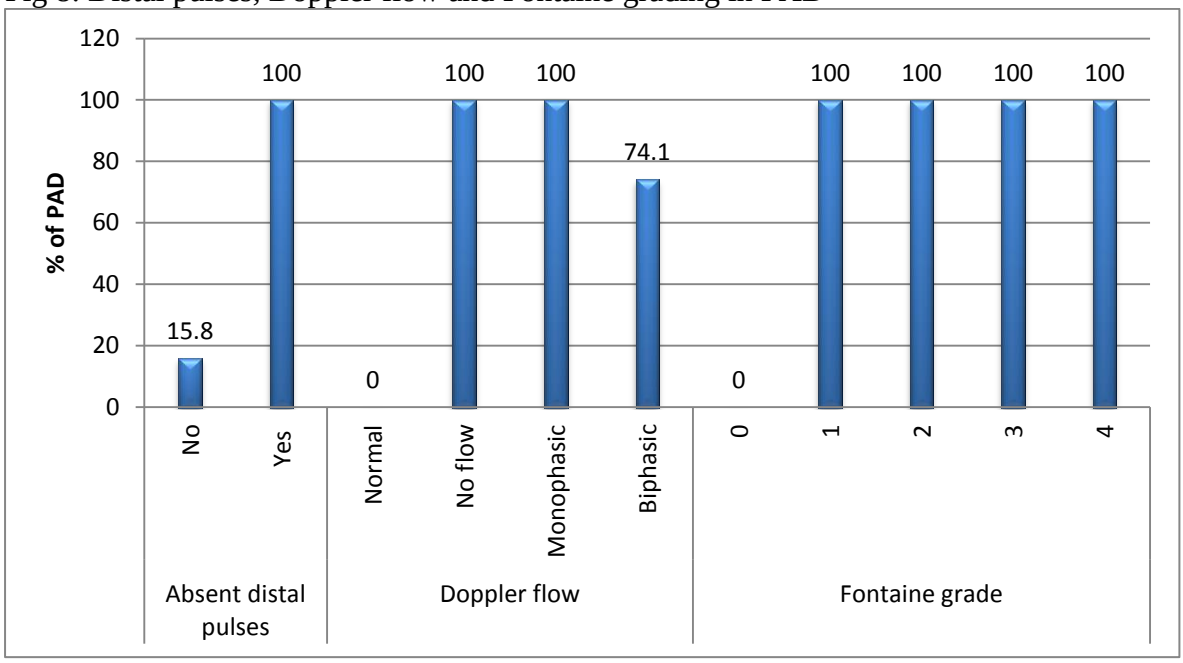
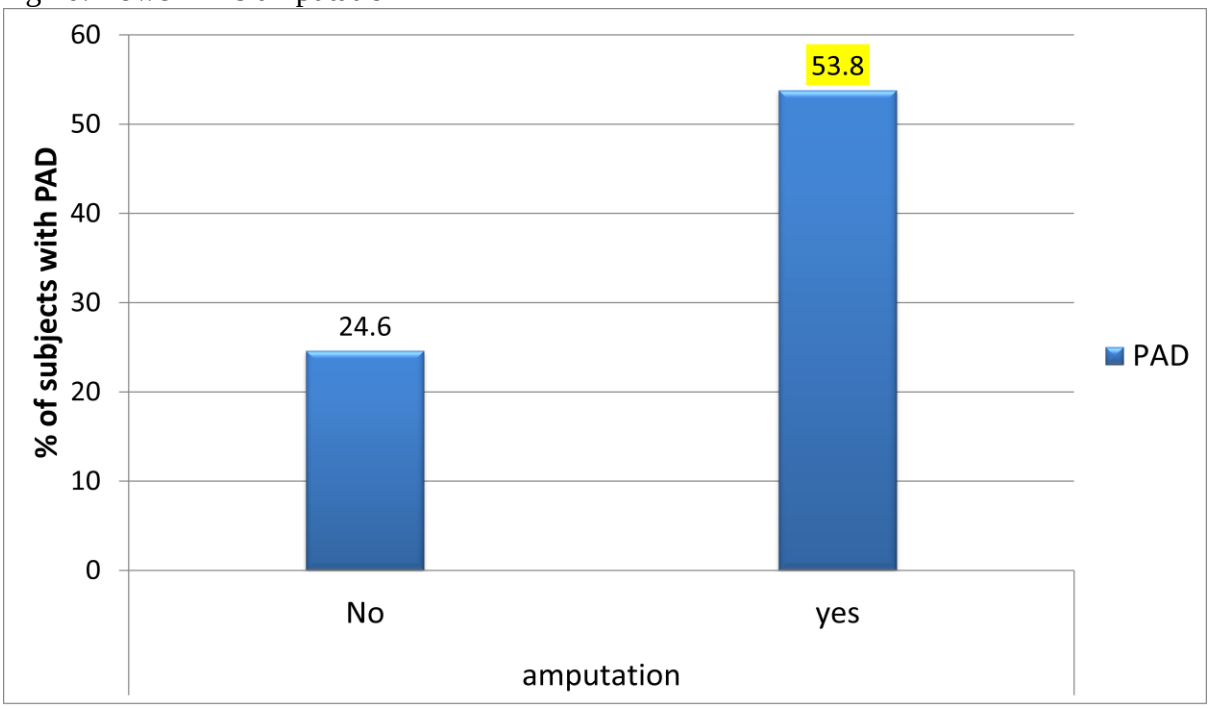


Table 15: Lower limb amputation in PAD

		PAD			
		NO PAD		PAD	
		Count	Row N %	Count	Row N %
amputation	no	46	75.4	15	24.6
	yes	18	46.2	21	53.8

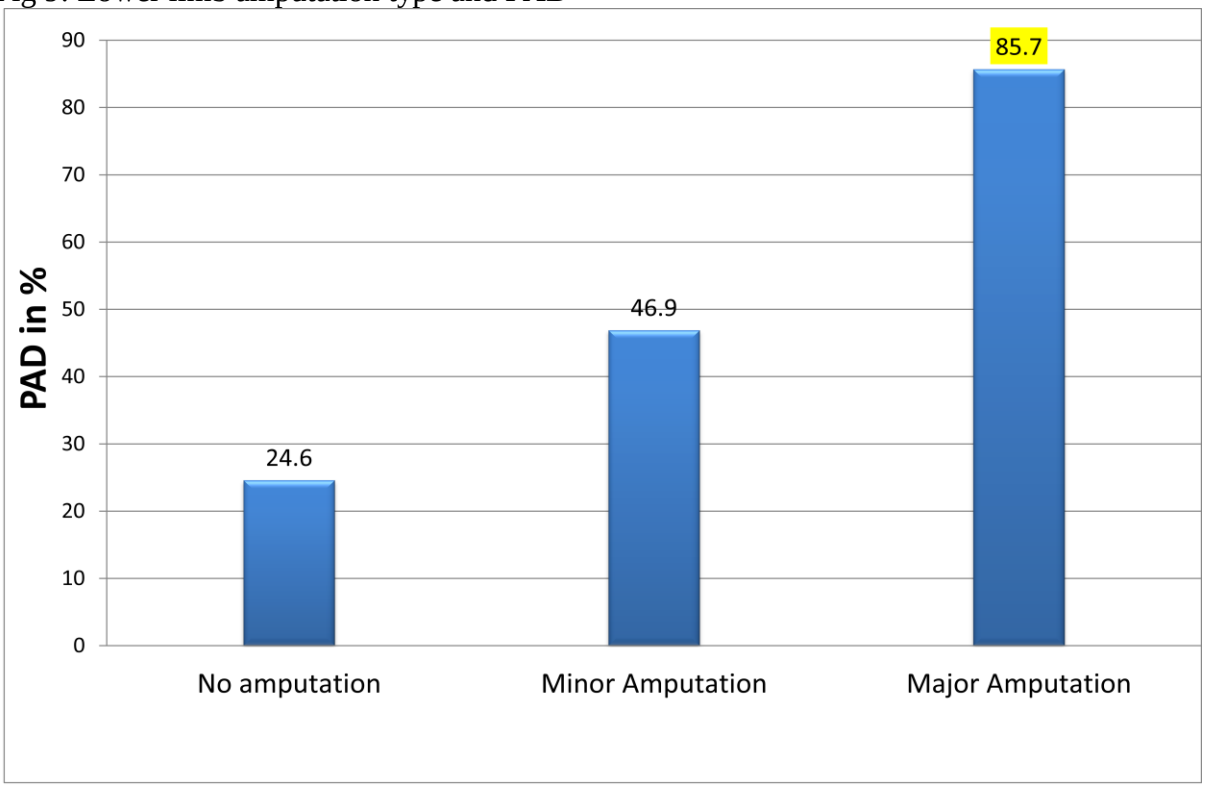
P=0.003

Fig 10: Lower limb amputation in PAD



PAD was associated with **higher amputation** rates(53.8%, p=0.003)

Fig 9: Lower limb amputation type and PAD



Of minor lower limb amputations, only 47% were associated with PAD whereas of all major amputations, **87%** were associated with PAD(p= 0.002)

Table 13: Lower limb amputation level in PAD

		PAD			
		NO PAD		PAD	
		Count	Row N %	Count	Row N %
Amputation	0	46	75.4	15	24.6
	Bka	1	16.7	5	83.3
	bka,aka	0	.0	1	100.0
	forefoot amputation	4	57.1	3	42.9
	toe amputation	13	52.0	12	48.0

P=0.01

Table 14: Lower limb amputation type and PAD

		PAD			
		NO PAD		PAD	
		Count	Row N %	Count	Row N %
ampute	No amputation	46	75.4	15	24.6
	Minor Amputation	17	53.1	15	46.9
	Major Amputation	1	14.3	6	85.7

P=0.002

Table 11: Neuropathy and venous disease associated with DFU

		PAD				
		NO PAD		PAD		
		Count	Row N %	Count	Row N %	p
Neuropathy	no	48	66.7	24	33.3	0.4
	yes	16	57.1	12	42.9	
venous disease	no	61	63.5	35	36.5	0.7
	yes	3	75.0	1	25.0	
PulseU#L#	no	0	.0	1	100.0	
	yes	64	64.6	35	35.4	0.2

Table 16: Correlation of WIFI-G score and diabetic duration with lower limb amputation in DFU

	Amputation						
	no			yes			
	Mean	Median	Standard Deviation	Mean	Median	SD	p
WIFIG Score	2.98	2.00	1.27	7.15	7.00	2.11	**<0.0001**
Diabetes_durationyears	7.6	5.0	5.9	9.8	8.0	6.5	0.06

Fig 11: Correlation of WIFI-G score with lower limb amputation in DFU

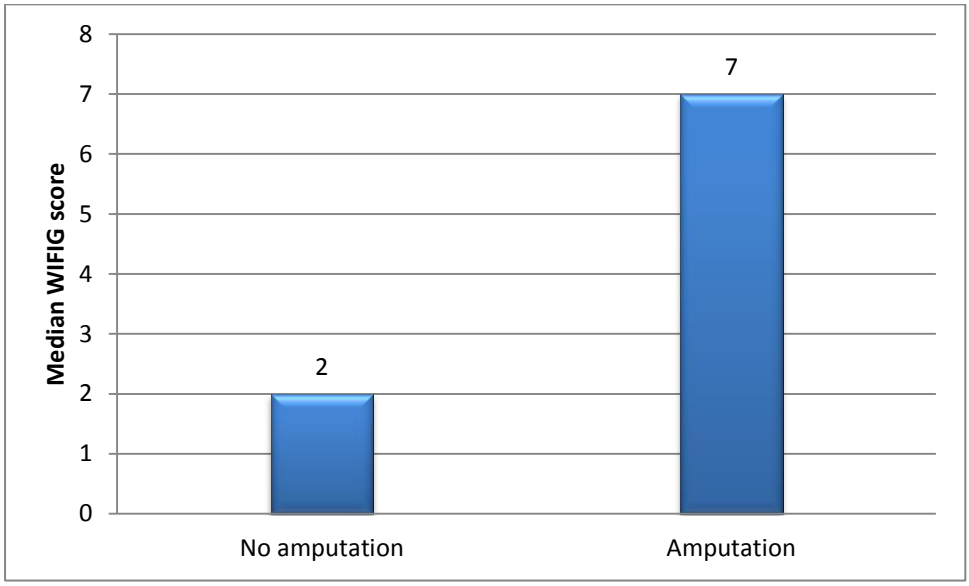


Table 17: Association of risk factors with lower limb amputation in DFU

		Amputation				
		No		Yes		
		n	%	n	%	P
Age	<40	2	40.0	3	60.0	0.2
	41-50	11	57.9	8	42.1	
	51-60	34	72.3	13	27.7	
	>61	14	48.3	15	51.7	
Sex	Male	43	64.2	24	35.8	0.6
	Female	18	54.5	15	45.5	
Fontaine grade	.00	46	71.9	18	28.1	
	1.00	5	71.4	2	28.6	**0.02**
	2.00	3	30.0	7	70.0	
	3.00	3	37.5	5	62.5	
	4.00	4	36.4	7	63.6	
CAD	no	57	61.3	36	38.7	0.9
	yes	4	57.1	3	42.9	
prev_amput	no	51	63.8	29	36.3	
	yes	10	50.0	10	50.0	0.3

Fig 12: Association of Fontaine grade with lower limb amputation in DFU

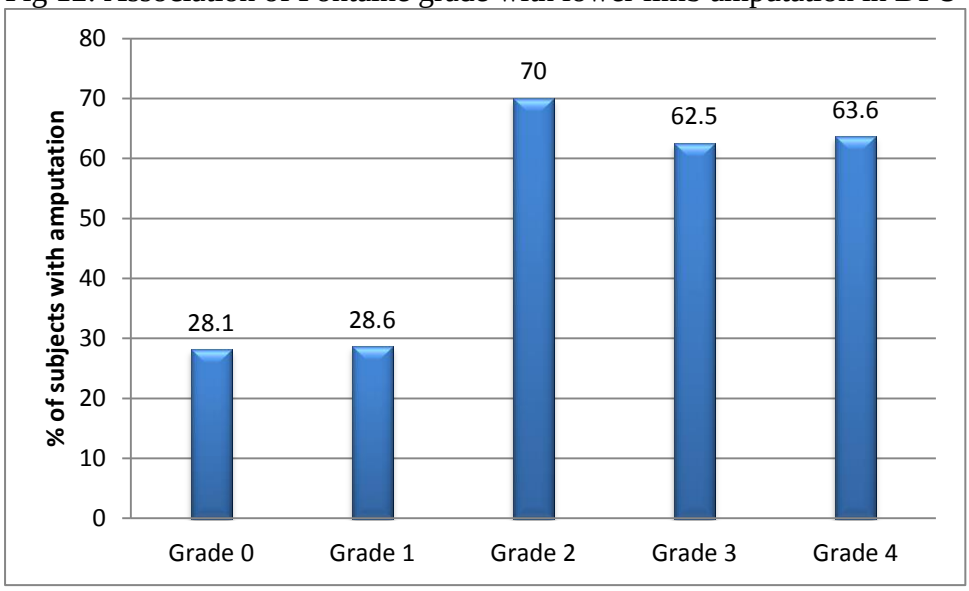


Table 18: Distribution of Osteomyelitis, lower limb amputation and bacteriology in DFU

		Count	Column N %
Osteomyelitis	no	73	73.0%
	yes	27	27.0%
amputat	no	61	61.0%
	yes	39	39.0%
organism	Polymicrobial	45	45.5%
	Klebsiella	47	47.5%
	Proteus	36	36.4%
	Ecoli	12	12.1%
	Pseudomonas	8	8.1%
	Acinetobacter	1	1.0%
sensitivity	Cefotaxime	14	14.3%
	ceftriaxone	10	10.2%
	cefperazone	4	4.1%
	ceftazidime	2	2.0%
	amikacin	64	65.3%
	imipenem	5	5.1%
	piptaz	6	6.1%

Table 20: Site of ulcer and amputation

		dorsum_plantar			
		dorsum		plantar	
		Count	Column N %	Count	Column N %
ampute	No amputation	50	61.7%	11	57.9%
	Minor Amputation	27	33.3%	5	26.3%
	Major Amputation	4	4.9%	3	15.8%

P=0.2

Table 21: Site of ulcer and amputation type

		locationoffoot							
		forefoot		hindfoot		midfoot		whole foot	
		Count	Column N %	Count	Column N %	Count	Column N %	Count	Column N %
ampute	No amputation	48	63.2%	5	100.0%	3	75.0%	5	33.3%
	Minor Amputation	28	36.8%	0	.0%	1	25.0%	3	20.0%
	Major Amputation	0	.0%	0	.0%	0	.0%	7	46.7%

P<0.0001

Table 22: Revascularization and amputation

		Revascular			
		No		Yes	
		Count	Column N %	Count	Column N %
ampute	No amputation	53	58.9%	8	80.0%
	Minor Amputation	30	33.3%	2	20.0%
	Major Amputation	7	7.8%	0	.0%

P=0.4

3 month follow up

PAD	Revascularization for severe ischemia(vascular consult)	Complete healing without amputation	Toe amputation	BKA	Expired (MI)	Lost to follow up	LIMB SALVAGE
36	10	3	2	1	1	2	60%

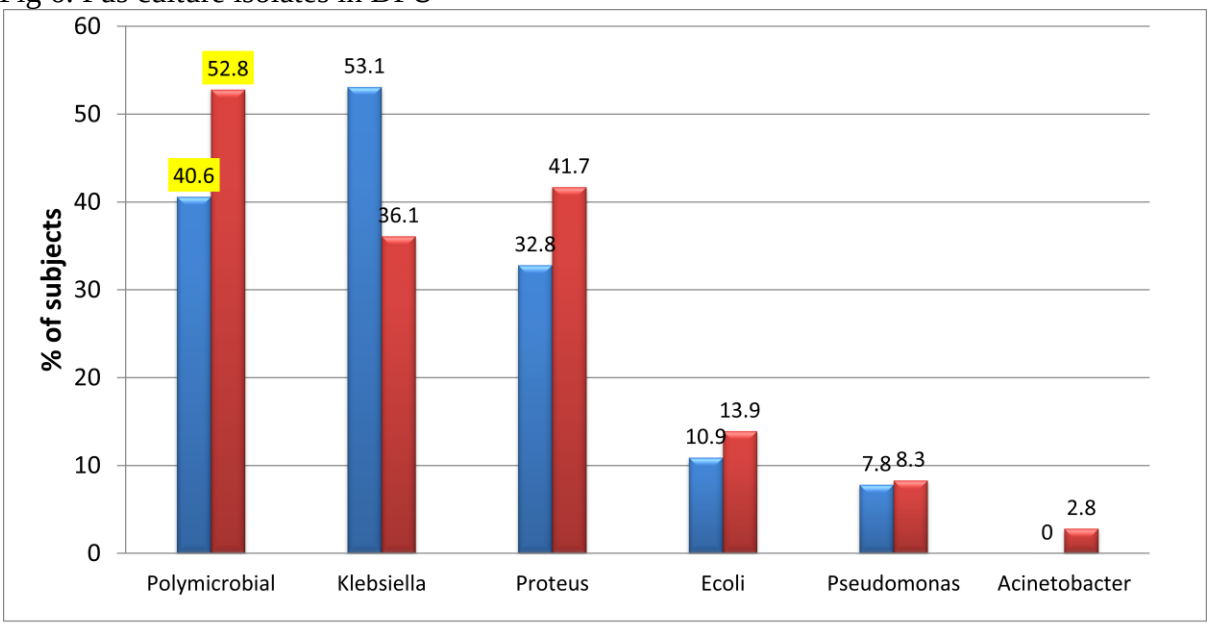
PAD	Best medical management	toe amputation	Forefoot	BKA	AKA	Lost to follow up	AMPUTATION RATE
26	6	6	1	9	3	1	65%

Since it is a tertiary setting, the **referral was late** and hence primary amputation was done as a life saving measure in these patients.

Table 9: Pus culture isolates in DFU

Organism	PAD				
	NO PAD		PAD		
	Count	Column Total N %	Count	Column Total N %	P
Polymicrobial	26	40.6	19	52.8	**0.3**
Klebsiella	34	53.1	13	36.1	0.2
Proteus	21	32.8	15	41.7	0.4
Ecoli	7	10.9	5	13.9	0.8
Pseudomonas	5	7.8	3	8.3	1
Acinetobacter	0	.0	1	2.8	0.4

Fig 6: Pus culture isolates in DFU



Wound culture most commonly revealed a **polymicrobial isolate** followed **by gram negative** aerobes sensitive to Aminogycosides.

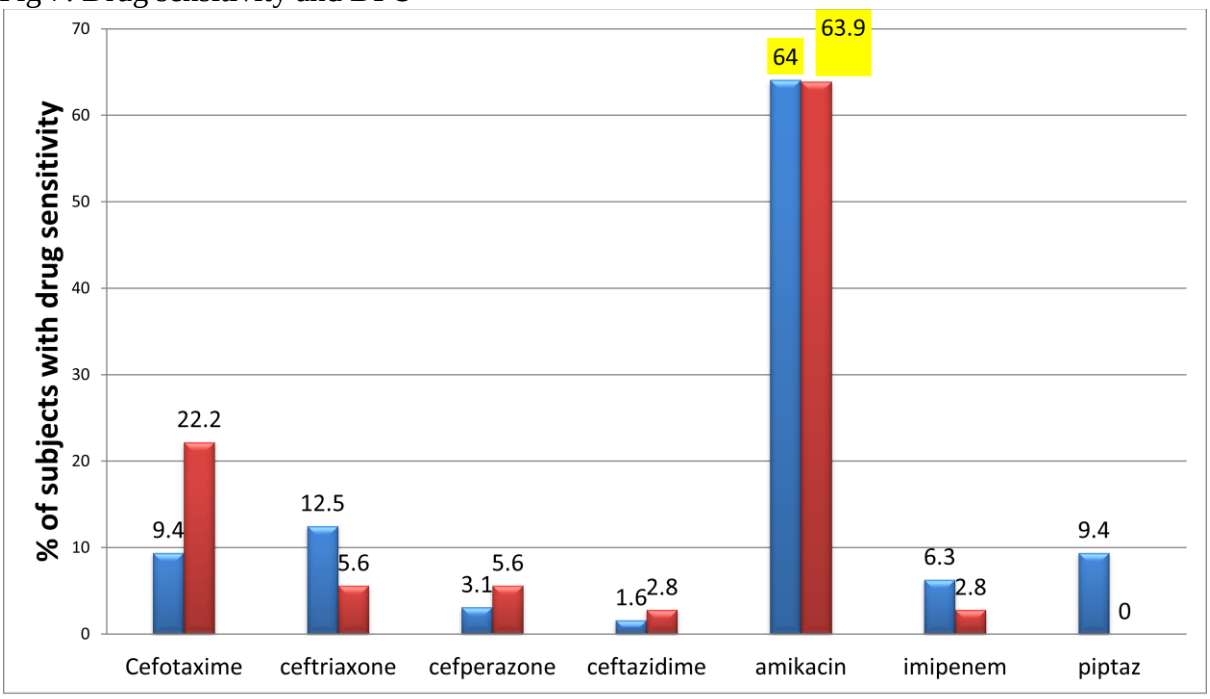
Table 19: Distribution of drug sensitivity in common culture isolates

		Sensitivity													
		Cefotaxime		ceftriaxone		cefperazone		ceftazidime		Amikacin		imipenem		piptaz	
		n	%	n	%	n	%	n	%	n	%	n	%	n	%
organism	Polymicrobial	7	16.3	2	4.7	1	2.3	1	2.3	27	62.8	5	11.6	3	7.0
	Klebsiella	9	19.6	5	10.9	2	4.3	1	2.2	32	69.6	2	4.3	0	.0
	Proteus	5	14.3	3	8.6	1	2.9	0	.0	22	62.9	2	5.7	4	11.4
	Ecoli	0	.0	2	16.7	0	.0	0	.0	8	66.7	1	8.3	1	8.3
	Pseudomonas	0	.0	0	.0	3	37.5	1	12.5	2	25.0	1	12.5	2	25.0
	Acinetobacter	0	.0	0	.0	0	.0	0	.0	1	100.0	0	.0	0	.0

Table 10: Drug sensitivity and DFU

Drug sensitivity	PAD				
	NO PAD		PAD		
	Count	%	Count	%	p
Cefotaxime	6	9.4	8	22.2	**0.1**
ceftriaxone	8	12.5	2	5.6	0.3
cefperazone	2	3.1	2	5.6	0.6
ceftazidime	1	1.6	1	2.8	1
amikacin	41	64.1	23	63.9	01
imipenem	4	6.3	1	2.8	0.7
piptaz	6	9.4	0	.0	**0.09**

Fig 7: Drug sensitivity and DFU



Amikacin was sensitive in almost all cases. Antibiotic resistance to Cefotaxime and Ceftriaxone was reported in few cases.

DISCUSSION

Previous studies aimed to study prevalence of PAD in diabetic population irrespective of foot ulcer. This study aimed to assess the burden of PAD in specific subset(DFU) of diabetic patients and the prevalence is 36%. A south Indian study(CUP) reported prevalence of peripheral arterial disease as 11.8%.

Mohan et al have reported on the prevalence of PAD in South Indian diabetics to be 3.9%. They compared their results with that of western studies, where the prevalence ranged between 22% and 45%.

Another study from South India reported a lesser prevalence of PAD (13%) among Indians.

A similar study from Greece reported prevalence of 42%.

In a study based on a tertiary setting in Eastern India by Sahana et al, 141 (34.4%) patients were diagnosed with PAD(ABI<0.9). PAD occurred in 46% of subjects with foot ulcerations compared to 32.7% of subjects without foot ulceration.

Mohan et al concluded that prevalence of PAD in diabetics increased with advancing age, with diabetic duration, from 15% at 10 years to 45% at 20 years from the diagnosis of diabetes.

In a study among Central Indian population by Pendsey et al, Diabetic duration<10 yrs had 30% associated PAD, whereas when duration was >10yrs, the association doubled to 60%.

Study	**Year**	**Occurrence of PAD**	
		In diabetes	**In diabetic foot ulcers**
Shojaie Fard et al.	2007		30%
Probal K. Moulik et al	2003		41%
Ikem R et al	2010		**25.7%**(>2 absent distal pulses) **55.4%** (ABI with hand held Doppler)
UKPDS	2008 U.K.	1.2%(at diagnosis) 11%(after 6yrs)	
Mohan et al	1995 South India	3.9%	
		X (10yrs) **3X** (20yrs)	
Pendsey et al	1997 Central India	3.9% X (10yrs) **2X** (>10yrs)	
Sahana et al	Eastern India	34.4%	46%

In our study, PAD prevalence was 36% which was higher in comparison to most of the other studies. This could be attributed to many causes including large percentage of patients with foot complications attending a **tertiary** care centre like our hospital, **specific** target population of DFU and **triple** assessment criteria.

	absent distal pulses	ABI	ABI + Duplex
PAD	24%	28%	**36%**
Non-compressibility		7%	
	Edema, Collaterals, human error		

In a study by Jyothylekshmy et al, Culture report on foot ulcer patients revealed that Gram-positive Staphylococcus species (18.8%) and the Gram-negative Pseudomonas species (18.2%) were the predominant organisms.

Gram-positive aerobic bacteria was reported to be the commonest isolate in diabetic foot infections in many studies but recent investigations reported a predominance of Gram-negative aerobes. Fluoroquinolones were the most commonly used empirical antibiotics in this study as it has broad spectrum of

activity, relatively safe, and cost effective antibiotic compared to the other antibiotics.

However in some studies, monomicrobial isolates were most common. This kind of discrepancy may be attributed to varying geography or infection severity in different hospitals included in the studies.

	Jyothylekshmi et al	Gadipalli	X	OUR STUDY
Isolate	Gram positive aerobic	Gram negative aerobic	Monomicrobial	**Polymicrobial** **Gram negative aerobic**

In this study, Cephalosporins followed by flouroquinolones was the most prescribed empirical antibiotic . The most common culture isolates were polymicrobial consisting of gram negative aerobes.

Taylor et al reported that ischemic patients without revascularization had the worst outcomes. Chang TY et al in a study in Taiwan reported a limb salvage rate of 36% in revascularized patients.

In a 3 month follow up in our study, the ulcer healing was delayed in patients with PAD compared to non-PAD. 60% limb salvage was possible with revascularization. The amputation rates were higher(65%) in the non-revascularized group.

Ulcer healing occurred in revascularized and non-revascularized group of patients and was quicker in the revascularized group(PAD). The mean hospital stay duration and drug expenditure was lower in the revascularized patients.

- Taylor et al reported that ischemic patients without revascularization had the **worst** outcomes.
- Chang TY et al in a study in Taiwan reported a limb salvage rate of 36% in revascularized patients.

- The **amputation rates were higher**(65%) in the non-revascularized group.
- **60% limb salvage** was possible with revascularization.

- **ulcer healing was delayed** in patients with PAD compared to non-PAD.
- Ulcer healing **occurred in both** revascularized and non-revascularized group of patients but was **quicker** in the revascularized group(PAD).

CONCLUSION

The prevalence of PAD in DFU is high in South Indian**.** The present study analyzed occurrence of PAD and various factors coexisting with DFU and PAD.

Increasing age and longer duration of diabetes are risk factors for peripheral arterial disease. High prevalence puts DFU at a higher risk of amputation.

The prevalence of PAD is found to be 36%. Patients with high risk factors associated with PAD like male, age>40 yrs, ulcer involving whole of foot, high WIFI-G score with diabetic duration> 10yrs, should be dealt with a strong suspicion of PADs

Strong association with osteomyelitis, higher amputation rates and longer hospital stay warrants a screening for PAD in all DFU.

WIFI-G scoring should be incorporated in the clinical assessment of wound due to its positive correlation with likelihood of amputation and PAD.

The use of hand held Doppler will aid early diagnosis in a reliable and cost-effective manner. Moreover, a triple assessment criteria involving distal pulse charting, ABI and Doppler study should be followed to properly diagnose a PAD at the earliest.

Based on the bacteriological data acquired by doing hospital based study, institutional antibiotic protocol should be formed for empirical treatment of the diabetic foot to counter the antibiotic resistance.

Once PAD diagnosed, patient should be counselled and categorized according to the appropriate management, and if found fit for revascularization should be done at the earliest onset to attain the benefit of limb salvage.

The patients who underwent revascularization had lower rates of major amputation and limb loss.

Nonetheless, in patients not fit for revascularization, the rates of major amputation were appreciable and ulcer healing was delayed.

The results conclude that peripheral arterial disease is a potential risk factor for delayed wound healing and major limb amputations in DFU.

Our data emphasizes the need for further dedicated research to identify and target this high risk population of DFU.

Strength and weakness of the study

- Since the study was conducted in a tertiary care centre, the number of patients with diabetic foot complications were high.
- Randomization was easier

- Neuropathic component was ignored
- Emergency revascularization procedures were not carried out.
- Not all patients were revascularized due to lack of awareness and ignorance, individual patient counselling was difficult.
- Follow up period was only 3 months.

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INSTITUTIONAL ETHICAL COMMITTEE, STANLEY MEDICAL COLLEGE, CHENNAI-1

Title of the Work : Assessment of peripheral arterial disease in diabetic foot ulcer : A prospective Study.

Principal Investigator : Dr. Vijayasarathy S.

Designation : PG MS (General Surgery)

Department : Department of General Surgery, Government Stanley Medical College, Chennai-01.

The request for an approval from the Institutional Ethical Committee (IEC) was considered on the IEC meeting held on 14.06.2016 at the Council Hall, Stanley Medical College, Chennai-1 at 2PM

The members of the Committee, the secretary and the Chairman are pleased to approve the proposed work mentioned above, submitted by the principal investigator.

The Principal investigator and their team are directed to adhere to the guidelines given below:

1. You should inform the IEC in case of changes in study procedure, site investigator investigation or guide or any other changes.
2. You should not deviate from the area of the work for which you applied for ethical clearance.
3. You should inform the IEC immediately, in case of any adverse events or serious adverse reaction.
4. You should abide to the rules and regulation of the institution(s).
5. You should complete the work within the specified period and if any extension of time is required, you should apply for permission again and do the work.
6. You should submit the summary of the work to the ethical committee on completion of the work.

MEMBER SECRETARY, 19/9/16
IEC, SMC, CHENNAI

MEMBER SECRETARY
ETHICAL COMMITTEE,
STANLEY MEDICAL COLLEGE
CHENNAI-600 001.

PROFORMA

S. No. DOA Contact:

Name Age/Sex IP. No.

Occupation

Education Religion Socioeconomic status

Site of ulcer:

Size of ulcer:

Duration of ulcer:

Wound depth Ischemia Foot infection Gangrene

WIFI-G score:

Previous hospitalization:

Trauma Smoking Hypertension CAD CVA

Retinopathy Nephropathy DM x

Footwear use

X-ray foot:

Wound culture: Organisms

Antibiotic

Bone culture: Organisms

Antibiotic

MRI Foot:

Neuropathy: Vibration sense 2-point discrimination:

Venous system: Superficial

Deep

Pulses: Right Bruit Pressure Left Bruit Pressure

U.L.

STA

CCA

SCA

AA

BA

RA

UA

L.L.

CFA

PA

PT

AT

DPA

ABI

Doppler/Duplex Scan:

CT Angiogram:

Limb ischemia/Fontaine:

Medical management: Heparin( ) Aspirin/Clopidogrel( ) Acitrom( )

Antibiotics:

Total duration:

Angioplasty/Stenting:

Debridements:

Amputation:

Minor

Major

Duration of hospital stay:

FOLLOW UP Clinical Ulcer ABI

Intervention

1st month

2nd month

3rd month

Limb Salvage:

Amputation free survival(3 months):

MALE:

MACE:

MASTER CHART

Name	Age	Sex	IP No	Ulcer Site	WIFI-G Score	DM(years)	Osteomyelitis	Culture yield	Polymicrobial
nelson	51	m	1673929	plantar,whole foot	9	20	n	Proteus	y
meenatchi	68	f	1608601	dorsum,forefoot	2	2	n	klebsiella	y
manjula	25	f	1604250	dorsum,whole foot	2	5	n	klebsiella	n
shanthakumari	41	f	1604067	plantar,midfoot	6	2	n	klebsiella	n
durairaj	60	m	1601581	dorsum,whole foot	12	5	y	klebsiella	n
velayudam	75	m	1601775	dorsum,forefoot	5	2	n	klebsiella	n
mangai	53	f	1604756	dorum,forefoot	9	15	y	Proteus	y
sakkubai	60	f	1687299	dorsum,forefoot	7	8	y	Proteus	n
chanbee	67	f	1611103	plantar,midfoot	8	8	n	klebsiella	n
sumathy	56	f	1611221	plantar,wholefoot	11	20	y	Proteus	y
govindaraj	37	m	1607832	dorsum,forefoot	8	4	y	klebsiella	y
kannabiran	38	m	1610375	dorsum,whole foot	6	8	n	Proteus	y
velayudam	50	m	1600518	dorsum,forefoot	6	5	y	Proteus	y
balakrishnan	48	m	1619793	dorsum,forefoot	6	8	y	klebsiella	n
vasu	55	m	1618161	plantar,hindfoot	2	10	n	klebsiella	n
mani	55	m	1614947	dorsum,forefoot	2	10	n	Proteus	n
mohan	63	m	1607396	dorsum,whole foot	2	13	n	Proteus	n
dharmaraj	60	m	1607823	dorsum,2nd toe	6	1	n	E.coli	n
henry	50	m	1600213	dorsum,5th toe	4	6	n	E.coli	n
munusamy	60	m	1611425	dorsum,forefoot	9	20	y	Acinetobacter	n
krishnamurthy	57	m	1609315	plantar,whole foot	9	10	y	E.coli	y
thomas	62	m	1608141	dorsum,forefoot	2	4	n	Proteus	n
mohan	55	m	1609456	dorsum,forefoot	2	5	n	E.coli	n
vasu	55	m	1627272	dorsum,hind foot	8	15	y	Proteus	n
jaya	64	f	1638102	dorsum,forefoot	4	3	n	klebsiella	n
yashoda	55	f	1610303	dorsum,forefoot	3	10	n	klebsiella	y
samikannu	85	m	1654353	plantar,hindfoot	2	10	n	E.coli	y
panneerselvam	56	m	1608995	plantar,forefoot	2	22	n	E.coli	y
anwar ali	42	m	1606588	dorsum,forefoot	5	5	n	Pseudomonas	y
rajendran	49	m	1689278	dosum,whole foot	8	8	n	Proteus	n
karunanidhi	59	m	1604972	dorsum,forefoot	3	18	n	Proteus	y

asothi	70	f	1608599	dorsum,great toe	9	5	y	E.coli	y
amsa	48	f	1609181	plantar,hindfoot	3	3	n	E.coli	y
anjalai	63	f	1673279	dorsum,whole foot	8	18	y	klebsiella	n
prema	65	f	1608102	dorsum,forefoot	6	8	y	klebsiella	n
sulochana	65	f	1673838	dorsum,great toe	7	10	n	Pseudomonas	n
avadiammal	72	f	1618835	dorsum,whole foot	9	14	y	Pseudomonas	n
ranjitham	63	f	1654232	dorsum,great toe	2	10	n	Pseudomonas	n
subamma	63	f	1653424	dorsum,great toe	4	10	n	Proteus	y
mary susila	54	f	1654322	dorsum,forefoot	3	5	n	Proteus	n
poornima	43	f	1678898	dorsum,forefoot	5	13	y	klebsiella	n
abdul	52	m	1604275	dorsum,forefoot	3	13	n	Proteus	y
alexander	65	m	1653433	plantar,whole foot	8	14	y	Proteus	y
nagarathnam	60	m	1638387	dorsum,forefoot	6	10	y	klebsiella	n
raniyammal	60	f	1613625	dorsum,whole foot	9	10	y	klebsiella	y
kanagammal	80	f	1672628	dorsum,great toe	10	5	y	klebsiella	y
arumugam	57	m	1621294	dorsum,great toe	6	5	n	Proteus	n
ganesan	63	m	1616716	dorsum,2nd toe	9	20	y	klebsiella	n
rangarajan	57	m	1619782	dorsum,5th toe	3	4	n	klebsiella	n
mani	48	m	1618198	plantar,hindfoot	2	3	n	Proteus	n
govindaraj	37	m	1620225	dorsum,forefoot	8	4	y	Proteus	y
lakshmanan	65	m	1618170	dorsum,forefoot	9	20	y	Proteus	y
desingh	67	m	1617984	dorsum,great toe	6	4	n	klebsiella	y
tamilarasan	50	m	1619166	dorsum,forefoot	2	10	n	Proteus	y
panneerselvam	55	m	1608445	plantar,great toe	2	22	n	Proteus	n
kuppan	45	m	1619907	dorsum,2nd toe	6	1	y	Pseudomonas	n
ragupathy	60	m	1615313	plantar,midfoot	4	25	n	Proteus	n
ganesan	52	m	1618851	dorsum,5th toe	2	15	n	klebsiella	n
vasudevan	64	m	1619942	dorsum,forefoot	2	12	n	klebsiella	y
kanniayappan	65	m	1672828	dorsum,forefoot	6	6	y	Pseudomonas	y
kannamal	60	f	1622780	dorsum,great toe	3	3	n	Proteus	y
Pushpa	53	f	1676551	dorsum,2nd toe	3	3	n	klebsiella	y
kamalanathan	70	m	1628937	dosum,hindfoot	9	2	n	klebsiella	y
balasubramaniam	58	m	1634232	dorsum,whole foot	4	8	n	klebsiella	y

sampath kumar	56	m	1629232	dorsum,forefoot	4	4	n	klebsiella,proteus	n
kannan	48	m	1626774	dorsum,forefoot	4	1	n	citrobacter	y
vasudevan	64	m	1619947	dorsum,forefoot	2	1	n	proteus	y
jayaraj	70	m	1629379	dorsum,forefoot	5	20	n	klebsiella	y
samidurai	60	m	1605804	dorsum,forefoot	2	3	n	klebsiella	y
hussain	55	m	1631559	dorsum,great toe	2	4	n	klebsiella	y
mahendran	51	m	1627078	dorsum,forefoot	2	20	n	klebsiella	n
kaliyamurthy	68	m	1631607	dorsum,forefoot	7	6	n	proteus	n
ellapan	63	m	1651216	dorsum,forefoot	2	3	n	klebsiella	y
meena	52	f	1676611	plantart,midfoot	5	1	y	proteus	n
maheshwari	40	f	1612820	dorsum,forefoot	4	5	n	klebsiella	n
meenakshi	60	f	1688800	dorsum,great toe	2	15	n	E.coli	n
govindamal	47	f	1630045	dorsum,forefoot	2	1	n	klebsiella	n
mary	55	f	1631445	dorsum,forefoot	2	15	n	klebsiella	n
prabakaran	55	m	1623422	plantar,forefoot	7	3	n	proteus,Ecoli	y
govindan	53	m	1627219	dorsum,forefoot	2	8	n	proteus	n
balasubramanian	54	m	1677282	dorsum,forefoot	2	15	n	klebsiella	n
saroja	70	f	1688732	dorsum,whole foot	11	8	y	Ecoli	y
devaraj	50	m	1677282	dorsum,forefoot	3	10	n	proteus	n
jaishankar	61	m	1626059	plantar,whole foot	7	25	y	klebsiella	n
sigamani	55	m	1673383	dorsum,forefoot	2	1	n	klebsiella	n
ameer basha	69	m	1689483	dorsum,forefoot	2	3	n	klebsiella	n
vatchala	55	f	1678283	dorsum,forefoot	3	7	n	klebsiella	n
prabakar	50	m	1683838	dorsum,forefoot	2	10	n	klebsiella	y
govind	45	m	1676262	dorsum,forefoot	2	2	n	klebsiella	n
palani	55	m	1637373	dorsum,forefoot	2	3	n	proteus	n
kuppurani	50	f	1679292	dorsum,forefoot	4	4	n	klebsiella	n
prabakaran	54	m	1633255	dorsum,forefoot	2	4	n	klebsiella	y
balasubramani	54	m	1654233	dorsum,forefoot	8	10	n	proteus	y
velu	60	m	1672822	dorsum,forefoot	2	5	n	proteus	y
nagamani	48	m	1673938	dorsum,great toe	4	6	n	klebsiella	y
bala	54	m	1683844	dorsum,forefoot	2	4	n	klebsiella	n
prabhu	55	m	1673637	plantar,forefoot	2	3	n	proteus	y
chellamal	55	f	1678992	plantar,whole foot	4	5	n	proteus	n
Basha	55	m	1653335	dorsum,forefoot	4	3	n	klebsiella	n
dhanalakshmi	45	f	1634234	plantar,forefoot	4	12	n	proteus	y

Name	absent distal pulses	ABI	Doppler study	PAD	Amputation	Revascularization	1st month	2nd month
nelson	y	0	no flow	y	aka	n		
meenatchi	n	nc	normal	n	n	n		healed
manjula	n	1	normal	n	n	n		healed
shanthakumari	y	0.8	biphasic flow in PT,AT	y	n	n		healed
durairaj	y	0	no flow	y	bka,aka	n		
velayudam	n	nc	normal	n	n	n		healed
mangai	y	0.6	biphasic flow in PT,AT	y	bka	n		
sakkubai	y	0.8	biphasic flow in PT,AT	y	toe amputation	n		healed
chanbee	y	nc	no flow	y	forefoot amputation	n		healed
sumathy	y	0.3	monophasic flow	y	bka	n		
govindaraj	n	1	normal	n	forefoot amputation	n		healed
kannabiran	y	0	no flow	y	n	y,	healed	
velayudam	n	nc	biphasic flow in PT,AT	n	toe amputation	n		healed
balakrishnan	n	1.3	normal	n	toe amputation	n		healed
vasu	n	1.2	normal	n	n	n		healed
mani	n	1	normal	n	n	n		healed
mohan	n	1.1	normal	n	n	n		healed
dharmaraj	y	0	no flow	y	n	y,	healed	
henry	y	0.5	monophasic flow	y	n	y,	healed	
munusamy	y	0	no flow	y	toe amputation	n		healed
krishnamurthy	n	0.8	biphasic flow in PT,AT	y	bka	n		
thomas	n	1	normal	n	n	n		healed
mohan	n	0.9	normal	n	n	n		healed
vasu	y	nc	biphasic flow	y	aka	n		
jaya	n	1.3	normal	n	n	n		healed
yashoda	n	nc	biphasic	n	n	n		healed
samikannu	n	1.3	biphasic flow in PT,AT	n	n	n		healed
panneerselvam	n	1	normal	n	n	n		healed
anwar ali	n	1	normal	n	toe amputation	n		healed
rajendran	y	nc	biphasic	y	bka	n		
karunanidhi	n	0.8	biphasic flow in PT,AT	y	n	n		healed

asothi	y	0	no flow	y	toe amputation	n		healed
amsa	n	1	normal	n	n	n		healed
anjalai	n	1	biphasic flow	n	bka	n		
prema	n	1.3	normal	n	toe amputation	n		healed
sulochana	y	0	no flow	y	toe amputation	n		healed
avadiammal	y	0.8	biphasic flow	y	bka	n		
ranjitham	n	1	normal	n	n	n		healed
subamma	y	0.6	biphasic flow	y	n	y,	healed	
mary susila	n	1	normal	n	n	n		healed
poornima	n	1.3	normal	n	n	n		healed
abdul	n	0.6	biphasic flow in AT,PT	y	n	y,	healed	
alexander	n	0.6	biphasic flow	y	toe amputation	n		healed
nagarathnam	n	1	normal	n	toe amputation	n		healed
raniyammal	n	0.8	biphasic flow	y	bka	n		
kanagammal	y	0	no flow	y	n	n		healed
arumugam	y	0	no flow	y	n	y,	healed	
ganesan	y	0.7	biphasic flow in PT,AT	y	forefoot amputation	y,	healed	
rangarajan	y	0.9	monophasic flow	y	n	y,	healed	
mani	y	1	monophasic flow	y	n	y,	healed	
govindaraj	n	1	biphasic flow	n	forefoot amputation	n		healed
lakshmanan	n	nc	biphasic	y	forefoot amputation	y,	healed	
desingh	y	0	no flow	y	n	n		healed
tamilarasan	n	1	normal	n	n	n	healed	
panneerselvam	n	1	normal	n	n	n	healed	
kuppan	n	nc	normal	n	toe amputation	n	healed	
ragupathy	n	nc	normal	n	n	n	healed	
ganesan	n	1	normal	n	n	n	healed	
vasudevan	n	1	normal	n	n	n	healed	
kanniayappan	n	0.9	normal	n	toe amputation	n	healed	
kannamal	n	1.1		n	n	n		healed
Pushpa	n	1		n	n	n		healed
kamalanathan	n	0.8	biphasic	y	bka	n		
balasubramaniam	n	0.8	biphasic dorsalis pedis	y	n	n		healed

sampath kumar	n	1.1	normal	n	toe amputation	n	healed
kannan	n	0.9	normal	n	toe amputation	n	healed
vasudevan	n	0.9	normal	n	n	n	healed
jayaraj	n	1	biphasic	y	toe amputation	n	healed
samidurai	n	1	normal	n	n	n	healed
hussain	n	0.9	normal	n	n	n	healed
mahendran	n	1	normal	n	n	n	healed
kaliyamurthy	n	nc	normal	n	forefoot amputation	n	healed
ellapan	n	1	normal	n	n	n	healed
meena	n	nc	normal	n	n	n	healed
maheshwari	n	1	normal	n	toe amputation	n	healed
meenakshi	n	nc	normal	n	n	n	healed
govindamal	n	1	normal	n	n	n	healed
mary	n	nc	normal	n	n	n	healed
prabakaran	n	1	normal	n	forefoot amputation	n	healed
govindan	n	1	normal	n	n	n	healed
balasubramanian	n	nc	biphasic	n	n	n	healed
saroja	y	0	no flow	y	bka	n	
devaraj	n	1	normal	n	n	n	healed
jaishankar	n	0.7	biphasic	y	toe amputation	n	healed
sigamani	n	1	normal	n	n	n	healed
ameer basha	n	1	normal	n	n	n	healed
vatchala	n	0.8	biphasic	y	n	n	healed
prabakar	n	nc	biphasic	n	n	n	healed
govind	n	1	normal	n	n	n	healed
palani	n	1	normal	n	n	n	healed
kuppurani	n	0.9	normal	n	toe amputation	n	healed
prabakaran	n	1	normal	n	n	n	healed
balasubramani	n	0.7	biphasic	y	bka	n	healed
velu	n	1	normal	n	n	n	healed
nagamani	n	1	normal	n	toe amputation	n	healed
bala	n	nc	normal	n	n	n	healed
prabhu	n	1	normal	n	n	n	healed
chellamal	n	1	normal	n	n	n	healed
Basha	n	nc	normal	n	toe amputation	n	healed
dhanalakshmi	n	nc	normal	n	n	n	healed

Name	3rd month				
nelson	healed	asothi		sampath kumar	
meenatchi		amsa		kannan	
manjula		anjalai	healed	vasudevan	
shanthakumari		prema		jayaraj	
durairaj	healed	sulochana		samidurai	
velayudam		avadiammal	healed	hussain	
mangai	healed	ranjitham		mahendran	
sakkubai		subamma		kaliyamurthy	
chanbee		mary susila		ellapan	
sumathy	healed	poornima		meena	
govindaraj		abdul		maheshwari	
kannabiran		alexander		meenakshi	
velayudam		nagarathnam		govindamal	
balakrishnan		raniyammal	healed	mary	
vasu		kanagammal		prabakaran	
mani		arumugam		govindan	
mohan		ganesan		balasubramanian	
dharmaraj		rangarajan		saroja	healed
henry		mani		devaraj	
munusamy		govindaraj		jaishankar	
krishnamurthy	healed	lakshmanan		sigamani	
thomas		desingh		ameer basha	
mohan		tamilarasan		vatchala	
vasu	healed	panneerselvam		prabakar	
jaya		kuppan		govind	
yashoda		ragupathy		palani	
samikannu		ganesan		kuppurani	
panneerselvam		vasudevan		prabakaran	
anwar ali		kanniayappan		balasubramani	
rajendran	healed	kannamal		velu	
karunanidhi		Pushpa		nagamani	
		kamalanathan	healed	bala	
		balasubramaniam		prabhu	

GOVT.STANLEY MEDICAL COLLEGE, CHENNAI- 600 001

INFORMED CONSENT

"Assessment of peripheral arterial disease in diabetic foot ulcer: A prospective study"

PLACE OF STUDY: GOVT. STANLEY MEDICAL COLLEGE, CHENNAI

NAME AND ADDRESS OF PATIENT:

I, _____ have been informed about the details of the study in my own language.
I have completely understood the details of the study.
I am aware of the possible risks and benefits, while taking part in the study.
I understand that I can withdraw from the study at any point of time and even then, I will continue to receive the medical treatment as usual.
I understand that I will not get any payment for taking part in this study.
I will not object if the results of this study are getting published in any medical journal, provided my personal identity is not revealed.
I know what I am supposed to do by taking part in this study and I assure that I would extend my full co-operation for this study.

Name and Address of the Volunteer: Name and signature of investigator:

Signature/Thumb impression of the Volunteer Date:

Date:

Witnesses:
(Signature, Name & Address)

Date:

அரசு ஸ்டான்லி மருத்துவகல்லூரி, சென்னை – 600001

Assessment of peripheral arterial disease in diabetic foot ulcer: A prospective study

நான் இந்த ஆராய்ச்சியில்விவரங்களை முற்றிலும் புரிந்து கொண்டேன்.

ஆய்வில் பங்கு எடுத்துபோது, சாத்தியமான அபாயங்கள் மற்றும் பயன்களை பற்றி நான் அறிந்துள்ளேன்.

நான் எந்தவொரு வேளையிலும் ஆய்வில் இருந்துதிரும்பமுடியும், அதன்பின்னர், நான்வழக்கம்போல் மருத்துவசிகிச்சை பெற முடியும் என்று புரிந்து கொள்கிறேன்

நான் ஆய்வில் பங்கு எடுத்து பணம் எதையும் பெறமுடியாது என்று அறிந்துள்ளேன். இந்த ஆய்வின் முடிவுகள் எந்த மெடிக்கல் ஜர்னலில் வெளியிடப்பட இருந்தால் நான் எதிர்க்கவில்லை, என் தனிப்பட்ட அடையாளத்தை வெளிப்படுத்தப்பட்டு இருக்ககூடாது.

நான் இந்த ஆய்வில் பங்கெடுப்பதன் மூலம் நான் என்ன செய்ய போகிறேன் என்று தெரியும்

நான் இந்த ஆய்வில் என் முழு ஒத்துழைப்பையும் கொடுப்பேன் என்று உறுதியளிக்கிறேன்.

தன்னார்வளர்	சாட்சி
பெயர் மற்றும் முகவரி	பெயர் மற்றும் முகவரி
கையொப்பம் / விரல் ரேகை	கையொப்பம் / விரல் ரேகை:

ஆராய்ச்சியாளராக
கையொப்பம் மற்றும் தேதி