DISSERTATION ON
A STUDY ON THE EFFECT OF SPLIT SKIN GRAFT IN THE WOUND HEALING OF DIABETIC FOOT ULCERS

Dissertation submitted To

TN.DR.M.G.R MEDICAL UNIVERSITY

In partial fulfillment for the award of

M.S-I BRANCH

(General Surgery)

SUBMITTED TO

THE TAMILANU DR. M.G.R MEDICAL UNIVERSITY

CHENNAI- TAMILNADU

MARCH-2010

CERTIFICATE

This is to certify that the dissertation entitled "**DISSERTATION ON A STUDY ON THE EFFECT OF SPLIT SKIN GRAFT IN THE WOUND HEALING OF DIABETIC FOOT ULCERS "** is the bonafide original work **l** of **Dr.Priyadharshini.C** a post graduate of General surgery of Kilpauk medical college and hospital chennai under my Lord's guidance in partial fulfillment of the requirements for MS (General Surgery) branch I examination of the Tamil Nadu Dr. M.G.R Medical university to be held in March 2010. The period of postgraduate study and training was from May 2007 to March 2010.

PROF DR.A.PERIYASAMY ,MS.,
Professor and Chief
Department of General Surgery
Kilpauk Medical College and Hospital
Chennai -10

PROF.DR.S.UDAIYAKUMAR.M.S
Professor And Head
Department of General Surgery
Govt. Royapettah Hospital
Chennai -600 014

Dr.V.KANAGASABAI M.D
THE DEAN
Kilpauk Medical College and Hospital
Chennai – 600 010

ACKNOWLEDGEMENT

I am extremely grateful to my LORD JESUS who has guided me in doing this study.

I am thankful to **Dr. A.KANAGASABAI, MD,** Dean, Kilapuk Medical College and Hospital, Chennai for permitting me to use the hospital materials for this study.

I thank **Prof.Dr.S.UDAIYAKUMAR.M.S** General surgery H.O.D of Department of General surgery Govt.Royapettah hospital.chennai

I thank Prof. **Dr.A.PERIYASAMY , MS.,** Gen surgery professor, HOD department of general surgery KMCH Chennai for allowing me to do this study.

I thank **Prof.Dr.BHASKAR M.S** General surgery chief SII unit KMCH.

I Thank Prof. **Dr. BHAVANI SHANKAR MS**., General surgery chief SIII unit KMCH,

I thank all the Assistant civil surgeons for their help in doing this study

I thank my mother for her extreme support in doing this study.

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INTRODUCTION

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The diabetes Mellitus is rapidly increasing in prevalence worldwide. Many people with diabetes develop complications that seriously affect their quality and length of life.

Most common complications seen with them are foot ulcers and amputations. The diabetic foot is often an inching painless surprise that holds in its dark portals a soon rising flood of complications.

It is a quiet dread of disability causing recurrent hospitalisation and prolonged hospital stay mounting impossible expenses.

Foot lesions in a person with diabetes is indeed an expensive proposal.

The costs of treating these complications account for about 25% of the hospital costs of diabetes. The indirect costs in loss of eamings and productivity can only be guessed as it is thought these amount to about 50% of the total direct costs of treating the diabetes.

While progress has been made in the treatment of diabetic foot ulceration particularly by the establishment of dedicated diabetic foot clinics which have reduced bed usage by up to 38% there still remains much morbidity and mortality.

AIM OF THE STUDY

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AIM AND OBJECTIVE:

This study was done to know about the effect of split skin graft graft in treating the diabetic foot ulcers.

The objectives of this study are

- To study the effect of split skin graft in the wound healing of diabetic foot ulcers
- To know the length of hospital stay
- To know the donor site infections.

REVIEW OF LITERATURE

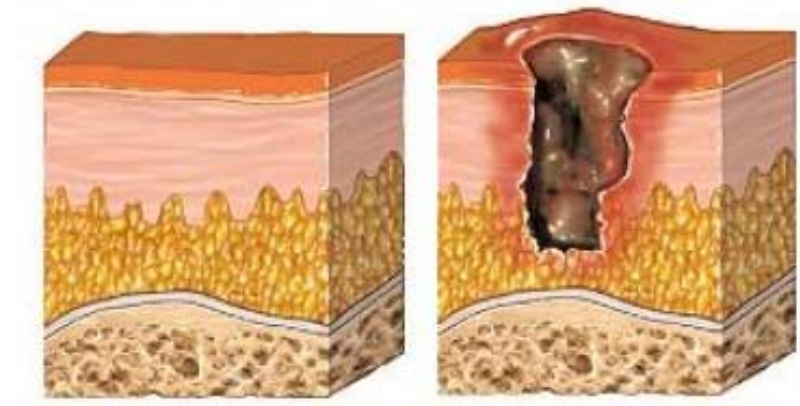
REVIEW OF LITERATURE

DIABETIC FOOT ULCERS

DEFINITION :

Diabetic foot ulcers are defined as full thickness wound below the ankle irrespective of the duration.

Diabetic foot ulcers are wound that occur on the feet of the people with type 1 and 2 diabetes mellitus. These ulcers usually occur on the bottom of the foot.



EPIDEMIOLOGY:

The global burden of diabetes is projected to increase form the current 246 million people to over 380 million people by the year 2025.

Among the people with diabetes 15% will experience a foot ulcer in their lifetime. Foot ulceration with infection is one of the leading cause of hospitalisation for patients with diabetes mellitus .

It is now appreciated that 15-20% of patients with such foot ulcers go on to need an amputation, especially major lower extremity amputation. Similarly 85% of major lower limb amputation are preceded by a foot ulcer.

Once one limb has been amputated not only is there an increased risk that the other limb will also require amputation but the patient's 5 year survival rate is only about 30%.

Since they are the major cause of lower limb amputations in these people they should be treated aggressively in order to have a better outcome.

ETIOLOGY :

It is firstly important to appreciate that the etiology of diabetic foot disease is truly multifactorial.

Within any individual patient one factor may predominate over all or some of the others but generally foot disease arises from more than one cause.

Diabetic foot ulcers are contributed by

1. Sensory, motor and autonomic, neuropathy
2. Macrovascular and Microvascular diseases
3. Infections
4. Connective tissue abnormalities
5. Haematological disturbances.

PATHOGENESIS:

NEUROPATHIC ULCERS:

Neuropathic ulcers may be

Due to – Sensory

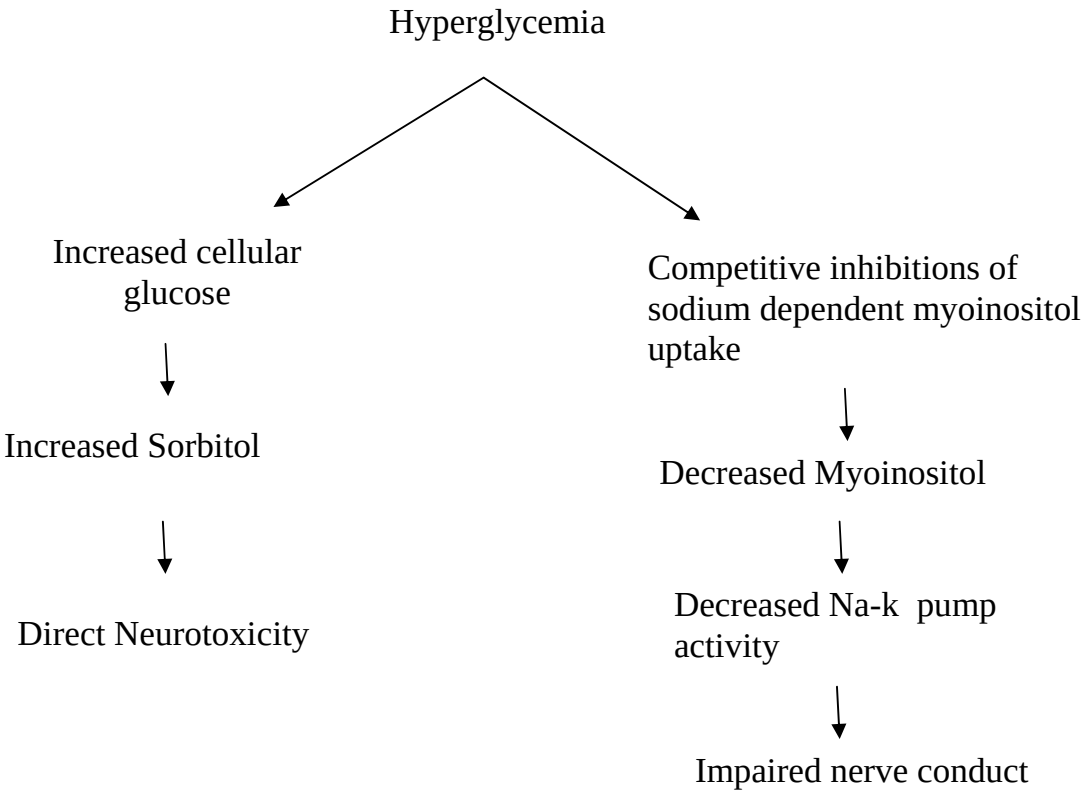
- Motor
- Autonomic

CAUSES OF NEUROPATHY :

There are essentially two theories as to the causation of diabetic peripheral neuropathy- one related to metabolic factors and the other associated with microvasular disease.

The metabolic theory suggests that peripheral nerve damage arises from abnormalities of sugar-alcohol metabolism. Hyperglycemia results in increased levels of intraneural sorbitol which may be directly toxic to the neural tissue.

Hyperglycemia also reduces the sodium-dependent uptake of myoinositol by competitive inhibition.



The reduction in myoinositol levels impair the action of the membrane bound sodium –pottassium dependent ATPase.

This results in the reduction of activity of the sodium –potassium pump and a reduction of nerve conduction velocity.

Microcirculatory changes also have an adverse effect on nerve metabolism as it is discussed below.

EFFECTS OF NEUROPATHY :

Loss of somatic sensation over the plantar aspect of the foot can lead to extrinsic neuropathic foot ulceration following trauma. The trauma can be varied – ill fitting footwear, thermal, foreign bodies in shoes and toenail cutting are merely examples.

The initial trauma is often minor and a person with intact sensation would naturally tend to protect the injury until it is healed.

In the absence of somatic sensation, however areas, which would normally be painful are not perceived as such so allowing tissue damage to continue, once started, an established ulcer is the end point of the this process.

The somatic motor neuropathy, results in weakness of the intrinsic muscles of the foot which in turn allows abnormal movement of the small bones of the foot and joint subluxation occurs. Weakness of foot ligaments due to abnormalities of collagen metabolism contributes to this effect.

Visceral sensory neuropathy recluces or abolishes proprioception. Though the early bony deformity is small, as patient continues to walk,

ligaments and joint capsules are stretched further and the bony structures of the foot are altered permanently.

As time goes on these changes lead to foot deformities such as a claw foot with prominent metatarsal heads or a Rocker-bottom foot with collapse of the longitudinal arch and prominence of the tarsal bones. The inflammation of the subluxed joints leads to charcot's arthropathy.

HOW DOES AN ULCER FORM:

The above bony changes produce localized areas of high pressure on the sole of the foot particularly under the metatarsal heads, on the tips of toes, on the heel and under the mid foot.

These high pressure areas are assoicated with ulceration around three quarters of neuropathic ulcers occur in the forefoot, while, the remainder occur under the midfoot and on the heel.

The initial response to this high pressure is the formation of protective callus. In addition to the vertical load force resulting from the patient's weight acting on the callus, the transverse and longitudinal shear forces are also established.

The shear forces particularly those in the longitudinal plane traumatise the subcutaneous tissues between the underlying bone and overlying callus producing cavities containing serum or blood.

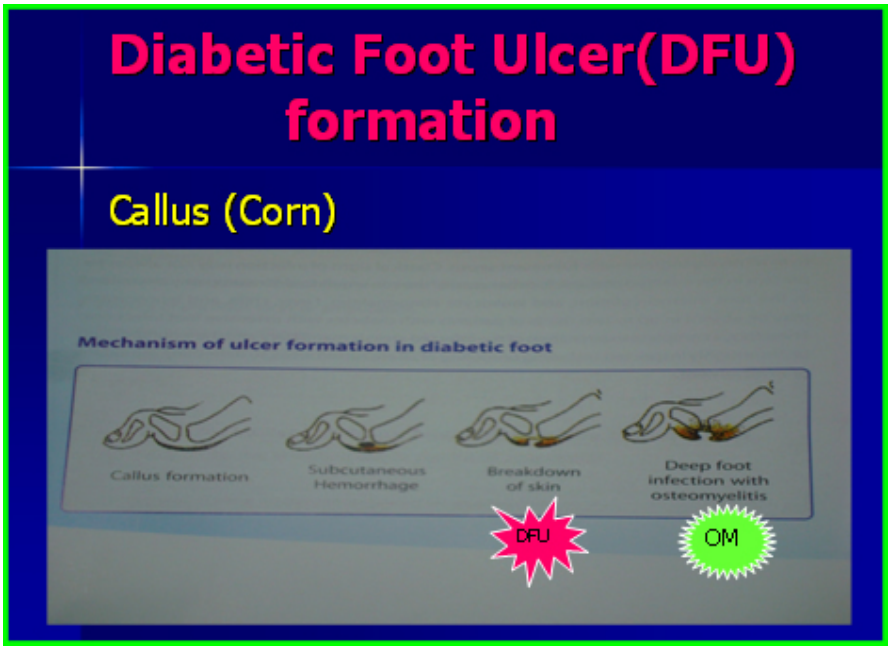
The cavities under the callus coalesce and eventually the callus beaks down resulting in an ulcer. The breakdown tends to occur centrally and the defect in the callus is much smaller than the cavity underneath.

This pattern of deep tissue destruction preceding epithelial breakdown is typical of neuropathic ulceration and differs from most other foms of ulcer.

Autonomic neuropathy also contributes to the formation of calluses through a reduction in sweating. Sweat contains keratinolytic enzymes which helps break down of hyperkeratotic areas.

In their absence the callus shows unimpeded growth. The absence or reduction of sweating also results in skin that is dry, inelastic and prone to trauma.

DIABETIC FOOT ULCER FORMATION



STAGES OF ULCER FOFMATION

1. CALLUS FORMATION
2. COLLECTION BENEATH THE CALLUS
3. BREAKDOWN OF CALLUS
4. ULCER

NEUROPATHIC ULCERS

ISCHEMIC ULCERS:

DIABETIC MICROVASCULAR DISEASE :

There are structural abnormalities of the capillary basement membrane in diabetes. The basement membrane is thickened as part of the general abnormality of extracellular matrix components and its composition is altered by excessive glycosylation of collagen and proteoglycans.

This glycosylation reduces the charge on the membrane . These changes may explain the increase in capillary permeability to highly charged molecules such as albumin.

It also affects transcapillary movement of leucocytes and macromolecules. Diabetics have shown to have abnormal function of increased flow through the distal arterio-venous shunts proximal to the dermal capillary bed.

There is an increased capillary luminal diameter secondary to basement membrane thickening and there is also an increase in capillary flow.

Although there is, increased capillary flow there is less ability to vasodilate and increase blood flow in response to various stimuli like trauma or infection. Furthermore the vasoconstrictor response to the vertical posture is reduced with the result that capillary pressure rises. This increases edema formation with the effect of impairing tissue perfusion.

Endothelial functions are also disturbed in diabetes. There is normally a fine balance in favor of vasodilatation over vasoconstriction and an anti thrombotic tendency brought about by reactions involving nitric oxide (Endothelium derived relaxing factor). Diabetes impair this function of the endothelium, which results in impaired microcirculation.

All of the above changes seen in the diabetes have a real effect on tissue perfusion and potentially play an important role in ulceration.

DIABETIC MACROVASCULAR DISEASE :

Diabetes are four to seven times more prone to atherosclerosis than non – diabetics and the process appears to be accelerated in diabetic patients requiring vascular surgery are therefore likely to be younger than non – diabetics.

Atherosclerosis in diabetes is the same condition as it is in the absence of diabetes, where it differs is in the prevalence and distribution of disease.

Atherosclerosis tends to affect the tibial and peroneal arteries in diabetes in contrast to non – diabetics where the affected vessels tend to be above the knee. This difference in disease distribution demands more distal bypasses.

One caveat to this is vascular calcification. The media of arteries in diabetes tends to become calcified, often in excess of the degree of

atherosclerosis that is present. The calcification is often visible on plain radiography or an angiography.

While this calcification per se does not result in vascular obstruction, it increases the impedance of the vessel can making surgery difficult and makes measurement of ankle brachial indices unreliable. Thus the ankle brachial indices in these patients are unreliable.

The pressure required to obliterate the Ankle-Doppler signal in such cases is often falsely high and in some patients it is not possible to obliterate the signal.

A more realistic measure of perfusion pressure may be obtained by using toe digital artery pressure the vessels within the toes can be spared the effects of calcification.

It may be thought that measurement of tissue oxygen partial pressure would overcome these difficulties of non-invasive arterial pressure assessment.

If there is any doubt regarding the peripheral circulation in a diabetic patient, angiography can be performed.

NEURO ISCHEMIC ULCERS:

Neuro ischemic ulcers (Mixed Etiology ulcers) have both neuropathy and ischemia.

INFECTION:

Infection is not generally a primary cause of foot lesions in diabetes with the exception of fungal infection between the toes which can lead to skin breakdown and secondary bacterial infection.

Once a lesion has developed infection plays an important role in determining its outcome whether the primary etiology is neuropathic, ischemic or a combination of the two. (ie neuroischemic lesion)

There are several reasons for an increased propensity to infections in diabetes.

These include abnormalities of the inmune system with deficiencies in cell mediated immunity, impaired leukocyte chemotaxis, phagocytosis intracellular bactericidal activity and serum opsonization. There is reduction of granulcyte motility and activity.

The infection is virtually always polymicrobial with gram positive and gram Negative aerobes and anaerobes including staphylococcus aureus, Bacterioides, Proteus, Enterococus, Clostridia and Escherichia coli being present.

Bacteria that overcome the host defences quickely colonise the wound, increasing in density until their cell signailing increases and gene expression alters.

This bacterial response produces a three- dimensional polysaccharide matrix, forming a 'BIOFILM' which increases virulence, lessens the host response to infection and thereby increasing the antimicrobial resistance.

This 'BIOBURDEN' may be responsible for delayed wound healing where bacterial numbers reach "CRITICAL COLONISATION' with no overt host responses when the 'Bioburden' reaches beyond the point of critical coloinsaiton. **(10^5 bacteria/g)** and bacterial invade the tissues causing direct cell damage.

Necrotic tissues should be debrided to reduce the bioburden . similarly infected bone requires proper surgical debridement to enable healing.

When infection is secondary to a primary neuropathic or ischemic lesion it may remain superficial and localized.

A Spreading cellulitis can also develop however or the infection can spread into the deeper tissues. Drainage is the most problematic in neuropathic ulcers with a small defect in the overlying callus.

If this defect is blocked a either by hyperkeratinization or by inspissated contents, then the infection is direly to spread into the deeper tissues and bones. This may results in abscess formation or osteomyelitis.

In this way infection is responsible for most of the tissue destruction seen in complicated diabetic foot lesions. It's actually a vicious cycle which helps in spreading the infection to surrounding areas, by compromising the blood flow due to edema formation.

The small vessels within this area are prone to thrombosis and occlusion as a results of sluggish flow due to platelet and leucocyte adhesion to vessel walls. These two factors may combine to produce localized tissue ischemia and even gangrene particularly in presence of macrovascular disease.

CONNECTIVE TISSUE ABNORMALITY:

The hyperglycemia of diabetes can significantly affect the structure and functions of proteins. This is most commonly brought about by non –enzymatic glycosylation, a process in which glucoses first binds reversibly with amino groups on proteins. These then irreversibly bind more glucose to form advanced glycosylation end products.

These can then form covalent cross –links with amino groups on others matrix proteins or on extravasated plasma proteins. These reactions are seen with haemoglobin , where the glycosylated product is hemoglobin AIc – a well known marker of the plasma glucose level over the previous 6 weeks.

The structural proteins collagen and keratin are all affected. As a results the inter-molecular cross linking produces tissues that are rigid, inflexible and resistant to digestion by proteases.

The rigidity of the subcutaneous tissue between the callus and underlying bone renders it more likely to be torn by the shear forces referred above. The resistance of the keratin to keratinases helps explain the production of callus both at sites of high pressure and at edges of open ulcers.

The protein cross linking makes this callus hard and at the edge of an ulcer which delays the wound healing by preventing wound contraction.

The collagen of the ligaments and joint capsules of the foot is affected in the sameway. These structures then become weak and inelastic and the process contributes to the deformation of the bony structure of the foot.

HAEMATOLOGICAL DISTURBANCES

Rheological abnormalities in diabetes contribute to ischemic ulcer formation and to the spread of infection. Red cells are less deformable possibly due to glycosylation of their cell membrane.

This along with a tendency towards hypercoagulability and increased plasma viscosity may play a part in reducing capillary circulation so contributing to any ischemia that is present.

FOOT DEFORMITY

X-RAY SHOWING FOOT DEFORMITY

“AT RISK FOOT”

All diabetics should undergo daily examination of their feet, either by themselves or by a relative, and especially by a trained health care professional.

The examination is done to assess the degree of risk of developing foot ulceration. This introduces the concept of the ‘**at risk**’ foot. Assessing this degree of risk is central to the examination of a diabetic patient.

During this assessment, attention must be paid to determining the presence and degree of

- Neuropathy,
- Peripheral vascular disease
- History of previous ulceration
- Foot’s appearence

With this information a patients foot can be described as normal, ischaemic, neuropathic or neuroischemic. This is essential for appropriate treatment plan.

NEUROPATHY :

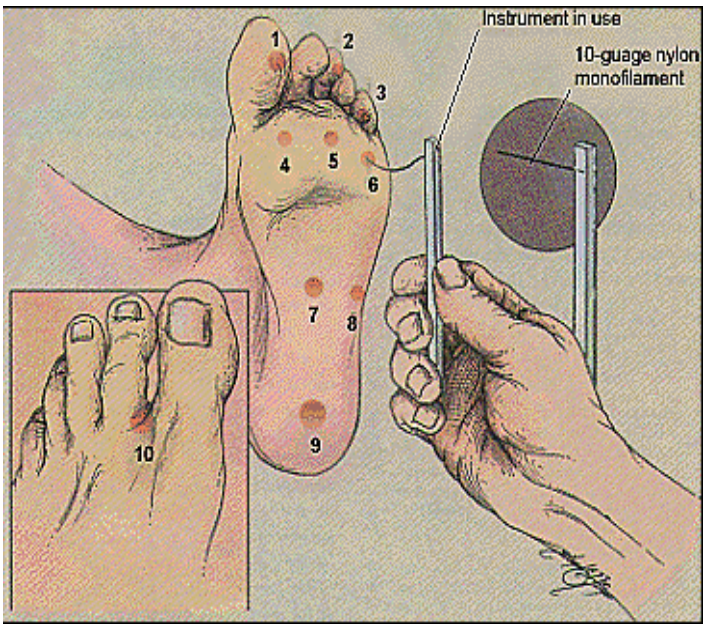
Neuropathy may be tested by the traditional clinical methods examining the various sensory modalities, muscle power and the knee and ankle reflexes.

The pain sensation can be assessed with nylon monofilaments and vibration sense with a biosthesiometer.

Nylon monofilaments are 5-10cm lengths of nylons of two differing thicknesses one which will buckle when a load of 80g is applied and the other at 10g.

The 10g monofilament is pressed onto the skin fo the foot until it buckles if the patient is unable to feel this then it is assumed that protective sensation is lost. If the paitent is unable to feel the 80g monofilament then a severe neuropathy is present.

TEST TO FIND OUT THE SENSATION



The biosthesiometer allows the vibration perception threshold to be measured . The device is placed on the great toe tip or onto a malleolus and the voltage increased until the patient can feel the machine vibrating. A threshold greater than 25v indicates significant neuropathy.

PERIPHERAL VASCULAR DISEASE:

Peripheral vascular status is assessed clinically by eliciting any history of claudication or rest pain and by examination of foot pulses and any foot colour changes, with elevation and dependency.

The presence of gangrene and the appearance of any ulcers are noted. This is supplemented by Ankle Brachial pressure index (ABPI) measurements.

However upto 10% of diabetic patients will have falsely high ABPI due to vascular calcification in the arterial media and so caution is required in their interpretation.

Absence of foot pulses make peripheral vascular disease likely and the foot is labeled as ischemic

If there is any doubt aobut the blood flow of the foot, Doppler study, duplex scanning and angiography can be done to know the adequacy of the blood flow. Magnetic Resonance angiography may demonstrate a patent vessel not seen on catheter angiography.

TRANSCUTANEOUS OXYGEN PRESSURE MAPPING:

Transcutanous oxygen pressure ($TcPo_2$) mapping can be used to determine the severity of foot ischemia, thus aiding selection of appropriate treatment and decreasing the total cost of care.

Studies show that if transmetatarsal ($TcPo_2$) level is 30mm Hg or greater treatment should be conservative comprising local wound care, debridement or a more minor ablative procedure. If the transmetatarsal ($TcPo_2$) level is below 30mm Hg it will anticipate the need for vascular reconstruction.

ANGIOGRAPHY:

Angiography remains the gold standard for assessment of the lower arterial system prior to any intervention. It can be performed via femoral or brachial catheterization, with iodine based contrast used to visualise the blood vessels.

MAGNETIC RESONANCE ANGIOGRAPHY:

Magnetic resonance angiography (MRA) is likely to be the future investigation of choice. Current systems give clear visualisation of vessels above knee level but tend to give rather fuzzy images of crural vessels distally.Diabetic patients with poor renal function may benefit form MRA.

MR imaging is also extremely valuable as it shows even early infection, with changes in bone marrow, edema of soft tissues, cavitations and sinus formation.

PREVIOUS ULCERATION:

Previous ulceration is one of the most significant risk factors for the development of subsequent ulcers.

A careful history regarding the development of location, treatment and eventual healing of the ulcer will give strong pointers as to its etiology and to the pattern of possible future ulceration.

FOOT APPEARENCE :

The feet should be examined for any change in shape which may lead to the development of areas of high pressure, prominent metatarsal heads, hammer toes or a collapsed midfoot.

Such areas may be identified by the presence of callus or by excessive wear on the sole of the shoe at that site. These areas are particularly prone to ulceration and should be watched carefully.

Any dryness of the skin is also noted .The toes and interdigital spaces are examined for fungal infection and for small ulcers which may not be immediately apparent.

The shoes should be looked at to determine their fit and any sites which may rub. Because ill-fitting shoes can produce Bunions, Hammertoes, Callouses, Blisters, Ulcers.

RISK ASSESSMENT:

It is useful to stratify the information obtained and grade the degree of risk of ulcer development for individual patients.

This helps determine how intensive foot observation has to be and can impress on high risk patients the need for vigilance.The following is a pragmatic stratification

RISK OF ULCER DEVELOPMENT FOR INDIVIDUAL PATIENTS

FEATURES	**LOW RISK**	**MODERATE RISK**	**HIGH RISK**
Sensation Vascularity History Appearence Vision / Mobility	Normal Palpable pulses No previous ulcer No deformity Normal	Neuropathy or/ Absent pulses No previous ulcer No deformity Normal	Neuropathy or/ Absent pulses Previous ulcer Deformity Impaired, with any of the above

CLASSIFICATION OF DIABETIC FOOT ULCERS:

There are various classifications they are :

(i) WAGNER MEGGITT CLASSIFICATION

(ii) UNIVERSITY OF TEXAS SYSTEM CLASSIFICATION

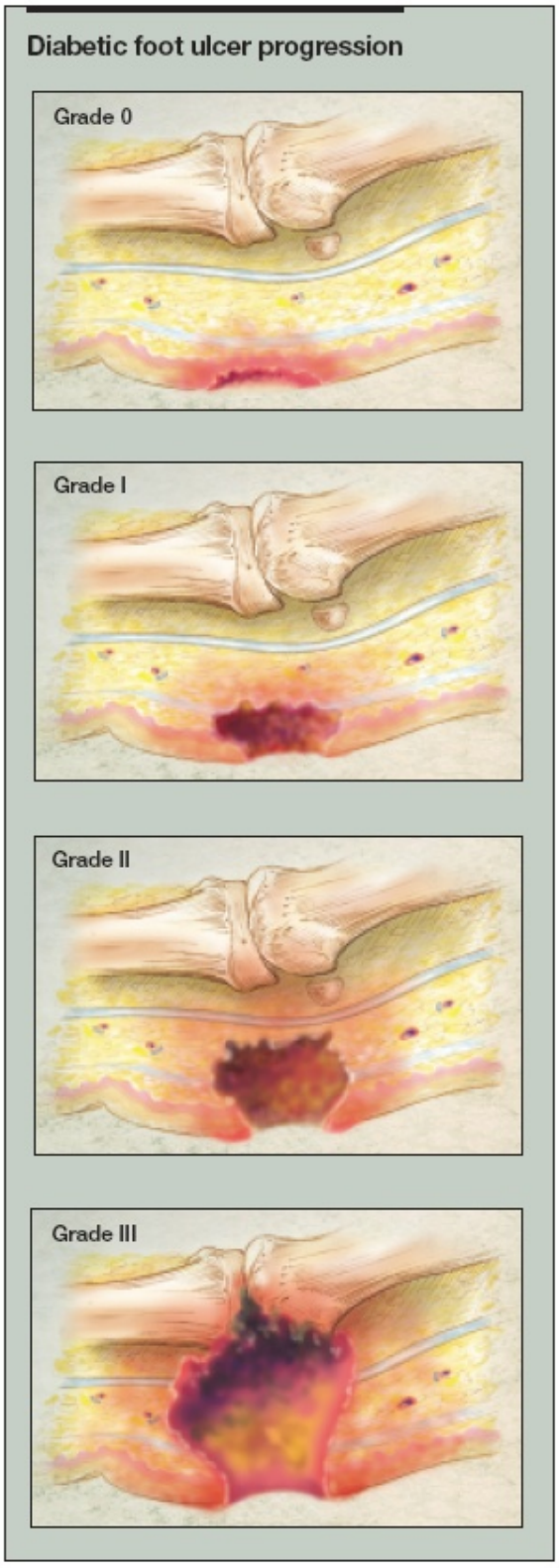
(iii) BRODSKY DEPTH ISCHAEMIA CLASSIFICATION

The most common classification followed is WAGNER – MEGGITT CLASSIFICATION.

WAGNER MEGGITT CLASSIFICATION

GRADES	ULCERS
Grade O	Preulcerative lesion
Grade 1	Partical thickness wound upto but not through the dermis
Grade 2	Full thickness wound extending to Tendons are deeper subcutaneous tissue but without bony involvement or osteomyelitis
Grade 3	Full thickness wound extending to and involving the bone
Grade 4	Localized gangrene
Grade 5	Gangrene of the whole foot

GRADING OF DIABETIC ULCER



UNIVERSITY OF TEXAS SYSTEM CLASSIFICATION

STAGE	O	1	2	3
A	Pre / Post ulcerative lesion. Completely epithelialised	Superficial wound not involving tendon, capsule bone	Wound penetrating to tendon or capsule	Wound penetrating to bone or joint
B	+Infection	+Infection	+Infection	+Infection
C	+Ischemia	+Ischemia	+Ischemia	+Ischemia
D	+Ischemia and infection	+Ischemia and infection	+ Ischemia and Infection	+ Ischemia and Infection

BROAKSKY DEPTH ISCHEMIA CLASSIFICATION

This is a modification of WAGNER MEGGITT CLASSIFICATION

BASED ON DEPTH :
GRADES

O - Previous ulcer / Neuropathy with deformity that may cause new ulceration

1 - Superficial ulceration not infected

2 - Deep ulceration exposing a tendon/ joint

3 - Extensive ulceration with exposed bone and / or deep infection (eg. Osteomyelitis, abscess)

ISCHEMIA CLASSIFICATION:

A - Not Ischemia

B - Ischemic without gangrene

C - partial gangrene of the foot

D - Complete foot gangrene with grades 1-3

PRESENTATION OF DIABETIC FOOT:

There is a spectrum of presentation of diabetic foot problem ranging from a mere pain to gangrene. They are :

(i) Pain in the foot

(ii) Sores, ulcers, Blisters

(iii) Absence of sensation

(iv) Absence of pulsation in the foot

(v) Loss of joints movements

(vi) Abscess formation

(vii) Change in colour and temperature when gangrene sets in

(viii) Patient may succumb to ketoacidosis septicemia, or myocardial infarction.

INVESTIGATIONS:

The investigations are done to improve the general condition of the patient, to attain glycemic control, to control the infection, to assess the vascularity of the leg and for appropriate management.

They are

- Complete Dblood count
- Blood glucose

  Fasting

  Postprandial
- Renal parameters
- Wound culture and sensitivity
- X-ray foot (osteomyelitis, foot deformity)
- Chest X-ray
- Cardiac evaluation –ECHO
- Doppler study
- Duplex scanning, angiography – Adequate blood flow
- CT scan /MRI (to see for pockets of pus / abscess)

- **TREATMENT :**

Here again we have so many options like medical management surgical interventions and also includes the patients education .

These are

ANTIBIOTICS:

It is effective when the infection is local or superficial. The choice of the drug should take account of the polymicrobial nature of these lesions. Currently, no specific antibiotic regimen has superior for treating these diabetic foot infections.

Clinical trials suggest that fluroquinolones, cephalosporins, beta-lactam inhibitor penicillins, and carbapenams are effective .

The following drugs can be used depending upon the degree of infection and pathogens.

For mild infections drugs like cephalexin 250-500 mg qid, cefazolin 1-2 g iv tds or ampicillin-sulbactam 3 g iv qid or amoxicillin-clavulanate (augmentin) can be used.

For complicated cases who are treated as in-patients who have both aerobes and anaerobes organisms , drugs like ciprofloxacin750 mg bd with metronidazole 500 mg tds or clindamycin 300 mg qid can be used. In severe

cases fourth generation cephalosporins like cefotaxime 2 g iv, ceftriaxone 2 g iv with metronidazole 500 mg iv or piperacillin-tazobactam 4 g iv or ticarcillin-clavulanic acid 3 g iv tds can be used.

For patients with life threatening infections imipenam500 mg iv qid or meropenam 1 g iv tds or vancomycin 1 g with aztreonam 2 g iv tds with metronidazole 500 mg iv or clindamycin or fourth generation cephalosporins can also be used.

OFF LOADING AND PRESENT APPLIANCES :

Removing or re-distributing mechanical force' off loading' – from a diabetic foot is an important factor in wound care and can alter skin physiology.

Orthotics play a very important role in achieving the healing of neuropathic ulcer by providing the footwear which reduces the pressure at the site of ulceration.There are several devices and techniques used for off loading:

1. Clinical padding , such as semi compressed felt
2. Orthotic management using various types of foam sponge which can be added to pre-fabricated insoles used in clinic or layered in bespoke insoles.
3. Footwear with adjustable insoles such a the 'DH boot'

4. Total contact castings (TCC) is a specialist technique using felts, stockinette, plaster of paris bandage and fiberglass plaster, to prevent movement at the ankle and transfer the weight to the lower leg.
5. The Scotch cast boot is similar to the TCC, but finishes below the ankle and after contains a' window' in the cast
6. Pneumatic walker (Air cast) is a full length plastic boot that works in a similar way to TTC, using internal balloons to achieve weight transfer.
7. Pressure Relieving Ankle Foot Orthotic (PRAFO) is a device for off loading utilizing a metal frame with adjustable strapping and footing.
8. Charcot's Restraint Orthotic Walker (CROW) is removable lined , plastic full lengths boot which is moulded to a cast of the patient's leg and foot. It tend to be used for patients with foot deformity.
9. Crutches, Zimmer frames or wheelchairs can be used to aid off loading under the supervision of a physiotherapist.

CONTACT PLASTER CAST

DIABETIC SHOE

DIABETIC SHOE

Diabetic shoes, sometimes referred to as extra depth or therapeutic shoes, are specially designed shoes or inserts intended to reduce the risk of skin breakdown in diabetics with co-existing foot disease

The primary goal of therapeutic footwear is to prevent complication such as strain ulcers, calluses or even amputation for patients with diabetes and poor circulation.

The shoes must be prescribed by the physician and fit by a qualified individual such as certified pedorthist

The shoes must also be equipped with a removable orthotic foot. Orthotics are devices such as shoe inserts, arch supports, or shoe filters such a lifts, wedges and heels.

The diabetic shoes help these patients to avoid foot injuries and improve mobility.

Types of shoes:

There are two types of diabetic shoes.

1. CUSTOM MOLDED

2. DEPTH SHOES

Custom molded shoes are shoes that are:

- Constructed over a positive model of the wearer's foot
- Made from leather or other material of equal quality
- Have removable inserts that can be altered or placed as the wears' s condition warrants
- Have some form of shoe closure such as lace or Velcro

Depth shoes are shoes that

- Have a full length heel to toe liner that when removed provides a minimum of 3/16 inch of additional depth used to accommodate custom molded or customized inserts.
- Made from leather or other material of equal quality

- Have some form of shoe closor, such as lace or Velcro
- are available in full and half sizes Velcro with a minimum of three widths.

DRESSINGS :

Although a wide variety of dressings are available none is particularly superior to any other with the possible exception of enzymatic preparation (Streptokinase).

The important principle is that the dressing should absorb any exudates which may be produced and it should be non adherent.

HYPERBARIC OXYGEN:

In an hypoxic environment wound healing is halted by decreased fibroblast proliferation collagen production and capillary angiogenesis.

Hypoxia also allows growth of anerobic organisms further complicating wound healing. Hyperbaric oxygen therapy(HBO) provides a siginificant increase in tissue oxygenation in the hypoperfused infected wounds. It influences the rate of collagen deposition angiogenesis and bacterial clearance in wound. The greatest benefits are achieved in tissue with compromised blood flow and oxygen supply.

IN DIABETIC PATIENTS:

The increased wound oxygen tension achieved with HBO promotes wound heating. Increases the host atimicrobial defenses and has a direct bacteriostatic effect on anaerobic microorganisms.

TREATMENT:

Patients undergoing hyperbaric oxygen therapy rest in a chamber which is pressurized to 2-3 times the atmospheric pressure with 100% oxygen for a duration of 90 to 120 minutes. The initial treatment is dictated by the severity of the disease process.

In the presence of limb-threatening infection, after debridement or compromised surgical flaps following amputation the patient should be treated twice daily.

When the infection in under control and the soft tissue envelope improves once daily treatment are adequate.

This increases the patients haemoglobin and plasma oxygen concentration. Thus poorly perfused tissues receive increased oxygen reducing ischemic damage and promotes microvascular angiogenesis. It also helps in fibroblast division necessary for matrix formation.

THE ROLE OF CHIROPODY:

In addition to cutting toenails and foot inspection for medium and high risk patients removal of callus is an important part of diabetic foot treatment. This in done with a scalpel and has been such to reduce the pressure exerted at these sites.

By doing this the risk of ulcerations is reduced. Padding for prominent areas or overlapping toes is provided. If a neuropathic ulcer develops then removal of callus form its edge is important to allow free drainage and also to enable wound contraction because the callus can be so hard as to prevent this.

SURGICAL PROCEDURES:

Certain factors in wound healing can be altered through surgical interventions. Diabetic foot ulcers are chronic ulcers, which are actually difficult to be treated

SURGICAL GOALS :

The following are the goals of surgery in treating the diabetic foot ulcers.

(i) Reduces the risk of ulceration /Amputation

(ii) Reduces the foot deformity

(iii) Provide stable foot for ambulation

(iv) To reduce the pain

(v) Improve the appearence of the foot

The following are the surgical interventions done in these patients to promote the wound healing.

1. Wound debridement
2. Reconstructive procedures Flaps/ Skin grafts
3. Endovascular procedures
4. Bypass procedures
5. Amputation.

WOUND DEBRIDEMENT:

Debridement is carried out mainly in patients with deep ulcers often associated with tissue necrosis and abscess formation.

So there is a need to open all the deep abscesses and excise all the necrotic tissue. Along with this broad spectrum intravenous antibiotics are required.

RECONSTRUCTIVE FOOT SURGERY :

Certain factors in wound healing can be altered through the surgical interventions.

In principle the primary benefit of reconstructive surgery for non healing wounds is the introduction of tissues with good vascular supply to deliver oxygen, nutrients and growth factors.

For reconstructive purpose, either flaps or skin grafts are used depending on the nature of the wound.

Management of soft tissue defects of the foot demands attention to specific features of regional anatomy and function as well as general principles of wound repair.

In diabetes Mellitus, devotion to these basic principles is crucial. The effects of neuropathy, peripheral vascular disease, and susceptibility to infection manifest as increased risk of skin ulceration and decreased capacity for wound healing .

The complex wounds encountered in patients with diabetes may contian exposed bone or tendon or they may lie in weight bearing areas of the foot .

Thus by placing the flaps / grafts it will reduce the wound tension and provide mechanical and thermal protection as well as hydration.

(i) FREE FLAP TRANSFER:

Flap covers are mainly done in regions of weight bearing areas of the foot [eg heel of the foot] with larger defects where skin grafts are not possible. This micrvascular free tissue transfer may allow wound healing and hence helps in limb preservation.

This may also be combined with arterial revascularisation procedure. The advantage of this techniques is that it provides immediate soft tissue coverage, limiting the amputation level and improving the healing time resulting in early ambulation. The following are the types of flaps.

THE TYPES OF FREE TISSUE TRANSFER ARE

1. **FASCIOCUTANEOUS FLAP**

    Free radial forearmflap

    Free lateral arm flap

    Free tenosr fascia latae flap

2. **MUSCLE FLAP WITH OVERLYING SKIN GRAFT**

    - Free rectus abdominis muscle flap
    - Free gracilis muscle flap
    - Free latissimus flap
    - Free serratus anterior flap
    - Free vastus lateralis flap

SKIN GRAFTS

Skin grafts are appropriate for partial thickness wounds or for deeper wounds that are well perfused. Sis Astely Cooper was the first to perform successful skin graft in 1817.

TYPES OF GRAFTS

- PARTIAL THICKNESS SKINGRAFTS
- FULL THICKNESS GRAFTS
- COMPOSITE GRAFTS

A.PARTIAL THICKNESS SKIN GRFATS (SPLIT SKIN GRAFT):

Also called as **THIERSCH** graft. Split skin graft consist of epidermis and a variable thickness of dermis. There remains some dermis on the donor site that heals by epithelialisation from the cut ends of hair follies and sweat glands in a manner similar to the healing of a superficial burn.

PROCEDURE :

The thigh is most frequently used as a donor site, but almost anywhere else can be used. Grafts are harvested using a skin graft knife or a called as **HUMBY'S KNIFE** or a power dermatome and a guard that can be adjusted to determine the thickness of the graft.

This graft may be thin or thick. The survival of the graft on the recipient area is known as **TAKE.**

STAGES OF GRAFT TAKE:

STAGE OF PLASMATIC IMBIBITION:

A uniform layer of plasma forms between recipient bed and the graft

STAGE OF INOSCULATION :

Linking of host and graft which is temporary

STAGE OF NEOVASCULARISATION:

New capillaries proliferate into the graft from the recipient bed which attains circulation later.

TECHNICLA ASPECTS :

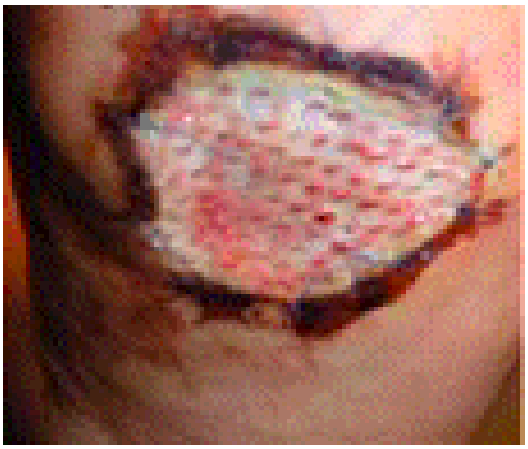
Since a graft in contrast to the flap does not derive its blood supply from the donor site it must depend on the vascularity of the recipient site.

As noted even tendon with intact paratenon and bone with periosteum will take a skin graft. It can not be applied over bare bone or bare tendon or cartilage.

HARVESTING SKIN GRAFT FROM THIGH

PRE AND POST OPERATIVE PICTURES

POST OPERATIVE PICTURES



POST OPERATIVE PERIOD :

Post operatively, the percentage of graft taken are assesed during the fifth day initially and the donor site is also examined on the tenth day for any signs of infection.

B.FULL THICKNESS GRAFTS :

It is also called as **WOLFE GRAFT**. It includes both the epidermis and full dermis. Here donor site is closed by primary suturing

C.COMPOSITE GRAFT :

Composite grafts consists of skin and some underlying tissue such as fat or cartilage.

PREOPERATIVE REQUIREMENTS :

Preparation of a wound for surgical repair requires attention to several factors that may be contributing to impaired healing.

These include infection, vascular insufficiency, neuropathy and edema. Systemic factors such as inadequate controls of blood gulcose or nutritional deficits should also be corrected.

As with any wound a foot wound in a patient with diabetes should be debrided of infected and necrotic tissue. This is usually accomplished most efficiently by sharp excision.

VASCULAR RECONSTRUCTIVE PROCEDURES:

The following are the procedures done in patients with poor vascularity. These procedures help in preventing amputation by restoring the blood flow or it avoids high level of amputations. They are in the form of either endovascular therapy, by pass surgery or a combination of both .

(i) ENDOVASCULAR PROCEDURES :

Endovascular procedures which are usually done are balloon angioplasty with or without stent placement. They are mostly used for inflow vessels and femoro popilteal lesions.

Currently , stents are metallic and permanent and may be self expanding or require balloon expansion.

Balloon angioplasty for distal poplitieal and crural run-off vessels is achievable using a small balloon for a short stenosis.

(ii)BYPASS SURGERY:

Distal bypasses onto tibial or pedal vessels are often required . In-flow vessels disease is common in diabetic patients.

In a recent study diabetic patients comprised a greater proportion of the total number of patients requiring in-flow bypass as well as a greater proportion of patients requiring subsequent outflow bypass for unresolved ischemic symptoms.

Inflow reconstruction can be achieved surgically with an aorta-bifemoral or axillo-bifemoral bypass in cases of bilateral disease. Femoro-Femoro crossover or ilio-femoral bypass can be used in unilateral disease. Femoral endartectomy , with or without a profundoplasty, remains a very useful simple procedure. Autogenous vein should be used whenever possible .

The results of by passing in patients with diabetes are as good as those in non diabetics.

AMPUTATION:

Amputation is done in limbs which are not salvagabel. Amputations of lower extremities can be classified into major limb amputation and foot amputation Major limb amputations can be above knee or below knee with trans femoral, Gritti- stokes and knee disarticulation.

Foot amputations can be a done alone or in combination with by pass surgery These are classified as ray amputation for toe necrosis, chopart's mid tarsal amputations, lisfranc's tarso-metatarsal joint amputation, syme's amputation or through ankle.

Total calcanectomy is described as an alterative procedure to trans-tibial amputation in patients with chronic osteomyelitis of the calcaneus, achieving eradication of infection and the preservation of improved functional ambulation.

NEW TRENDS

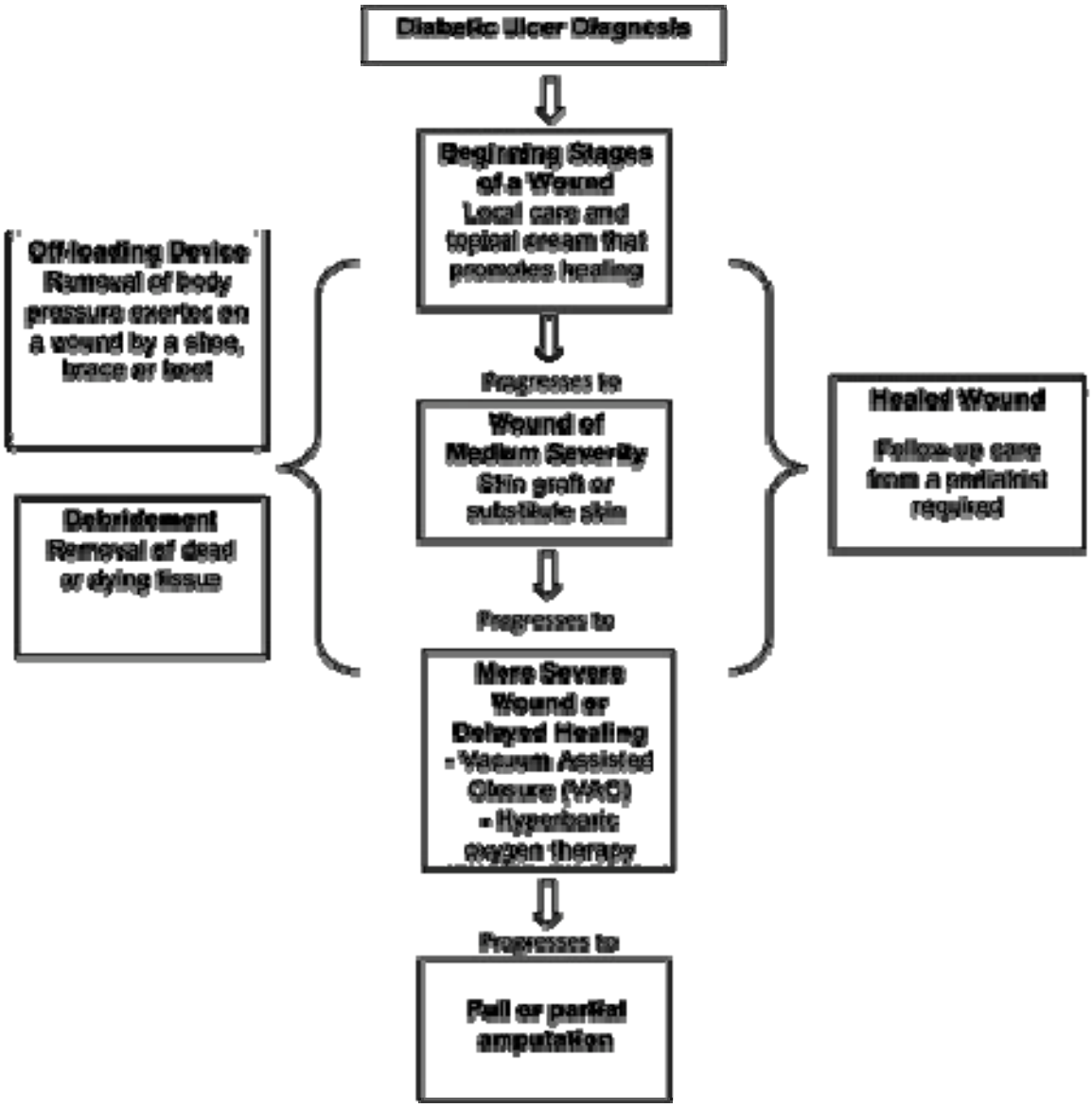
New methods which are followed recently are as follows,

1) **REMOVAL OF NECROTIC TISSUE WITH DESLOUGHING AGENTS :**

Hydrocolloids ,larvae, enzymes

SCHEMATIC REPRESENTATION OF

MANAGEMENT OF DFU



2) MANAGING EXUDATES:

Polyurethane foams, capillarity dressing, kerraboot, vacuum assisted closure (VAC), alginates, cellulose composites, non adherent dressings, film dressing

3) SKIN EQUIVALENTS:

Allograft (cultured keratinocytes, or fibroblasts)

Xerografts

Bioengineered skin

4) GROWTH FACTOR:

Platelet devived growth factor PDGF

5)AIDING CELL MOTILITY: Hyaluronan

6) REDUCING THE BACTERIAL BIOBURDEN:

Topical antiseptics, topical antibiotics, honey, chlorhexadine charcoal-based dressings.

LATEST PRODUCTS :

1) SILVER IMPREGNATED DRESSINGS :

With the evolution of organisms with multi resistance of antibiotics, the introduction of silver impregnated dressings have emerged.

Silver can be added to many dressing types. Thereby combining the effectiveness of the dressing with antimicrobial activity .

2) THE KERRABOOT:

The kerraboot is a non contact wound care device comprising a plastic boot with a super absorbant foot pad . It maintains warmth and moisture at the wound site necessary to promote angiogenesis and epithelialisation.

Odour is virtually eliminated by use of a ‘trapping’ layer foming part of the boot’s overall construction.

PREVENTIVE MEASUERS:

The following are some of the preventive measures to be followed by the diabetic patients to prevent ulcer formation or any major complications

- Cleansing the feet and keeping it dry
- Check the feet for any ulcers /sores
- Check for the sensation of the foot
- Properly fitting shoes
- Avoid smoking
- Regular exercise
- Removal of the callus.
- **PATIENT’S EDUCATION:**

Patient education is central to the management of the diabetic foot with the emphasis on prevention. The importance of daily self examination and careful hygiene is stressed along with the need for good, well fitting shoes with sufficient depth.

The patient should always look and feel in the shoe before putting it on in case any object has fallen in which may cause foot trauma.

Patients with foot deformities should have shoes which accommodate the foot without creating abnormally high pressure areas. This may mean simple obtaining extra depth shoes or formally fitted orthoses may be required.

Hand cream without perfume or colouring should be used daily or twice daily to prevent dry skin from cracking

Toenail cutting should only be carried out by patients if they are low risk, for the all others regular chiropody is advised

MATERIALS AND METHODS

MATERIALS AND METHODS

This is a prospective study conducted on 20 patients with diabetic foot ulcers who have undergone split skin graft procedure in the surgical department of Kilpauk Medical College and Hospital during the period of September 07-August 09.

Detailed History of the patients and thorough clinical examination was done in all cases. Documentation was done using a stratified proforma which included dermographic data of the patients studied. For all patients hematological , biochemical, microbiological and radiological investigations were carried out as enumerated in the proforma. Blood sugar both fasting and postprandial were done. Renal parameters were also done. X ray of the affected foot, Chest Xray, ECG and cardiac evaluation were done. The vascular status of the patients were also assessed.

Wound preparation was done for all the patients by wound debridement, all the patients were put on broad spectrum antibiotics according to their respective wound culture reports. Their glycemic status was assessed and all of them were put on Injection. Human insulin both monotard and actrapid according to their blood sugar level. After getting assessed by the anesthetist they were taken up for surgery- Split Skin Graft.

Post operatively, on fifth day, the graft take percentage was assessed followed by on tenth day. On tenth day, the donor site was also examined for any infection. The results were analysed.

The mean stay of the patiens in the hospital during the preoperative and postoperative period were also analysed

RESULTS AND OBSERVATION

RESULTS AND OBSERVATION

20 patients with diabetic foot ulcers have undergone split skin graft after attaining all the preoperative requirements of which the percentage of graft take was assessed during the fifth and tenth post operative day, and the length of the stay in the hospital during the pre operative and post operative period were assessed.The donor site infections were also observed. The following results were observed.

ON THE FIFTH POSTOPERATIVE DAY

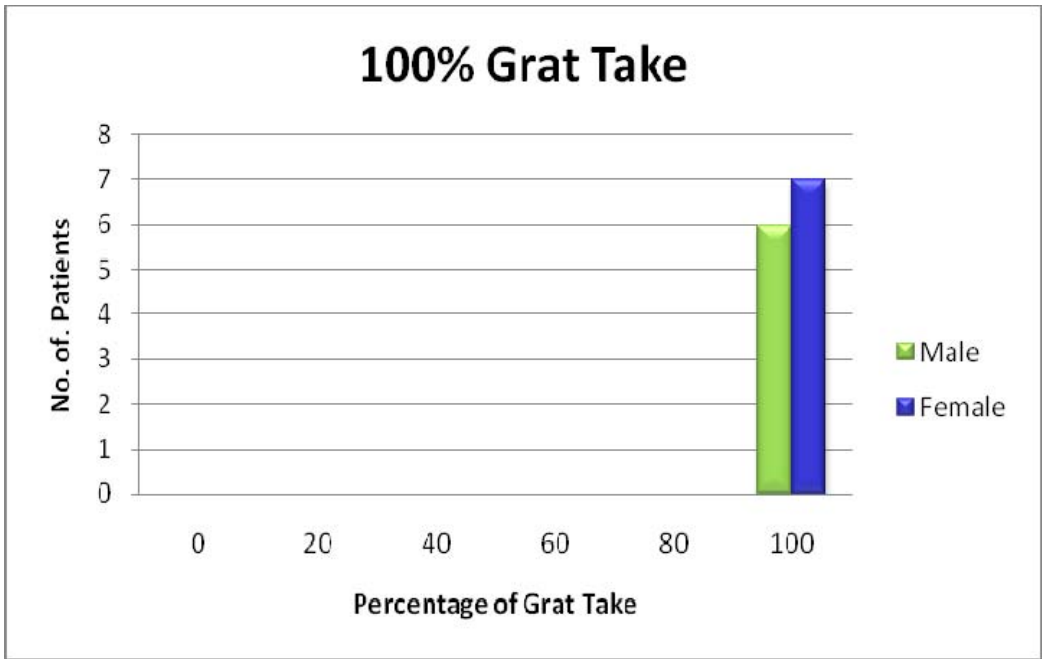
NO OF PEOPLE SHOWED 100% GRAFT TAKE

	Male	Female	T	Frequency
100% graft take	6	7	13	65%

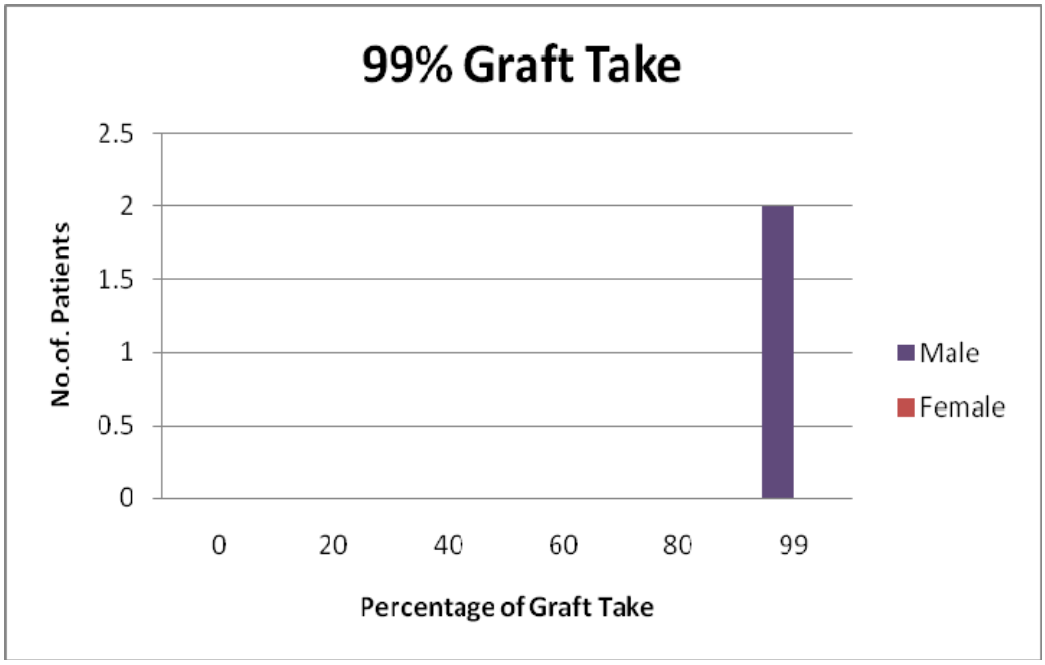
NO. OF PEOPLE SHOWED 99% GRAFT TAKE

	Male	Female	T	Frequency
99% graft take	2	0	2	10%

FIFTH POST OPERATIVE DAY



FIFTH POST OPERATIVE DAY



OF PEOPLE SHOWED 98% GRAFT TAKE

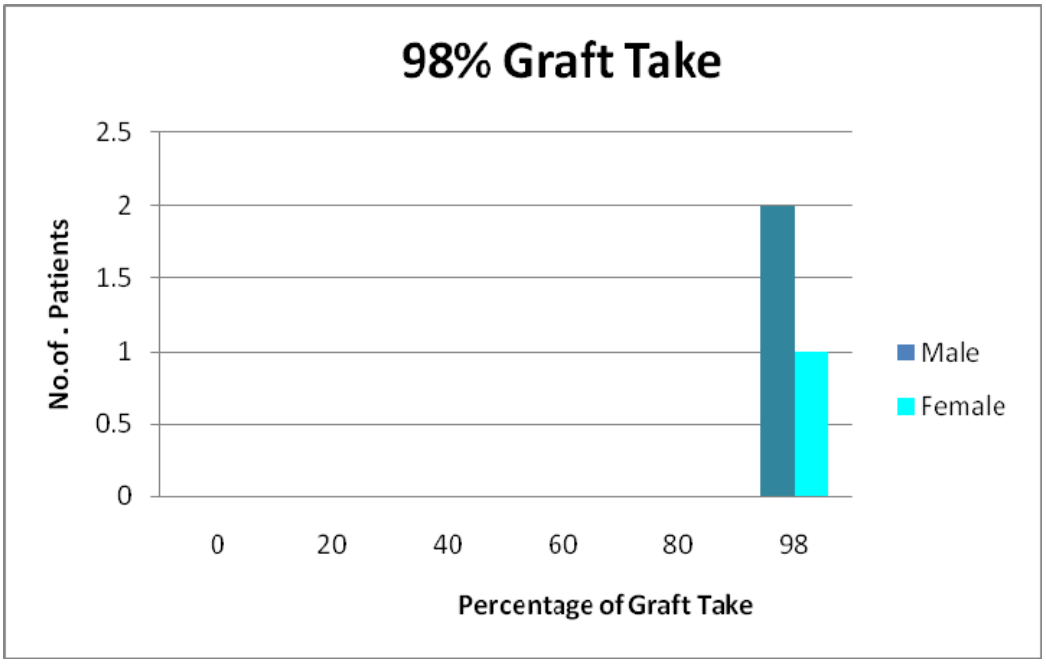
	Male	Female	T	Frequency
98% graft take	1	1	2	10%

NO

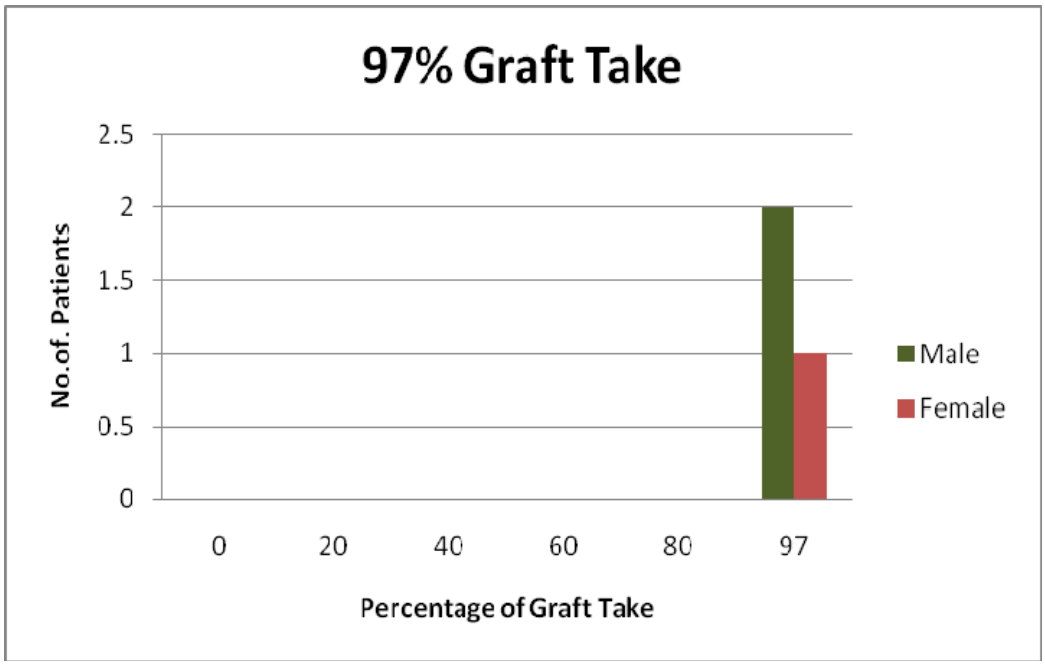
. OF PEOPLE SHOWED 97% GRAFT TAKE

	Male	Female	T	Frequency
97% graft take	2	1	3	15%

FIFTH POST OPERATIVE DAY



FIFTH POST OPERATIVE DAY



ON THE TENTH POSTOPERATIVE PERIOD

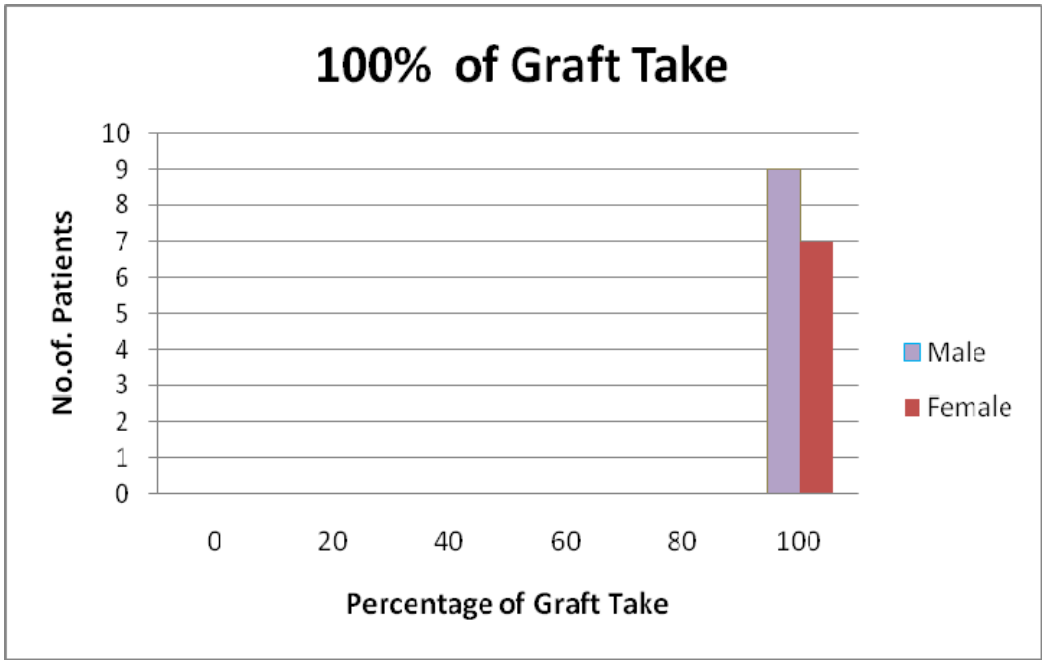
NO. OF PEOPLE SHOWED 100% GRAFT TAKE

		Male	Female	T	Frequency
N	100% graft take	9	7	16	80%

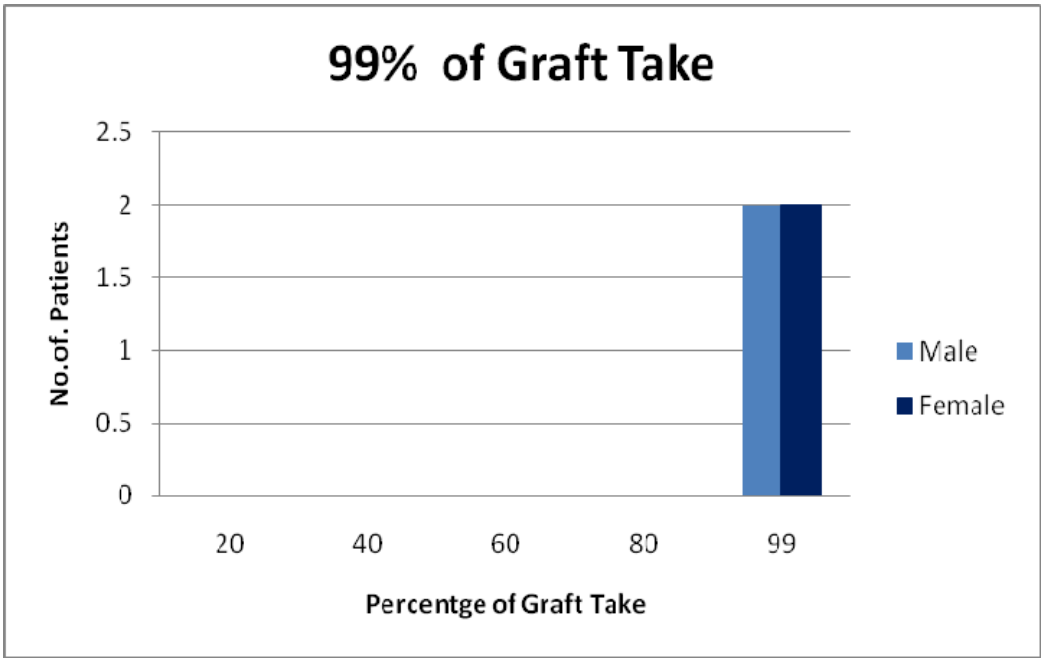
O. OF PEOPLE SHOWED 99% GRAFT TAKE

	Male	Female	T	Frequency
99% graft take	2	2	4	20%

TENTH POST OPERATIVE DAY



TENTH POST OPERATIVE DAY

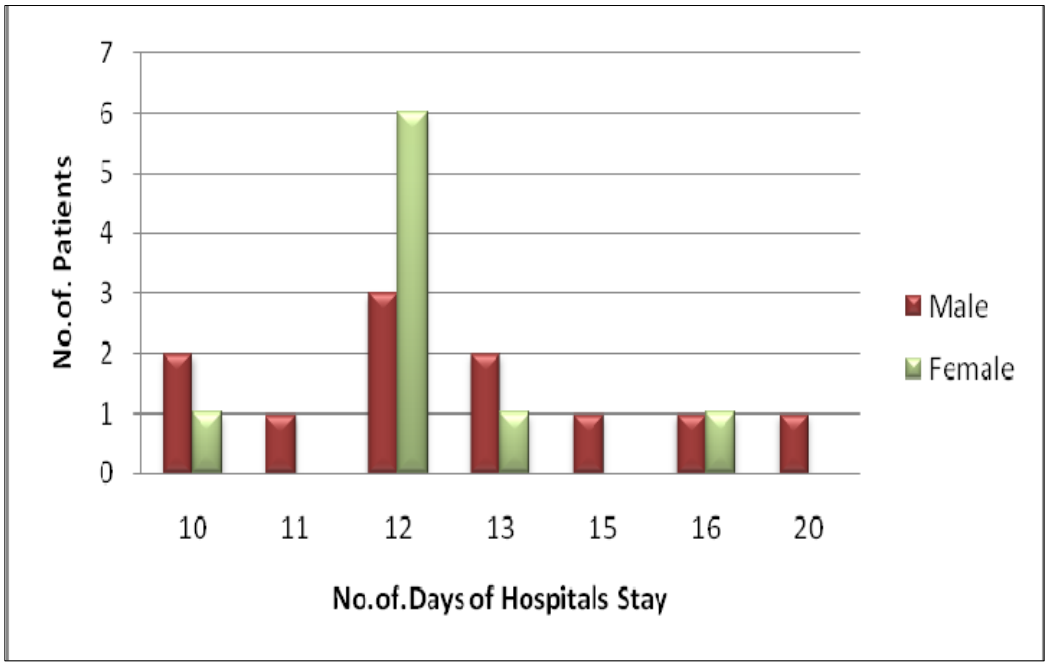


LENGTH OF STAY IN THE POSTOPERATIVE PERIOD

Average no of days of stay in the hospital in the postoperative period - 13 days

No. of. Days of stay	No. of . people			Frequency
	M	F	T	
10	2	1	3	15%
11	1	0	1	5%
12	3	6	9	45%
13	2	1	3	15%
15	1	0	1	5%
16	1	1	2	10%
20	1	0	1	5%

POST OPERATIVE HOSPITAL STAY

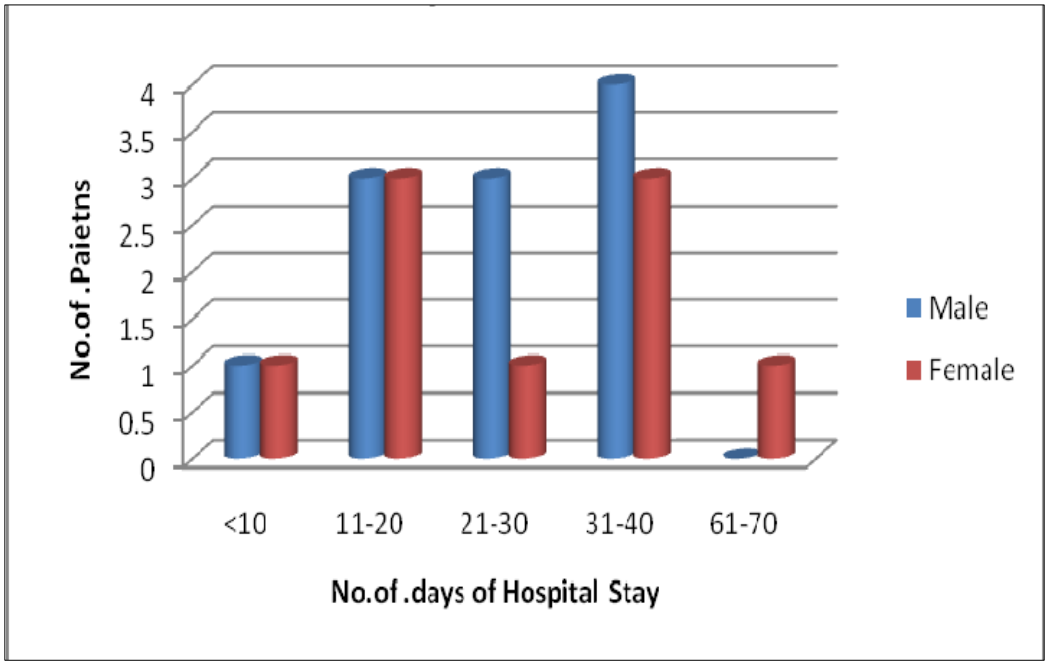


GH OF HOSPITAL STAY IN THE PREOPERATIVE PERIOD

No. of. Days of stay	**No. of . people**			**Frequency**
	M	F	T	
<10	1	1	2	10%
11-20	3	3	6	30%
21-30	3	1	4	20%
31-40	4	3	7	35%
61-70	0	1	1	5%
Average No.of Days of Hospital stay-26.15 days				

Average no days of hospital stay in the pre operative period is 26.15 days

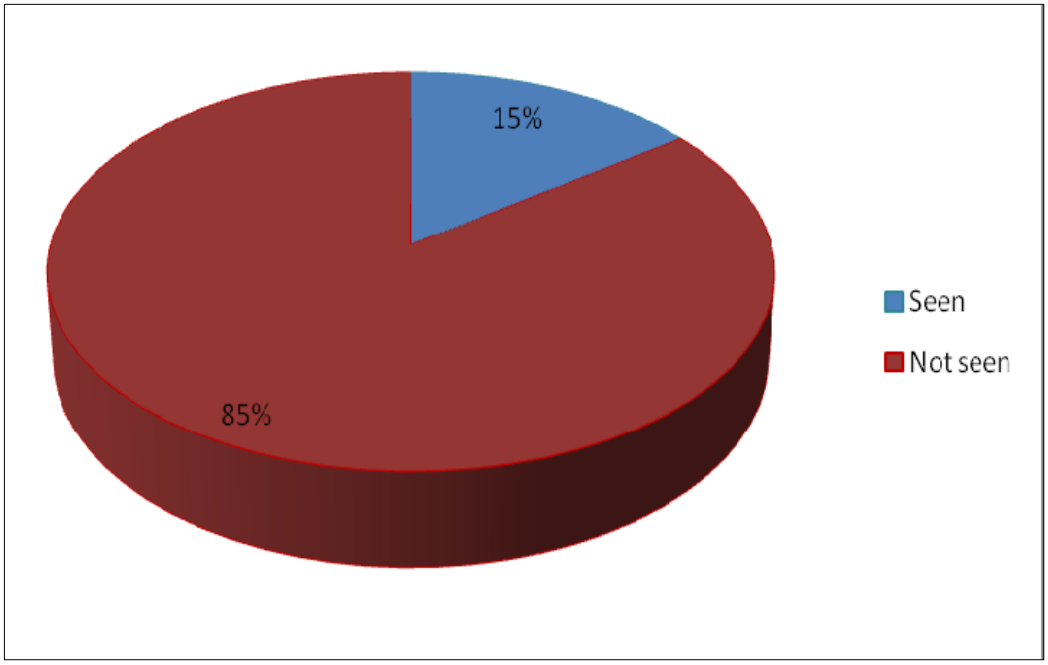
PRE. OPERATIVE PERIOD



DONOR SITE INFECTION

No. of . Patients showed minimal donor site infection -3

Soner site infection	No. of . patients			Frequency
	M	F	T	
Seen	2	1	3	15%
Not seen	9	8	17	85%



DISCUSSION

DISCUSSION

In this prospective study conducted on 20 patients with diabetic foot ulcers, who have undergone split skin graft, we are able to observe the following results.

The percentage of graft take the length of hospital stay and donor site infectionS were assessed from the study. It was found that 65% of patients showedd 100% graft take on the fifth postoperative day, followed by 10% of patients showed 99% graft take on fifth post operative day. Another 10% of patients showed 98% graft take on fifth post operative day and 15% of patients showed 97% graft take on the fifth postoperative day.

From the study it was observed that 80% of patient showed 100% graft take on tenth post operative day and 20% of them showed 99% graft take on tenth post operative day.

According to the literature, 80% of patients have shown 100% graft take on fifth postoperative day and remaining 20% of patients have shown 100% graft take on second week of surgery.

From the above study it was observed that the length of stay in the postoperative period has come down to aobut 50% of the preoperative stay.

In the preoperative period, the average number of days of hospital stay was about 26.15 days whereas in the postoperative period it was about 13 days. According to the literature the preoperative stay is about 21 days and the postoperative stay in the hospital is about 12 days.

From the above study it was found that 15% of paitents showed minimal donor site infections. According to the literature it is about 10%. This were the results observed.

CONCLUSION

CONCLUSION

As we know that the prevalence of the diabetes mellitus is increasing world wide and many people with diabetes develop the foot ulcers, which are difficult to heal on its own due to the various etiological factors and it has become the major cause of long hospitalisation and it also casts an economical burden for both the hospital and the individual.

It is important to treat these ulcers earlier to prevent major complication like amputation. Certain factors can be altered by surgery to promote the wound healing. One such procedure is this application of split skin graft.

From this study we could observe the effect of split skin graft in promoting the wound healing and it has also shown to reduce the length of hospital stay thereby it reduce the expenses spent on treating these ulcers. Moreover it helps the patient to walk again and also prevents major complications.

From the above study, it was observed that the length of hospital stay in the post operative period , has came down to about 50% of the preoperative operative**.**

As per the literatwe, the mean post operative stay is about 12 days. In the above study it was observed that 45% of patients have stayed for 12 day in the hospital after surgery.

Among the patients in this study group, it was observed that 15% of patients have shown minimal donor site infection. According to the Literature it is about 10%

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CLINICAL PROFORMA

CLINICAL PROFORMA

NAME

AGE

SEX

IPNO

D.O.A

D.O.S

D.O.D

DURATION OF STAY

- PREOPERATIVE PERIOD
- POST OPERATIVE PERIOD

HISTORY

- ULCER
  - SPONTANEOUS / TRAUMATIC
  - SITE OF ULCER

H/O CLAUDICATION

PREVIOUS H/O ULCER

SMOKING H/O

ALCOHOL H/O

FAMILY H/O DIABETES MELLITUS

TREATMENT FOR DIABETES MELLITUS

LOCAL EXAMINATION OF ULCER

ULCER

SITE

SIZE

EDGES

MARGIN

FLOOR

BASE

COLOUR OF THE FOOT

ANY DEFORMITY OF FOOT

PERIPHERAL PULSES

INVESTIGATIONS

HB%

TC

DC

ESR

BT

CT

BLOOD SUGAR

FASTING

POSTPRANDIAL

BLOOD UREA

SR. CREATININE

URINE – ALBUMIN

SUGAR

ACETONE

WOUND – CULTURE AND SENSITIVITY

CHEST X-RAY

X-RAY FOOT

- ANTERO POSTERIOR VIEW
- OBLIQUE VIEW

ELECTRO CARDIOGRAM

ECHO

TREATMENT – SPLIT SKIN GRAFT

MASTER CHART

KEY TO THE MASTER CHART

A	Absent
P	Present
M	Male
F	Female
FOD	Family H/O Of Diabetes
DOA	Date of Admission
DOS	Date of Surgery
DOD	Date of Discharge
PGT	Percentage of Grafttaken
DSI	Donor site infection
POD	Post operative day
NS	Notseen

MASTER CHART

S.No	IPNO	NAME	AGE	SEX	FOD	DOA	DOS	DOD	PGT		DSI	Pre OS	POS
									5 POD	10 POD			
1	8435	Anand	50	M	A	24/9/2007	8/10/2007	18/10/2007	99	100	NS	38	10
2	1387	Paneersel vam	55	M	A	19/1/2008	30/1/2008	11/2/2008	100	100	NS	13	12
3	9218	venkatesan	50	M	A	2/5/2008	23/5/2008	3/6/2008	100	100	NS	20	11
4	6053	Kuppan	51	M	A	22/3/2008	17/4/2008	2/5/2008	97	99	NS	40	15
5	19463	Mani	56	M	P	7/9/2008	14/10/2008	30/10/2008	98	100	MSI	36	16
6	4409	Sarabee	60	F	P	21/0/2008	4/4/2008	15/4/2008	98	99	NS	15	12
7	25027	Sasi	35	F	A	18/11/2008	20/11/2008	8/12/2008	100	100	NS	7	12
8	20935	Rukmani	45	F	A	26/9/2008	4/11/2008	18/11/2008	100	100	NS	38	13

9	17195	Shanihi	60	F	A	10/8/2008	18/10/2008	3/11/2008	97	99	NS	68	16
10	517	Prema	60	F	P	19/9/2008	23/11/2008	4/11/2008	100	100	NS	34	12
11	10441	Revathy	40	F	P	20/5/2008	2/6/2008	14/6/2008	100	100	NS	12	12
12	1515	Gangammal	40	F	A	29/1/2008	3/3/2008	4/3/2008	100	100	NS	33	12
13	9456	Gopal	45	F	P	5/5/2008	11/6/2008	24/6/2008	100	100	NS	36	13
14	3277	Hariram	40	M	A	14/2/2009	13/3/2009	23/3/2009	100	100	NS	23	12
15	15327	Ramasamy	60	M	A	8/7/2009	29/7/2009	18/8/2009	97	99	MSI	21	20
16	19407	Chittibabu	45	M	P	19/8/2009	12/9/2009	24/9/2009	100	100	NS	21	12
17	19290	Sekar	45	M	A	18/8/09	31/8/2009	13/9/2009	100	100	NS	13	13

18	1387	Premkumar	55	M	A	20/1/09	30/1/2009	9/2/2009	99	100	NS	10	10
19	2904	Amsa	37	F	P	10/2/2009	2/3/2009	14/3/2009	100	100	NS	19	12
20	604	Vasantha	40	F	A	8/1/2009	4/2/2009	14/2/2009	99	100	NS	26	10