"RECONSTRUCTION IN REVASCULARISED DIABETIC FOOT"

Dissertation submitted to

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In partial fulfillment of the regulation for the award

of the degree of

M.Ch.(PLASTIC AND RECONSTRUCTIVE SURGERY)

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CERTIFICATE

This is to certify that the dissertation entitled **"Reconstruction in Revascularised Diabetic Foot"** is a bonafide work done **DR.K.Adhil Ahamed Yameen**, post graduate (2011-2014) in the Department of Plastic, Reconstructive & Faciomaxillary Surgery, Madras Medical College & Rajiv Gandhi Government General Hospital, Chennai – 03, in partial fulfillment of the University rules and regulations for award of **Master of Chirurgiae**, **Plastic & Reconstructive Surgery (branch III)** degree under my guidance and supervision during the academic year 2011-2014.

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DECLARATION

I solemnly declare that this dissertation was "**RECONSTRUCTION IN REVASCULARISED DIABETIC FOOT**" one by me in the Department of Plastic, Reconstructive & Faciomaxillary Surgery, Madras Medical College & Rajiv Gandhi Government General Hospital, Chennai-03 between 2011 and 2014.

This dissertation is submitted to **THE TAMILNADU DR.M.G.R. MEDICAL UNIVERSITY, GUINDY, CHENNAI-32** in partial fulfillment of the university requirements for the award of degree of **M.Ch. PLASTIC & RECONTRUCTIVE SURGERY**.

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LIST OF ABBREVIATIONS

DNA	–	DeoxyriboNucleic Acid
HbA1c	–	Glycosylated Hemoglobin
PAD	–	Peripheral Arterial Disease
LDL	–	Low Density Lipoproteins
HDL	–	High Density Lipoproteins
RBC	–	Red Blood Cells
WBC	–	White Blood Cells
AGES	–	Advanced Glycation End Products
MP	–	Metatarso Phalangeal
IP	–	Inter Phalangeal
KOH	–	Potassium Hydroxide
ABI	–	Ankle Bachial pressure Index
DSA	–	Digital Subtraction Angiography
SSG	–	Split thickness Skin Graft

ALT	–	Antero Lateral Thigh
PT	–	Prothrombin Time
MRA	–	Magnetic Resonance Angiogram
LD	–	Latissimus Dorsi
RFF	–	Radial Free Forearm
G	–	Gracilis
PSF	–	Parascapular Flap
ALT	–	Anterolateral Thigh Flap
MP	–	Medial Plantar artery
SFC	–	Superiorly based Fasciocutaneous
IFC	–	Inferiorly Based Fasciocutaneous
RSSA	–	Reverse Superfical Sural Artery
PF	–	Perforator Flap

CONTENTS

S.NO	CONTENT	PAGE NO
1.	INTRODUCTION	1
2.	REVIEW OF LITERATURE	4
3.	MATERIALS AND METHODS	46
4.	RESULTS	53
5.	DISCUSSION	66
6.	CONCLUSION	71
7.	BIBLIOGRAPHY	
8.	ANNEXURES	
	PROFORMA MASTER CHART ETHICAL COMMITTEE APPROVAL CERTIFICATE PLAGIARISM DIGITAL RECEIPT PLAGIARISM SCREEN SHOT	

INTRODUCTION

Diabetes Mellitus is a disease with neurovascular and metabolic elements. These elements are related to each other. Changes in the metabolism of carbohydrate, proteins and lipids are secondary to absent or decreased secretion of insulin or due to ineffective action of insulin. Vascular syndrome is due to abnormalities in small vessels (microangiopathy).Neuropathic changes occur in peripheral nervous system due to metabolic and vascular causes.

About 366 million persons were affected by Diabetes Mellitus in the year 2011 and this will increase to about 552 million by the year 2030[1].One of the most common cause of hospitalization in diabetics is due to foot infections. Foot lesions develop in 25% of this group during their period of life[2]. One of the largest diabetic populations in the world is currently in India. Overall incidence of diabetes in India is 1.2 %[3] of which incidence in urban population is 4 to 11% and incidence in rural population is 2.4%[4].Diabetes Mellitus Prevalence increases after the age of 40 years,15 to 20% increase after the age of 65 years.

Diabetics battle numerous complications related to their underlying disease but none is more devastating both psychologically and economically than gangrene of extremity with associated risk of amputation. Diabetic peripheral polyneuropathy is the major cause of diabetic foot wounds. In patients with neuropathy non healing ulcers precede to 80% of amputation. The risk of amputation increases by eight times in patients with diabetes of age more than 45 years and twelve times more in patients aged above 65 years [3,4.]If an amputation is done in one limb, the incidence of second amputation in contralateral limb is about 50% in 2 years. In India most of the people are barefoot walkers. Without knowing that they have lost sensation and protective reflexes, they walk and have repeated trauma and foot gets infected. These groups have to be counseled regularly to prevent the recurrence of the wounds on the foot. Charcot's deformity of the joints of the foot occurs in 2.5% of the diabetic population.

The reconstruction of diabetic foot after the revascularisation is a challenge. To do this a complete knowledge of the vascular anatomy, disease pathology, nature of rehabilitation and reconstructive procedures is necessary. Functional reconstruction means to reconstruct the mechanical design to bear weight. This

includes reconstruction of skin to the bones of the foot. The decreased vascularity of the foot should be recognized first as this delays the normal healing of the wound. Next step is revascularization and establishment of wound healing potential.Revascularisation is done by endovascular or surgical techniques. The first option is endovascular. Regional Vascular deficiency following revascularization is corrected and then the functional reconstruction is carried out. Regional Vascular Insufficiency is failure of wound healing due to perfusion deficiency attributed to diabetic functional micro angiopathy in the presence of good macro vascular blood flow after revascularization.

Limb loss makes the life difficult for Indian people by reducing their income. The cost of health care and burden over the families of diabetic foot patients is huge therefore it is necessary to study the methods of foot salvage by reconstruction procedures instead of amputation.

REVIEW OF LITERATURE

HISTORY OF DIABETES MELLITUS

History of Diabetes can be divided into four periods[5].

1. Ancient period
2. Diagnostic period
3. Empirical treatment period
4. Modern period

1. ANCIENT PERIOD (UPTO 1675 AD)[6]

An Egyptian Papyrus (1500 BC), was discovered at Luxor in Egypt in the year 1872 by George Ebees.This journal has record the symptoms of passing frequent and large quantity of urine.Diabetes was understood in ancient India. Charaka(300 BC) in his charaka samhita mentioned about the sweetness of urine in addition to the symptom of polyuria.Sushruta in 500 BC described Diabetes as “Madhu Meha” – Honey urine. He also described the symptoms of polyphagia,polydipsia and polyuria. They noted that this disease was prevalent in people who were indolent, overweight and gluttons.

Chen Chen in 7 th century described as sugary urine. The first description of diabetic gangrene was given by Avicenna (980 – 1037),an Arabian physician. He described the condition as sugary urine and causing impotence and gangrene.Aretaecus of Cappodocia(2 AD) gave description of the disease as melting down of flesh and limbs into urine.Aretaeus described the term "Diabetes" means siphon in Greek. This disease causes fluid to go out of the body and uses the body like a channel to exit.

2. DIAGNOSTIC PERIOD (1675 – 1796)

Thomas Willis in the year 1675 AD described that the urine of a diabetic patient was wonderfully sweet.Mortan in the year 1678 AD described the hereditary nature of Diabetes.Mathew Dobson in the year 1784 proved that sweetness of urine is due to sugar.Thomas Cawly in the year 1778 described in fatal cases of Diabetes had abnormal changes in pancreas.Cullen (1709 – 1790) added the adjective "Mellitus" to the disease to distinguish it from Diabetes Insipidus .Sugar from urine was isolated by adding yeast to urine by Francis.

3. EMPIRICAL TREATMENT PERIOD (1796 – 1850 AD)

In the year 1796 Rollo advised a diet which was restricted to low food value foods of green vegetables and animal fat. Little

bread and milk was permitted occasionally. This was done to combat diabetes. Smell of acetone in the breath of young diabetic patients was noticed by Kussmaul.In the year 1850 Michel Eugene Chevreul showed that the sugar in diabetics urine is glucose.

4. MODERN PERIOD (1850 ONWARDS)

M.Gregor in this period demonstrated fermentable substance in diabetic patients.Qualitative test for sugar in the urine of diabetics was described by Trammer.Acetone was discovered in the urine of diabetic coma patients by Peter in the year 1857 AD.All diabetics are due to pancreatic abnormality was Baumel's hypothesis.Bouchardet (1806 – 1886) advised the daily testing of urine for sugar. The features of diabetes mellitus was demonstrated in two pancreatomised dogs in the year 1889 by Joseph,Yon Mering and Oscar Minkowski.

Paul Langerhans,a German medical student noted that the pancreas contains two different groups of cells.The acinar cells which secrete the digestive enzymes and cells that are clustered in islands. These cells served a second function. In the year 1891 – 1894, Laguesse described the collection of interacinar cells as a gland of secretion within the pancreas. This was later named as "Islets of Langerhans". In the year 1850 Fehling reported his

quantitative test for urine sugar. In the year 1908,Benedict introduced Benedict's test for diagnosing urine sugar in diabetics.Belgram Meyers first named the secretion as "Insulin" in 1909.The classical treatment for diabetes mellitus was written by Elliot .P. Joslin in the year 1915.

Fredrick Banting, a Canadian orthopedic surgeon who was interested in diabetes along with Charles H.Best, a second year medical student worked in the laboratory of Prof.J.J.R.Macleod in Toronto[7]. Insulin was discovered in the year 1922 by Banting and Best and they won Nobel Prize for it.Banting and Best with the help of J.B.Collip, a skilled chemist produced consistently successful preparation of insulin for the treatment of diabetes in humans. This revolutionized the treatment of diabetes mellitus and the complications. The action of insulin was prolonged by adding protamine and thereby reducing its immediate action by Hagedorn Era (1937 – 43).Zinc was added to insulin by Fischer and Scott. Lente insulin was developed by Halease and Moller et al.In the year 1982, Human insulin with recombinant DNA technology was prepared by Elli Lilly and Goeddal. Oral hypoglycemic drugs came into use around 1957.

During the 19th and 20th centuries lower limb disease in the diabetic patients was described as ‘gangrene in the diabetic foot’ or ‘diabetic gangrene’. The differentiation between gangrene with infection in a limb with normal vascularity and gangrene due to vascular insufficiency was made around 1893.Before the advent of insulin, surgery was a hazardous task in diabetics. Insulin discovery and its use reduced the need for surgical intervention in diabetics. Insulin therapy replaced hyperglycemic coma with diabetic foot as the major cause of mortality in diabetics. Some hospitals in North America initiated prophylactic care and patient education of diabetic foot. The role of neuropathy in diabetic foot was neglected as ischemia and infection was considered to be the cause of diabetic foot at that time[8].

PATHOPHYSIOLOGY OF DIABETIC FOOT

Diabetics are more prone for foot problems.

Predisposing factors

- Changes in vascularity
- Neuropathic changes
- Hyperglycemia
- Infections

HYPERGLYCEMIA

Hyperglycemia causes the complications to develop in the diabetics through the pathways.

Polyol pathway

Due to hyperglycemia, the sorbitol increases and accumulates in the cell. It inhibits competitively in the uptake of myoinositol.Glucose is shunted through this pathway leading to the poor utilization of pyruvate thereby decreasing the production of energy. This is called hyperglycemia causing pseudo hypoxia.

Protein Glycation

The reaction between sugars and amino groups produces complex glycation products known as Advanced End products of glycation.This causes damages to the endothelium increases the permeability of the endothelium. This also promotes chemotaxis of macrophages and increases the growth factors. Increased cross linking of collagen, serum protein trapping leading to enzymatic degradation is seen.

2. CHANGES IN VASCULARITY

Atherosclerosis of blood vessels causes ischemia in diabetic foot. Peripheral vascular disease of lower limb is a common factor causing poor wound healing leading to ulcers, gangrene, and amputation[9].

It commonly occurs as

- Peripheral Arterial Disease
- Functional Microangiopathy
- Hemorrheological abnormalities

Peripheral Arterial Disease

Peripheral Arterial Disease is accelerated by hyperglycemia. Every 1% increase in levels of HbA1c leads to 25 to 28 % increase in the relative risk of PAD.Long standing diabetics for more than 10 to 15 years have atherosclerotic changes in arteries below the level of Profunda Femoris characterised by multiple segmental involvement.Peroneal and Tibial arteries are mostly affected between knee and ankle.Dorsalis Pedis artery and other foot vessels are not usually involved. Establishment of collateral circulation in arteries around knee joint is decreased in diabetes [9].

Risk factors

- Hypercholesterolemia – Hypertriglyceridemia,

Decreased levels of HDL

Increase in cholesterol lecithin ratio

- Hypertension
- Tobacco use

PATHOGENESIS

Increased non enzymatic glycosylation of lipoproteins causes impairment of LDL binding to the receptors. Glycosylation of LDL increases the formation of cholesterol esters and human macrophage accumulation which is characterized by formation of foam cells in early atheromatous lesions[10]

Functional Microangiopathy

Hyperglcemia leads to thickened basement membrane in the capillaries and small vessels. This induction of enzymes glycosyl and galactosyl transferase incorporates carbohydrates in basement membrane. Increased hydrostatic pressure in the most dependent portion causes basement membrane thickening. Though basement membrane thickening is seen in glomerulus but not evident in small vessels of foot. Failure to dilate and undergo compensatory hyperplasia (Functional microangiopathy) is the only abnormality made out in diabetic foot patients.

Chronic hyperglycemia causes nonenzymatic glycation of hemoglobin causes increased affinity for oxygen and decreased oxygen release. This causes tissue ischemia leading to endothelial damage in capillaries .Non enzymatic glycation of spectrin caused by hyperglycemia leads to decreased flexibility of RBC negation of FARAH LINDQVIST effect leading to accumulation of RBCs and thereby increasing blood viscosity at arteriolar end leading to decreased perfusion causing endothelial damage in capillaries.

Due to persistent hyperglycemia endothelial cell dysfunction and smooth cell abnormalities develop in peripheral arteries[11] resulting in the decrease in endothelium-derived vasodilators which causes vasoconstriction. This hyperglycemic in diabetes also causes increased thromboxane A2 levels which are a vasoconstrictor and platelet aggregator. This in turn increases the risk for plasma hypercoagulability[12].

Hemorrheological abnormalities

Functional alterations in capillary blood flow are seen in diabetics which leads to diabetic complications. Blood and serum viscosity is increased. This causes flow abnormalities in WBCs,RBCs and platelets. Altered flow characteristics due to stiffened RBC membrane and increased RBC aggregation changes

the viscosity and causes compensatory rise in perfusion pressure leading to increased transudation across capillary beds increasing the viscosity further.

NEUROPATHIC CHANGES

55% of diabetics are affected by neuropathy. Neuropathy incidence increases along with duration of disease. Neuropathic episode increases with episodes of hyperglycemia and duration of disease. Neuropathy has sensory, motor and autonomic components.

Sensory neuropathy

Proposed mechanisms are

i) Nitric oxide blocking

Hyperglycemia, dyslipidemia, insulin resistance and stress oxidation all lead to cell damage and endothelial dysfunction and other diabetic complications. Hyperglycemia blocks endothelial nitric oxide synthetase activation. This leads to increase in the levels of reactive oxygen – superoxide. Superoxide dismutases enzymatically convert the superoxide into hydrogen peroxide. Hydrogen peroxide is converted to hydroxyl radical which is highly reactive and damaging in the presence of ferrous or cuprous ions.Peroxy nitrate is produced by the binding of superoxide anion

to nitric oxide, which is a potent vasodilator. This decreases the bioavailability of endothelium derived potent vasodilator.Sulfhydrl groups in proteins is oxidized by peroxy nitrate anion which also does lipid peroxidation and generation of reactive aldehydes/N2O along with the proatherogenic low density lipoproteins production. Endothelium regulated vascular function disruption causes vasoconstriction, platelet aggregation, intimal growth abnormalities, thrombus formation, inflammation[13].Vascular wall structural protein lipoxidation and glucoxidation causes increase of atherogeny through inflammatory cytokine interaction and vessel wall characteristic effect. Neuropathy is caused by peripheral nerve small vessel (vaso nervorum) atherogenesis.

(ii)Mailard reaction

It is slow but complex reaction that leads in production of Advanced Glycation End products (AGES), by reacting between biomolecules of amino groups and reducing sugars. This reaction is considered in diabetic pathophysiology complications to be important. This reaction links to protein modification in diabetics and in aging process. Lipoproteins and proteins modified by Advanced Glycation End products causes' atherosclerosis. The polyol metabolic pathway converts excess glucose into sorbitol by

aldose reductase. This consumes NADPH (Nicotinamide Adenide Dinucleotide Phosphate) Hexosamine biosynthetic pathway activation reduces Nicotinamide Adenide Dinucleotide Phosphate. This limits NADPH conversion from Nicotinamide Adenide Dinucleotide through inhibition of Glucose 6 Phosphate Dehydrogenase enzymatic activity causing decreased Nicotinamide Adenide Dinucleotide Phosphate. Decreased NADPH reduces glutathione synthesis which is an important antioxidant[13]

Increased production of reactive 02 and antioxidant depletion causes diabetic foot complications. Neuropathy causes limited joint mobility leading to foot deformity. Neuropathy results in callus formation due to increase in foot pressure on the pressure points. This causes increase in local pressure which along with repeated injuries causes local necrosis and ulceration.

(iii)Neuropeptides

Nerve Growth substance factor P, Calcitonin gene related peptide are neuropeptides that are affected by diabetic neuropathy. Growth factor production, cell chemotaxis is caused by these neuropeptides which increases the proliferation of cells. Immune defense mechanism is modulated by sensory nerves. This is evident by decreased leukocyte infiltration in denervated skin.

Screening of sensory neuropathy is by physical examination, monofilament test for sensory perception –Testing of ten sites with monofilament placed on intact skin. Absence of sensation in 3 to 4 sites indicates sensory neuropathy. Tuning fork test with 128 Hz fork for vibration sense perception.

Motor neuropathy

Motor neuropathy is due to unequal muscle pull. First muscle to be affected is plantar muscle. Distal motor loss causes muscles of the dorsum of foot to be pulled unequally and the difference in pressure causes cocked up toe. Additional deformity like claw toe and hammer toes are also caused by difference in pressure. Claw toe is proximal phalangeal dorsiflexion on the lesser metatarsophalangeal joints along with proximal and distal interphalangeal joint flexion. Hammer toe has proximal MP joint deformity and IP joint deformity resulting in a hammer like flexure. Hammer toe deformity increases the prominence of metatarsal head on the plantar side with claw toes.

Ill-fitting shoes on these deformities causes break down of skin on the increased pressure points. Due to dropping of the metatarsal heads there is herniation of the corresponding fat pads distally into the base of the toes. Collapsed bone on the plantar side

acts as pressure point causing formation of callus on metatarsal heads. Risk of ulceration is greatly increased with callus. Connective tissue stiffness due to hyperglycemia causing joint function impairment and reduces the range of motion as seen in equinus deformity.Equinus deformity with ankle joint dorsiflexion restriction causes fixed toes. This increases the pressure in the forefoot and toe on the plantar side. Motor neuropathy is assessed by ankle reflex test.

Autonomic neuropathy

Autonomic neuropathy involves sympathetic nervous system causing decreased sweating, opening of arterio venous shunts leading to altered blood flow regulation and precapillary sphincter malfunction. Features of autonomic neuropathy are skin changes, nail changes. This reduces blood flow and presents as warm, dry skin. This leads to skin break down.

Skin changes

Anhidrosis with dry skin and fissures, this should be differentiated from fungal infection and other dry plantar skin etiologies. Fungal intertrigo is commonly seen in fourth and fifth web space.

Nail changes

Distal streaking of nail are changes seen in fungal infections like complete asymmetry of the nail plate. Fungal infections are assessed by KOH microscopic examination and culture.

Charcot foot

It is the late complication of peripheral neuropathy due to repeated trauma of insensitive bones and joints in diabetic foot.

MECHANISMS

Neurotrauma:

Repeated micro trauma due to peripheral sensory loss and loss of proprioception damages joint. This is not noticed by the diabetic neuropathic patient.This causes inflammatory resorption of bone that is traumatised leading that region to weakness Decreased motor control increases the pressure on certain joints causing microtrauma.

Neurovascular:

Autonomic neuropathy in diabetic foot patients along with sensory neuropathy in joints causes dysvascularity leading to hyperemia. This increases osteoclastic resorption of bone leads to destruction of bone.

Joint Involvement

Most common joint involved in charcot foot is tarsometatarsal joint wherein the medial joint is more affected than lateral joint in about 60 percentage. Metaatarsophalangeal involvement is about 30%.Ankle involvement in 10%. Most of them have undergone some form of minor trauma.

Clinical features

- Redness,
- Swelling,
- Deformity of foot.
- Increased temperature
- Foot ulcer
- Dislocation of bones and joints without trauma

Classification of Charcot foot

Sanders and Frykberg have classified the Charcot's foot based upon the neuropathic osteo-arthropathy.They described the pattern of joint and bone destruction.

1. Forefoot is mainly involved seen in 26 -67%, Metatarsal head ulcers are seen
2. Midfoot collapse.It is seen in 15 – 48%.It involves tarsometataarsal joints

3. Midfoot collapse.It is seen in 32%.It involves calcaneocuboid, Naviculocuneiform, and talonavicular joints.
4. Unstable Ankle seen in 3 to 10% of the patients .It involves subtalar and ankle joint.
5. Calcaneal involvement. It is rarely seen.

Dounis classification of neuro-osteoarthropathy(1997)

Type I : Forefoot involvement

Type II: Midfoot collapse

Type III: Rear foot is involved and is subclassified as :

IIIa: Ankle instability

IIIb : Varus deformity.Involves Subtalar joint

IIIc : Loss of weight bearing

In acute phase, it is difficult to distinguish cellulitis, Charcot's foot and osteomyelitis. Radiography of Charcot shows Fractures, Bone destruction, periosteal new bone formation. Healing is delayed taking about 6 to 9 months .In this period the foot becomes distorted into clinically visible Charcot foot – Rocker bottom foot deformity.

Eichenholtz's Classification describes the nature of joint destruction process clinic- radiologically.

Stage 0: Joint edema is seen clinically, but x rays are negative.

Stage 1(Developmental stage): Dislocation of joints with fragmentation of bones seen on x ray.

Stage 2(Coalescence stage): Local edema is decreased, Fragments coalese along with absorption of bony debris.

Stage 3(Reconstructive stage): Local edema is absent, but fracture fragments undergone consolidation and remodeling with deformity. The foot is stable.

4. INFECTIONS

Infection is defined as the presence of a pus secretion or 2 symptoms or signs of inflammation like palor, rubor, dolor, calor. Diabetic patients are more prone for infections than normal individuals. In normal people the flora in the leg and foot are restricted due to

I. Less than optimum skin temperature for the survival of human pathogens.
II. Antimicrobial effect of skin metabolic products
III. Dorsum of foot and leg have acidic surface
IV. Thick stratum corneum

Most common infections seen in diabetics are bacterial and fungal.

RISK FACTORS

- Vascular insufficiency
- Peripheral Neuropathy leads to repeated trauma
- Decreased host resistance
- Decreased leukocyte mobilisation
- Defect in formation of reactive oxygen metabolites

Common organisms are aerobes and anaerobes. Usually the infections in diabetes mellitus are polymicrobial.

1. Gram - ve bacilli

- Proteus mirabilis
- Escherichia coli
- Pseudomonas aeroginosa
- Enterobacter aerogenes

2. Gram +ve cocci

- Staphylococcus aureus
- Group B streptococcus
- Enterococcus
- Peptostreptococcus magnus
- Peptostreptococcus.anaerobes

3. Anaerobes

Gram negative bacilli

- Bacillus Fragilis
- Bacillus ovatus
- Bacillus ureolyticus

GRAM POSITIVE BACILLI

Clostridium bifirmentans Of all these organisms, Staph aureus and beta hemolytic strepytococcus,coagulase negative staphylococcus are most important. Ischemia and neuropathy both mimic inflammatory signs, so the following signs like friable tissue, undermined wound edge, foul smelling discharge indicate infection. Diabetic foot infections mostly do not cause systemic manifestations like pyrexia or leukocytosis. If they are present they indicate the severity of infections. Infection can be a major threat to limb and life if not treated aggressively. Superficial cultures from wound are not helpful .Tissue specimens are preferred to wound swabs as they are more sensitive and specific. Life threatening infections is characterised by bullae,ecchymoses, soft tissue crepitus and ascending infection progressing to necrotising fascitis caused by multiple bacteria,aerobes,anaerobes,microaerophilic bacteria.Necrotising fascitis spreads rapidly along the fascial plane

involving the perforator supplying the skin. This leads to ischemia and gangrene of skin and fascia. This is associated with high mortality due to septicemia. Other common problem in diabetic foot is osteomyelitis. It is difficult to distinguish osteomyelitis from arthropathy.X ray changes in osteomyelitis are focal osteopenia,lucency of the cortex or medullary bone. In Chronic osteomyelitis sequestration of dead bones is seen.

CLASSIFICATIONS OF DIABETIC FOOT

Once diabetic foot ulcers develop clinical staging is important. Most common classification used for diabetic foot ulcer is Meggit Wagner classification.This classification is based on wound depth, presence and location of wound infections. Grades range from 0 to 6.University of Texas classification is used commonly in wound care clinics[13]

Meggit Wagner classification

Grade 0 – No ulcer in high – risk foot

Grade 1 – Superficial ulcer involving the full skin thickness but not underlying tissues

Grade 2 – Deep ulcer, penetrating down to ligaments and muscle, but no bone involvement or abscess formation

Grade 3 – Deep ulcer with cellulitis or abscess formation, often with osteomyelitis

Grade 4 – Localized gangrene

Grade 5 – Extensive gangrene involving the entire foot

University of Texas Classification

Grade I A – Noninfected,nonischemic superficial ulceration

Grade I B – infected, nonischemic superficial ulceration

Grade I C – ischemic, noninfected superficial ulceration

Grade I D – Ischemic and infected superficial ulceration

Grade II A – Noninfected, nonischemic ulcer that penetrates to capsule or bone

Grade II B – Infected,nonischemic ulcer that penetrates to capsule or bone

Grade II C – Ischemic, noninfected ulcer that penetrates to capsule or bone

Grade II D – Ischemic and infected ulcer that penetrates to capsule or bone

Grade III A - Noninfected, nonischemic ulcer that penetrates to bone or a deep abscess

Grade III B – Infected, nonischemic ulcer that penetrates to bone or a deep abscess

Grade III C – Ischemic, noninfected ulcer that penetrates to bone or a deep abscess

Grade III D - Ischemic and infected ulcer that penetrates to bone or a deep abscess

PEDIS classification

It is developed by International Working Group of Diabetic Foot based on the following categories.(Research oriented)

- Perfusion
- Extent
- Depth/tissue loss
- Infection
- Sensation

Infectious Diseases Society of America 2004 guidelines

Diabetic foot wound with infections are sub classified as mild, moderate and severe.

Mild – Skin and subcutaneous tissue only involved with more than 2 manifestations of inflammation (purulence or erythema,pain,tenderness,warmth or induration);any cellulitis or erythema extends < 2cm around the ulcer; no local complications or systemic illness

Moderate – affecting deeper tissues or extensive involvement. Infection in a patient who is systematically well and metabolically stable but has more than 1 of the following: cellulitis extending >2cm;lymphangitis;spread beneath fascia; deep tissue abscess;gangrene;muscle,tendon,joint, or bone involvement.

Severe – Systemic signs of infection or metabolic instability. Infection in a patient with systemic toxicity or metabolic instability (eg, fever, chills, tachycardia, hypotension, confusion, vomiting, leukocytosis, acidosis, hyperglycemia or azotemia)

S(AD)AD classification grades 5 categories – Size, (Area,Depth), Sepsis, Arteriopathy and Denervation on a four point scale – 0 to 3.

INVESTIGATIONS

Blood tests – Fasting blood sugar > 120 mg

Postprandial blood sugar >180 mg indicative of diabetes

Glycosylated Hemoglobin assay - HbA1c – Hemoglobin undergoes nonenzymatic condensation of glucose of free amino groups of globin molecule,due to chronic hyperglycemia.So higher the level of hyperglycemia higher the level of glycosylated hemoglobin. Since the half-life of RBC is 120 days this reflects the metabolic control of diabetes over the last 4 months.Major part is HbA1c about 6% remaining HbA1a and HbA1b.The normal range is 5 – 8%..Anything more than 12 – 15% reflects poor control of diabetes.

Serum Fructosamine

This is due to glycosylation of serum proteins .This reflects the glycemic control of last 2 weeks.

Culture and sensitivity tests – pus from infected area cultured for microorganisms and sensitivity to antibiotic is tested.

X–ray of foot

X- ray detects the presence of osteomyelitis , bone destruction commonly seen at metatarsophalangeal joints or interphalangeal joint of great toe.Gas in tissues indicates anaerobic infection.Osteoporosis of bones can be made out.Calcification of vessels can be made out.

Ankle Brachial Index (ABI)

ABI measured with pocket Doppler probe of 4 to 10 MHz.Brachial systolic pressure of both arms is measured. Ankle systolic pressure measured by applying BP cuff over the ankle above the malleolus and both posterior tibial and Dorsalis pedis pressure is measured by not applying pressure on the probe and maintaining a beam vessel angle of 60 degree. The highest ankle pressure divided by highest brachial pressure – Ankle Brachial Index. Normal value is 1 – 1.2. Less than 0.9 ABI indicates occlusive arterial disease.ABI less than 0.3 or ankle systolic pressure less than 50 mm Hg indicates critical limb ischemia. Change of ABI value more than 0.15 indicates significant narrowing and angiography evaluation needed. Medial calcification of vessels make arteries incompressible resulting in high ABI values more than 1.2.In these cases toe pressure is used.

Duplex scanning with ultrasound analysis

Duplex scanner use the combination of real time B mode USG imaging of the arterial wall along with pulsed Doppler imaging and examine the flow patterns in the arterial lumen at specific sites. It is a simple, low cost and useful method. It

determines the site and degree of obstruction. Patency of vessel after revascularisation is seen.

ANGIOGRAPHY

Percutaneous femoral angiography

It is used for evaluating the vascular supply of leg and foot.Angiography gives information regarding the patency of the vessel ,direction of blood flow, site of stenosis. It shows diffuse atherosclerotic disease of tibial and peroneal arteries, site of stenosis. Complications are thrombosis, peripheral embolization and allergic reactions to contrast.

Digital Subtraction Angiography

It is a digital fluoroscopic technique for clearly seeing blood vessels in bony or soft tissue background. The images are taken after injecting contrast by subtracting pre – contrast image or mask image. Digital Subtraction Angiography can be done on OPD basis with iv contrast.DSA has better contrast resolution. Cost is less compared with conventional angiography.DSA demonstrates small vessels distal to an obstruction .DSA has poor spatial resolution.DSA has advantage of simultaneous endovascular intervention also. Disadvantage in DSA is in diabetics the risk of contrast induced nephropathy is high in the prevalence of renal

insufficiency. Therefore patients with renal impairment are given pre – procedural intravenous volume expansion, Metformin is stopped prior to Digital Subtraction angiography as it causes lactic acidosis. In patients with severe occlusive disease when the concentration of contrast is less; visualization of pedal vessels is limited.

TREATMENT

Management of diabetic foot

Control of risk factors

1. Stop smoking

2. Control of hypertension,hypercholesterolemia

Infection

In management of diabetic patients with infected feet, following factors are to be considered – extent of tissue necrosis and gangrene, extent of bone involvement, vascularity of foot, peripheral neuropathy, presence of foot deformity, metabolic control of diabetes .Metabolic control of diabetic state to be aggressive and achieved by insulin therapy. Wound debridement – all necrotic tissues should be debrided until healthy bleeding is reached. Wound cleansing after surgical debridement acts as a complement to systemic antibiotics and reduces the incidence of

continued infection. The irrigation solution used are normal saline,povidone iodine solution. Normal saline alone is effective in reducing bacterial counts[16].All purulent material should be drained; copious normal saline wash should be given. Daily debridement should be done if needed.

Antimicrobial therapy based on culture reports is started and multiple debridements may be necessary in the diabetic foot prior to reconstruction. Adequate removal of all necrotic tissues is associated with quicker healing times.Debridements increases the rate of limb salvage to 89.8%[17]. In presence of severe infection with sepsis higher antibiotics like Imipenem,Ticarcillin clavulanate,ampicillin sulbactum have activity against anaerobic bacteria. Metronidazole has activity against gram negative anaerobic bacilli.B.fragilis is treated with clindamycin. With severe infections in presence of septicemia, combination of 2 antimicrobial agents is started as preliminary empirical therapy, later the antibiotics are changed according to the culture and sensitivity reports.

Offloading

Pressure relief on ulcers known as offloading is always part of the treatment plan. Bony deformity and displacement of soft

tissues leads to high plantar pressure and causes non-healing ulcer. Measurement of pressure between the foot and ground and between the foot and shoe tells whether the patient has adequate load relief during and after ulcer healing. Offloading with total contact cast accelerates ulcer healing. Neuropathic ulcers that have resisted healing for many months heal in 6 weeks of time with total contact cast. Total contact cast is successful when properly applied and changed weekly.Crutches,bed rest, wheel chairs are not effective for offloading without direct intervention of the foot. Different kind of braces, sandals, modified shoes are available for offloading the plantar surface and immobilizing the foot and ankle. Once the infection is controlled, and if the ulcer is small in size, protective and therapeutic footwear insoles, orthosis,total contact cast, bed rest[15].

Revascularization

Revascularization in diabetic foot patients with peripheral arterial disease has a limb salvage rate of 85%[18].Patients with ABI less than 0.5 after appropriate vascular imaging with infection and non-healing ulcers, earlier vascular intervention is required. Digital Subtraction Angiography is the standard imaging modality for evaluating Peripheral Arterial Disease when revascularization is

planned. The important task for any revascularization procedure is to achieve at least one open infrapopliteal artery down to the foot. Standard treatment for patients is femorodistal bypass with autogenous saphenous vein graft. Prosthetic grafts can also be used but with decreased patency rates. Endovascular treatment is preferred in patients with borderline toe pressures and short lesions.

Bypass is used in patients with long occlusions. Endovascular therapy in infrapopliteal arterial disease is used as the first line revascularization method for improving ulcer healing and to salvage the limb. There are good results and patency rates with endovascular treatment of PAD with critical ischemia[19].In distal bypass procedures, the clamping of vessels should be avoided to prevent any lesions distal to the anastomosis. This makes distal anastomosis challenging in the distal outflow vessels which are heavily calcified. In these cases balloon obstruction or tourniquet ischemia improve visibility during the anastomosis. The best graft material in distal bypass is autologous saphenous vein graft as it has better patency rates and also resists infection in comparison to a prosthetic graft. Timing of the revascularization inpatients with infection is usually delayed 2 to 5 days to control the infection. The debridement is done first and revascularization later. Simultaneous

revascularization at the time of initial debridement helps in increasing the blood flow.

Negative pressure wound therapy is used to fasten healing. The prerequisite for negative pressure wound therapy is that there is sufficient blood supply for ulcer healing. Negative pressure wound therapy does not replace surgical wound debridement and measures to improve blood circulation. Negative pressure wound therapy is effective than other available local wound treatments in patients without significant infection.

ADJUVANT PROCEDURES

Tendoachilles lengthening

The insensate foot gets deformed by the action of the Gastronemius soleus muscles which is the most powerful muscle in the leg represented by the tendoachilles.Contracture of the tendoachilles results in the equinus deformity of the foot. This prevents dorsiflexion in ankle joint. While walking the angle between the tibia and foot is about 11 degrees. The midfoot collapses due to loss of normal movements.This causes rise in the pressure from the great toe ball to the midfoot. The heel contact phase is reduced along with the midstance.This caused the ball of the foot to be in contact with the floor for a longer duration.In

patients who have lost the foot sensation, increases the risk for forefoot ulceration leading to non-healing ulcer unless equinus deformity is corrected.

In patients affected with diabetes for many years, neuropathy develops. Electron microscopic studies of tendoachilles shows collagen changes which causes the contracture of the tendoachilles. Tendoachilles lengthening is done and tendon is repaired by nonabsorbable sutures in the open procedure. Percutaneous lengthening is done by small incision and the foot is manipulated to bring about a dorsiflexion of 5 to 10 %.

When performing tendoachilles tenotomy or combining tendoachilles lengthening with plantar midfoot exostectomy, Achilles tendon work is done first followed by osseous work. For hind foot or midfoot reconstructive arthrodesis osseous work is done first and tendoachilles lengthening is done later. The arthrodesis procedure is done in the supine position and the patient turned in prone position and tendoachilles lengthening is performed. Achilles tendon lengthening or tenotomy is also recommended for recurrent plantar ulcer following trans metatarsal amputations. In Trans metatarsal amputations excessive dynamic forces acts on the short foot to cause recurrent plantar ulcers.

Achilles tendon lengthening or tenotomy reduces the propulsive force stride length and shear force that contribute to development of ulcer. Stride length is a contributing factor for the development of ulcers in neuropathic foot. There is less pressure and force transmitted to the distal aspect of the plantar foot when the stride length is shortened to 14 inches. Achilles tendon lengthening or tenotomy results in shortening of stride length.

Subtotal calcanectomy

In ulcers around the hind foot with osteomyelitis of the calcaneum,resection of the calcaneum upto one third of the posterior calcaneum is performed so that the soft tissues is freed and the defect can be closed primarily.Resection of the major portion of calcaneum does not prevent the patient from bearing weight on the foot. Subtotal calcanectomy is reported to be superior to total calcanectomy because it provides a stable foot.Subtotal calcanectomy is a reliable adjunctive procedure for maintaining the foot stability.Tendoacilles is erased from the calcaneum and attached back to the stump of the calcaneum.If there is osteomyelitis of the calcaneum the tendoachilles is removed circumferentially from the calcaneum,this does not allow the tendon to retract back. Immobilising the leg for 6 weeks causes

fibrosis of the tendon to the surrounding tissues and the function of tendoachilles is maintained.

Ostectomy for Charcot's joint

Plantar ulcerations are common in charcot's joint disease. Large portions of the bone migrate to the plantar aspect of the foot with secondary prolapse of the medial or longitudinal arches producing the classic Rocker Bottom foot. This foot concentrates enormous amount of pressure in isolated areas causing pressure points that results in ulcerations. Typical midfoot break down associated with Charcot joint disease is classified as type I Charcot's joint based on Brodsky classification. Patients that are candidates for surgery fall in Eichenholtz stage III Charcot's disease when bony healing is taking place[20]. Midfoot ostectomies with saucerisation of bone is done in cases of plantar deformities – Charcot midfoot collapse (Charcot's Rocker Bottom foot). The defect can be closed primarily.Saucerisation prevents local recurrence when performed along with tendoachilles lengthening.

Charcot joint stabilization

Arthrodesis for stabilization of charcot joint done are midfoot procedures and hind foot procedures. When joints in mid foot coalesce bony prominences are removed with ostectomies. In

Eichenholtz stage II Charcot bony prominence is removed to eliminate non healing plantar ulce .Non healing ulcer in stage II can be treated with Arthrodesis of mid foot.Arthrodesis is considered once ostectomy fails. In stage II disease there is instability so arthrodesis helps. In stage III Eichenholtz, arthrodesis is rarely necessary because there is bony consolidation. In severe hind foot deformity which is not corrected by ankle foot orthoses or braces with chronic ulceration; arthrodesis is indicated in hind foot. The goal in this surgery is to maintain structural stability and to maintain plantargrade position of the foot. Success of arthrodesis depends on adequate vascularity, absence of infection, nonweight bearing and removal of pressure points. These procedures may be used as an alternative to amputation in salvaging the foot.

Great toe ulcers

The paralysis of the EHL muscles leads to ulcer on the great toe in neuropathic patients.These are corrected by fusion of the interphalangeal joint.The great toe is incised by two elliptical incision about an inch over the dorsal aspect of the Ip joints.EHL tendon is cut and the joint is exposed.The cartilage is excised from the base and head of the phalanx.The osteotomy is done.The bones are then aligned with two k wires in a criss cross manner. This is

kept for about 3 weeks. The fibrosis which results is satisfactory. The digits are fixed in improvised position.Localised procedures to close the wound on the tip of the toe can be performed now.

Hallux arthroplasty – two incisions which intersect each other on the dorsal aspect of the great toe are put.EHL tendon is divided and the Ppx head is delivered and resected and wound is closed with a toe bandage in straight alignment. This bandage is continued till the fibrosis occurs for about 6 weeks. This correction results in healing of the ulcers on the great toe. Ulcers beneath the head of the 1st metatarsal caused due to prominence of the 1st metatarsal bone due to high instep foot. This is repaired by removal of one or both the sesamoid bones. This reduces the pressure on the head of the 1st metatarsal and decreases the recurrence of the ulcer. The sesamoid can be directly approached through the wound or through a medial incision in the MP joint.The capsule is opened by an incision and median digital nerves and long flexor tendon are to be protected.

Flap cover

Soft tissue reconstruction in the diabetic foot is challenging as the area is characterized by tight skin and poor blood supply. SSG on the weight bearing areas are unrealistic and mostly fails.

Flap selection is based on multiple factors like location, size of the defect to be covered, type of deficient tissue components, presence of infection, weight bearing, sensory needs, donor site and its morbidity and logistics of operation[21]. Muscle flaps have been preferred for decades; they conform better to three dimensional wounds obliterating the dead spaces and decreasing the risk of infections. Muscle flaps are believed to improve vascularity. Muscle flaps bring in antibiotics and oxygen delivery to the contaminated wounds.Fasciocutaneous flaps have also been used successfully for the coverage of chronic wounds.

Local flaps are used when possible as they reduce the operating time as microsurgical anastomosis is not required, patient can be operated under regional anaesthesia.Potential risks in local or regional flap are regional hypertension and distal tip necrosis[22]. The use of local flaps to cover the defect has been applied but there use is limited by the availability of healthy well vascularized tissue in a diseased foot and the size of the defect[23]. First successfully performed free groin flap to reconstruct dorsal foot defect by O Brien and Daniel Taylor four decades ago. Microsurgical techniques have become one of the essential pillars of foot and ankle reconstructive procedures. The successful treatment with diabetic foot ulcers by microsurgical

procedures are now done with a better understanding of biomechanical requirements concern for durability, donor site morbidity and shoe fit.

A transferred flap with micro vascular anastomosis is highly vascularized with its own intrinsic blood vessels in contrast to local flaps which are relatively ischemic in their distal portion. Transposition flap is most frequently used flap to cover exposed bone, joint or tendon only. Rest of the defect is covered with SSG.The flap is taken from the site that is most mobile and that has a definable perforator at its base. The rotation flap is useful on the plantar aspect of the foot where the flap is elevated off the plantar fascia and rotated into position.

V – Y plantar flap – forefoot skin, fat and fascia is advanced in a V-Y fashion either singly or in pairs. Wounds up to 4 to 5 $cm^{3.}$Medial plantar artery flap is useful foe heel pad reconstruction. The donor site must be skin grafted. The instep should be non-weight bearing and unlike a Charcot's midfoot collapse where the skin graft will not be taken. The instep of the foot is elevated as a fasciocutaneous flap based on the medial plantar artery and transposed posteriorly to cover the heel.Sural artery flap (Retrograde sural artery) is used for ankle and posterior heel

defects. A median sural artery is seen coursing with the sural nerve in most cases the arterial component is a plexus of vessels with the sural nerve and receives retrograde flow from a peroneal perforator 5 cm above the lateral malleolus. The artery first courses above the fascia and then goes deep to the fascia at the mid-calf level while accompanying lesser saphenous vein. The insert of the flap is critical to avoid kinking of the pedicle. During the healing period, the flap and heel is offloaded. Donor defect is skin grafted; there is a sensory loss on the lateral aspect of the foot.

Micro vascular composite tissue transplantation (free flaps) – Large foot wounds with bone or tendon exposure are best reconstructed with free flaps. Anterolateral thigh flap is a septocutaneous flap based on perforators originating from descending branch of lateral circumflex femoral artery. The ALT flap is well accepted and supplies large amount of subcutaneous fat and skin on a safe and reliable pedicle with no functional donor site morbidity. The flap can be raised as a sensate flap with the lateral femoral cutaneous nerve and also as a flow through flap. The flap can be thinned and tailored for the particular defect.

Radial forearm flap – It is a good option for dorsal foot wounds. It is a thin and pliable and can be harvested with a sensory

nerve. The radial artery with venaecomitantes provides vascular pedicle of about 14 cm length. The donor site is skin grafted.

Para scapular flap is based on the circumflex scapular artery. It is used for large defects. It is an insensate flap and also bulky. The flap has to be thinned at a later date.

Gracilis muscle flap – it is a thin flat muscle located on the medial thigh extending between the symphysis pubis and medial tibial condyle lying between the adductor longus and Sartorius muscle anteriorly and semimembranosus posteriorly. Functions as an adductor of the thigh but is expendable due to compensation made by adductor magnus and longus.Vascular supply is from the terminal branch of the medial circumflex femoral artery. Motor innervation is from the anterior branch of the obturator nerve.Gracilis muscle can be tailored to any size and is supple and pliable. Contour improvement occurs early.

Latissimus dorsi muscle flap – It is a large flat muscle covering mid and lower back. It is the first choice in large defects of the foot involving entire sole or complex defects. The muscle is bulky but contour correction over time is satisfactory. It has a long reliable pedicle based on the thoracodorsal artery which enters the

undersurface of the muscle 5 cm from the posterior axillary fold. Donor site morbidity can include shoulder dysfunction, weakness and pain, so it is used with caution in patients with crutch dependence

MATERIALS AND METHODS

TYPE OF STUDY

It is a retrospective clinical study.

STUDY SETTING

The study was conducted in the Department of Plastic and Reconstructive Surgery, Rajiv Gandhi Government General Hospital and Madras Medical College, Chennai over a period from January 2003 to February 2014.

SAMPLE SIZE

All the diabetic foot patients who got admitted and had undergone revascularization and subsequently had foot defects in need of reconstruction of foot or foot remnants from January 2003 to February 2014 were included in our study which includes 78 cases and it includes 10 females and 68 males.

INCLUSION CRITERIA

1. All the diabetic foot patients who had undergone revascularization and had foot defects, subsequently in need of reconstruction of foot or foot remnants.
2. All revascularised diabetic foot patients with PT/ INR around 1.5.

EXCLUSION CRITERIA

1. Those who have undergone revascularization but with co-morbid illness complicating anesthesia for foot reconstruction

2. Patients with renal failure

3. Age more than 70 years

METHODOLOGY

Patients who underwent revascularization of diabetic foot and in need of foot reconstruction were selected in the period from January 2003 to February 2014. Prior permission and no objection certificate were obtained from the Head of Department and the study details were presented to the Institutional Ethical Committee and permission obtained. A detailed proforma is made which includes general information of the patient, any co- morbid illness, smoking history, treatment history, provisional diagnosis, general and local examination and investigations undergone. Relative information was filled in a proforma and information was taken from case records.

Following information are noted from the case records –

1. Type of revascularization procedure

2. Latency period (period before embarking on reconstruction following revascularization)

3. Number of debridement done prior to reconstruction

4. Adjuvant procedures performed

5. Foot reconstruction procedure

Various types of flaps and other reconstructive procedures were done by the experienced surgeons only after detailed analysis of the case by the reconstructive surgery team after taking into consideration of all the factors. Patients were well informed about the advantages and complications of the procedure. Those patients who were assessed by the expert team to have higher probability of failures were not taken up for reconstructive procedure and were asked to undergo primary amputation.

All the patients are preoperatively evaluated with following investigations

- Complete Blood count,
- Renal Function Tests - *Blood Urea

*Serum Creatinine,

- Blood sugar
    * Fasting Blood Sugar
    * Post Prandial Blood Sugar
    * HbA1c
- PT - INR
- Screening for Sexually Transmittable diseases
    * HIV,
    * HbsAg,
    * VDRL, and
- MR Angiogram / Digital Subtraction Angiogram (optional)

Before taking up reconstructive procedures in these patients their critical limb ischemia was addressed by revascularization either angioplasty or a bypass procedure and then reconstruction was planned.

All the patients had ulcers with poor potential for healing before the revascularization procedure. Once the revascularization was done, the patients developed regional vascular insufficiency characterized by poor wound granulation,this was treated with dressings,debridements.Negative pressure wound therapy was used in this period . Off- loading techniques were also used which took many days and that period was used to improve the patient’s

general condition both physically and mentally. They were transfused with blood and blood products if needed.

Culture and sensitivity of the wounds were done and appropriate antibiotics were administered and once the infection was controlled, with a favourable wound bed reconstruction was planned and the perforators of the flaps and recipient vessels were assessed by hand held Doppler of 8 to 10 MHz.The need for adjunctive procedures like Tendoachilles lengthening, Ostectomies, gap arthroplasties and tenotomies was also assessed as adjuvant procedures form an integral part of any definitive reconstructive procedures. Procedures were done using regional or general anesthesia under cardiac monitoring. Postprocedure, Inj.Dexamethasone, low molecular weight Dextran 40 and a proton pump blocker were given to all the patients for 48 hours and the limb of the patients were immobilized with posterior popliteal slab. The patients were discharged after 1 week and then followed up periodically to check for postoperative complications.

Follow up Procedure:

At weekly interval for one month and followed by once a month for 6 months and twice a year thereafter.

Assessment of Outcome:

Post - operative complications were assessed. Complications include

- wound infection,
- hematoma and seroma
- flap necrosis,
- wound dehiscence,
- ulcer recurrence

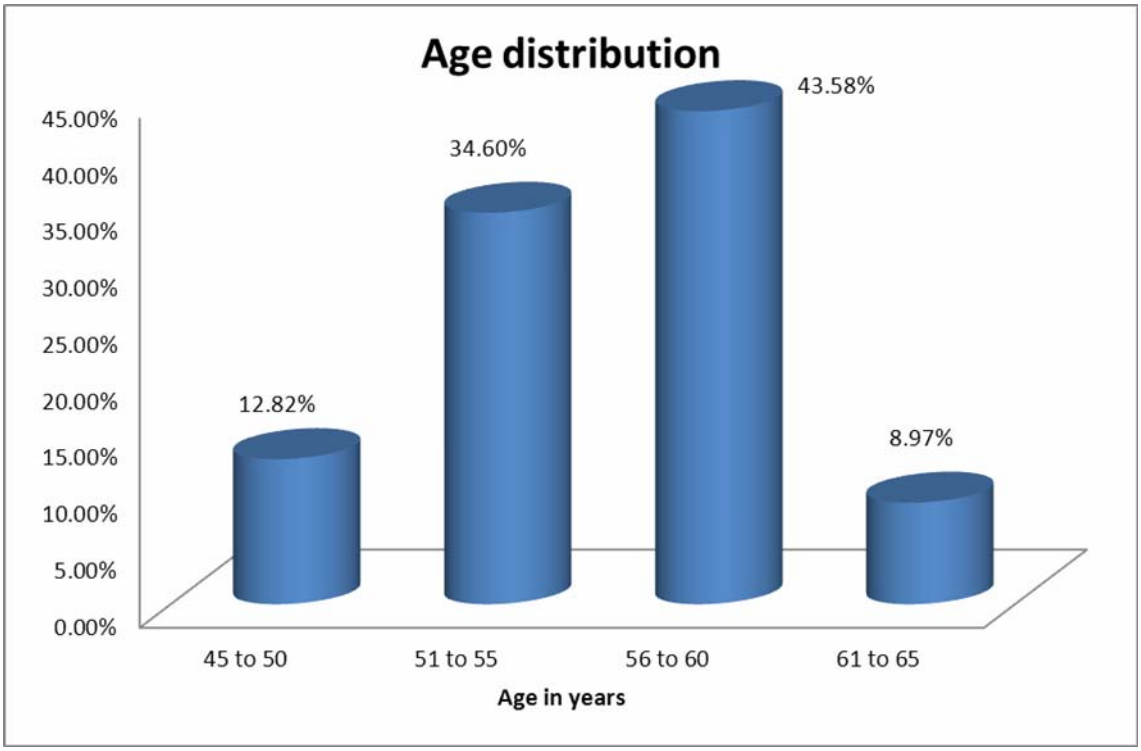
DATA MANAGEMENT AND ANALYSIS

All data were reviewed at periodic intervals and all raw data are finally computed at the end of the study to find out the outcomes of surgery in terms of post-operative complications**.** Statistical analysis was done .The graphs were produced using Microsoft Excel.

RESULTS

TABLE 1 – AGE DISTRIBUTION

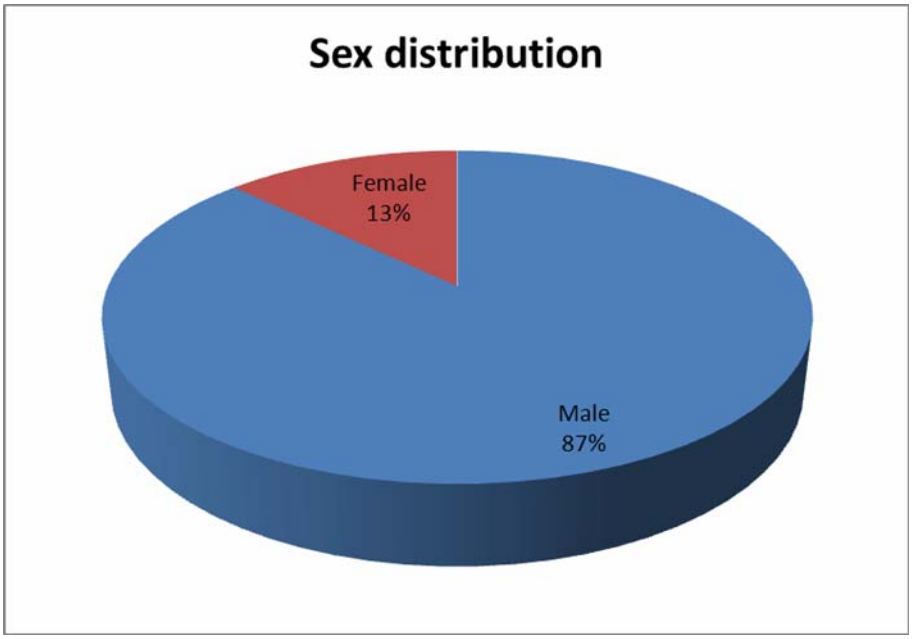
Age in years	Number	Percentage
45 to 50	10	12.82%
51 to 55	27	34.6%
56 to 60	34	43.58%
61 to 65	7	8.97%



Out of 78 patients included in the study, Maximum number of patients i.e., 34(43.58%) were in the age group between 56 to 60 years, 27(34.6%) were in the age group of 51 to 55 years, 10(12.82%) were in the age group of 45 to 50 years and the remaining 7 (8.97%) fall in the age group between 61 to 65 years. Mean age found in the study is 57.2 years

TABLE – 2 SEX DISTRIBUTION

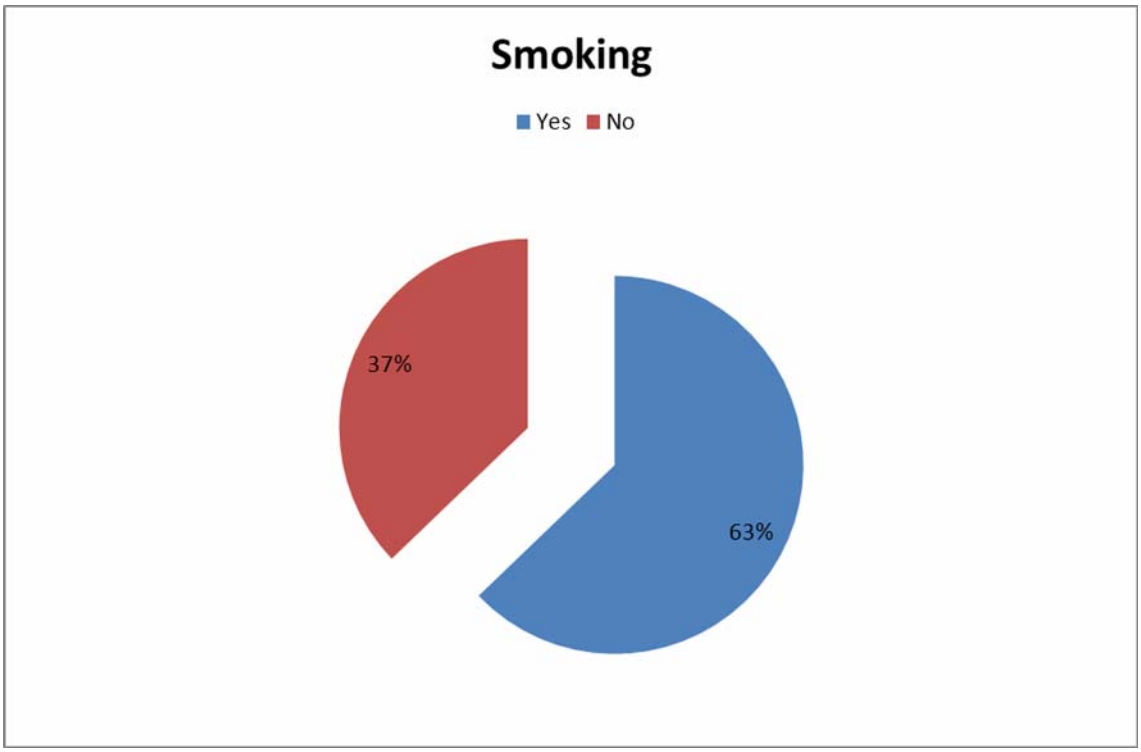
Sex	Number	Percentage
Male	68	87.17%
Female	10	12.82%



In our study, majority of the patients i.e., 68(87%) were males and the rest 10(13%) were females

TABLE – 3.SMOKING

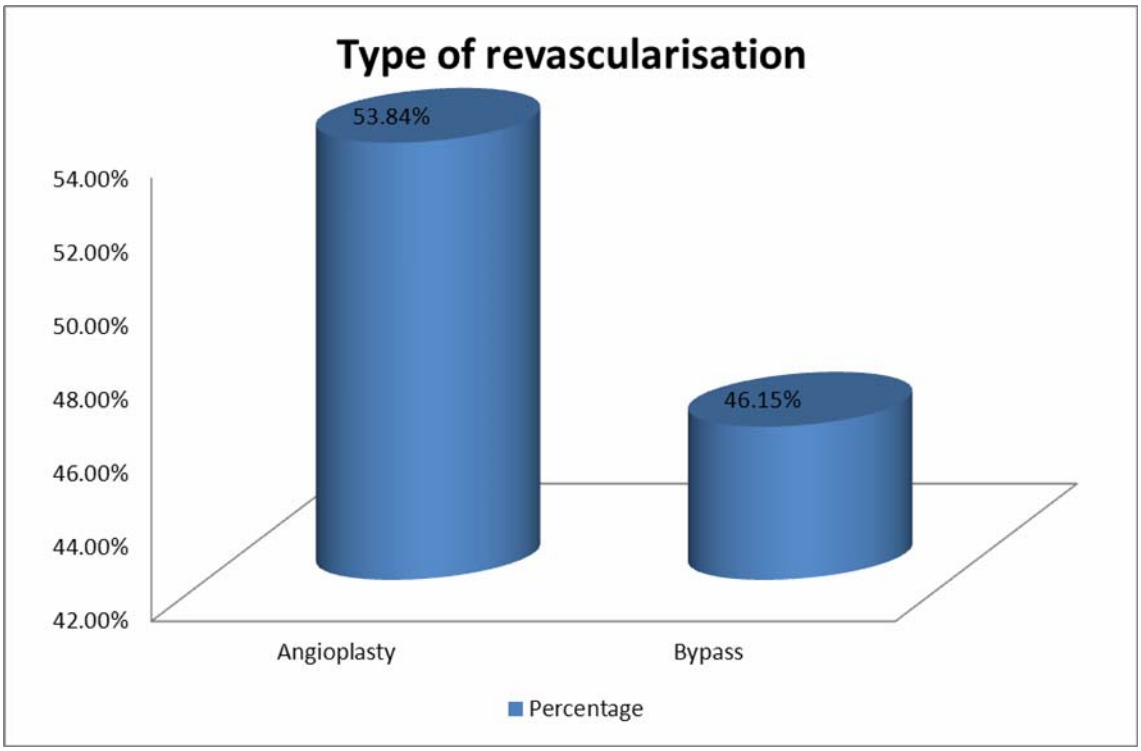
Smoking	Number	Percentage
Yes	49	62.82%
No	29	37.17%



Majority of the patients were smokers i.e., 49(62.82%) and 29(37.17%) were non smokers.

TABLE – 4- TYPE OF REVASCULARISATION

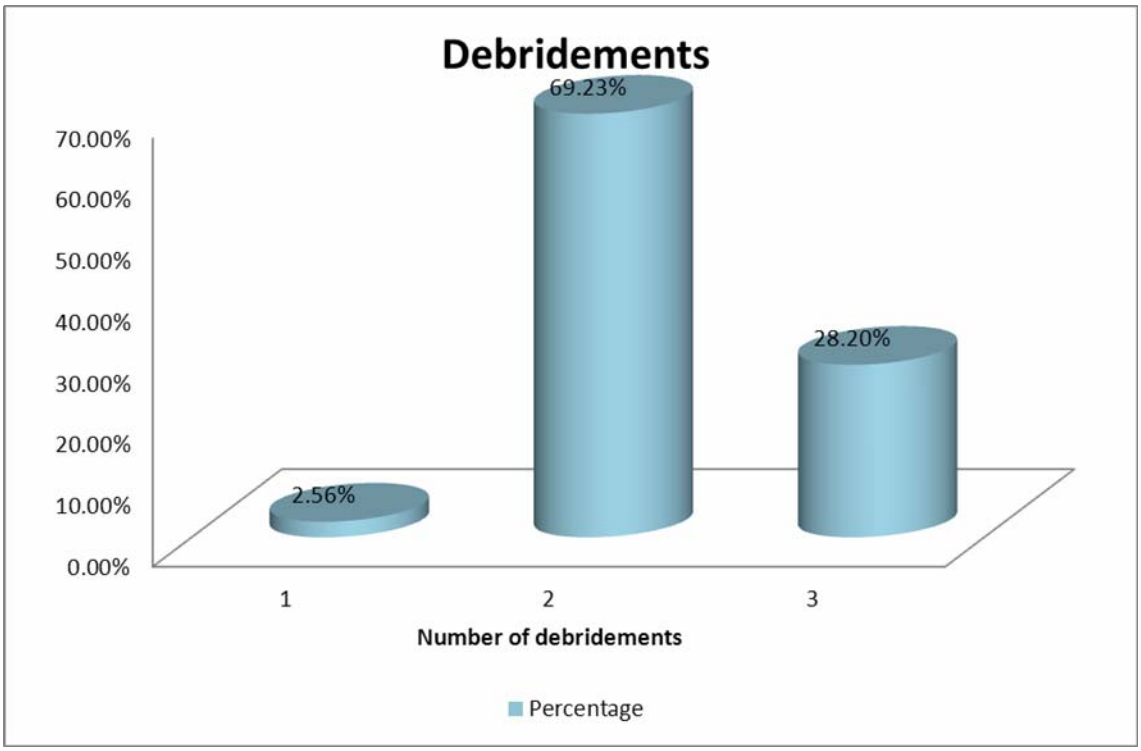
Type of revascularisation	Number	Percentage
Angioplasty	42	53.84%
Bypass	36	46.15%



Of the 78 patients, 42(53.84%) underwent angioplasty and 36(46.15%) underwent bypass procedures for revascularisation

TABLE – 5 – NUMBER OF DEBRIDEMENTS

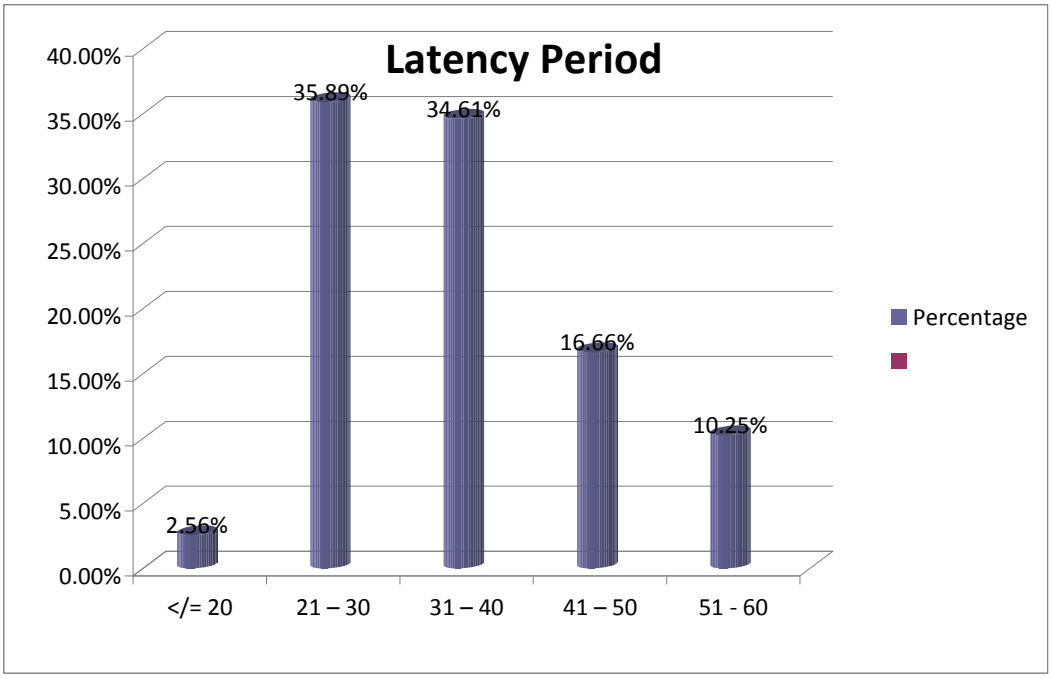
Number of debridements	Number	Percentage
1	2	2.56%
2	54	69.23%
3	22	28.2%



Most of the patients in our study has underwent two debridements 54(69.23%), while 22 (28.2%) has underwent 3 debridements and 2 of them has undergone single debridement.

TABLE -6- LATENCY PERIOD

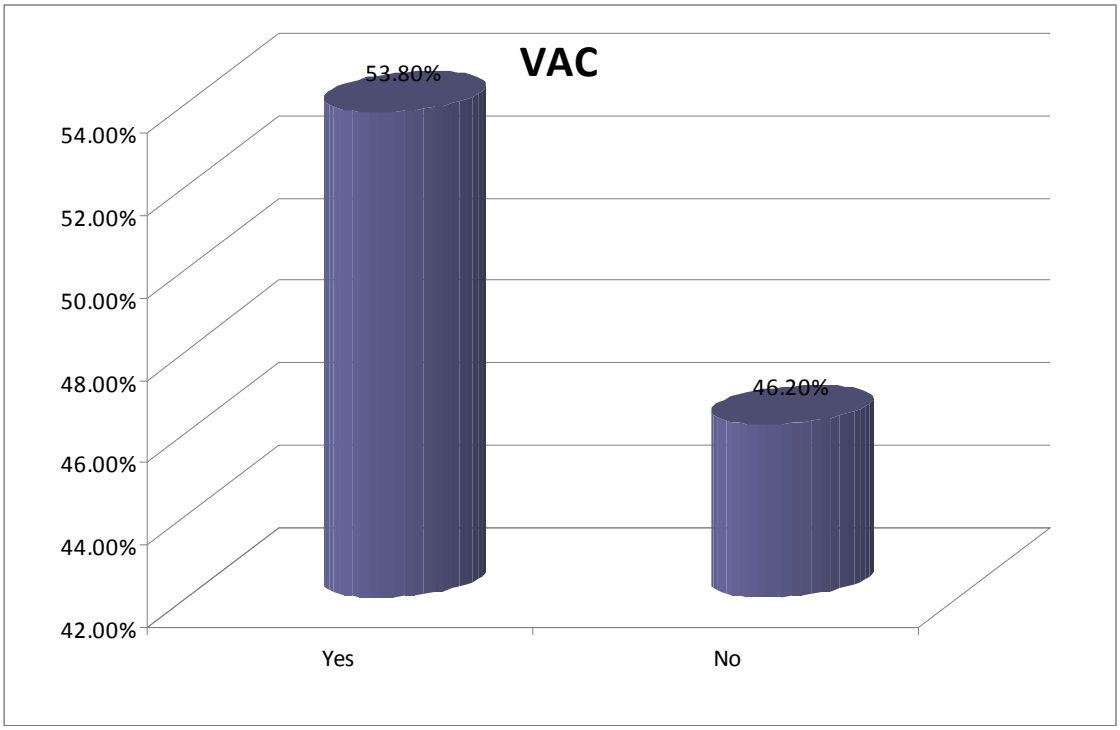
Latency period in days	Number	Percentage
</= 20	2	2.56%
21 – 30	28	35.89%
31 – 40♣	27	34.61%
41 – 50	13	16.66%
51 – 60	8	10.25%



The average latency period found in our study is 35.36 days. Most of the patients 28(35.89%) in our study show a latency period between 21 to 30 days

TABLE 7 – VAC

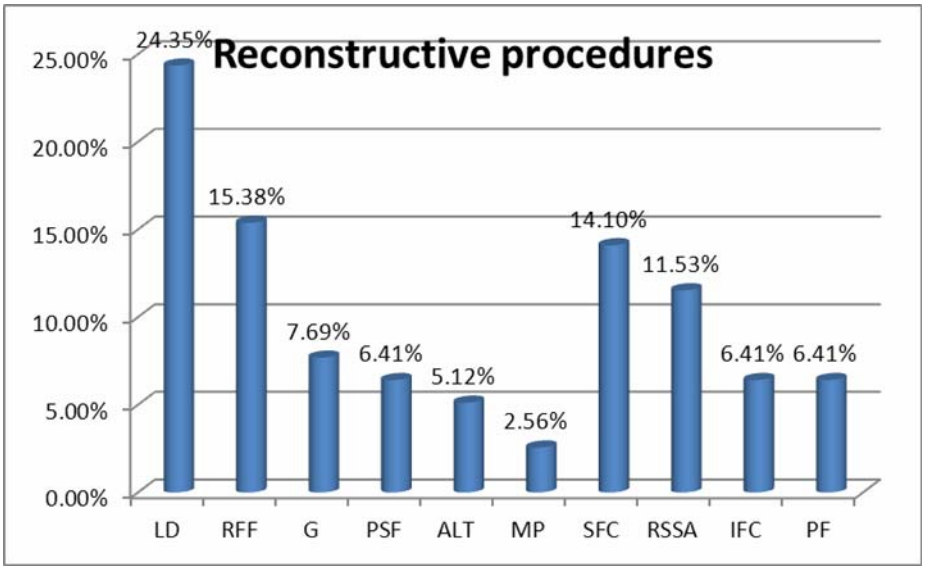
VAC	Number	Percentage
Yes	42	53.8%
No	36	46.2%



In our study 42(53.8%) of the cases underwent Negative Pressure Wound Therapy in the latency period.

TABLE 8 – RECONSTRUCTION PROCEDURES

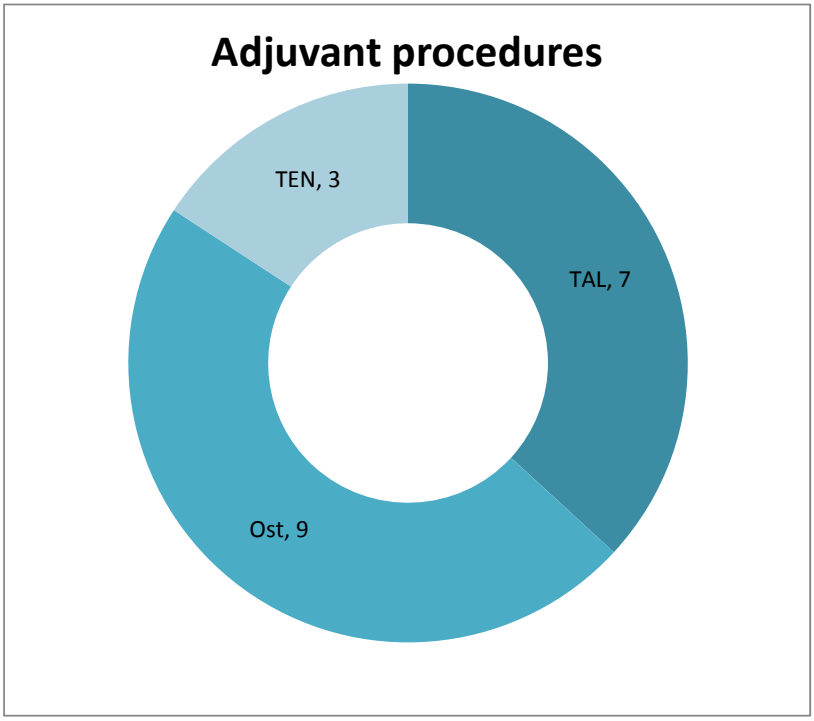
Type of procedures	No. of patients	Percentage
LD	19	24.35%
RFF	12	15.38%
G	6	7.69%
PSF	5	6.41%
ALT	4	5.12%
MP	2	2.56%
SFC	11	14.10%
RSSA	9	11.53%
IFC	5	6.41%
PF	5	6.41%



Of the 78 patients 19(24.35%), 12(15.38%), 6(7.69%), 5(6.41%), 4(5.12%), 2(2.56%), 11(14.10%), 9(11.53%), 5(6.41%), 5(6.41%) underwent Latissimus Dorsi flap,Radial free Forearm flap, Gracilis flap, Parascapular flap, Anterolateral thigh flap, Medial Plantar artery flap, Superior Fasciocutaneous Flap, Reverse Superficial Sural Artery flap, Inferiorly based fasciocutaneous flap, Perforator flap.

TABLE 9 – ADJUVANT PROCEDURES

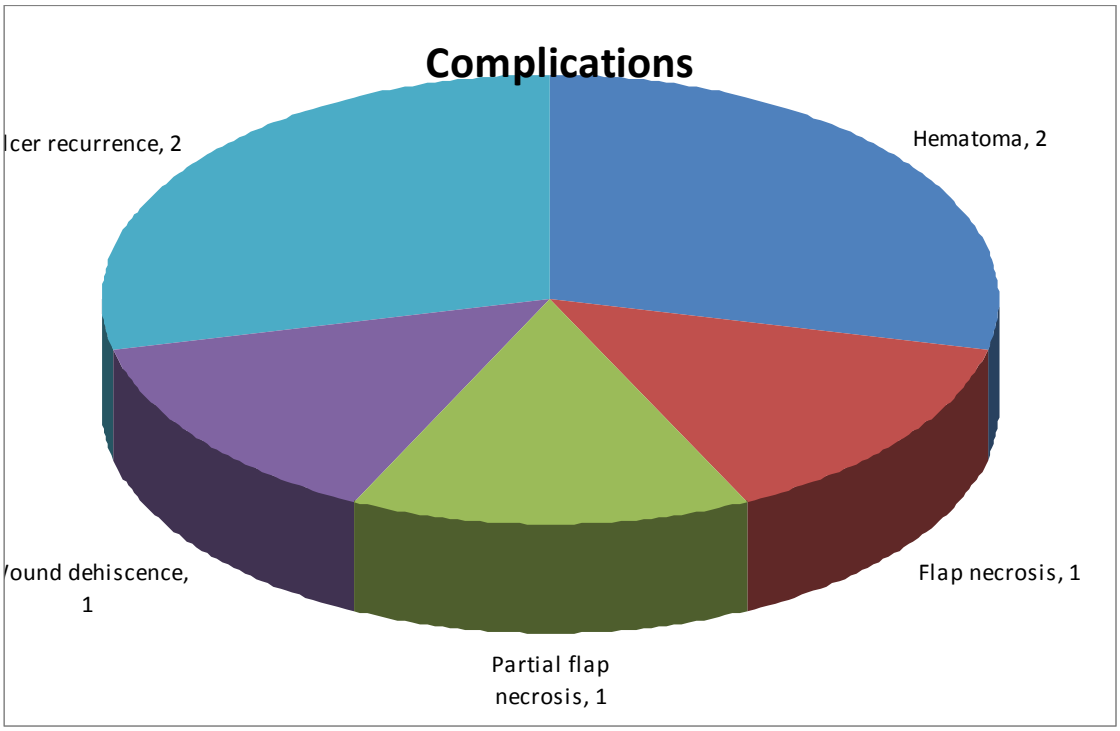
Adjuvant procedures	No.of patients	Percentage
TAL	7	8.97%
Ost	9	11.53%
TEN	3	3.84%
Nil	59	75.64%



Of the 78 cases, 59 did not undergo any adjuvant procedures, while 7(8.97%),9(11.53%),3(3.84%) has undergone Tendo Achilles Lengthening, Ostectomy, Tenotomy.

TABLE 10 – COMPLICATIONS

Complications	Number
Hematoma	2
Flap necrosis	1
Partial flap necrosis	1
Wound dehiscence	1
Ulcer recurrence	2
Total	**7(9%)**



Complications were found in 7(9%) of the patients, of which 2 of them had ulcer recurrence, 2 of them had hematoma,1 had wound dehiscence,1 had partial flap necrosis,1 had flap necrosis.

DISCUSSION

The mean age found in our study group is 57.2 years. This is similar to other studies like Tae Suk Oh et al, C.Randon et al,Baek Kyu Kim et al.Tae Suk Oh et al conducted a study in 121 patients in Asan Medical center, South Korea which included the patients of age group 26 to 78 years and found the mean age to be 54.6 years in their study on Diabetic foot reconstruction using free flaps[24].Randon et al in his study conducted at Ghant University hospital,Belgium found the average age to be 50 years in a case series of 76 patients aged between 18 to 80 years[25].Kyu Kim et al in their study conducted in Seoul National University.Bundang Hospital found the average age of the patients to be 60.3 years in 30 men and 3 women[26].This is in correlation with the factor that most of the diabetics develop peripheral neuropathy 10 years after the onset of the disease leading to the maximum incidence of diabetic ulcers from 50 years onwards.

Majority of the patients were males 68(87.17%) and 10 (12.82%) were females in our study .This is in accordance with Randon et al study[25].In Indian scenario males are mostly the bread winners working in fields or as laborers mostly barefoot

predisposing to repeated trauma in the neuropathic foot leading to ulcerations.

Majority of the patients i.e., 49(62.82%) are found to be smokers in our study .In the study conducted by Tae Suk Oh et al, 34.25 were found to be smokers [24]. Smoking habits are higher in Indian men which predisposes to the Peripheral Arterial Disease, moreso in diabetic patients leading to ulcerations.

Revascularization procedures are needed to improve the vascularity to the critically ischemic limb in the diabetics. In nonhealing ulcers failing conservative therapy revascularization aids in limb salvage. In our study of the 78 patients, 42(53.84%) had undergone angioplasty and 36(46.15%) had undergone bypass procedures.Revascularisation improves the limb salvage rate by more than 50 %(27 – Karl A Illig).

In our study, majority of patients underwent 2 or more debridements to achieve a good wound bed and to reduce infection preceding the reconstructive procedure.Latency period is the time period between the revascularization and reconstruction.This is the period in which wound healing is established due to the

improvement in vascularity of the foot. In our study the average latency period is 35.36 days ranging between 20 to 60 days.

Negative pressure wound therapy was used in our study in 42(53.8%) of patients. Negative pressure wound therapy was found to reduce the time taken for wound closure and increase healing [18].

Reconstruction of the foot after the revascularization is necessary as revascularization alone is not sufficient for the healing and to prevent limb loss. In our study, the reconstruction was carried out by locoregional flaps and free flaps.Microvascular free flap surgery is proven to be safe procedure for providing stable cover to the wounds after revascularization.In our study,30 patients underwent locoregional flaps and 48 of them underwent free flaps for reconstruction.L.Scott Lewin,Duke University medical center,Durham have found regional flaps to be viable option in treating foot defects with impaired vascularity[21].In our study the locoregional flaps done were Superiorly based Fasciocutaneous flaps were done in 11cases,Inferiorly based Fasciocutaneous flaps were done in 5 cases,Reverse Superficial Sural Artery flap in 9 cases,Perforator flap in 5 cases.

Catherine De Blacam in her study conducted in Royal College of Surgeons, Dublin found a flap loss of about 3.2% with Reverse Superficial Sural Artery flap. They also found that distal tip necrosis problem in the Reverse Superficial Sural Artery flap in 15.3%[28].Brenda K. Cohen conducted a study in 33 patients with medial plantar artery flap and found that it provides excellent like tissue reconstruction of the foot providing good weight bearing ability.6 cases had complications in their study[29].

Free flaps used for reconstruction in our study are Free Latissimus Dorsi muscle flap used in 19 cases, free Gracilis muscle flap in 6 cases, Radial Free Forearm Flap in 12 cases,Parascapular free flap in 5 cases, Free Anterolateral thigh flap in 4 cases. Free Medial Plantar artery taken as a free flap from the opposite foot was used in 2 cases to give a like tissue reconstruction. H.Sunar et al in the study conducted in Trakya University,Turkey found that the free flap done after revascularization in a delayed setting had benefits like assessment of the patency of the grafts improving wound conditions and reducing the operating time[30].Tay Suk Oh et al conducted a study of 121 cases with 90 Free Anterolateral Thigh Flaps and reported a complication rate of 9%[24].Lukas Prantl et al conducted a study in University of Regensburg,Germany found that

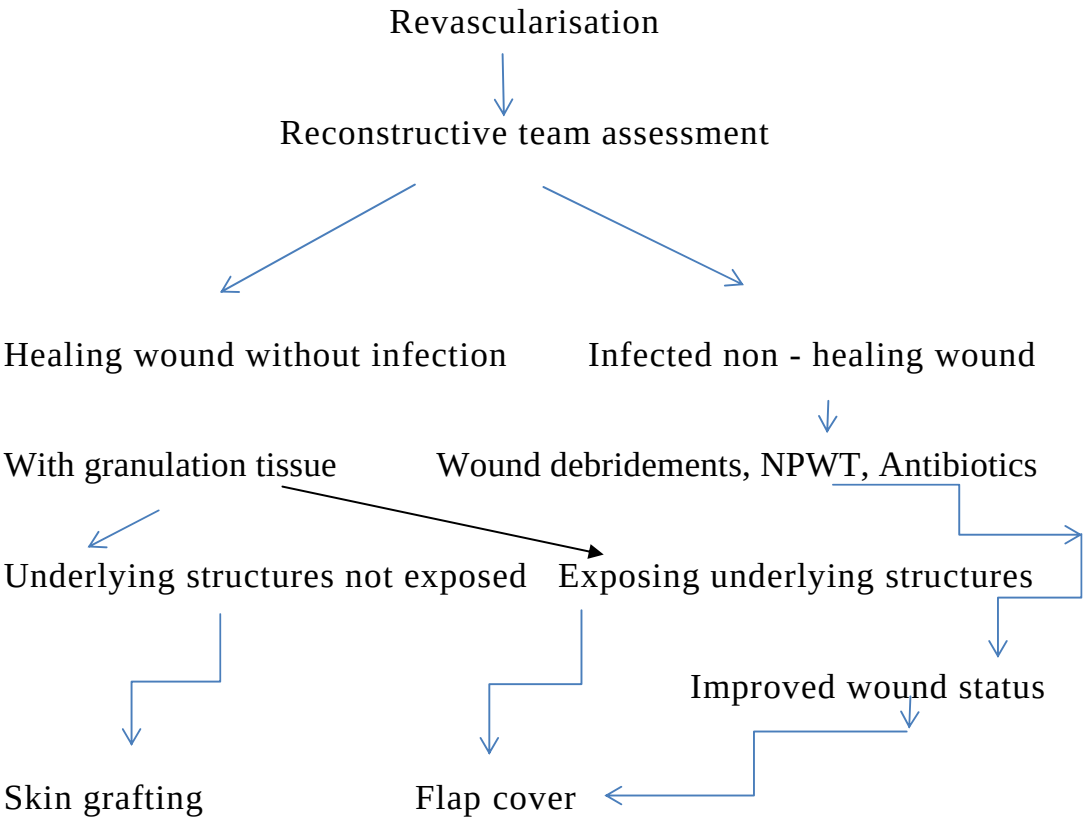
the parascapular flap to be useful to cover defects in the weight bearing area[31].M.Czerny et al University Medical school,Vienna,Austria conducted study and found the Gracilis muscle flap to be a good option as it can be done under regional anaesthesia and minimal morbidity of the donor site[32].In case of large foot defects Latissimus Dorsi Free Muscle flap is the preferred flap.

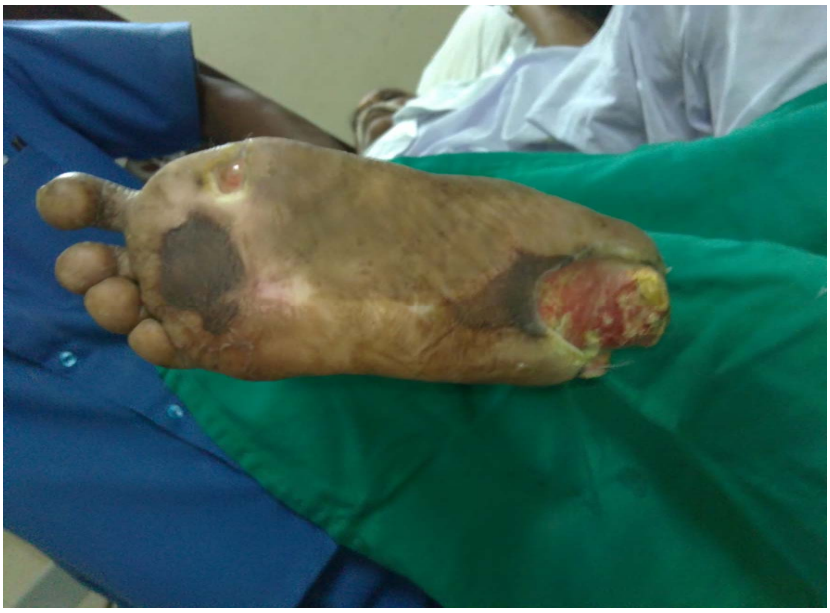
In our study, complications like hematoma were seen in 2 cases; flap necrosis is seen in Reverse Superficial Sural artery Flap. Partial flap necrosis is seen in 1 Antero Lateral Thigh Flap, Wound dehiscence in 1 perforator flap, Ulcer recurrence was found in 2 Latissimus Dorsi Flaps. The overall complication rates was 9%.This is similar to the study done by Tae Suk Oh et al [24].

CONCLUSION

Following revascularization of the diabetic foot which is key to establish the vascularity in ischemic foot and helps in controlling the infection .Infection is also controlled by appropriate antibiotics and wound debridements to remove the necrotic tissues which help in the healing by formation of granulation tissue. Negative pressure wound therapy is used in the latency period to help in the faster healing.Microvascular free flaps bring in well vascularized tissue for the stable cover of the diabetic foot with minimal complications where there is a paucity of locally available flaps. Hand held Doppler is adequate for planning of the reconstructive procedures. Angiogram may not be needed. Adjuvant procedures form an important part in the reconstruction process. Prevention of recurrences is by proper education which is most important. Use of orthotic foot wear ,silicone gel insoles and proper off-loading helps in preventing the recurrences.

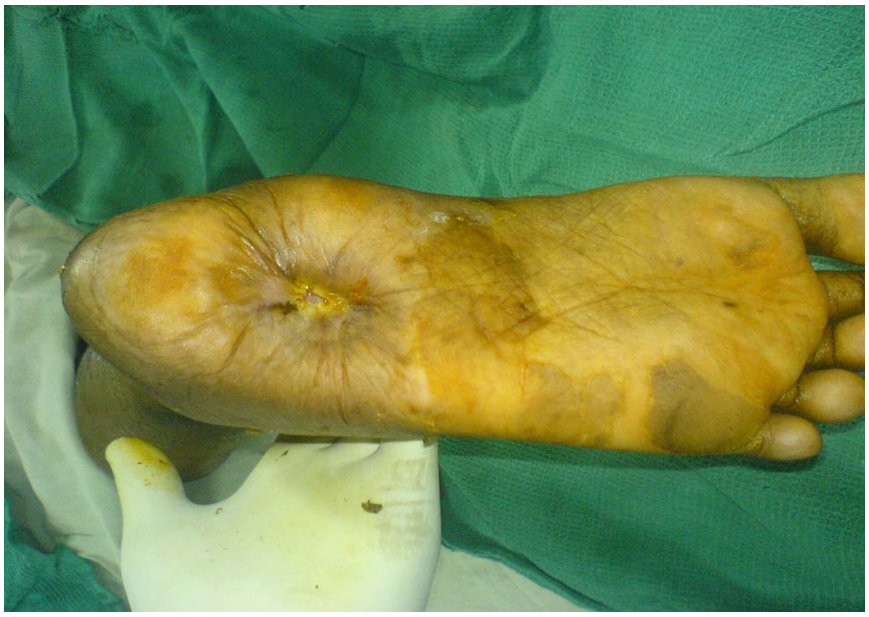
PROTOCOL IN THE MANAGEMENT OF DIABETIC FOOT AFTER REVASCULARIZATION



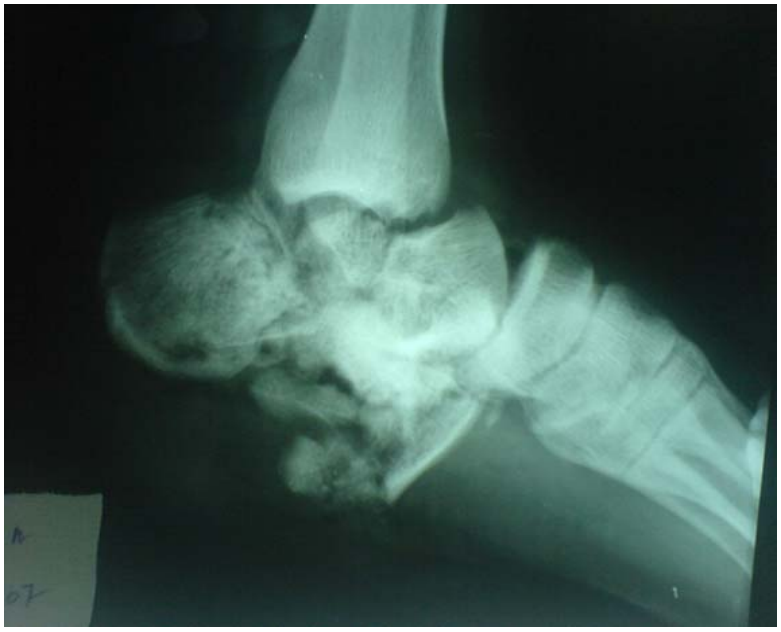


A case of distal femoral block.Femoropopliteal bypass done

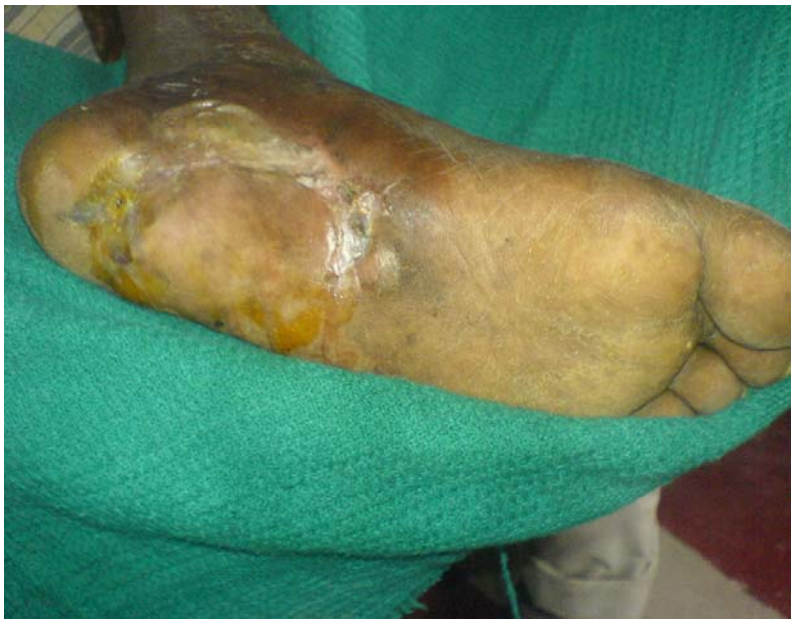
Femoropopliteal bypass scar, well settled SSG over LD flap



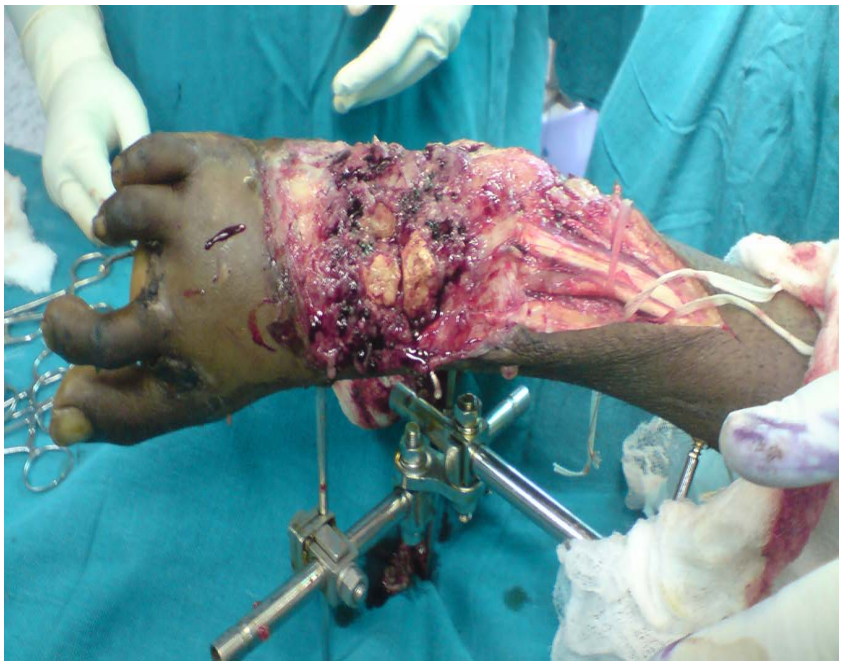
A case of like tissue reconstruction. Underlying calcaneal degeneration .With good preserved arch on the opposite foot treated with free medial plantar artery flap from opposite foot.



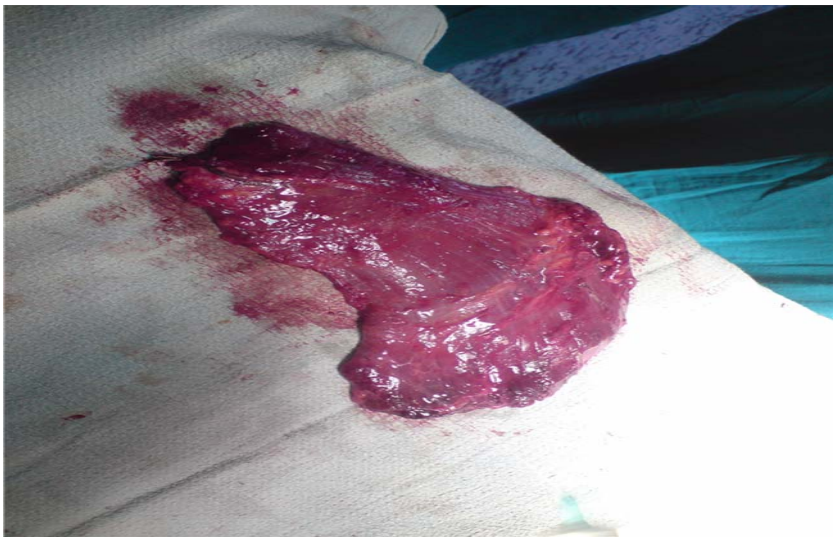
Charcot's degeneration of calcaneum



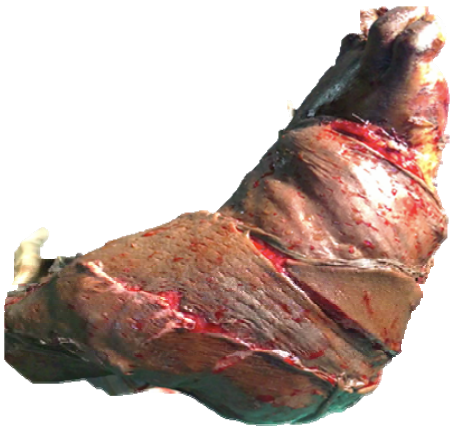
1 ½ year follow up of well settled Medial Plantar Artery Flap from opposite leg

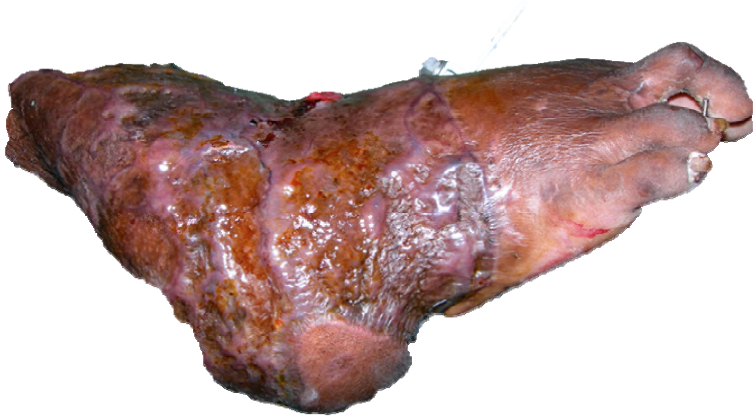


Complex composite reconstruction. Debridement done. 3rd toe removed. Instability of tarsum fixed by External Fixator

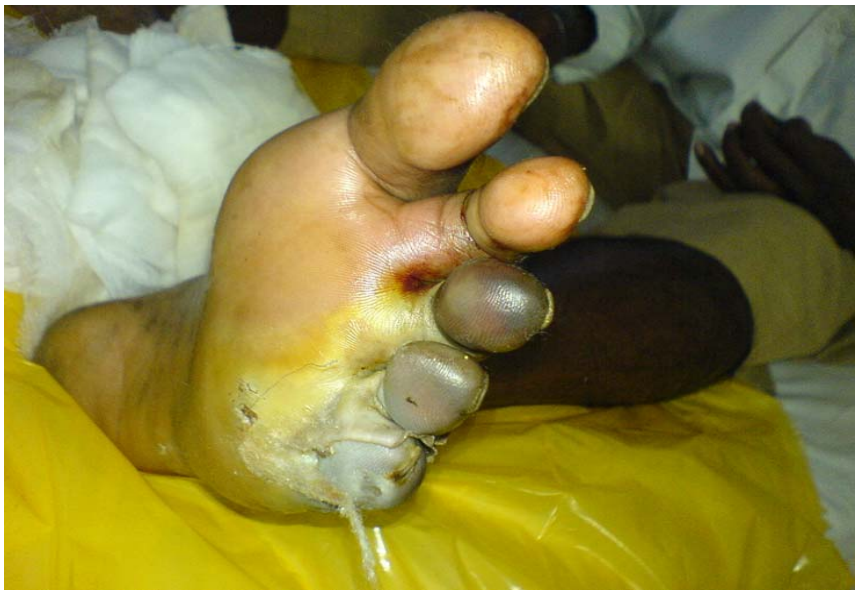


Latissimus Dorsi Flap

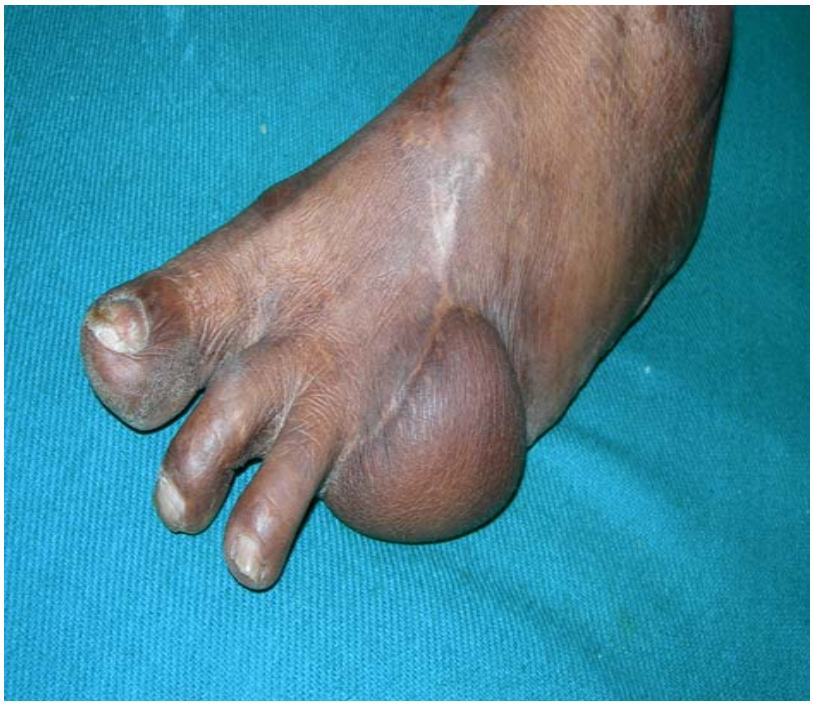




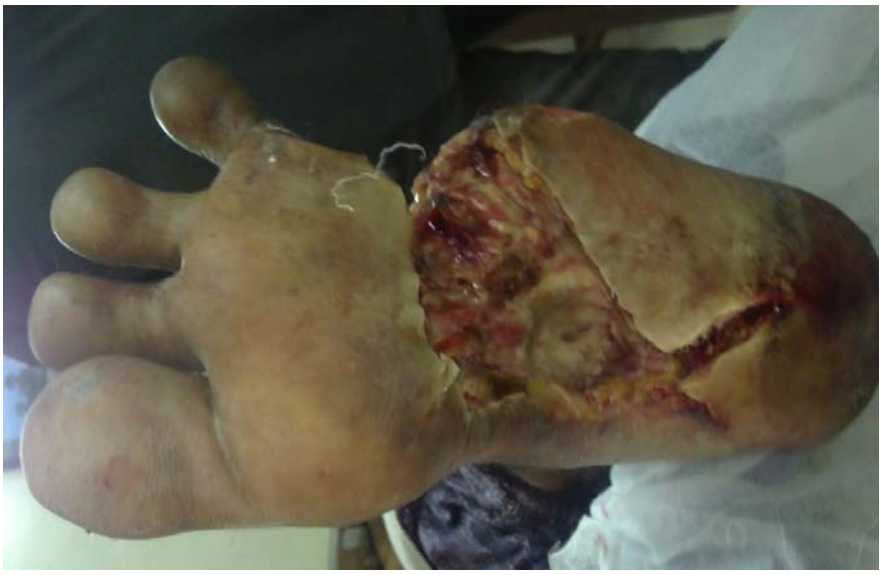
Latissimus Dorsi flap



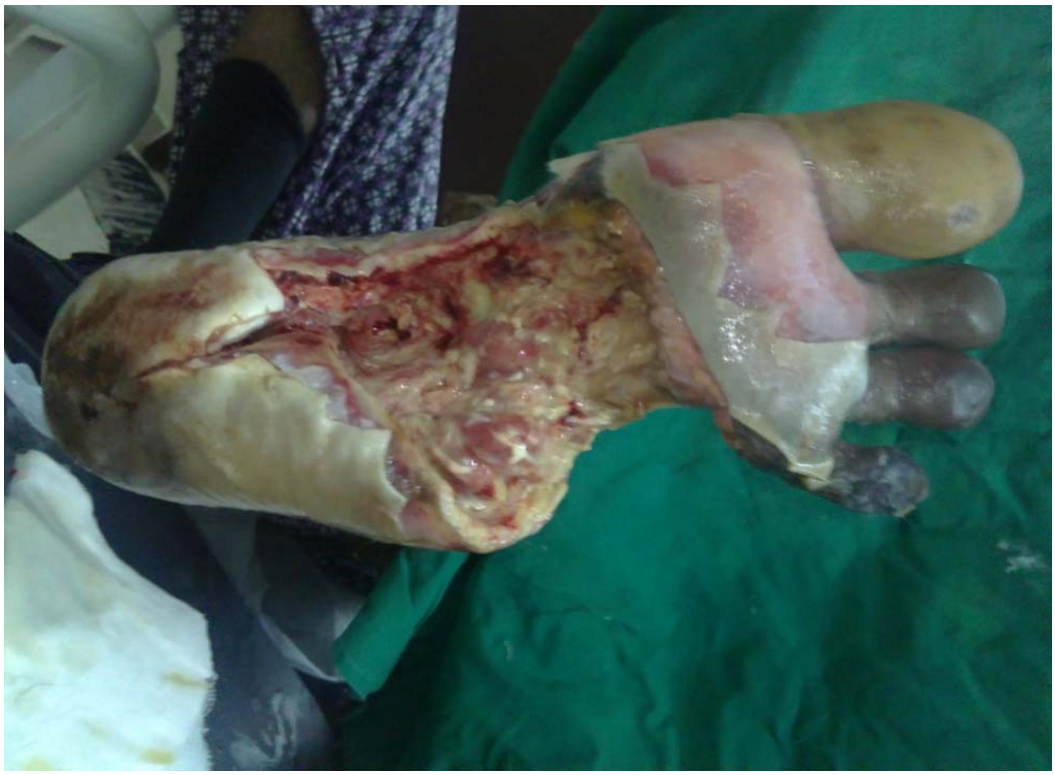
A case of severe tibial disease with inflow problem.Angioplasty and distal bypass done



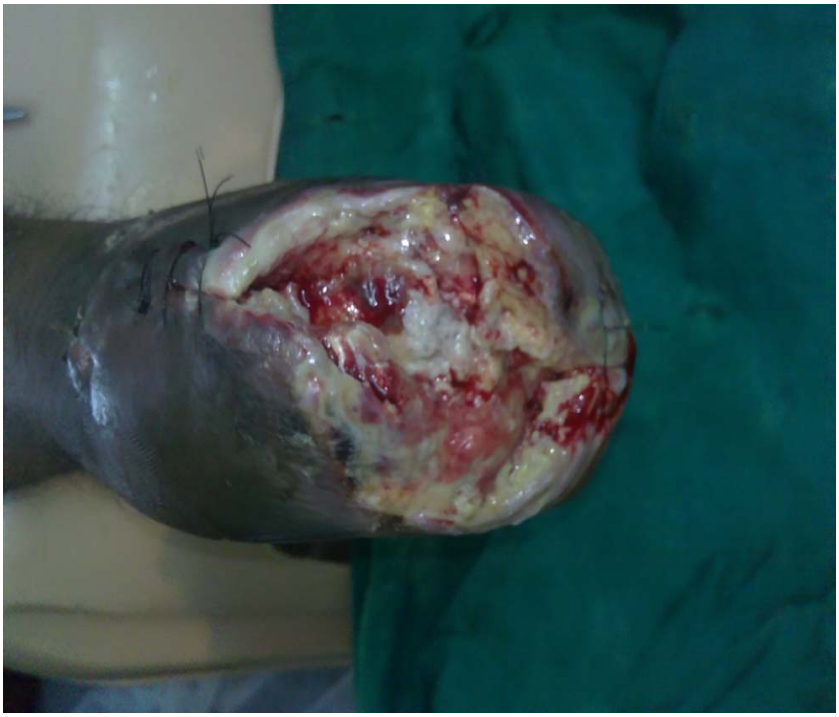
2 1/2 yrs late postop after Debridement and ALT flap cover



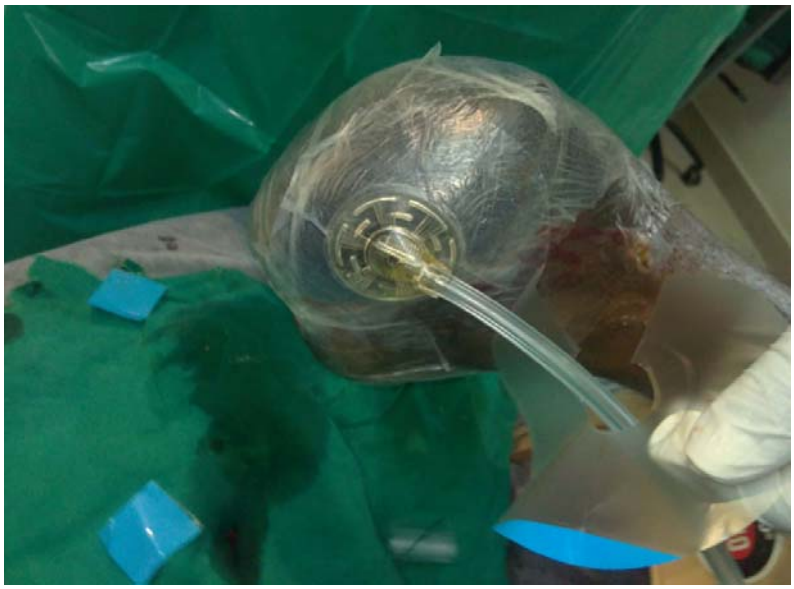
Femoro popliteal disease with debridement on table. RVI after 24 hours of revascularization



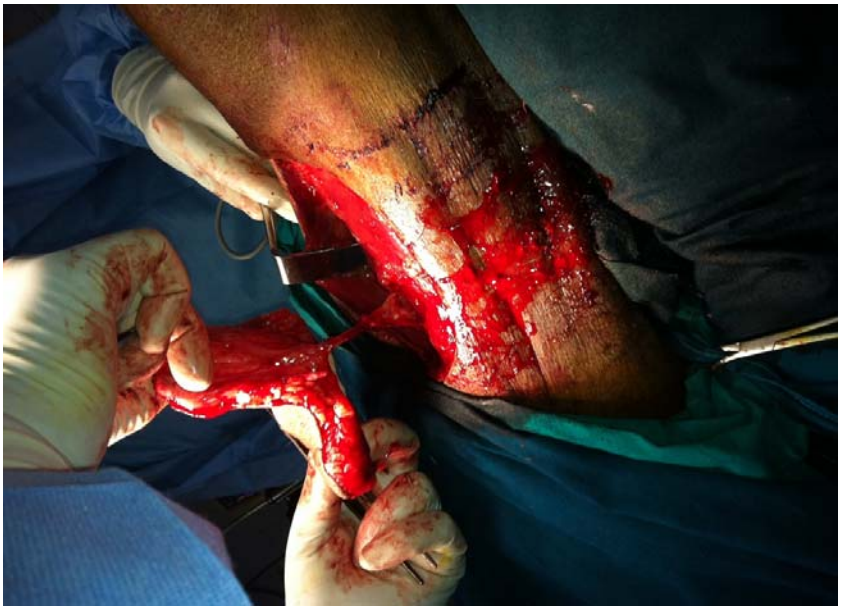
Presented with RVI which is evident with further debridement



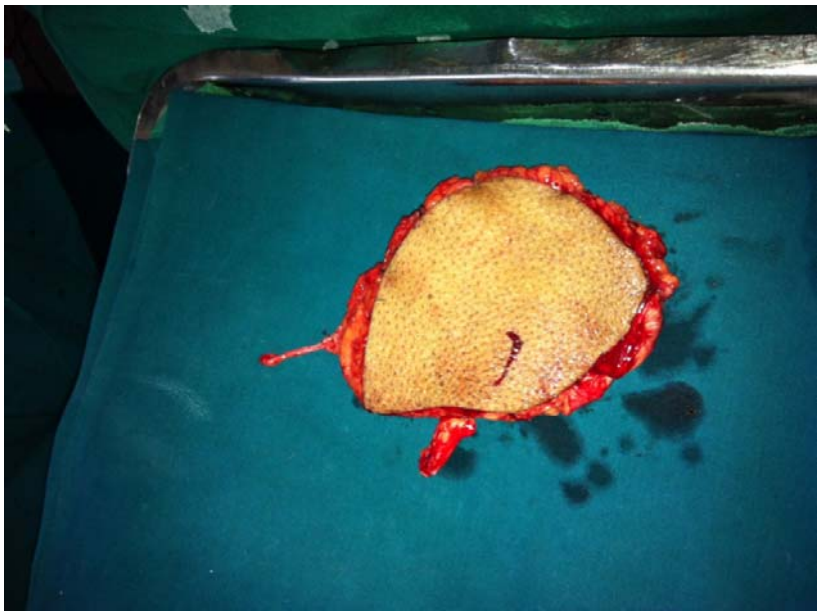
After failure of stump in modified Pirograff amputation



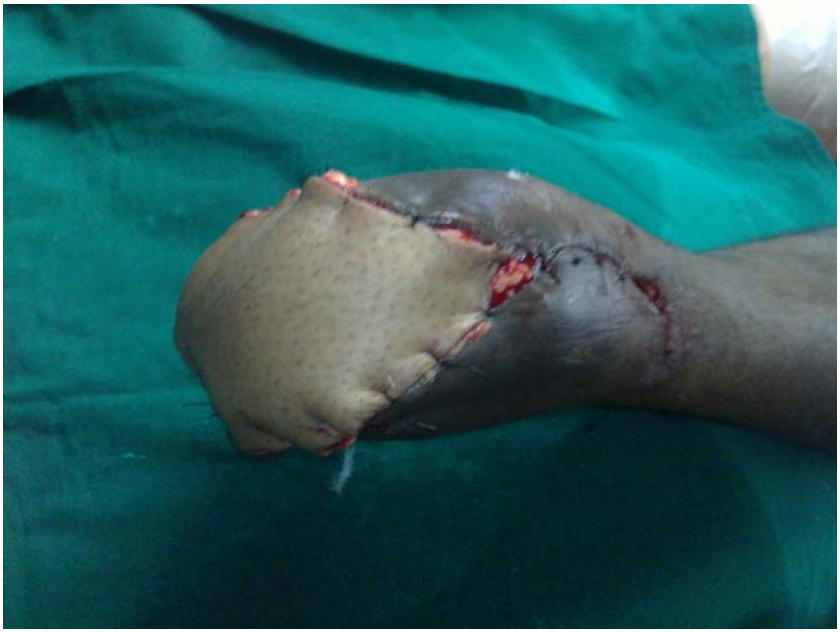
Wound treated with VAC



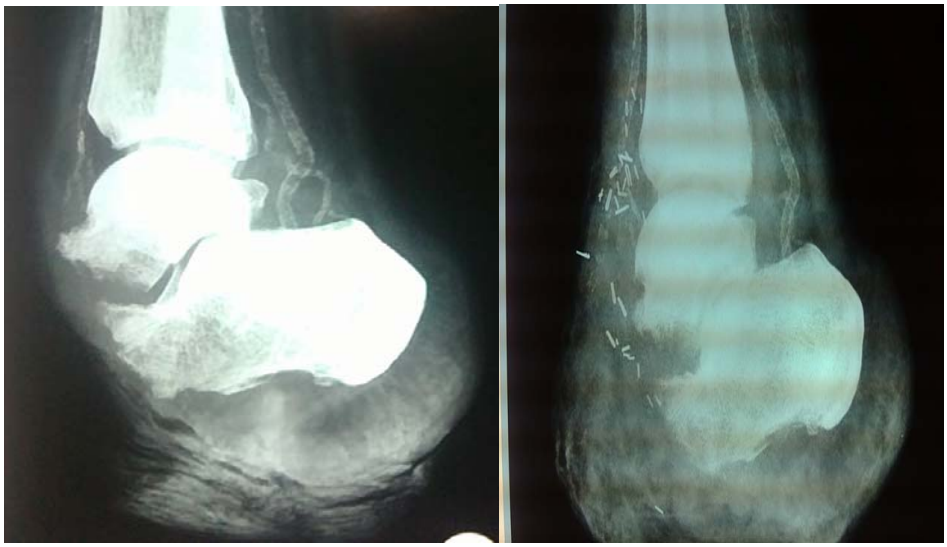
Microvascular composite tissue harvest



Free ALT flap with pedicle and lateral cutaneous nerve for neurotization

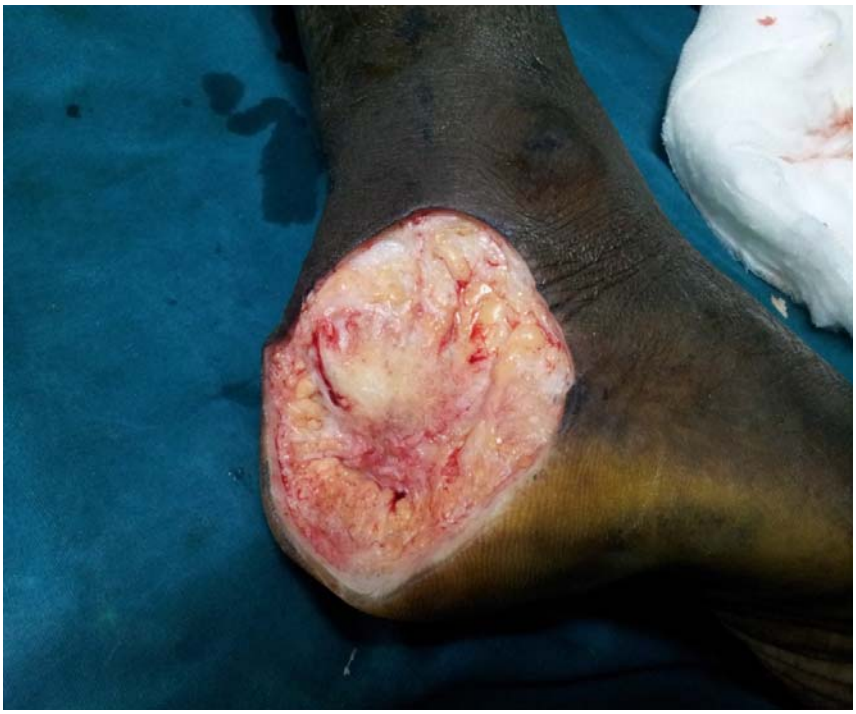


After ALT transfer(4th POD)

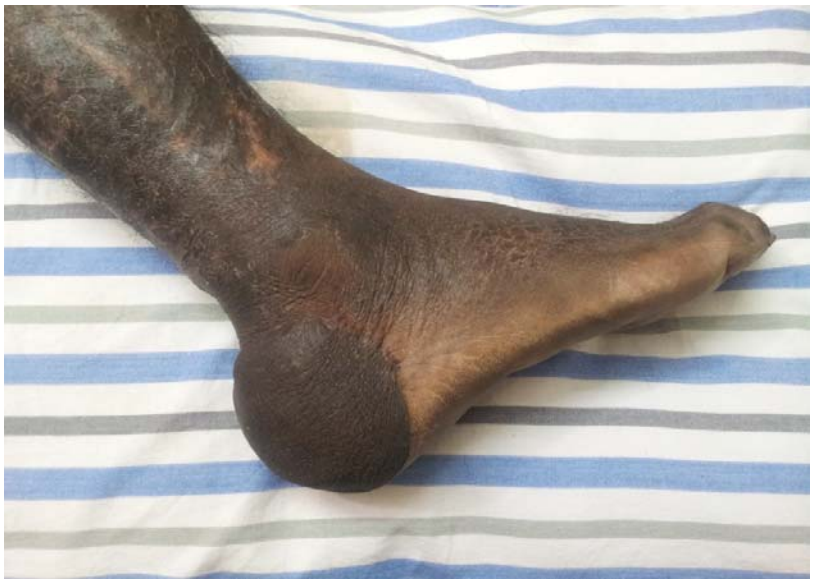


Monckebergs sclerosis pre op ***Post op***

1 1/2 years postop



Heel Defect



After Reverse Superficial Sural Artery Flap after 1 yr postop

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INSTITUTIONAL ETHICS COMMITTEE
MADRAS MEDICAL COLLEGE, CHENNAI-3

EC Reg No.ECR/270/Inst./TN/2013
Telephone No : 044 25305301
Fax : 044 25363970

CERTIFICATE OF APPROVAL

To
Dr. Adhil Ahamed Yameen.K,
PG in Plastic Surgery,
Department of Plastic Surgery,
Madras Medical College, Chennai-3.

Dear Dr. Adhil Ahamed Yameen.K,

The Institutional Ethics Committee of Madras Medical College, reviewed and discussed your application for approval of the proposal entitled **"Reconstruction in Revascularised Diabetic Foot"** No.22032014

The following members of Ethics Committee were present in the meeting held on 11.03.2014 conducted at Madras Medical College, Chennai-3.

1.	Dr. C. Rajendran, M.D.	-- Chairperson
2.	Dr. R. Vimala, M.D. Dean, MMC, Ch-3.	-- Deputy Chairperson
3.	Prof. Kalaiselvi, MD Vice-Principal, MMC, Ch-3	-- Member Secretary
4.	Prof. Nandhini, M.D. Inst. of Pharmacology, MMC, Ch-3.	-- Member
5.	Prof. Bhavani Shankar, M.S. Prof & HOD of General Surgery, MMC, Ch-3.	-- Member
6.	Prof. V. Padmavathi, M.D. I/c Director of Pathology, MMC, Ch-3.	-- Member
7.	Thiru. S. Govindasamy, BABL	-- Lawyer
8.	Tmt. Arnold Saulina, MA MSW	-- Social Scientist
9.	Thiru. S. Ramesh Kumar, Administrative Officer, MMC, Ch-3.	-- Layperson

We approve the proposal to be conducted in its presented form.

Sd/Chairman & Other Members

The Institutional Ethics Committee expects to be informed about the progress of the study, and SAE occurring in the course of the study, any changes in the protocol and patients information / informed consent and asks to be provided a copy of the final report.

13/3/14

Member Secretary, Ethics Committee

MEMBER SECRETARY
INSTITUTIONAL ETHICS COMMITTEE
MADRAS MEDICAL COLLEGE
CHENNAI-600 003

13/3/14

PROFORMA FOR CLINICAL CASE STUDIES

NAME: PS No:

AGE: ADM No:

SEX: D.O.Adm:

ADDRESS: D.O.Surg:

D.O.Dis:

Ph no:

PRESENTING COMPLAINTS:

HISTORY OF PRESENT ILLNESS

PAST HISTORY:

Co-morbidity:

PERSONAL HISTORY

Smoker/ non-smoker

TREATMENT HISTORY

GENERAL EXAMINATION

LOCAL EXAMINATION

PROVISIONAL DIAGNOSIS:

INVESTIGATIONS

TYPE OF REVASCULARISATION

NUMBER OF DEBRIDEMENTS

LATENCY PERIOD

ADJUVANT PROCEDURE

PLAN OF MANAGEMENT

OPERATION: Date:

(Photographs)

FOLLOW UP:

PATIENT'S SATISFACTION:

PATIENT CONSENT FORM

Study Title: Reconstruction in Revascularised Diabetic Foot

Study centre: Department of Plastic Reconstructive and Maxillofacial Surgery,
Madras Medical College and Government Hospital, Chennai – 600003

Patient's Name:

Patient's Age:

Identification Number:

Patients may check ( ) these boxes

I confirm that I have understood the purpose of procedure for the above study. I have the opportunity to ask the questions and all my questions and doubts have been answered to my complete satisfaction.

I understand that my participation in the study is voluntary and that I am free to withdraw at any time without giving reason, without my legal right being affected.

I understand that sponsor of the clinical study; others working on the sponsor's behalf, the ethical committee and the regulatory authorities will not need my permission to look at my health records both in respect of the current study and any further research

that may be conducted in relation to it, even if I withdraw from the study. I agree to this access; however I understand that my identity would not be revealed. In any information released to third parties or published, unless as required under the law. I agree not to restrict the use of any data or results that arise from this study.

I agree to take part in the above study and to comply with the instruction given during the study and faithfully to cooperate with the study team, and to immediately inform the study staff if I suffer from any deterioration in my health or my well - being or any unexpected or unusual symptoms.

I hereby give consent to participate in this study.

Signature/ Thumb impression of the patient:

……

Place:

……

Patient’s name and address:

……

Signature of the Investigator:

..……Place…… Date……..

Name of the Investigator:

……

INFORMATION SHEET

We are conducting study “Reconstruction in Revascularised Diabetic Foot” on patients attending Plastic and Reconstructive and Maxillofacial Surgery Department at Government General Hospital, Chennai.

The purpose of this study:

1. To study various methods to reconstruct revascularised diabetic foot to provide useful shoeable and stable foot or remnant of foot that renders him/her ambulant.

2. To form an evolving protocol in terms of timing of reconstruction and the best reconstructive flap techniques.

3. To identify complications and pitfalls related to revascularised diabetic foot reconstruction and describe how to avoid them.

The privacy of the patients in the research will be maintained throughout the study. In the event of any publication or presentation resulting from the research, no personally identifiable information will be shared.

Taking part in this study is voluntary. You are free to decide whether to participate in this study or withdraw at any time; your decision will not result in any loss of benefits to which you are otherwise entitled.

The results of the special study may be intimated to you at the end of the study period or during the study if anything is found abnormal which may aid in the management or treatment.

Signature of Investigator

Signature of Participant

<u>ஆராய்ச்சி ஒப்புதல் படிவம்</u>

ஆராய்ச்சி தலைப்பு
சர்க்கரை நோயால் பாதிக்கப்பட்ட காலில் இரத்த நாளங்கள் அறுவைசிகிச்சைக்குப் பின்னர் கால் மறுசீரமைப்பு பற்றிய ஓர் ஆய்வு

பெயர்	:	தேதி	:
வயது	:	உள் நோயாளி எண்	:
பால்	:	ஆராய்ச்சி சேர்க்கை எண்	:

இந்த ஆராய்ச்சியின் விவரங்களும் அதன் நோக்கமும் முழுமையாக எனக்கு தெளிவாக விளக்கப்பட்டது.

எனக்கு விளக்கப்பட்ட விஷயங்களை புரிந்துகொண்டு நான் எனது சம்மதத்தை தெரிவிக்கிறேன்.

நான் இந்த சர்க்கரை நோயால் பாதிக்கப்பட்ட காலில் இரத்த நாளங்கள் அறுவைசிகிச்சைக்குப் பின்னர் கால் மறுசீரமைப்பு ஆராய்ச்சி தாளை பெற்றுக் கொண்டேன்.

இந்த ஆராய்ச்சியில் பிறரின் நிர்பந்தமின்றி என் சொந்த விருப்பத்தின்பேரில் நான் பங்கு பெறுகின்றேன். இந்த ஆராய்ச்சியில் இருந்து நான் எந்நேரமும் பின் வாங்கலாம் என்பதையும் அதனால் எந்த பாதிப்பும் ஏற்படாது என்பதையும் நான் புரிந்துகொண்டேன்.

அறுவை சிகிச்சை செய்வதற்கு முன் வலி தெரியாமல் இருக்க மயக்க ஊசி முதுகில் போடுவதற்கு சம்மதம் தெரிவிக்கிறேன்.

மேற்கண்ட ஊசி போடும்போதோ அல்லது அறுவைசிகிச்சை செய்யும்போதோ ஏதேனும் பின்விளைவுகள் ஏற்படலாம் என்பதையும் மருத்துவர் மூலம் அறிந்து கொண்டேன்.

நான் என்னுடைய சுய நினைவுடனும் மற்றும் முழு சுதந்திரத்துடனும் இந்த மருத்துவ ஆராய்ச்சியில் என்னை சேர்த்துக்கொள்ள சம்மதிக்கிறேன்.

ஆராய்ச்சியாளர் கையொப்பம் **பங்கேற்பாளர் கையொப்பம்**

நாள் :
இடம் :

<u>ஆராய்ச்சி தகவல் தாள்</u>

சென்னை இராஜீவ்காந்தி அரசு பொது மருத்துவனையில் ஒட்டுறுப்பு அறுவை சிக்சைப்பிரிவில் சர்க்கரை நோயால் பாதிக்கப்பட்ட காலில் இரத்த நாளங்கள் அறுவைசிகிச்சைக்குப் பின்னர் கால் மறுசீரமைப்பு முறை ஆராய்ச்சி இங்கு நடைபெறுகிறது.

நீங்களும் இந்த ஆராய்ச்சியில் பங்கேற்க நாங்கள் விரும்புகிறோம். இந்த ஆராய்ச்சியில் உங்களுடைய இரத்தம், சிறுநீர் மற்றும் ஸ்கேன் ஆகியவற்றை சிறப்பு பரிசோதனைக்கு உட்படுத்தி அதன் தகவல்களை ஆராய்வோம். அதனால் தங்களது நோயின் ஆய்வறிக்கையோ அல்லது சிகிச்சையோ பாதிப்பு ஏற்படாது என்பதையும் தெரிவித்துக்கொள்கிறோம்.

முடிவுகளை அல்லது கருத்துகளை வெளியிடும்போதோ அல்லது ஆராய்ச்சியின் போதோ தங்களது பெயரையோ அல்லது அடையாளங்களையோ வெளியிடமாட்டோம் என்பதையும் தெரிவித்துக் கொள்கிறோம்.

இந்த சிறப்பு சிகிச்சையின் முடிவுகளை ஆராய்ச்சியின்போது அல்லது ஆராய்ச்சியின் முடிவின் போது தங்களுக்கு அறிவிக்கப்படும் என்பதையும் தெரிவித்துக் கொள்கிறோம்.

இந்த ஆராய்ச்சியில் பங்கேற்பது தங்களுடைய விருப்பத்தின் பேரில் தான் இருக்கிறது. மேலும் நீங்கள் எந்நேரமும் இந்த ஆராய்ச்சியிலிருந்து பின்வாங்கலாம் என்பதையும் தெரிவித்துக் கொள்கிறோம்.

ஆராய்ச்சியாளர் கையொப்பம் **பங்கேற்பாளர் கையொப்பம்**

நாள் :
இடம் :

S.No	Age	Sex	IP.No	Smoking	Revas cularisation	No.of debridements	Latency period	VAC	Reconstructive procedures	Adjuvant procedures	Complications	Follow up
1	45	M	138109	N	B	3	30	N	SFC	Nil	Nil	Y
2	48	M	54381	Y	B	3	38	N	SFC	Nil	Nil	Y
3	54	M	181171	Y	B	2	48	N	RSSA	Nil	Nil	Y
4	61	M	58341	Y	B	3	60	N	SFC	Nil	Nil	Y
5	60	M	33942	Y	B	3	54	N	SFC	Nil	Nil	Y
6	58	M	87542	Y	A	2	35	Y	LD	Os	Nil	Y
7	54	M	125944	Y	A	2	30	Y	LD	Nil	UR	Y
8	50	M	98634	Y	A	2	34	Y	IFC	Nil	Nil	Y
9	54	M	167543	N	A	3	20	N	SFC	Nil	Nil	Y
10	58	M	56743	N	A	2	25	N	SFC	Nil	Nil	Y
11	54	M	987652	Y	B	2	35	N	RSSA	Nil	FN	Y
12	56	M	453261	Y	B	2	38	Y	SFC	TAL	Nil	Y
13	58	M	567453	Y	A	2	25	Y	RSSA	Nil	Nil	Y
14	61	M	76548	Y	A	2	35	Y	IFC	Nil	Nil	Y
15	64	M	89765	Y	A	2	20	N	RSSA	Nil	Nil	Y
16	60	M	52987	Y	B	3	36	Y	SFC	TAL	Nil	Y
17	58	M	7864211	Y	B	2	21	Y	LD	Nil	Nil	Y
18	59	F	897453	N	A	2	28	Y	RFF	Nil	Nil	Y
19	55	M	908745	Y	A	2	22	Y	LD	Os	Nil	Y
20	56	M	564198	Y	A	3	29	N	SFC	Nil	Nil	Y
21	54	M	986754	Y	A	2	35	Y	LD	TEN	Nil	Y
22	57	F	390876	N	B	2	40	Y	RSSA	Nil	Nil	Y
23	56	M	467523	Y	B	3	45	Y	LD	Nil	UR	Y
24	58	M	980675	Y	A	2	22	Y	LD	Nil	Nil	Y
25	59	M	45632	Y	A	2	28	Y	RSSA	Nil	Nil	Y
26	55	M	34786	Y	A	2	24	Y	IFC	Os	Nil	Y
27	56	M	56432	N	A	3	45	N	LD	Os	Nil	Y
28	54	M	98765	Y	B	2	28	Y	RFF	TEN	Nil	Y

29	58	M	890756	Y	B	3	35	Y	LD	Os	Nil	Y
30	59	F	908765	N	A	2	27	Y	PF	Nil	Nil	Y
31	52	M	456732	Y	A	2	28	Y	RSSA	Nil	Nil	Y
32	48	M	456387	N	A	2	35	N	LD	Nil	Nil	Y
33	50	M	89076	N	A	1	26	N	RFF	TAL	Nil	Y
34	49	M	78654	N	B	2	30	N	RSSA	Nil	Nil	Y
35	56	F	12564	N	B	2	22	N	IFC	Nil	H	Y
36	59	M	567489	Y	B	3	56	N	RFF	Nil	Nil	Y
37	52	M	908765	Y	A	2	28	Y	G	Nil	Nil	Y
38	59	M	567211	Y	A	2	32	Y	RFF	Nil	Nil	Y
39	48	M	432115	N	A	2	40	N	LD	TAL	Nil	Y
40	54	M	907654	N	B	3	56	N	PF	Nil	Nil	Y
41	59	M	34256	N	A	2	31	N	RSSA	Nil	Nil	Y
42	64	M	564782	Y	B	3	49	N	SFC	TAL	Nil	Y
43	53	M	432562	N	B	3	58	N	RFF	Nil	Nil	Y
44	47	M	764890	N	B	2	35	N	FMP	Os	Nil	Y
45	51	M	765847	N	A	2	28	N	PSF	Nil	Nil	Y
46	57	F	468829	N	A	2	35	N	G	Os	Nil	Y
47	61	M	806778	Y	A	3	45	Y	SFC	Nil	Nil	Y
48	59	F	789934	N	B	2	32	N	RFF	Nil	Nil	Y
49	62	M	68888	N	B	2	42	N	PF	Nil	Nil	Y
50	54	F	466373	Y	B	3	52	N	LD	Nil	Nil	Y
51	58	M	378327	Y	B	2	47	N	RFF	Nil	Nil	Y
52	56	M	378838	N	A	2	33	N	ALT	Nil	PFN	Y
53	55	M	838637	Y	A	2	25	N	RFF	Nil	Nil	Y
54	59	F	635433	N	A	2	42	N	LD	Nil	Nil	Y
55	51	M	65672	N	A	2	26	N	RFF	Nil	Nil	Y
56	53	M	33889	Y	A	2	32	N	ALT	Nil	Nil	Y
57	49	M	64432	Y	A	2	26	N	ALT	Nil	Nil	Y
58	56	M	777666	N	B	3	44	N	PSF	Nil	Nil	Y

59	51	M	543287	Y	B	2	28	Y	PF	TEN	Nil	Y
60	52	M	53897	Y	B	1	22	Y	LD	Os	Nil	Y
61	57	F	67545	N	A	2	36	Y	RFF	Nil	Nil	Y
62	54	M	432521	Y	A	2	39	Y	G	TAL	Nil	Y
63	59	M	87628	N	A	2	28	Y	PSF	Nil	Nil	Y
64	61	M	423525	Y	A	3	48	Y	LD	Os	Nil	Y
65	56	M	776556	Y	A	2	26	Y	RFF	Nil	Nil	Y
66	52	M	89899	Y	B	3	41	Y	LD	TAL	Nil	Y
67	56	M	676787	N	B	2	38	Y	PSF	Nil	H	Y
68	59	M	54456	Y	B	3	51	Y	G	Nil	Nil	Y
69	53	M	87654	Y	B	2	42	Y	LD	Nil	Nil	Y
70	51	M	987653	Y	B	3	44	Y	PF	Nil	WD	Y
71	55	F	54289	Y	B	2	28	Y	LD	Nil	Nil	Y
72	56	M	88272	Y	B	2	34	Y	MP	Nil	Nil	Y
73	48	M	676755	Y	B	2	58	Y	PSF	Nil	Nil	Y
74	53	M	57682	Y	B	3	39	Y	ALT	Nil	Nil	Y
75	56	M	44345	Y	A	2	32	Y	G	Nil	Nil	Y
76	59	M	77666	Y	A	2	28	Y	ALT	Nil	Nil	Y
77	54	M	45788	N	A	2	34	Y	G	Nil	Nil	Y
78	57	M	987652	N	A	2	36	Y	LD	Nil	Nil	Y
							35					

KEY FOR MASTER CHART

TYPE OF REVASCULARIZATION

A – Angioplasty

B- Bypass procedures

Reconstruction Procedures

SFC - Superiorly based Fascio Cutaneous flap

IFC - Inferiorly based Fascio Cutaneous flap

RSSA – Reverse Superficial Sural Artery Flap

LD - Latissimus Dorsi flap

RFF - Radial Free Forearm flap

PF - Perforator Flap

G - Gracilis muscle flap

PSF - Para Scapular Flap

FMP -Free Medial Plantar artery flap

ALT – Antero Lateral Thigh flap

Adjuvant procedures

Os – Ostectomy

TAL – Tendo Achilles Lengthening

TEN – Tenotomy

Complications

FN – Flap Necrosis

PFN – Partial Flap Necrosis

WD – Wound Dehiscence

H – Hematoma

UR – Ulcer Recurrence

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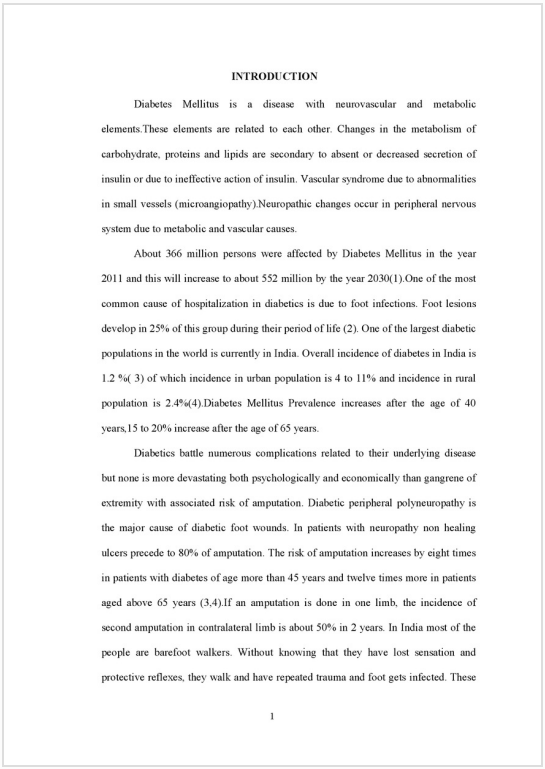

INTRODUCTION

Diabetes Mellitus is a disease with neurovascular and metabolic elements.These elements are related to each other. Changes in the metabolism of carbohydrate, proteins and lipids are secondary to absent or decreased secretion of insulin or due to ineffective action of insulin. Vascular syndrome due to abnormalities in small vessels (microangiopathy).Neuropathic changes occur in peripheral nervous system due to metabolic and vascular causes.

About 366 million persons were affected by Diabetes Mellitus in the year 2011 and this will increase to about 552 million by the year 2030(1).One of the most common cause of hospitalization in diabetics is due to foot infections. Foot lesions develop in 25% of this group during their period of life (2). One of the largest diabetic populations in the world is currently in India. Overall incidence of diabetes in India is 1.2 %(3) of which incidence in urban population is 4 to 11% and incidence in rural population is 2.4%(4).Diabetes Mellitus Prevalence increases after the age of 40 years,15 to 20% increase after the age of 65 years.

Diabetics battle numerous complications related to their underlying disease but none is more devastating both psychologically and economically than gangrene of extremity with associated risk of amputation. Diabetic peripheral polyneuropathy is the major cause of diabetic foot wounds. In patients with neuropathy non healing ulcers precede to 80% of amputation. The risk of amputation increases by eight times in patients with diabetes of age more than 45 years and twelve times more in patients aged above 65 years (3,4).If an amputation is done in one limb, the incidence of second amputation in contralateral limb is about 50% in 2 years. In India most of the people are barefoot walkers. Without knowing that they have lost sensation and protective reflexes, they walk and have repeated trauma and foot gets infected. These

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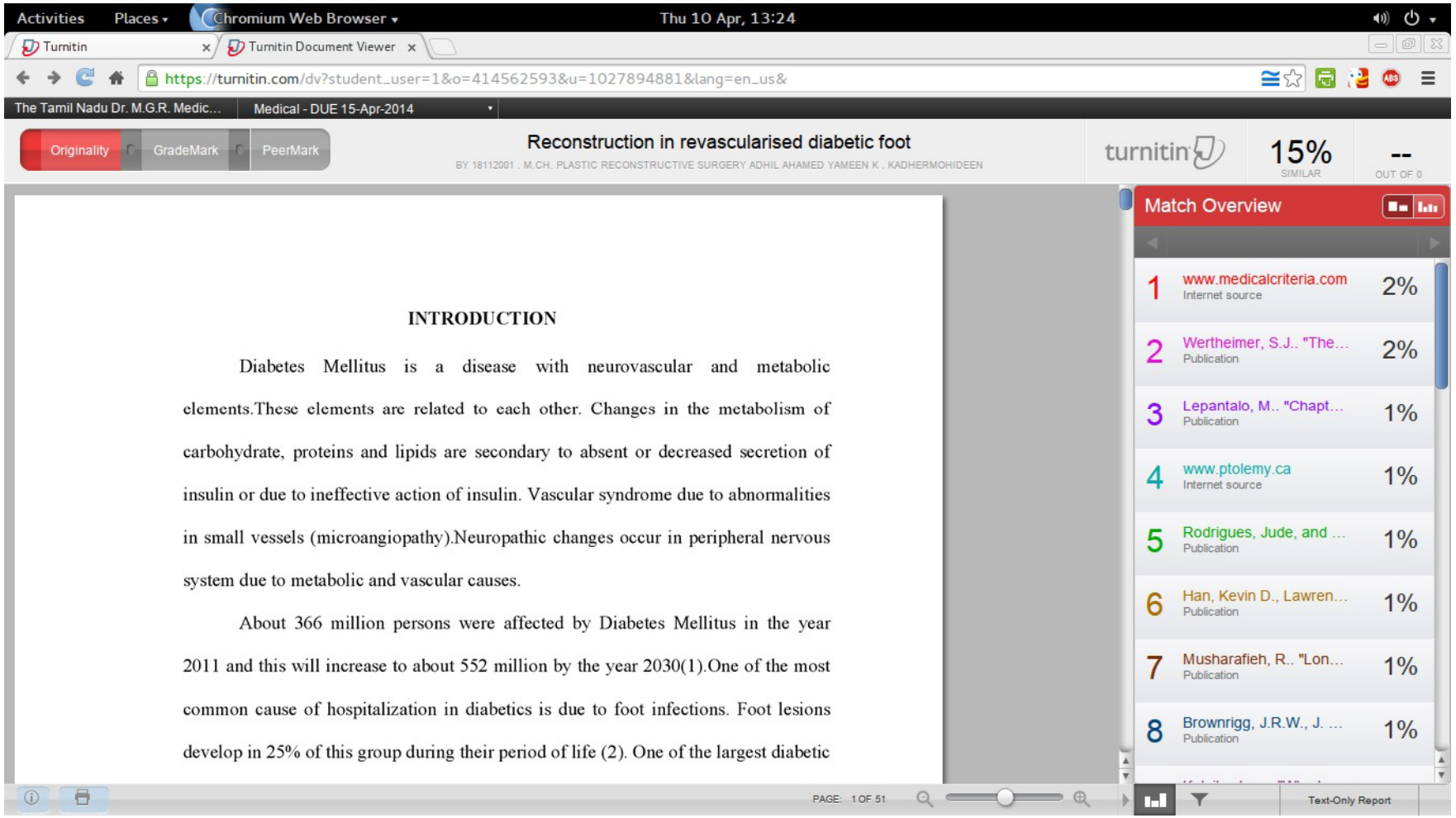
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INTRODUCTION
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