A CLINICAL STUDY OF COMPARISION BETWEEN EFFICACY OF TOPICAL SUCRALFATE AND CONVENTIONAL DRESSING IN THE MANAGMENT OF DIABETIC ULCER IN TIRUNELVELI MEDICAL COLLEGE

DISSERTATION SUBMITTED TO

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

CHENNAI

in partial fulfilment of

the requirements for the degree of

MASTER OF SURGERY

In

GENERAL SURGERY

DEPARTMENT OF GENERAL SURGERY

TIRUNELVELI MEDICAL COLLEGE

TIRUNELVELI

APRIL-2016

CERTIFICATE

This is to certify that the dissertation entitled "**A CLINICAL STUDY OF COMPARISION BETWEEN EFFICACY OF TOPICAL SUCRALFATE AND CONVENTIONAL DRESSING IN THE MANAGMENT OF DIABETIC ULCER IN TIRUNELVELI MEDICAL COLLEGE**" is a bonafide research work done by **Dr.A.JOHN AMALAN** in fulfilment of the requirement for the degree of Master of Surgery in General Surgery

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NAME OF PRINCIPAL INVESTIGATOR: Dr. JOHN AMALAN, MBBS.,
DESIGNATION OF PRINCIPAL INVESTIGATOR: Post Graduate in MS General Surgery
DEPARTMENT & INSTITUTION: Department of General Surgery, Tirunelveli Medical College

Dear Dr. John Amalan, *The Tirunelveli Medical College Institutional Ethics Committee (TIREC) reviewed and discussed your application during the IEC meeting held on 08.10.2014.*

THE FOLLOWING DOCUMENTS WERE REVIEWED AND APPROVED

1.	TIREC Application Form	☑
2.	Study Protocol	☑
3.	Department Research Committee Approval	☑
4.	Patient Information Document and Consent Form in English and Vernacular Language	☑
5.	Investigator's Brochure	☐
6.	Proposed Methods for Patient Accrual Proposed	☐
7.	Curriculum Vitae of the Principal Investigator	☐
8.	Insurance /Compensation Policy	☐
9.	Investigator's Agreement with Sponsor	☐
10.	Investigator's Undertaking	☐
11.	DCGI/DGFT approval	☐
12.	Clinical Trial Agreement (CTA)	☐
13.	Memorandum of Understanding (MOU)/Material Transfer Agreement (MTA)	☐
14.	Clinical Trials Registry-India (CTRI) Registration	☐

THE PROTOCOL IS APPROVED IN ITS PRESENTED FORM ON THE FOLLOWING CONDITIONS

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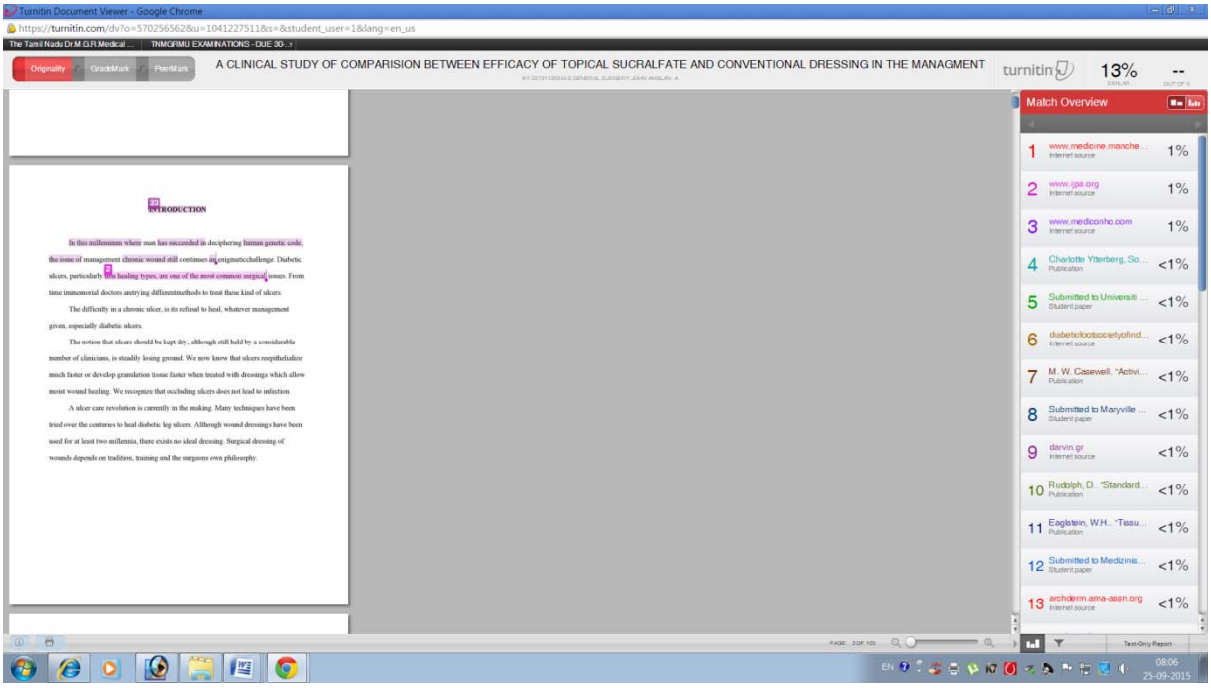
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INTRODUCTION
In this millennium where man has succeeded in deciphering human genetic code, the issue of management chronic wound still continues in enigmaticchallenge. Diabetic ulcers, particularly the healing types, are one of the most common surgical issues. From time immemorial doctors aretrying differentmethods to treat these kind of ulcers.
The difficulty in a chronic ulcer, is its refusal to heal, whatever management given, especially diabetic ulcers.
The notion that ulcers should be kept dry, although still held by a considerable number of clinicians, is steadily losing ground. We now know that ulcers reepithelialize much faster or develop granulation tissue faster when treated with dressings which allow moist wound healing. We recognize that occluding ulcers does not lead to infection.
A ulcer care revolution is currently in the making. Many techniques have been tried over the centuries to heal diabetic leg ulcers. Although wound dressings have been used for at least two millennia, there exists no ideal dressing. Surgical dressing of wounds depends on tradition, training and the surgeons own philosophy.
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DECLARATION BY THE CANDIDATE

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Lastly, I express my thanks to my patients without whom this study would not have been possible.

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Place:

LIST OF ABBREVIATIONS

DM	:	Diabetes Mellitus
Hb	:	Haemoglobin
Vs	:	Versus
PR	:	Pulse Rate
BP	:	Blood Pressure
GF	:	Growth Factor
FBS	:	Fasting blood sugar
CBC	:	Complete blood count
UKB	:	Urine ketone bodies
PVD	:	Peripheral vascular disease
DBP	:	Diastolic Blood Pressure
SBP	:	Systolic Blood Pressure
PDGF	:	Platelet derived growth factor
rh-PDGF	:	Recombinant human Platelet derived growth factor
US-FDA	:	United States Food and Drugs Administration

ABSTRACT

Background

Sulphated saccharides, primarily sucralfate, have previously been indicated for the treatment of gastric and duodenal ulcers. In radio-labelled form, sucralfate has also been used as a diagnostic agent for the imaging of gastrointestinal mucosa, since the substance binds selectively to ulcerated areas in the stomach and upper small intestine. It has been found that sucralfate exerts an anti-inflammatory effect when applied topically to the skin and to mucosal surfaces, and that sucralfate exerts a beneficial effect on wounds when applied topically on epithelial surfaces outside the digestive tract**.** Accordingly, the present study relates to the use of sucralfate for the preparation of a medication for topical application over the skin or to any nongastrointestinal surface**.** Although the evidence is not yet conclusive the clinical evidence tends to support the anti-inflammatory effect and wound healing effect of sucralfate.

Objectives of the study

- To compare the efficacy of topical sucralfate with that of a control group using
- conventional dressings, in the healing of diabetic ulcers in terms of: Number of days needed for healing
- To study the healing properties of sucralfate in management of nongastrointestinal wounds
- Rate of reduction in mean ulcer surface area

- To assess the effect of topical sucralfate on bacterial load by comparing the culture and sensitivity of wound swabs before and after application of sucralfate

Methodology:

This was a comparative study conducted at Tirunelveli Medical College Hospital,Tirunelveli.Total of 100 patients were studied. They were separated into 2 groups through computerized randomization. Patients in Control group were treated with conventional dressing and patients in Study group were treated with sucralfate dressing and watched for the wound size reduction.

Results:

The study group patients showed higher reduction in wound size of about 40.87%as against 15.63% of the control group with P value < 0.001.

Conclusion

Topical application of sucralfate once daily significantly increases incidence of

wound healing and reduce the time required for healing in diabetic ulcers**.**

Key words

Sucralfate, Topical application, Diabetic foot ulcer

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Introduction

INTRODUCTION

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The difficulty in a chronic ulcer, is its refusal to heal, whatever management given, especially diabetic ulcers.

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A ulcer care revolution is currently in the making. Many techniques have been tried over the centuries to heal diabetic leg ulcers. Although wound dressings have been used for at least two millennia, there exists no ideal dressing. Surgical dressing of wounds depends on tradition, training and the surgeons own philosophy.

During the last 2 ½ decades a wide range of innovative dressings have been introduced. People have tried various non-conventional topical therapies in wound healing, such as Aloe vera, Benzoyl peroxide, collagen, gentian violet,

impregnated gauze, topical phenytoin, mercurochrome, oxygen therapy, sugar and vinegar.

Studies have also proven that topical sucralfate promotes healing of decubitus ulcers, venous stasis ulcers, traumatic wounds, burns, trophic ulcers and was seen to be superior management of diabetic ulcers.

Sucralfate, an oral gastrointestinal medication primarily indicated for the treatment of active duodenal ulcers, is also used for the treating gastroesophageal reflux disease (GERD) and stress ulcers.

It shows potential utility in the healing of skin wounds. Sucralfate induces proliferation of dermal fibroblasts and keratinocytes. It also enhances prostaglandin E2 synthesis in basal keratinocytes, enhances interleukin-1-stimulated interleukin-6 release from fibroblasts. When applied to full-thickness wounds daily, sucralfate increased the thickness of granulation tissue. It also promotes rapid epithelialisation of 2nd degree burns

A series of studies in animals has shown that application of sucralfate to a wound enhances the wound repair process. Sucralfate has been demonstrated in preclinical studies to promote the granulation tissue formation and thus promoting cutaneous ulcer healing.

Various human cellular studies have definitely established the fact that for diabetic ulcers which are resistant to conventional treatment, topically applied sucralfate is a new pharmacologically active therapy Various studies showed the efficacy of sucralfate, that is complete healing of the wound, and the reducing the

size of the wound. Sucralfate stimulates granulation tissue and promotes cutaneous ulcer healing.

In view of other studies regarding the efficacy of sucralfate in diabetic ulcers, we undertook this study to know whether sucralfate applied topically over the diabetic ulcers reduces the size of the wound effectively compared to normal saline dressing(conventional treatment)

Aim and Objective

AIM AND OBJECTIVE OF THE STUDY

- To compare the efficacy of topical sucralfate with that of a control group using

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- Number of days needed for healing
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- Rate of reduction in mean ulcer surface area
- To assess the effect of topical sucralfate on bacterial load by comparing the culture and sensitivity of wound swabs before and after application of sucralfate
- From January 2014 to July 2015 are taken for study
- At Tirunelveli medical college, Department of surgery

Review of Literature

REVIEW OF LITERATURE

HISTORICAL BACKGROUND OF WOUND HEALING

- The treatment and healing of wounds are the oldest topics discussed in the medical literature and probably earliest problems of human race.
- Early surgeons like Ambrose, Pare, John Hunter, & Sir James Paget have given some scientific knowledge to their handling of wounds, particularly those resulted from war.
- Halsted was intensely interested in wound healing process.
- In the early 1900's Carrel & his associates made investigations with the scientific approach to wound healing. Later Carrel (1916), Harvey & Howe's (1930), studied incised wounds & contributed to the knowledge of wound healing.
- There is a saying; "If there were no regeneration, there would be no life; if everything regenerated, then, there would be no death".
- The earliest medical writings deal extensively with wound care. Seven of the 48 case reports included in the Edwin Smith Papyrus (1700 BC) describe wounds and their management.
- Empirically, in Egypt, Greece, India and Europe, the physicians developed gentle methods of treating wounds by removing foreign bodies, suturing, covering wounds with clean materials, and protecting injured tissues from corrosive agents

- More than 4000 years ago, the theory of the "three healing gestures" was formed,with earliest writing recorded on a clay tablet from 2200 BC.

  The tablet describes the three gestures as:

  - wound washing
  - plasters over the wound
  - application of bandage over the wound
- These gestures evolving into varying forms of today's same basic themes. The Greeks belief of dry healing came from Hippocrates, at a time when the only function of dressings was thought to be the protection of the wound from injury.
- During the fourteenth century, with the widespread use of gunpowder and the increasing frequency of bullet wounds, there was an increased need for surgeons assuming an aggressive posture, which was often done at the expense of aseptic precautions. Examples included applications of burning oil, scalding water, wine, turpentine, feathers, sugar, clay, bismuth, milk of magnesia to wounds. However, none of these have proven efficacy leased on sound scientific studies.
- The modern era of gentle wound care started in the mid-sixteenth century, when Ambroise Pare, the great French army surgeon, who during the Battle of Villaine, applied milder agents like digestive solution of egg yolk, rose oil, honey and turpentine to amputation stumps with dramatic results.

- John Hunter, William Stewart Halsted, Alexis Carrel and many other great clinical biologists demonstrated that minimizing tissue injury produces rapid and effective healing leading to the "minimal interference" concept of wound care. If the surgeon can remove all impediments, normal wound healing process will produce the best possible result.
- Joseph Lister advocated cleanliness in the hospital, the frequent use of soap and water on wounds and carbolic acid dressings of contaminated wounds. Later Semmelweis, Ehrlich, Fleming, and Florey also realized that bacteria were pathogens. Control of bacteria by asepsis, antiseptics and antimicrobials heralded a new era in wound management.
- Finally it is apt to say that advances of the previous decades are only a prelude to the changes in wound care management that will occur in the coming decades.

DIABETES MELLITUS

Definition:

"Diabetes mellitus is characterized by chronic hyperglycemia with disturbances

of carbohydrate, fat, and protein metabolism resulting from defects in insulin secretion, insulin action, or both"**.**

Hyperglycemias may be due to the following etiological factors:

- Reduced insulin secretion
- Decreased glucose utilization
- Increased glucose production

Classification

TYPE I

Type Pathology

I A : Autoimmune beta cell destruction Insulin Deficiency

I B : Develop insulin deficiency by unknown mechanism causing destructive process of beta cells Lack immunologic markers

Type II

Is characterized by :-

- Impaired insulin secretion
- Insulin resistance
- Excessive glucose formation

Type-2 DM is preceded by a period of abnormal glucose haemostasis classified as

- Impaired glucose tolerance (IGT)
- Impaired fasting glucose (IFG)

Diagnosis

Diagnostic criteria for Diabetes Mellitus type2(WHO & National diabetic Data Group):

- RBS ≥200 mgs / dL Or ≥11.1 m mol / L with symptoms of DM (Polyuria,Polydipsia, Polyphagia, Weight loss)
- FBS ≥126 mgs / dL or ≥ 7.0 m mol / L
- 2 Hr Plasma Glucose (During Oral GTT) ≥ 200 mgs / dL or ≥11.1 m mol/L
- Strong co-relation b/w ↑ FPG & ↑ HbA1c concentration but currently notrecommended for the diagnosis of DM.

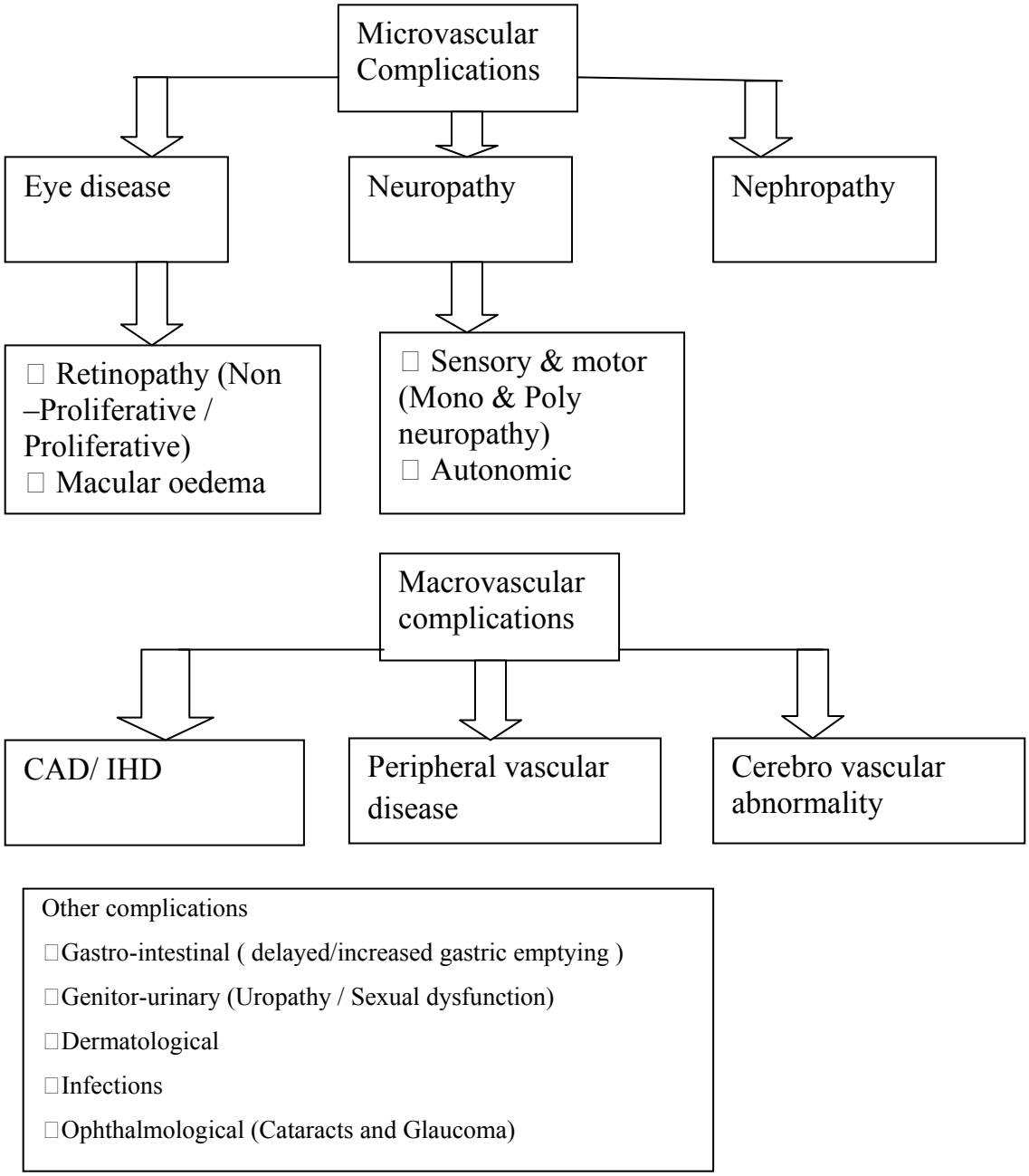
Table 3.1: Diagnosis of Diabetes Mellitus

Terms	**Definition**
Random Blood Glucose (RBS)	Blood Glucose levels without regard to time since last Meal
Fasting Blood Glucose (FBS)	Blood Glucose levels when there is no caloric intake forpast 8 Hrs
2 Hr Plasma Glucose (During Oral GTT)	75gm of glucose(anhydrous form) mixed with water.

Chronic Complications of DM

Mortality and morbidity asso with T2DM are mainly due to the chronic complications of DM

Fig. 2.1: Chronic Complications of DM

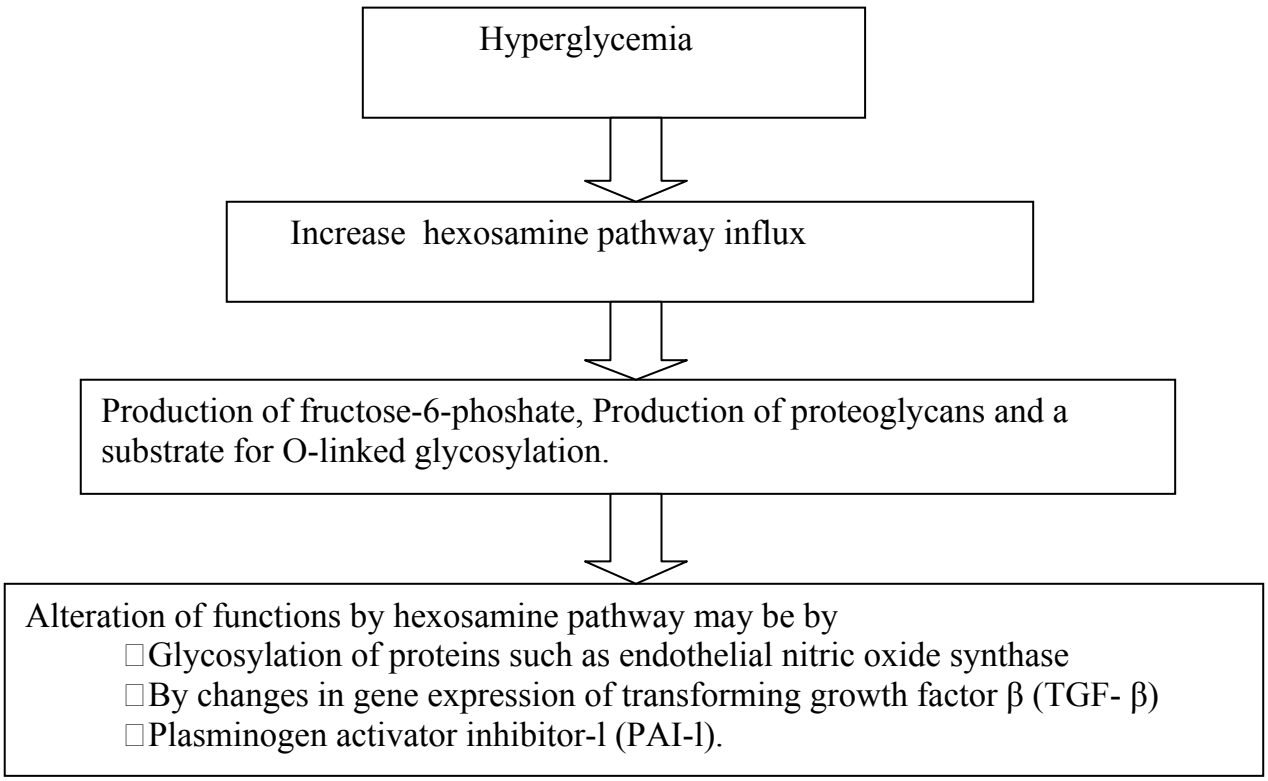


- With the increase in duration of the disease(hyperglycaemic state), the is associated increase in risk of chronic complications.
- At the time of diagnosis, the patient may present with complications because of the long asymptomatic periods of hyperglycemia.
- In both type 1 and type 2 diabetes mellitus, microvascular complications are seen as a result of chronic hyperglycemia.
- Many clinical trials shows that there is significant prevention/delay in retinopathy, neuropathy and nephropathy if chronic hyperglycaemic phase is reduced.
- In type 2 DM patients, there is two to four fold increase in incidence of coronary heart disease mortality.
- The above events have a positive correlation with FBS and PPBS glucose levels as well as with the Hb A 1 C.
- Macrovascular complications are also related to Dyslipidemia and increased blood pressure

Complications - mechanism

A hypothesis proposes that leading to :

Fig. 2.2: Mechanisms of Complication of Diabetes Mellitus



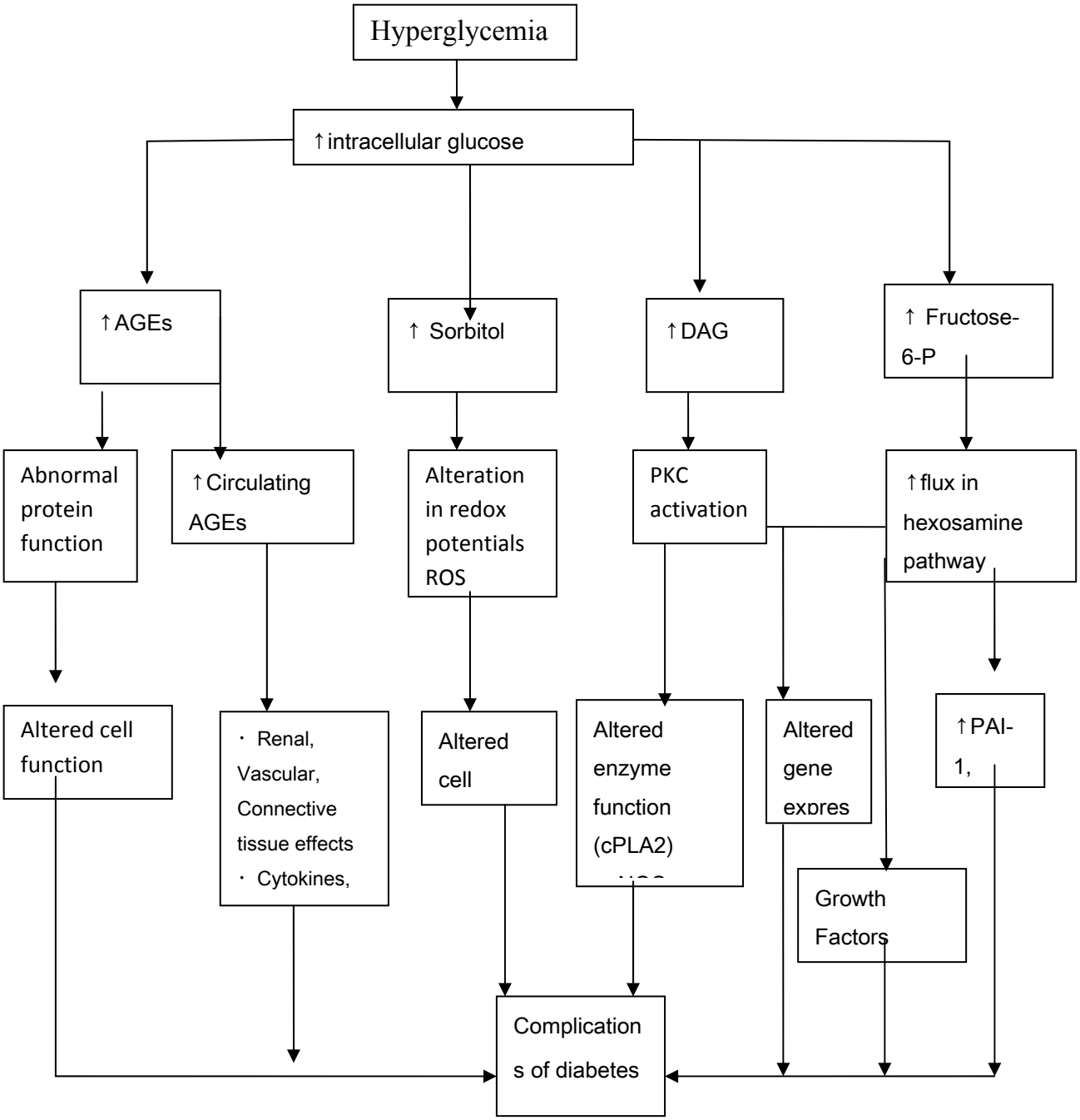


Figure showing - Possible molecular mechanism of diabetes-related complications

AGEs : Advanced glycosylation end products

PKC : Protein kinase – C

DAG : Diacylglycerol

c PLA 2 : Phospholipase A2

eNOS : Endothelial Nitric Oxide Synthase

ROS : Reactive oxygen species

PAI-l : Plasminogen activator inhibitor-l

Neuropathy And Diabetes Mellitus

- The prevalence of diabetic neuropathy in patients with type 2 diabetes is 32 percent overall and more than 50 percent in patients over 60 years of age.1,2
- Diabetic neuropathy have a positive correlation with the disease duration and control of hyperglycemia in type1 & 2 DM.
- May manifest as Polyneuropathy/ Mono-neuropathy/Autonomic Neuropathy
- All the nerve fibers (including myleinated and unmyelinated) are affected.
- Diabetic neuropathy have similar clinical features when comapering with other neuropathical diseases, therefore all other possible causes should be excluded before making a diagnosis

Poly-neuropathy / Mono-neuropathy :

- The most common form of diabetic neuropathy is distal symmetric
- polyneuropathy.
- It presents as:

    1. Distal sensory loss - most frequent presentation
    2. Hyperesthesia
    3. Paresthesia
    4. Dysesthesia

- Symptoms includes a sensation of following, which begins in the feet & spreads proximally.

  1. Numbness,
  2. Tingling
  3. Sharpness
  4. Burning

As neuropathy progresses,any of the following symptoms may develop

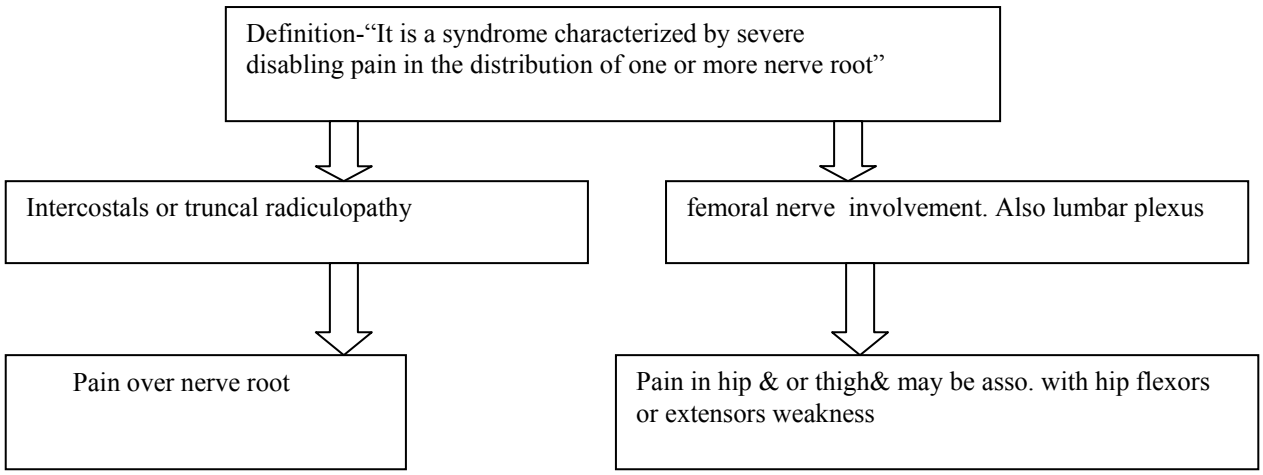
- Physical examination reveals

  1. Loss of sensation
  2. Ankle reflexes lost
  3. Position sense abnormality.

- Pain usually rest pain may be present in the lower limbs, which may increase at night.
- With the progression of the disease, there will be decrease in the intensity of the pain and eventually it disappears. But in the extremities, sensory loss may persists.
- In some patients, with the improvement in glycemic status, development of neuropathic pain is seen.

Diabetic Neuropathy :

It may be accompanied by - Motor weakness



Treatment of diabetic neuropathy :

- Nerve conduction velocity will be better if the glycemic control of the patient is improved.but there won't be any improvement in neuropathic symptoms.
- Alcohol and other neuro toxins should be avoided, vitamins for possible deficiencies (BI2, B6, folate) should be supplemented
- Treatment of symptoms.
- After the 1st year of the diabetic neuropathy, there will be substantial decrease in pain, due to the progressive neuronal damage from DM. Therefore analgesics may be discontinued after this.
- Chronic, painful diabetic retinopathy – treatment is usually difficult, but may respond to

  1. Tricyclic antidepressants - Amitryptiline, desipramine, nortriptyline

2. Gabapentin

3. NSAIDs (Avoid in renal dysfunctions)

4. Others (Mexilitine, Phenytoin, Carbamazepine, Capsaicin cream)

The patient should be reffered to any pain management clinic..

Lower Extremity Complications

- In diabetic patients, the major source of morbidity is ulcers over the foot and infetions over that region.
- Increased occurrence of lower limb complications in DM is due to interaction of may pathogenic factors
    - Neuropathy
    - Abnormal foot biomechanics
    - Peripheral arterial disease
    - Poor wound healing.

Neuropathy:

75% of patients having foot ulcers develop neuropathy.

Peripheral sensory neuropathy:

Due to this, patients will be having repeated minor injuries over the feet, leading to defect in normal protective mechanisms. These happen without the knowledge of the patients.

Motor and sensory neuropathy:

May cause abnormality in mechanism of foot muscles and may also cause change in structure of the foot (hammer toe, claw toe deformity, prominent metatarsal heads, Charcot joint)

Autonomic neuropathy :

Lead to dry skin, fissure etc. as a result of anhydrosis and blood flow alterations.

Peripheral arterial disease and poor wound healing :

Cause defective healing of minor injuries in skin, leading to enlargement of the wound and super added infection

Disordered proprioception :

Callus formation and ulceration is formed due to abnormal weight bearing while walking and standing.

Approximately 15% of individuals with DM develop a foot ulcer, and a significant subset will ultimately undergo amputation (14 to 24%) risk with that ulcer or subsequent ulceration.

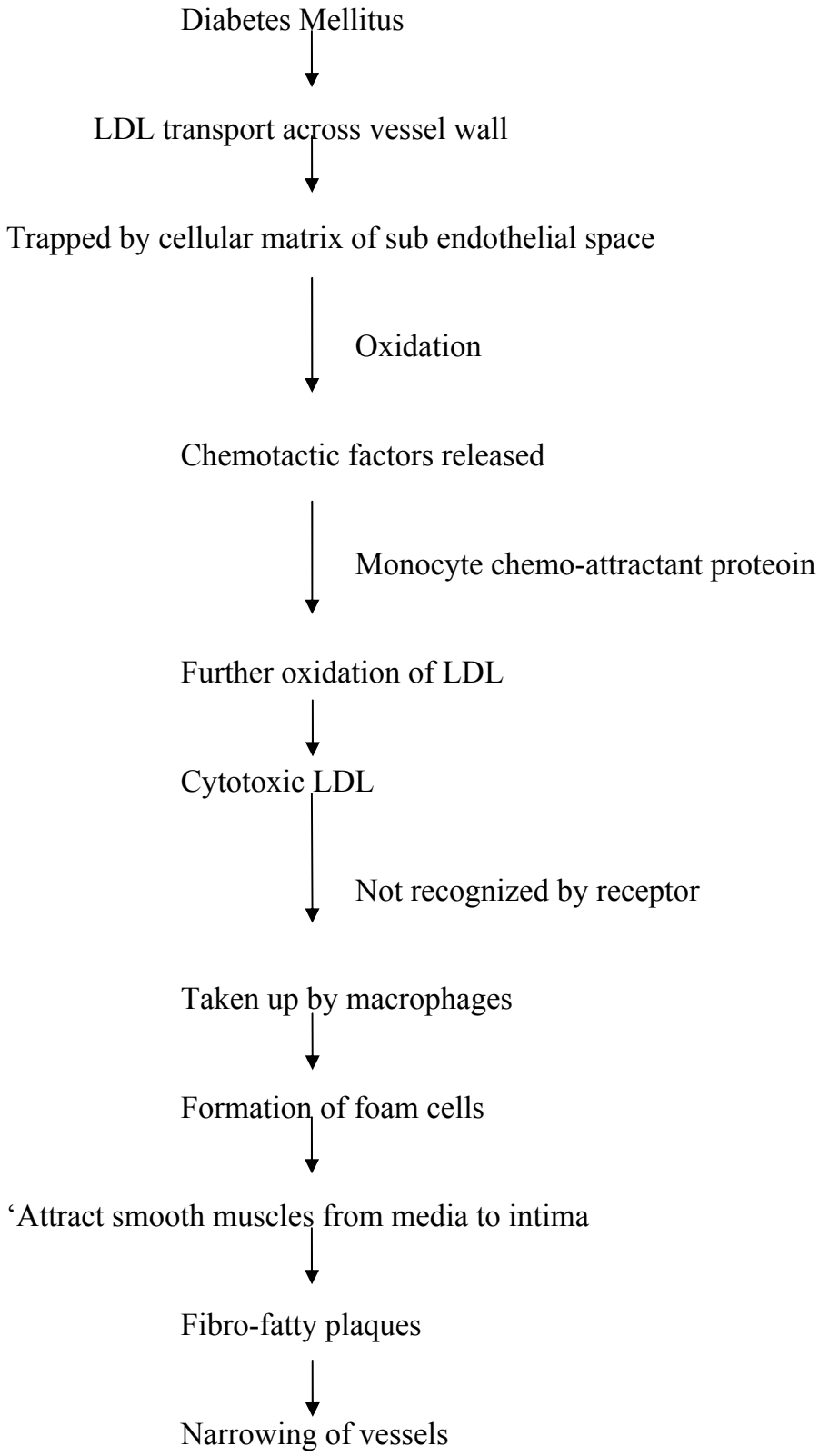
VASCULAR CHANGES IN DIABETES

1. Atherosclerosis: Chronic inflammatory process that can be converted into acute clinical event by plaque rupture.

Development of atherosclerosis is accelerated in DM leading to increased morbidity and mortality. All the large vessels are involved in this process and clinical manifestations are apparent as a result of atherosclerotic narrowing and thrombosis of coronary, cerebral and leg vessels

I Lipoproteins pathogenesis:

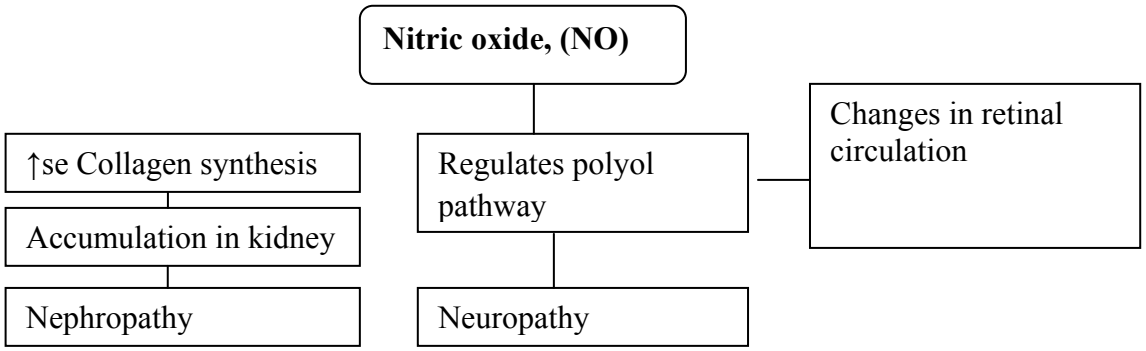
Fig 2.3 Pathophysiology Diabetic Vasculopathy



II Endothelium:

a. Nitric oxide, (NO): (EDRF-Endothelium derived relaxing factor)

Nitric oxide, (NO)



b. Prostacyclin (PGI2)

PGI2

↓

Potent vasodilator also inhibits

Platelet adhesion & aggression

↓

Prostacyclin stimulating factor

(Present in human arterial smooth muscle)

↓

↓se in Diabetics & arterial wall of patients with old MI

c Thromboxane A2(TX-A2):>>>>Vasoconstrictor- Counteracts effect of N.O

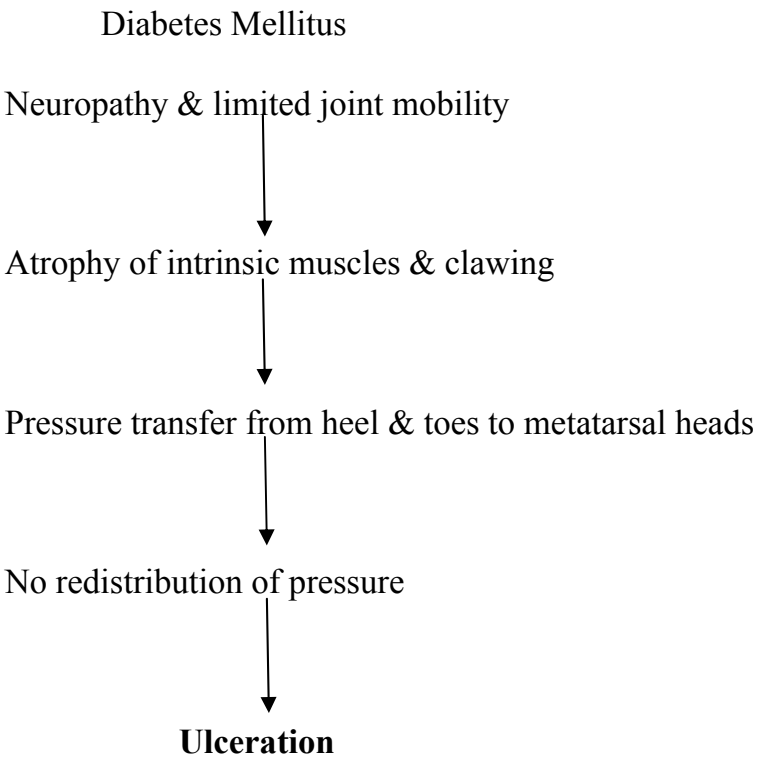
↑sed levels found in DM, HTN & hyperlipidemia

.

d Endothelin::>>>>Vasoconstrictor

↑sed levels found in DM around 3.5 times

Fig 2.4 Pathogenesis of Diabetic Ulcers.



Predisposing factors for ulceration:

1) Limited joint mobility.

2) Peripheral neuropathy.

3) High plantar pressure.

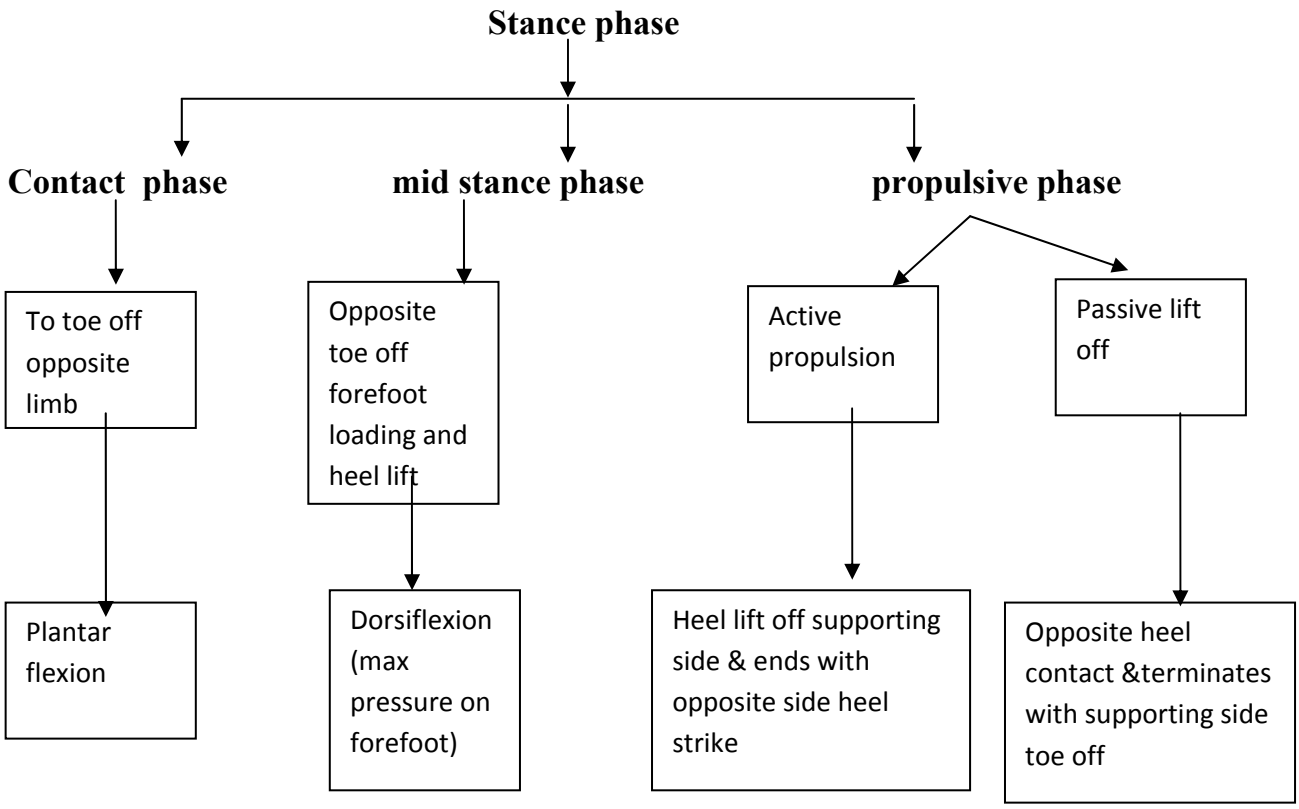
4) Vascular diseases.

Biomechanics of diabetic foot

Gait cycle:

1. **Stance phase**
2. **Swing phase**

Fig 2.5 Biomechanics of Diabetic Foot



Changes in foot caused by diabetes

1. Peripheral neuropathy

A. Dryness of skin

B. Callus formation

2. High pressure at bony prominences

↓se plantar tissue thickness

↓

Weak intrinsic muscles of foot

↓

Imbalances of flexors & extensors causing clawing of foot

↓

Pulling away fat padding from metatarsal heads

3 Limited joint mobility

Unknown etiology

↓

Collagen abnormality

↓

Thickening of skin, tendons & joint capsules

↓

Decreased tissue flexibility

↓

Increased plantar pressure

4 Trauma

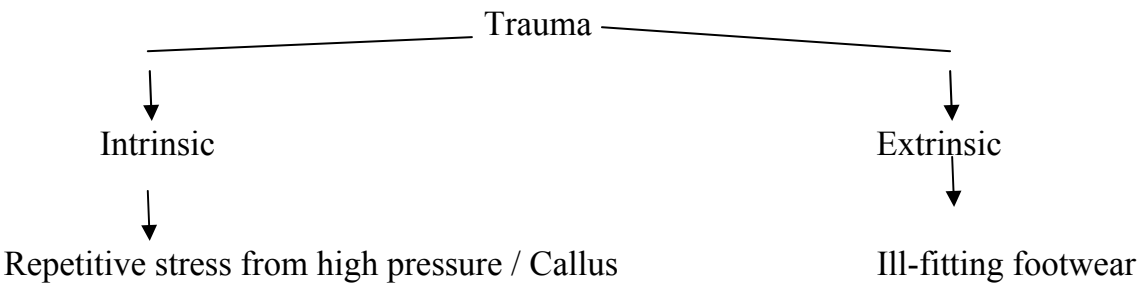
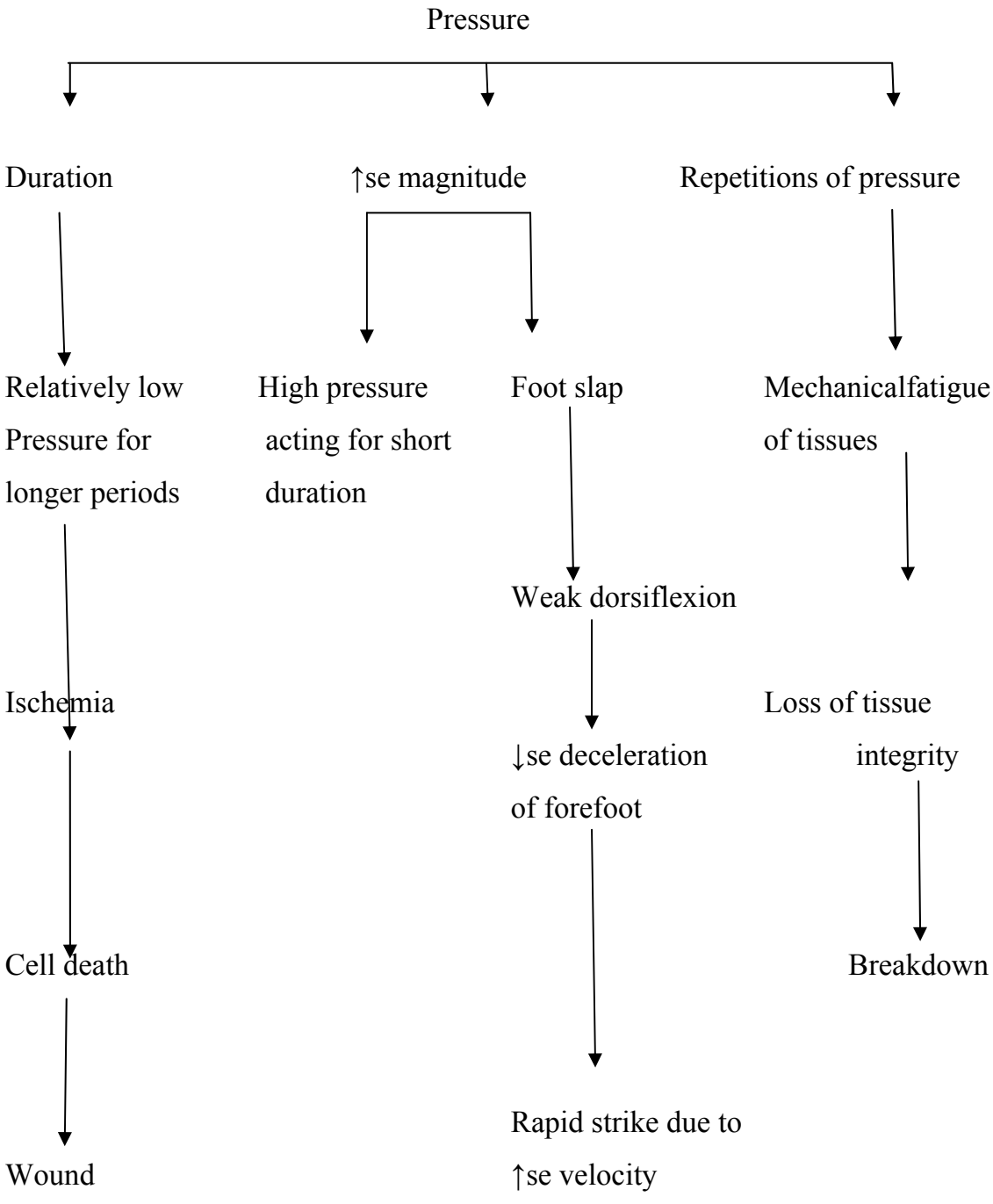


Fig 2.6 Causation of ulceration



CLASSIFICATION OF DIABETIC FOOT ULCERS.

The six grade Wagne- Meggit classification, which has been used for decades, classifies wounds by the depth of ulceration and extent of gangrene. The PEDIS classification (by International Working Group on Diabetic Foot)

- Perfusion (arterial supply)
- Extent (area)
- Depth
- Infection
- Sensation

WOUND DRESSING IN DIABETIC FOOT

In the management of diabetic foot, the main aspect is the dressing of the wound and wound managment. For the limb to be salvaged, proper scientific, cost effective method should be used for dressing the wound. The various functions of the dressings are:

- wound isolation
- edema limitation/reduction
- pain reduction
- exchange of gases from blood to tissue should improve.
- Inflammation should be limited
- exudates should be absorbed
- bacterial growth should not be promoted.
- contamination and dessication should be prevented

All the dressings can be classified as primary or secondary. Primary dressing is the one, which is in direct contact with the wound. Secondary dressing is of the material, which holds the primary dressing in place. It has function of compression, occlusion and additional protection.

Diabetic Foot Disease: An Understanding of The Disease:

Pathophysiology:

Knowledge of the pathophysiologic changes caused by diabetes Mellitus is essential for understanding and treatment of diabetic foot problems. Sustained hyperglycemia, neuropathy, infections and ischemia are the principal pathogenic factors.

Neuropathy:

Diabetic patients develop sensory, motor and autonomic neuropathy. The neuropathic foot is characteristically healthy. It is well nourished, hair will be present, has good dorsalis pedis pulsations and posterior tibial pulsation, has a high arch. Callus formation common on pressure points over the soles or toes, and sweating may be there. This shows lumbar sympathetic activity. Over the soles, thick calluses may act as foreign bodies and may lead to bruising of the subcutaneous tissues along with extravasation of blood and serum through the capillaries. This acts a culture medium for local bacteria to grow and leading to an abscess. The condition may not be known by the patient because of anesthesiacaused by the neuropathy and can expand without being detected till the patient develops generalized infection or the problem is noticed by it's foul smell.

Sometimes the overlying callus may be so hard that the infection can easily involve the joint capsule lying below or the metatarsal head below than the callus itself. Thus osteomyelitis associated with calluses can be explained and understood. The same process may occur over the top of hammer toes or in association with the deformed and collapsed bones and joints seen along with Neuro-osteoarthropathy / Charcot's foot.

Vasculopathy:

Both the Microvascular and Macrovascular disease is present in diabetics.

Atherosclerosis is speeded up in diabetics. Infrageniculate vessels (Posterior tibial, Anterior tibial, peroneal vessels) are affected severely. Perfusion is decreased due to occlusive disease withmicrocirculatory impairment of cellular exchange.

Ischemic foot is characteristically scaly dry atrophic and hairless, and undernourished. It is cold on touch. The nails are found to be thick and overgrown alongwith dry scales under the nail itself. Such ischemic feet usually have a small infection behind a nail or in the depth of a fissure. The ischemic foot may have small flat, dry atrophic ulcers with full thickness skin necrosis in the centre with a rim of erythema surrounding the ulcer. Wet gangrene is seen when a higher vessel in the thigh or calfbecomes suddenly occluded. The tissues are still wet and the impending gangrene is manifested by pallor, then rubor, and lastly the blistered skin withunderlying blue black tissue shining through. Only the ischemic foot has these features, and usually the flow into the patent major vessels is not enough or

notin time to revive the injured tissues. Therefore higher amputationsare necessary.

Diabetic Angiopathy is classified in to 2 types:

1. Macro Angiopathy

- Atherosclerosis - Characterized by formation of atheromatous plaque in vessel wall leading to thrombus formation.
- Monckeberg's Sclerosis / Medial calcific sclerosis- Characterized bycalcification of media of mascular artery
- Intimal fibrosis - a part of normal aging process**.**

2. Microangiopathy

Small vessel disease comprises of changes in arterioles and capillaries.

Occlusion of these capillaries leads to patchy areas of gangrene. The capillaries are a common site of diabetic microangiopathy. Homogenous periodic Acid Schiff positive thickening of capillary wall is considered as the hallmark of diabetic microangiopathy because of basement membrane thickening.

Banson and Lacy in their studies involving 18 diabetic & 17 non diabetics, foundthat capillary basement membrane thickened in 88% of the Small vessel disease consists ofchanges in arterioles and capillaries. Occlusion of these capillaries results to patchyarea of gangrene.

Infections:

Infection affects diabetic control. Uncontrolled diabetes causes infection because hyperglycemia at wound site that forms nidus for bacterial flora to grow. An abscess or cellulitis may produce hyperglycemia, conversely, hyperglycemia may affect the healing of abscess.

Mild infections are commonly due to surface organisms like staphylococcus andstreptococcus. Severe infections are polymicrobial that includes aerobic gram +ve, anaerobic gram negative rods like (E-Coli, Klebsiella, Pseudomonas and Proteus) and anaerobes. Identification using cultures and appropriate antibiotics is necessary for controlling infections. When infectious process dissects deep in to the soft tissue and enters the periosteum, osteomyelitis results. In acute stages of osteomyelitis, the process is a suppurative one. There is necrosis of both cortical and cancellous bone with formation of the sequestrum. Later chronic osteomylitis develops.

Guidelines In The Examination of The Diabetic Leg And Foot:

History: It isimportant to start by checking patients diabetic history. A long history of insulin dependent diabetes is often associated with a higher incidence of neuropathy, retinopathy and also nephropathy called the "Diabetic triopathy".

Severe triopathy is thought to be due to secondary to basement membrane thickening and in patients who have this, the wound healing appears to be delayed. This is particularly true in those patients with renal failure. Consideration

of patient’s renal function is also important in deciding whether they are candidates for arteriography for further evaluation of arterial insufficiency.

Hypertension, tobacco consumption, and Hypercholesterolemia are also important factors in the development of arterial insufficiency. Severe cardiac disease may be contraindication for aggressive management of the diabetic foot problems.

Patient's presenting problem discussed in detail. If the patient describes a rapidly enlarging lesion asso. with drainage or swelling, perhaps with red lines extendingupto the leg and fever or chills, then infection is very likely.

The classic distal polyneuropathy seen in diabetic patients may not only cause lesions on its own, but may also mask the symptoms of infection and arterial insufficiency and may lead to the development of painless ulcer over a pressure point.

Patients may complain of pain, burning sensation of feet, that the feet feel cold or feel as encased in concrete or of the sensation of walking on a glass. Sometimespatient may describes knife like shooting pains up the legs, usually both legs are involved and this may help in distinguishing neuropathic from ischemic pain. The history of ischemia is usually present in patients with diabetic foot lesion, although these symptoms may be masked by presence of neuropathy. A history of cardiac or carotid disease would definitely make the presence of peripheral vascular disease more likely. Claudication pain or pain in muscle

groups on exercise is the usual presenting symptom of peripheral vascular insufficiency.

Occasionally, particularly in smokers, inflow disease is responsible for ischemia, and then buttock and thigh claudication may be their presenting symptom. This is known as leriche syndrome due to obstruction of the aortoiliac vessels, may be asso. with impotence in men.

Physical Examination:

It is helpful to start examination away from the area of interest. The carotids should be checked for presence bruits and the presence of pulses in the arm. Atrial fibrillation as a possible source of embolisation can be noted. The abdomen should be thoroughly examined for an abdominal aortic aneurysm, which may also be a source of distal emboli.

Assessment of Diabetic foot:

Includes identification of:

A. . Infection

B. Vasculopathy

C. Osteopathy

D. Neuropathy

The feet should be looked for

o Dry skin, cracks, fissures

o Ulcers, cavity, sinuses

- o Calluses, hyperkeratosis
- o Infection
- o Gangrene.
- o Foot deformity

A. Neurologic Examination to assess the sensory and motor disturbances:

- o Monofilament testing
- o Vibration testing using tuning fork
- o Tendon reflexes and also pain

B. Vascularity is assessed by examination of the:

- o Distal pulses
- o Transcutaneous oxygen saturation
- o Ankle brachial pressure index
- o Duplex arterial study.

C. Osteomyelitis is assessed by the X - ray examination of foot

D. Infection is assessed whether superficial or deep**.**

It is usually common to have all three processes occurring at the same time. Thus an infected neuropathic lesion seen in a patient with poor circulation is not unusual. Significantly neuropathy may hide the signs and symptoms of ischemia, and the resultant failure to diagnose the underlying ischemia may result in mistreatments. Severe neuropathy may result in a pain free foot that is auto

sympathectomised and thus, warm, but at the same time, severely ischemic. A minor procedure on such a foot may result in gangrene.

Table no: 3.2 Grading of Diabetic foot: Wegner's Classification of diabetic foot.

Grade	Clinical features
Grade 0	Normal foot with variable degree of neuropathy. Joint deformity, foot at risk.
Grade 1	Superficial ulcer, cellulites
Grade 2	Uncomplicated deep ulcer
Grade 3	Complicated deep ulcer, deep infections, osteomyelitis
Grade 4	Limited necrotizing gangrene
Grade 5	Extensive gangrene

The Diabetic Foot: Medical and Surgical Management:

A. Baseline Approach in Managing the Acute Problem of the Diabetic Foot:

1. Appraise problem

    o Careful inspection with emphasis on web spaces and back of heels.

    o Record pulses, venous filling time, rubor

    o Record sensation.

2. Describe lesion

3. Describe Necrotic tissue, probe sinuses with sterile probe to determine the extent of disease.

4. Culture pus for aerobic and anaerobic organisms

5. Begin broad spectrum antibiotic until appropriate antibiotics can be given according to culture and sensitivity.

6. Medical Management of Diabetes - Blood sugar monitoring and antidiabetic measures to achieve good glycemic control, Doppler study of vessels.

7. X - ray both feet to exclude osteomyelitis.

8. No weight bearing (Hospitalize with absolute bed rest when indicated.

Crutches or walker when feasible.)

9. Surgical Management of the Problem

- No soaks
- Antibiotics
- Medical Management of diabetes
- Dressing change atleast once daily.
- Surgical debridement, frequently if necessary.
- Consideration for possible arterial reconstruction
- Drainage or open amputation.

10. Rehabilitation

- Podiatrist for patient education, preventive maintenance, orthotics, healing sandals and special shoes.
- Nutritionist to advice on diet needs.
- Surgeon to ensure proper wound healing and proper prosthetics

- Physician to make final decision about diabetes management.
- Psychiatrist to return to normal activity.

PREVENTION IS BETTER THAN CURE:

Prevention of ulceration and recurrence once ulceration has occurred are the ultimate goals of any modern team approach to the diabetic foot.

Wagner's Grade O: Foot are the patients who are potentially "at risk" to develop ulcer or infection due to varying degree of neuropathy and joint deformities. They need regular* assessment annually for neuropathy and vascular status. Hence the role of proper footwear and hygiene cannot be overemphasized. The diabetic patient and his family must establish a routine for daily foot and shoe inspection and hygiene. Every patient must be taught to shake his shoes at and inspect them prior to wearing. Proper hygiene must become a religion. Washing the feet everyday with mild < soap and rinsing and drying thoroughly especially between the toes are adviced.

The physician or health care provider must always set the example. Controlling blood glucose, weight, and blood pressure; eliminating smoking; encouraging daily exercises are important. Periodical neurological and vascular examinations are important. Early recognition and prompt reporting of a problem are encouraged.

Principles of Medical Management:

- Pus from ulcers sent for culture and sensitivity.
- Careful monitoring of the blood glucose levels.

- Appropriate antidiabetic measures either insulin preparations or oral hypoglycemic drugs.
- Broad spectrum antibiotics to be started at the onset and change over to other antibiotics depending on the culture and sensitivity report.
- Patients with limb threatening infections require hospitalization. It is most prudent, initially to administer antibiotics parenterally to ensure adequate serumlevels.

Principles of Surgical Management:

- Early recognition and prompt intervention.
- Control of blood glucose
- Complete rest of injured area.
- Careful but complete debridement and drainage of all involved areas.
- Appropriate antibiotic coverage
- Wound care and dressings
- Appropriate vascular reconstructions
- Careful follow up including podiatric appliances and modified footwear.
- More experienced consultation as necessary**.**

Wagner Grade 1 foot: These are patients with superficial ulcers and cellulitis.

Infection is controlled with appropriate antibiotics and debridement if required. Ulcers occur because of repetitive pressures. Pressure is relieved by complete bed rest, use of total contact cast, walker, braces etc. Associated vascular insufficiency has to be corrected by vascular reconstruction.

Wagner Grade 2 and Grade 3 feet : These are patients with deep ulcers, with or without complications like abscesses and osteomyelitis. Aggressive surgical debridement, excision of the infected bone and vascular reconstruction if necessary is the mainstay of the treatment. To avoid recurrence education about foot care is essential.

Wagner Grade 4 and 5 feet:

These are patients with localized or extensive gangrene. Management is by appropriate minor or major amputation followed by vascular reconstruction.

Local Treatment of Diabetic Foot:

Uncontrolled diabetes affects infection and infection adversely affects diabetes.

The basic rules in treating any foot infection are:

- o Absolute bed rest
- o Regulation of diabetes
- o Adequate culturing of wound

Administration of appropriate antibiotics

Adequate drainage of all infection

Appropriate wound care.

Drainage: Drainage means opening all abscesses, probing carefully, and laying open all sinus tracts, debriding all necrotic tissue and providing unhindered dependent drainage of pus in the resting foot. The pus must drain down and out.

Gas in the tissues can often be felt as crepitus or may be the first detected on x-ray film. This is a serious finding and must be treated immediately by open drainage of all infected spaces and prompt i.v. antibiotics.

Drainage of an infected area may involve amputation of a necrotic toe or toes or even an open amputation. Such amputations are drainage procedures primarily. The avascular joints tolerate infection badly, and ultimately the infected joints in the toes and the feet have to be removed. When an infected area has been enclosed, it is important to plan and attempt to salvage tissue for a possible definitive wound closure**.**

Dressings:

Most foot infections do not require extensive incisions debridement, yet the principles must always be remembered. Dressings are used to serve the following purposes.

- Contain wound drainage.
- Debride a wound
- Protect an area from trauma
- Protect an area from contamination
- Promote proper wound healing

The basic equipment necessary for bedside foot care is:

1. Sterile debridement set containing

- Sharp scissors for debriding
- Blunt ended needle wound probe

- Smooth forceps

2. Sterile toenail clippers

3. Sterile guaze dressings

4. Tube guage, paper tape, culture tubes

5. Medicines - Povidone iodine 2.5% - Bactericidal

    - Dakin's solution (chlorazene 0.25%)
    - Bactracin ointment - antibacterial
    - Vaseline guaze
    - Normal saline

Dakin's solution: is a chlorine releasing agent that is both bactericidal and active in loosening necrotic tissue to aid in local debridement. Dakin's also helps to control fetid odours from severely infected wounds.

Open wounds require packing using an unfilled guage moistened with atherapeutic solution. Changing packing two or three times a day is recommended for debridement of a necrotic wound. Allowing sufficient time between dressing changes gives the packing time to begin to dry and therefore provide gentle debridement as the packing is removed from the wound. Unfilled guaze is recommended for packing wounds. Care must be taken not to pack the wound too tightly as it tightly obstructs drainage.

A properly applied dressing will not constrict the foot or leg or slip, possibly causing wound trauma or exposure. Spiralling or wrapping the roller

guage in a figure of eight fashion is the best way to prevent a tourniquet effect and will decrease the risk of compromising the circulation of the foot.

Routine Foot Dressings:

- Moisten guaze with appropriate solution and pack the wound gently.
- Fashion a heel cup from cut, folded and taped abdominal pad.
- Fluff two 4 inch guaze sponges over toes
- Secure the primary dressing, including heel cup by using a spiral roller by wrapping in a figure of eight fashion.
- Apply paper tape to secure the roller guaze.

Casts / Splints:

A cast or splint may be applied to immobilize a limb after a skin graft or to protect the incision and reduce contractures after a below knee amputation. Applying a rigid plaster cast or splint to any neuropathic extremity can be hazardous and may cause pressure sores.

Amputation Stump Dressings:

The dressing applied to any amputation stump is fashioned to meet the needs of the wound. Since most amputation wounds do not have drains, the dressing is put on more for wound protection than to collect and contain blood and secretions. A first transmetatarsal amputation dressing is a bulky standard foot dressing. A posterior splint may be applied to prevent plantar flexion and thus avoid tension on the delicate suture line. A below knee amputation (BKA) requires an extra bulky initial.

Dressing to contain the initial expected bleeding. Below knee amputations are managed with a posterior splint that extends from the crease of the buttocks to beyond the end of the stump. A well padded knee immobilizer is the splint of choice. Knee flexion is a natural pain relieving action or reflex that, if allowed to occur, can lead to serious contracture. It is customary to have a patient with a BKA measured for a prosthesis on the 3rd or 4th post op day. Depending on the progression of stump healing, a patella bearing prosthesis may be fitted and patient begins mobilization eight to ten days postoperatively.

The initial dressing for an Above Knee Amputation (AKA) is bulky and similar to the BKA dressing. Splints are not used for AKA despite the tendency for patients to hold up and flex the painful thigh. The stump usually falls down with muscle fatigue, thus decreasing the tendency for a hip contracture.

Skin graft dressings are usually applied in accordance with the surgeon's preference. Mesh grafts are the most common split thickness skin graft. The mesh graft has proved to be the most successful because the open mesh allows adequate wound drainage.

Bed rest is the first thing in the care of a diabetic foot lesion. Bed rest must be absolute and continuous. A patient with diminished circulation who has a painful ischemic foot lesion may be helped by having the head of the bed elevated to 6-8 inches. This elevation allows gravity flow of blood to the feet and is known as arterial position or Reverse Trendlenberg position.

Non-Surgical Modalities to Enhance Healing:

1. Growth Factors

Greater understanding of the healing process at the cellular level has resulted in the use of growth factors like becaplermin, recombinant platelet-derived growth factor, are produced through recombinant DNA technology. According to a study by Steed et al, debridement enhances the effectiveness of becapiermin in healing chronic neuropathic ulcers.

2. Human Skin Equivalents

Modern human skin replacement dates back to the 1960s, when advances in tissue culture technologies led to the cultivation of human epidermal cells. These were obtained via biopsy of the tissue and treated with trypsin so that the dermis get separated from epidermis.

The keratinocytes were then grown in vitro to produce sheets of autologous epidermal tissue. These sheets were fragile, delicate to handle, and provided only 50 percent to 60 percent permanent take. New tissue required two to three weeks growth time, and lacked a dermal component, vital in skin grafting.

More dermis grafted means less wound contracture and scarring, more tensile strength and better cosmetic results. Refinements in the development of a matrix led to the development of Dermagraft, a living, metabolically active, immunologically inert dermal tissue.

Dermagraft contains normal dermal matrix proteins and cytokines, and is composed of cultural neonatal fibroblasts grown on a polyglycolic acid bioabsorbable mesh. As the tissue grows it produces extracellular proteins and closely resembles human skin. In two studies by Gentzkow et al and one by Pollak et al, patients were enrolled with full-thickness diabetic ulcers that had adequate perfusion. Pooled data showed that 51 percent of those who received a weekly application of Dermagraft for 12 weeks achieved complete healing, vs. 31.7 percent in the control group.

Apligraf, another living tissue equivalent, was approved by the Food and Drug Administration in 1998 for venous leg ulcers. Apligraf consists of bovine Collagen matrix containing fibroblasts and connected to a layer of stratified epithelium. The result is a sheet of tissue with both dermal and epidermal layers, metabolically and biochemically comparable to human skin. The dermoepidermal junction is flatter, however, and there are no melanocytes, Langerhans cells, lymphocyes or hair follicles present.

In a study by Falanga et al, 293 patients with non-healing venous ulcers received either compression therapy or Apligraf. At six months, 63 percent of the patients receiving Apligraf healed vs. 49 percent in the control group and did I so more quickly - than the control group - 61 vs. 181 days to closure**.**

3. Miscellaneous Topical Agent:

Collagen: Collagen is critical in the proliferative phase of wound healing. Exogenous sources of collagen primarily purified bovine extracts, are available as

gels, particles, and in an alginate dressing. Exogenous collagen provides additional protein for tissue repair. As a foreign agent it might also revert the chronic wound to an inflammatory phase, "jump-starting" the healing process.

Donaghue et al evaluated the alginate dressing (Fibracol, Johnson and Johnson, Arlington, Texas) in the treatment of diabetic foot ulcers. Seventy-five patients were randomly assigned to either a collagen-alginate dressing or gauze group. At the end of the study, the mean reduction in wound size was 80.6 percent for the collagen-alginate group and 61.1 percent for the gauze group. Complete healing was achieved in 48 percent of the collagen-alginate group and 36 percent in the gauze group.

Hyaluronic Acid: Hyaluronic acid is involved in the structure and organization of the extracellular matrix and is associated with increased mitotic activity. It is a highmolecular weight polysaccharide synthesized in the plasma membrane of fibroblasts and other cells. The ability of injured fetal tissues, which are high in Hyaluronic acid, to heal without scarring has prompted extensive research

Beta Glucan: It is a major cell-wall carbohydrate extracted from such grains as oats and barley. The biological activity of beta glucan results from its ability to bind macrophage beta-glucan receptors and promote macrophage stimulation.

Beta glucan products enhance the activities of not only macrophages but also neutrophils, natural killer cells, T cells and B cells. Beta glucan is thought to increase macrophage infiltration, speeding the onset of fibroplasia and fibrogenesis, stimulation of increased tissue granulation, and enhanced reepithelialization. Beta glucan is available as either BCG matrix or Glucan II.

Both are available in multifilament mesh dressings; BCG matrix is also impregnated with collagen.

Silver Arglaes: Silver compounds are powerful antimicrobials, useful in promoting healing. Arglaes is an inorganic phosphate similar to other compounds such as silver nitrate, silver oxide and silver chloride. It consists of fused sodium and calcium phosphates with small amounts of silver in the presence of water, these materials release free silver ions.

4. Pharmaceuticals:

Oxandrolone: Oxandrolone is an anabolic steroid with a high anabolic and low androgenic ratio, and has anticatabolic, protein-sparing properties. Exogenous anabolic agents clubbed with nutritional intervention can result in a threefold to fourfold higher rate of protein synthesis than with nutritional interventions alone.

Demling and De Santi studied eight patients with non-healing wounds and a 10 percent or greater loss of body weight. Nutrition was optimized over four weeks, without significant effect on weight gain or healing. Adding oxandrolone resulted in gains of approximately 4 pounds per week across 12 weeks. During this time, five wounds closed completely and three others were 75 percent closed.

5. Devices

Vacuum Assisted Closure (VAC): Argenta and Morykwas determined that intermittent negative pressure at 125 mmHg promoted wound healing by improving blood flow, granulation tissue growth rates and nutrient flow while reducing bacterial levels. Based on these findings, Kinetic Concepts (San

Antonio, Texas) developed the VAC system. The VAC consists of a wound dressing (a charcoal - impregnated sponge - like material) connected by tubing to a wound canister, with a pump that creates negative pressure. A transparent drape or film over the dressing establishes the seal needed to create a vacuum. The pump can be adjusted for various levels of intermittent or continuous pressure. Exudate is collected in the cainster. The VAC also is said to reduce edema.15

Radiant Heat Bandage: Heat therapy has long been employed, especially for musculoskeletal conditions, but it has not been widely used as a wound healing modality. Heat increases local blood flow, subcutaneous oxygen tenson which improve healing mechanisms. In clinical studies by Santilli and Robinson on patients with venous leg ulcers, those who used radiant heat bandage devices reported significant decreases in both wound size and pain across two weeks with no adverse effects.16

Topical Hyperbaric Oxygen Therapy:

The therapy is based on achieving an atmospheric pressure of 1.02 to 1.03 atmos, which is thought to stimulate fibroblast, growth, collagen formation andneoangiogenesis. This provides a lethal environment for anaerobes, often a normal part of the diabetic foot's flora. Topical hyperbaric oxygen is administered using a sealedpolyethylene bag over the affected area and administering 100 percent oxygen to apressure between 20 and 30 mmHg. Treatments last 2 to 2 and a halfhours.

In a study of Landau, 50 patients with diabetic ulcers were treated with topical hyperbaric therapy, alone or with a low-energy laser. On average, 25 treatments

were performed over three months. Forty-three of the 50 patients experienced resolution of their ulcers.

Classification of Dressings:

Wound dressings have evolved over the years on the principles of providing protection to wound raw surface, absorbing exudates, controlling infection and promoting granulation tissue formation and creating ideal environment for healing.

There are two major categories in dressings:

1. **Short term application:** we should replace these dressings frequently

2. **Long term or skin substitutes**:

- Temporary : these are applied till complete healing. Used in partial thikness wounds
- Semi-Permanent : these are used till autografting. Used over full thikness wounds.

They are classified as conventional, synthetic, biological, based on material used for preparation. Each further divided into :

o **Primary Dressing :**which is in physical contact to the wound bed.

o **Secondary Dressing :**primary dressings are covered with these dressings.

o **Island Dressing :** at the central region there is absorbent part, adhesive part surrounds the central portion.

A. Conventional Dressings:

Fabric materials like gauze are used, but these allows moisture to evaporate and dries the desiccated wound bed.Also causes exogenous bacteria to enter the wound. Some used paraffin soaked dressings. This also led to development of usage of antibacterial agents like polymixin,carbolic acid in combination with dressings.

B. Synthetic Dressings:

1. Films : these are polymer sheets with adhesive coated on one side. Polyurethane , polyethylene, dimethyl aminoethyl methacrylate, polytetra fluoroethylene are commonly used.used in superficial wounds. But causes accumulation of wound fluid, due to impermeability to water vapour and gases, and lack of absorbing capacity. Thus leading to leakage and entry of exogenous bacteria.

2. Foams and sprays: Polymers of polyvinyl alcohol and polyurethane are converted to foam solutions and are used for dressings. They are better than film dressings. They provide thermal insulation and keep the surface moist. They are permeable to gas. They are non adherent also. Silastic foam and lyofoam are examples. Spray dressings are co polymers of certain compounds, eg: hydroxyl vinyl chloride acetate modified maleic acid ester is polyeriesed to form Aeroplast.

3. Composite dressings: This dressing consists of more than one layer. Durabilty and elasticity maintained by outer layer. And inner layer maintains the adherence. They are classified as:

a. Hydrocolloid dressings: These contain mixture of gelling agents and elastomeric adhesive. Commonly used absorptive agent is Carboxymethyl cellulose.

b. Hydrogel sheets:these are hydrophilic polymers made into sheets of 3 dimensional networks.polyethylene oxide, polyacrylamide and polyvinylpyrrolidine are usually used.used in thermal burns because of their cooling ability. Eg: Vigilon.

c. Hydrogel Amorphous :they are similar to hydrogel, but there isn't any crosslinking between the polymers. Collagen or complex carbohydrates are present in small amounts. They give moisture to dry wound eschar and also promotes autolytic debridement

d. Super Absorbents : examples are Combiderm, Convatec.

e. Gels : examples are HEMA, Hydran, Geliperm etc.

Above mentioned dressing are usually act as a temporary covering. In large burns injuries these are combined with alternative wound closure techniques.

C. Biological Dressings:

They are obtained naturally from tissues and are combined with collagen lipid and elastin in various formulations. Their main advantages over synthetic dressings are:

1. Prevent dehydration of wound by restoring a water vapour barrier.
2. Lessen heat loss by evaporation
3. Exudative loss of protein and electrolytes are reduced.
4. Contamination of wound by organism are prevented.

5. Change of dressings are less painful.

6. Joint mobility is maintained.

7. Wound debridment can be done.

8. Autografting made easy by creating good granulation.

9. Reduce the healing time and

10. Healing quality is improved and contraction of tissues are decreased.

Other Biological dressings which are used are allografts,embryonic membranes, skin of foetus/neonate, fibrin, grafts from cultured epidermis/dermal matrix, bovine collagen is reconstituted to films. Herterografts from pigs and dogs are also used.

Cost consideration:

In this era of globalisation, the cost of treatment of chronic conditions has a major role to play and chronic wound management and its financial impacts are very important for a surgeon. The cost effectiveness of topical sucralfate :

o Lesser time required for wound to heal or granulate

o Better response to defenitive treatment modalities like graftings, flaps etc. After removal of topical therapy

o Low cost of the sucralfate, compared to conventional dressing material.

Thus topical sucralfate moist dressing is found to be an efficient and cost - effective modality for treatment of diabetic ulcers.

ABOUT THE DRESSING MATERIAL USED IN THIS STUDY:

TOPICAL SUCRALFATE DRESSINGS:

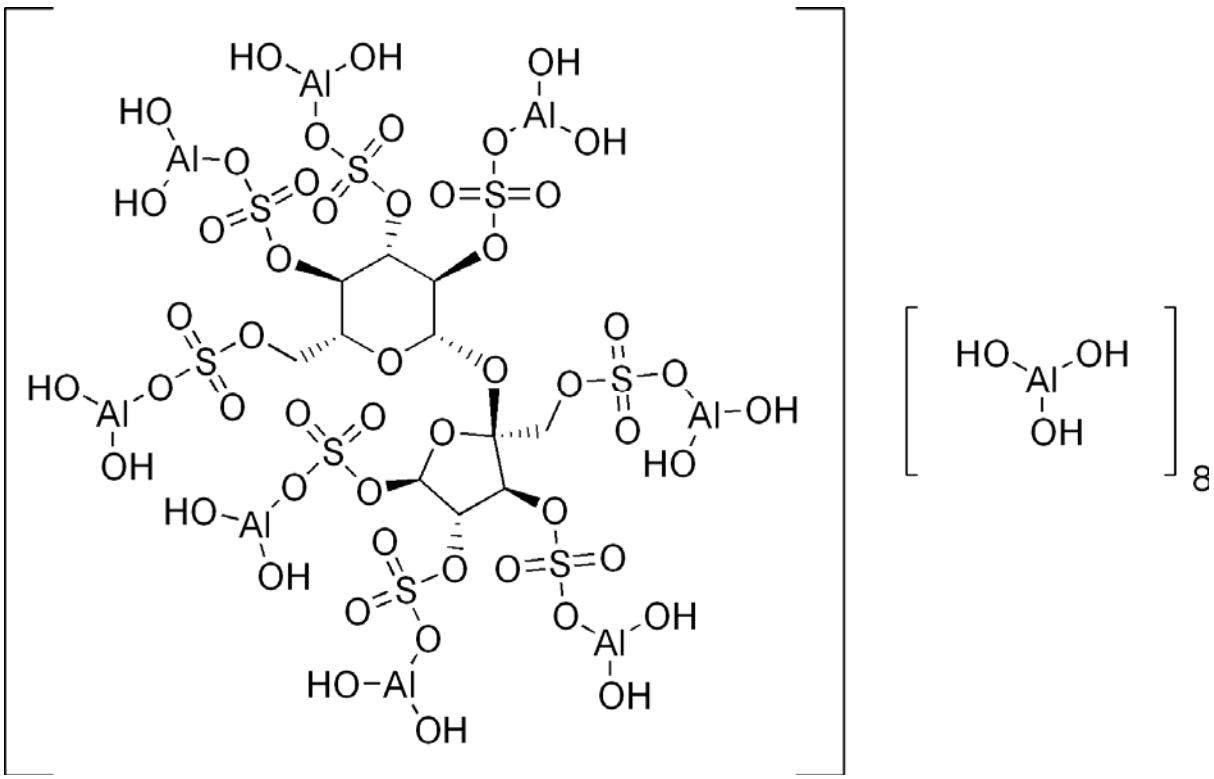
Sucralfate is an oral gastrointestinal medication primarily indicated for the treatment of active duodenal ulcers. Sucralfate is also used for the treatment of gastroesophageal reflux disease (GERD) and stress ulcers.

Unlike the other classes of medications used for treatment of peptic ulcers, sucralfate is a sucrose sulfate-aluminium complex that binds to the hydrochloric acid in the stomach and acts like an acid buffer with cytoprotective properties. Sucralfate was approved by the U.S. Food and Drug Administration (FDA) in 1981.

CHEMICAL DATA:

Formula: $C_{12}H_{54}Al_{16}O_{75}S_8$

Mol. mass: 2086.75 g/mol



SYSTEMATIC NAME: Hexadeca-μ-hydroxytetracosahydroxy[μ8-[1,3,4,6-tetra-*O*sulfo- β-Dfructofuranosyl-α-D-glucopyranoside tetrakis(hydrogen sulfato)8-)]]

hexadecaaluminum

Sucralfate has been investigated as a treatment of small wounds like scratches, cracks, burns, uninfected wounds and also in large wounds like decubitus ulcer, diabetic ulcers and other ulcers..

Sucralfate covers the damaged area and act as a barrier of protection.

It is known that in case of stomach and duodenal ulcers, sucralfate tablets and solutions are used for many decades. They form a protective covering over the damaged tissue.

In case of venous ulcers sucralfate help in wound healing by stimulating EGF expression and other factors which helps in tissue repairing and angiogenesis.

Mechanism Of Action:

The action of sucralfate can now be defined by the '1 x 1 x 1' mechanism of

action—1) acute prevention 2)healing of chronic ulcers 3) both for chronic and acute protection.

The important acute actions of sucralfate are, they maintains the flow of blood to the tissues and integrity of the vasculature, there by leading to quick repairing of superficial defects.

Sucralfate stimulates angiogenesis, epithelisation and formation of granulation tissue by binding to FGF and EGF.

Sucralfate wound healing mechanisms may include:

- Stimulation of fibroblast proliferation.
- Enhancing the formation of granulation tissue.
- Decreasing collagenase activity, inhibition of glucocorticoid activity
- Direct or indirect antibacterial activity by affecting inflammatorycells
- Neovascularization.

The in-vitro activity of sucralfate (sucrose octa-sulphate) in suspension was examined against 128 strains of Gram-negative bacilli. Inhibitory activity was demonstrated against all isolates and bactericidal activity was demonstrated for 68.

Sucralfate has inhibitory and bactericidal antibacterial activity which may contribute to its in-vivo clinical efficacy

CLINICAL EXPERIENCE:

STUDY-I

INTERNATIONAL JOURNAL OF MOLECULAR MEDICINE 22: 17-23, 2008 17 TOPICAL TREATMENT OF CHRONIC VENOUS ULCERS WITH SUCRALFATE: A PLACEBO CONTROLLED RANDOMIZED STUDY GIOVANNI TUMINO1, LAURA MASUELLI2, ROBERTO BEI3 LUCILLA SIMONELLI2, ALBERTO SANTORO1 AND SILVANA FRANCIPANE4 DEPARTMENTS OF 1SURGICAL SCIENCES, 2EXPERIMENTAL MEDICINE, UNIVERSITY OF ROME

This ultrastructural anaylitical study showed that topical use of SUC-LIS 95can improve the healing the chronic venous ulcers by promoting angiogenisis and re-epithelisation and also by reducing the inflammatory reaction. This also helps increasing the wound contraction.

STUDY-II

TOPICAL USE OF SUCRALFATE CREAM IN SECOND AND THIRD DEGREEBURNS Anjana Banatia, Siti Roy Chowdhurya, Saswati Mazumderb Accepted 6 November 2000.

Abstract

This clinical study showed that in second degree burns, sucralfate cream causes quick epithelisation of the wound area. And also showed minimal side effects in treating burns cases.

STUDY-III

ARCHIVES OF DERMATOLOGY THE USE OF SUCRALFATE SUSPENSION IN THE TREATMENT OF ORAL AND GENITAL ULCERATION OF BEHÇET DISEASE A RANDOMIZED, PLACEBO-CONTROLLED, DOUBLE-BLIND STUDY

Erkan Alpsoy, MD; Hanife Er, MD; Cicek Durusoy, MD; Ertan Yilmaz, MD

Arch Dermatol. 1999;135:529-532.

This study showed that in case of treatment of oral and genital ulcers in bechets disease, there is major decrease in pain caused by the ulcer and also time required for healing while comparing the same with the pre-treatment period.

The results proves that while using sucralfate topical solution in treatment of bechets disease, there is effective improvement in oral and genital ulcers. It is a safe, easy inexpensive treatment in bechets disease.

STUDY-IV

ANTIBACTERIAL ACTIVITY OF SUCRALFATE AGAINST ESCHERICHIA COLI, STAPHYLOCOCCUS AUREUS AND PSEUDOMONAS AERUGINOSA IN BATCH AND CONTINUOUS CULTURE. PUBLICATION: EUR J CLIN MICROBIOL INFECT DIS VOLUME: 12(11), PAGE NUMBERS: 869-71,YEAR PUBLISHED: 1993

ABSTRACT:

The antibacterial effect of varying concentrations of sucralfate was studied on Escherichia coli, Pseudomonas aeruginosa and Staphylococcus aureus grown

in both agitated batch and continuous culture. The minimum inhibitory concentrations estimated with the two methods were in close agreement, ranging from 5 to 15 mg sucralfate/ml, concentrations easily attainable in gastric juice after a standard adult dose. Batch culture results indicated a dose-dependent effect. This study provides further evidence for an antibacterial effect by sucralfate against a range of species associated with respiratory tract infection in ventilated patients

SIDE EFFECTS:

The most commonly reported side effect of sucralfate is constipation. Some of the other possible reactions include nausea, indigestion, and bloating . Although most problems are minor and are generally easy to treat, serious side effects may also occur.

Materials and Methods

METHODOLOGY

Study design **: Prospective** Randomized comparative trial

Source of Data **:**Patients with long standing diabetic ulcers (>2 weeks) admitted in surgery wards at Tirunelveli medical college hospital, Tirunelveli over a period from Jaunuary 2014 to July 2015

Sample Size:

100 patients

50-patients –study group

50-patients –control group

Inclusion criteria

1. Patients between 12 to 75 years of age.
2. Duration of the diabetic ulcer more than 2 weeks.
3. Size of ulcer less than 15 x 15 cm
4. Patients giving consent for topical sucralfate therapy

Exclusion criteria:

1. Pulse less limb
2. Immunocompromised patients
3. Associated septicaemia and osteomyelitis.
4. Skin malignancies
5. Diabetic Ketoacidosis.
6. Exposed bones, tendon
7. Charcot joint

Method

The present study was carried out in Tirunelveli medical college hospital, Tirunelveli for a period of one and a half year, where 100 patients with diabetic foot ulcer more than 2 weeks participated in the present study. Using a pretested and pre designed proforma the study population was randomized into either study group or control group using a computerized randomization chart. Out of 100, patients, 50 took treatment in the form of conventional normal saline dressings and 50 took treatment with sucralfate dressing. Off-loading of pressure from the affected area and no antibiotics were used in both the groups. Photographs of the ulcers before and after the dressings were taken, along with culture and sensitivity of the ulcers before and after the dressings. After undergoing a detailed clinical examination, and relevant investigations, the initial wound area was recorded after sharp debridement by Measuring length x width (ulcer should be less than 15 x 15 cm). Both the groups were subjected to once daily dressings. The patients were followed up on a daily basis for a period of 3 weeks in both the groups.

The outcome that is the area of the target ulcer was measured by Plannimetry using a transparent graph sheet .Results were calculated by using student‘t’ test.

DRESSING TECHNIQUE:

After allotting the dressing with the help of Random number table

For conventional dressing:

The ulcer was cleaned with normal saline and saline soaked gauze piece was kept over the ulcer which was covered with pad and roller bandage.

For topical sucralfate dressing:

The ulcer was cleaned with Normal Saline. A single one gram sucralfate tablet was crushed and powdered and placed in Soil of sterile normal saline to form a suspension. Sterile gauze was soaked in the suspension and placed over the wound at 20mg/cm2 TBSA.

At the end of 21 days the wounds in both the groups were inspected and the wounds were compared based on the following parameters. They are:

- Rate of granulation tissue formation as percentage of ulcer surface area
- Quality of ulcer bed
- Present dimensions and surface area of the ulcer

The dressings were changed everyday morning in both control and study groups for 21 days and appearance of healthy granulation tissue is observed and the final area is measured on 21st day by planimetry using a transparent graph sheet and subjected to statistical analysis.

Observations and Results

RESULTS

TABLE NO. 4.1 : AGE DISTRIBUTION

Age	Mean	S.D
CONTROL	62.28	6.94
STUDY	58.88	10.18

The mean age in the study group was 58.88 years and in the control group was 62.28 years

TABLE 4.2 :SEX DISTRIBUTION

Gender	Male	Female
CONTROL	31	19
STUDY	26	24

Incidence of diabetic ulcers were more in males (57.00%) as compared to females (43.00%).

TABLE 4.3 : FBS

	Area of Reduction	
	Correlation coefficient	P value
FBS	0.15	0.136

Pearson chi square test

Mean FBS in the control group was 137.06mg/dl and that in the study group was 156.96mg/dl

TABLE4.4: ONSET

ONSET	S	T
CONTROL	40	10
STUDY	33	17

In this study, 27.00% of the ulcers were traumatic in origin. 73.00% were spontaneous in origin

AREA OF REDUCTION

Onset	CONTROL		STUDY			
	Mean	S.D	Mean	S.D	P value	Independent Sample 't' test
S	19.08	14.75	41.93	8.35	<0.0001	
T	15.54	5.35	42.05	5.33	<0.0001	

TABLE NO 4.5: SITE

Site	D	P	MM	LM
CONTROL	16	26	5	3
STUDY	14	25	8	3

30.00% of the patients had ulcer on the dorsal surface of the forefoot and 13.00% had ulcers on the Medial malleoli. About 51.00% on the plantar aspect, and about 6.00% the lateral malleoli.

Area of Reduction	CONTROL		STUDY			
	Mean	S.D	Mean	S.D	P value	Independent Sample 't' test
D	18.99	15.44	43.62	9.49	<0.0001	
P	19.95	13.77	40.75	5.63	<0.0001	
MM	10.92	2.32	42.71	9.62	<0.0001	
LM	13.81	6.75	42.5	3.63	0.003	

TABLE NO 4.6: CULTURE & SENSITIVITY –AFTER

	CONTROL	STUDY
CULTURE **+ve**	49	4
CULTURE **-ve**	1	46

–ve culture in 46 patients in the study group, whereas 49 patients in the control group still had a +ve culture.

TABLE NO 4.7 : AREA OF REDUCTION

Area of Reduction	Mean	S.D	P value	Independent Sample 't' test
CONTROL	18.37	13.43		
STUDY	41.97	7.41	<0.0001	

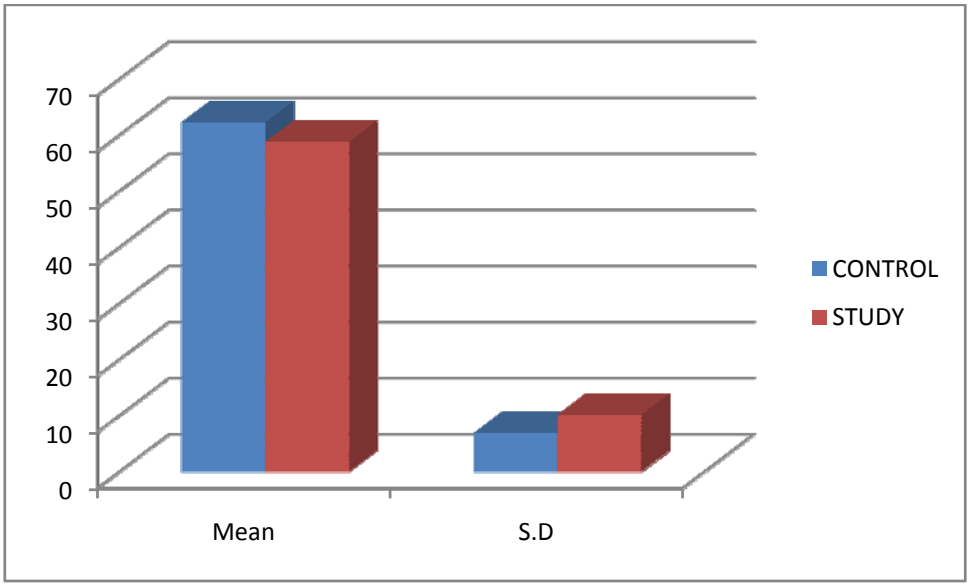
Study group had better area of reduction of 41.97% (S.D : 7.41) as compared to the control group, the mean area of reduction was 18.37%(S.D ; 13.43). These were found to be statistically significant on independent sample T test ($p<0.0001$)

TABLE NO 4.8: WEEKS FOR RECOVERY

Weeks	Mean	S.D	P value	Independent Sample 't' test
CONTROL	5.36	0.59		
STUDY	2.68	0.47	<0.0001	

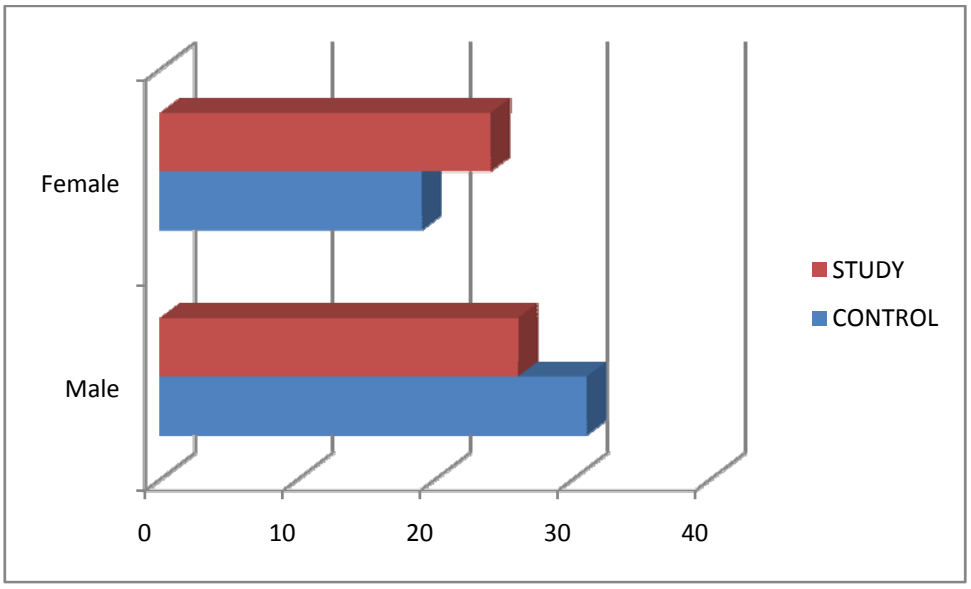
The mean time taken for complete healing of the ulcers were 2.68 weeks in the study group as compared to 5.36 weeks in the control group

GRAPH NO.1 AGE DISTRIBUTION



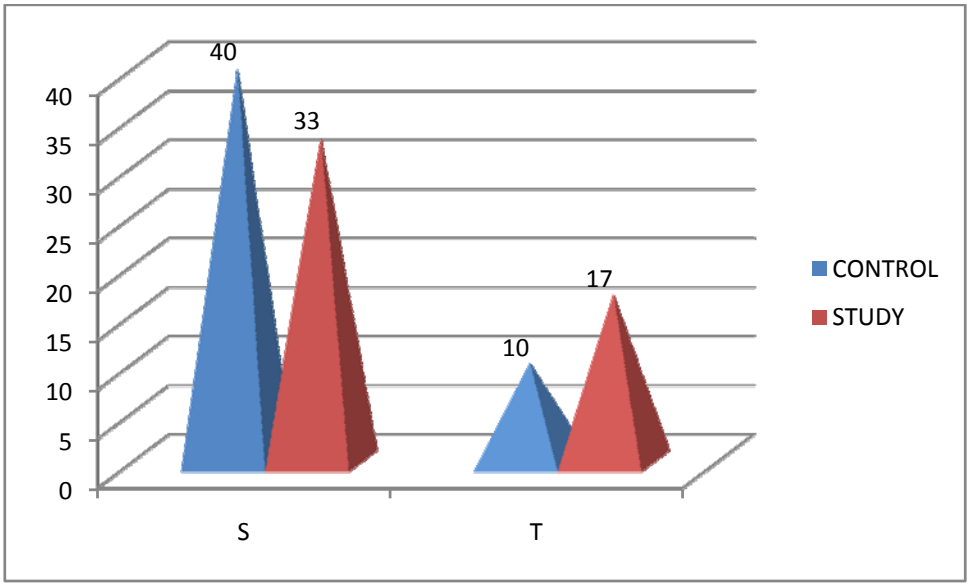
The mean age in the study group was 58.88 years and in the control group was 62.28 years

GRAPH NO. 2 : SEX DISTRIBUTION

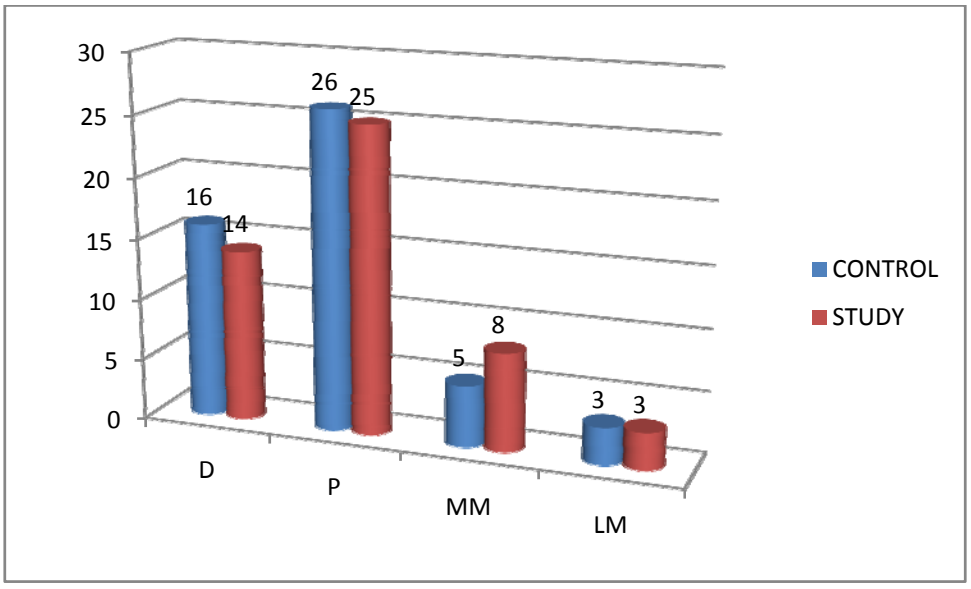


Incidence of diabetic ulcers were more in males (57.00%) as compared to females (43.00%).

GRAPH NO. 3: ONSET

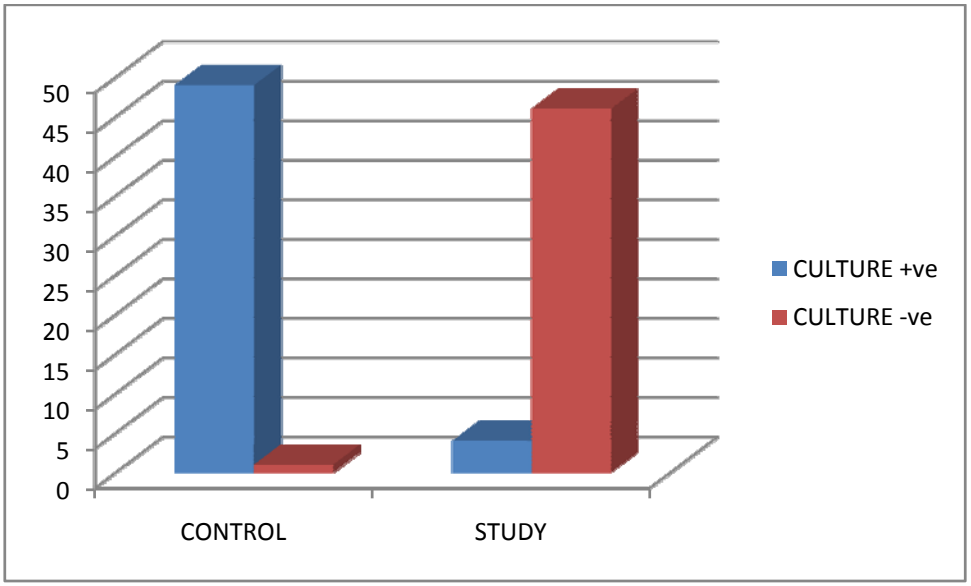


GRAPH NO.4: SITE



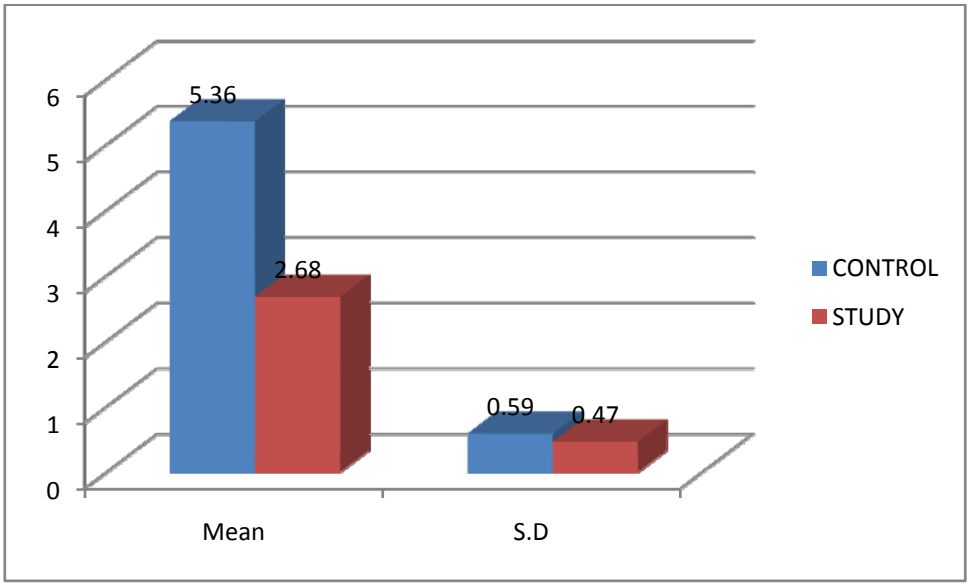
30.00% of the patients had ulcer on the dorsal surface of the forefoot and 13.00% had ulcers on the Medial malleoli. About 51.00% on the plantar aspect, and about 6.00% the lateral malleoli.

GRAPH NO. 5 : CULTURE AND SENSITIVITY – AFTER



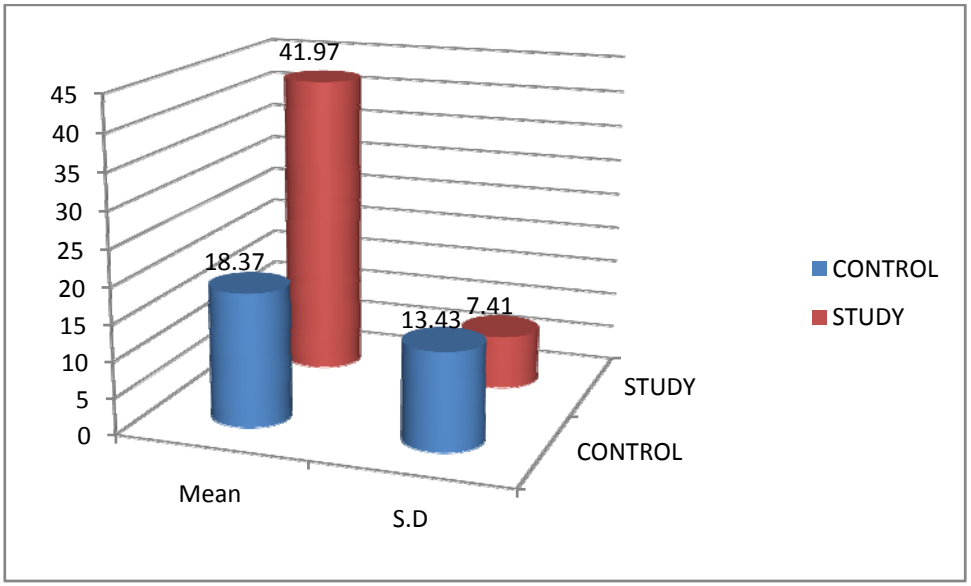
–ve culture in 46 patients in the study group, whereas 49 patients in the control group still had a +ve culture.

GRAPH NO.6 : WEEKS FOR RECOVERY



The mean time taken for complete healing of the ulcers were 2.68 weeks in the study group as compared to 5.36 weeks in the control group

GRAPH NO. 7 AREA OF REDUCTION



Study group had better area of reduction of 41.97% (S.D : 7.41) as compared to the control group, the mean area of reduction was 18.37%(S.D ; 13.43). These were found to be statistically significant on independent sample T test ($p<0.0001$)

STATISTICAL ANALYSIS

Statistical analysis was done by using Microsoft EXCEL software and SPSS computer program.

Diabetic ulcers in the study group had better mean % reduction of area 41.97% (S.D : 7.41) as compared to the control group which had mean % reduction of area was 18.37%(S.D ; 13.43)the difference in the mean 23.6% of reductionof area of the two groups where studied using independent sample T test was found to be significant ($p<0.0001$).

Discussion

DISCUSSION

An ideal dressing is every surgeon's desire, a dressing that promotes chronic ulcer healing without any complications. Successful wound dressing should keep the wound moist and be devoid of any adverse reactions such as infection, maceration and allergy.

Diabetic ulcers are chronic wounds, stuck in inflammation phase and shows cessation of epidermal growth.

The present study was conducted at Tirunelveli medical college hospital, Tirunelveli to study the effect on diabetic ulcer healing dynamics

In the present study it was seen that the incidence of diabetic ulcers were more in males (57.00%) as compared to females (43.00%).

The second national data source, NHDS documented higher hospital rates in males suffering from diabetic ulcers.

The mean age group in the study group with diabetic ulcers were 59 years and in the control group were 62 years.

In this study, 27.00% of the ulcers were traumatic in origin, trauma being the triggering factor secondary to neuropathy. 73.00% were spontaneous in origin secondary to blister rupture or unnoticed trivial trauma.

About 30.00% of the patients had ulcer on the dorsal surface of the forefoot and 13.00% had ulcers on the Medial malleoli. About half (51.00%) on the plantar aspect, and about 6.00% on the lateral malleoli.

Study conducted by Edmonds et al in 1986, (Edmonds) showed more foot ulcers were on plantar and fore foot areas. Most of the diabetic foot ulcers are invariably shoe related and due to gait abnormalities. They can be prevented by appropriate sized footwear. However in our study the incidence of ulcers over the plantar aspect of the foot were not as high as postulated by Edmonds et al.

In our study the culture and sensitivity of the ulcers before the commencement of sucralfate dressings were positive for many microorganisms. In the study group 18 were positive for SA, 7 patients for PM, 6 for PA, 1 patient showed EC.18 of them did not show any growth. In the control group 17 of them were positive for SA. 12 of them for PM. 4 for PA, 6 for KP and 2 of them for EC. 9 of the patients did not show any growth

After sucralfate dressings were given culture obtained on the 21st day surprisingly showed –ve culture in 46 patients in the study group, whereas 49 patients in the control group still had a +ve culture. This may account for the antimicrobial activity of Sucralfate.

In our study it was observed that participants receiving Sucralfate dressing had better area of reduction of 40.87% (S.D: 5.98) as compared to the control group receiving only conventional dressing (normal saline dressing) in whom the mean area of reduction was 15.62%(S.D ; 8.51).These were found to be statistically significant on independent sample T test ($p<0.0001$)suggesting that Sucralfate enhances wound healing in diabetic ulcers.

Also in the sucralfate group the mean time taken for complete healing of the ulcers were 2.68 weeks as compared to 5.36 weeks in the control group

Feasibility of this study:

In the present study we have taken 100 patients suffering from diabetic footulcers (>2 weeks). Patients were taken up for study based on inclusion and exclusion criteria. Out of 100 patients, 50 (26 males, 24 females) were study cases and 50 (31males and 19 females) were control. Participants included in the study group were treated with Sucralfate dressing from day 01 to day 21. All 50 patients selected for Sucralfate treatment complied for the 21 days period of the study. The initial area measurement was taken on day 01 and final area measurement on day 21 was taken on transparent sheet.

All 50 patients selected as a control complied for the 21 days duration period of the study. The initial area measurement on day 01 final area measurement on day 21 was taken on transparent sheet. The area measurement was done using planimetry.

We have applied the following formula to calculate % reduction in area of wound after 21 days period in both cases and control groups.

Rate of contraction of wound after 21 days of treatment =

$$\frac{(\text{Initial area} - \text{Final Area}) \quad X \quad 100}{\text{Initial area}}$$

We have found 18.37(S.D; 13.43) contraction of wounds in the control groups as compared to 41.97% (S.D:7.41) contraction of wounds in study group. Therefore, study groups have a better percentage of wound contraction as compared to the control group. On applying independent sample T test $p<0.0001$ which is significant.

From our study, we can say that Sucralfate dressing therapy facilitates wound healing in patients suffering from diabetic ulcers.

Limitations of our study:

Sample size is the main limitation in our study. For statistical analysis a sample of 100 induviduals is enough, but for further substantiation of findings and revealation of variations which weren't noticed in the present study, we may need to do a randomised controlled comparative study with a much larger population.

Various factors other than cost of dressings can influence the cost burden over the patient, so it also not analysed. Sucralfate dressing wasfound to be less expensive and easily available when compared to conventional moist dressings.

Summary

SUMMARY

The incidences of DIABETIC ulcers are on the rise. 15% of all diabetics develop diabetic ulcers, the most common site being the sole of foot. Diabetes has highest risk factor associated with limb threatening ischemia. Trivial trauma secondary to neuropathy and distorted pedal architecture causes ulcerations. 15% of all diabetics develop foot ulcer. 20% of admissions in diabetics are for foot problems.

Various modalities of treatment have been developed to aid faster healing of diabetic ulcers. Course of healing in diabetic ulcer patients is unpredictable and sometimes resistant to treatment.

100 patients of diabetic ulcers were studied. They were divided into two groups of 50 each.

One group received Sucralfate dressing and the control group received treatment in the form of conventional therapy. A comparative study was done between both groups regarding percentage area wound reduction.

The mean age group in the study group with diabetic ulcers was 59 years and in the control group were 62 years. Males were more affected than females. 57.00% males Vs 43.00% females. 73.00% were spontaneous in origin caused mainly due to blister rupture or unnoticed trivial trauma. About half (51.00%) of the patients had ulceron the plantar surface of the forefoot.

All patients in the study underwent X-ray of the affected foot, patients with stress fractures and osteomyelitis were excluded.

In our study it was observed that participants receiving Sucralfate dressing had better area of reduction of 41.97% (S.D : 7.41) as compared to the control group receiving only conventional dressing (normal saline dressing) in whom the mean area of reduction was 18.37(S.D ; 13.43).These were found to be statistically significant on independent sample T test ($p<0.0001$) suggesting that Sucralfate enhances wound healing in diabetic ulcers.

In our study the culture and sensitivity of the ulcers before the commencement of sucralfate dressings were positive for many microorganisms. IN the study group 18 were positive for SA,7 patients for PM, 6for PA, 1 patient showed EC. 18 of them did not show any growth. In the control group 17 of them were positive for SA. 12 of them for PM. 4 for PA, 6 for KPand 2 of them for EC. 9 of the patients did not showany growth

After sucralfate dressings were given culture obtained on the 21st day surprisingly showed –ve culture in 46 patients in the study group, whereas 49 patients in the control group still had a +ve culture.

This may account for the antimicrobial activity of Sucralfate. Also in the sucralfate group the mean time taken for complete healing of the ulcers were 2.68 weeks as compared to 5.36 weeks in the control group.

Increased rate of granulation tissue formation was seen in topical Sucralfate dressing group when compared to conventional dressing group.

Considerable effect on bacterial load was seen with the topical Sucralfate compared to conventional dressing group.

Shorter duration of hospital stay was observed in the topical Sucralfate dressing group.

Topical Sucralfate dressing appears to be the main therapeutic agent in wound healing which is effective, inexpensive and easily available

Thus, Sucralfate dressing therapy in the treatment of DIABETIC ulcers was found to be more effective, safe, promoter of wound healing, and hence can be recommended for the treatment of DIABETIC ulcers as an adjuvant to the conventional mode of treatment, reducing the progression of the pathology and hence the amputation.

Conclusion

CONCLUSION

The wounds in subjects treated with Sucralfate dressing contracted more than the wounds in the control group (41.97% Vs 18.37% ; P = < 0.0001→Significant) which indicates Sucralfate dressing is an effective modality to **FACILITATE** area of reduction of wound in patients suffering from DIABETIC footulcers and can be used as an adjunct to conventional mode of treatment (conventional dressings and debridement) for faster and better healing of DIABETIC ulcers.

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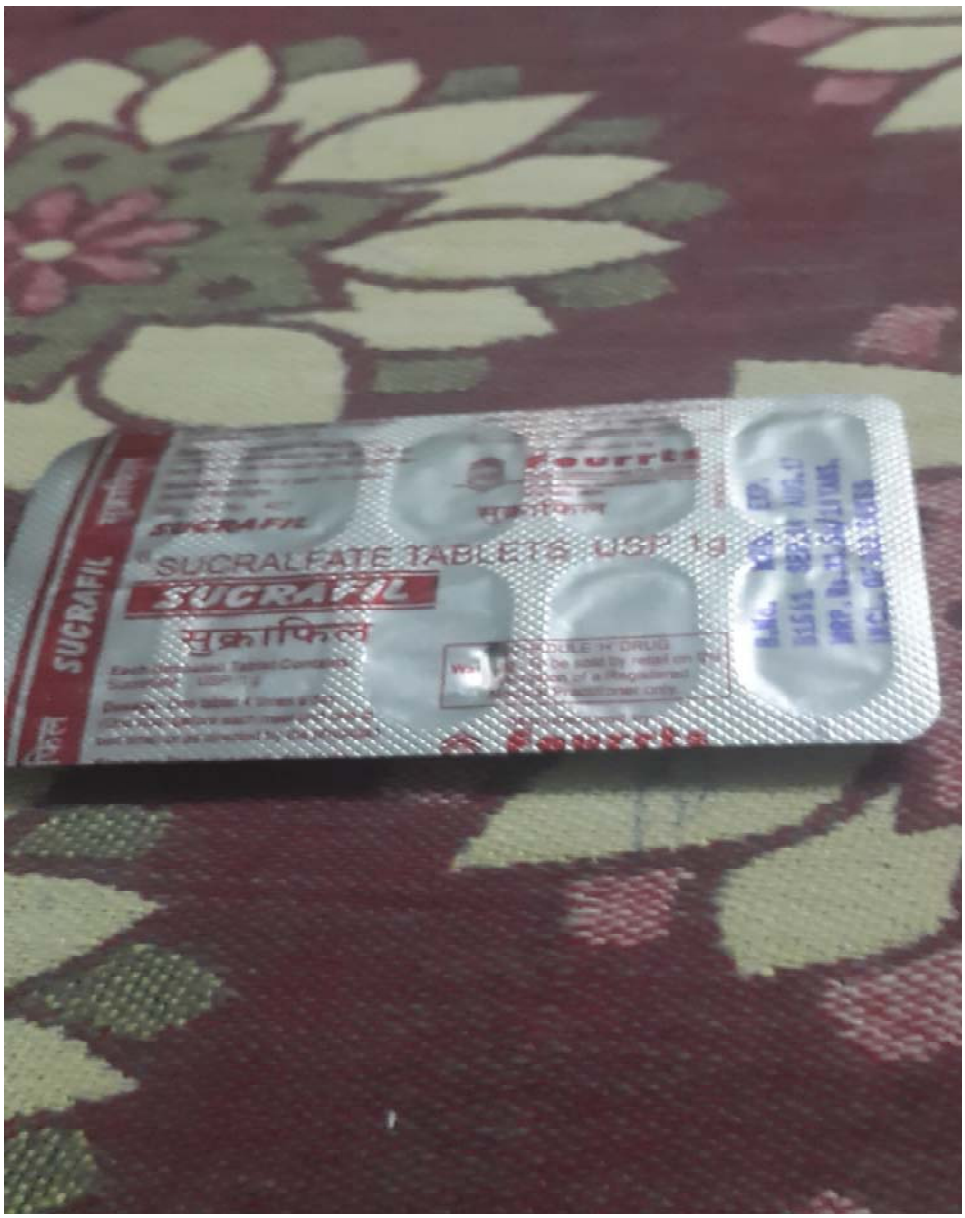
Annexures

ANNEXURE-I



SUCRALFATE TABLET 1gm

BEFORE

AFTER

BEFORE

AFTER

BEFORE

AFTER

BEFORE

AFTER

ANNEXURE-II

RESEARCH PARTICIPANT INFORMATION AND CONSENT FORM

Mr. /Miss/ Mrs. _____

You are invited to participate in our research study that is **"A CLINICAL STUDY OF COMPARISION BETWEEN EFFICACY OF TOPICAL SUCRALFATE AND CONVENTIONAL DRESSING IN THE MANAGMENT OF DIABETIC ULCER"**

Since you are suffering from Diabetic ulcer, which is not healing since a long time and will be requiring treatment for the same, you are eligible to be part of the study and hence asked to participate. This research is about the beneficial effects of Sucralfate dressing therapy on your foot ulcer and the result of this research will help in a better treatment of similar participants in the future.

If you agree to be a part of this research, we would ask you some relevant clinical history. You are free to not to answer to whichever question u think are not relevant. Aclinical examination will be done and then you will be treated with either Sucralfate dressing therapy or the normal saline dressing for 21 days. On the first day the area of the ulcer will be measured and this will be repeated at the end of the therapy that is21stday. Also culture and sensitivity, and photographs of the ulcers before and after the dressings will be taken. There are chances you may have a speedy and better recovery with this therapy and it will also help in the treatment of participants with similar complaints in the future.

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

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

This study does not have any damaging aspect and there are no chances of injury during the course of the study, but if injured the investigator is not responsible. There will be extra cost incurred by you. However you will have to pay for the routine investigations, which are a part of the existing management protocol for the treatment of ulcers, and diabetes. There is no commitment for any reimbursement or any compensation for the participant. The participation in this study is entirely voluntary and you may withdraw from the study at any time. At any time during or after the study, for any information you may contact the researcher.

Dr.John Amalan.A
P.G hostel, Room no: 19
Tirunelveli medical college hospital
High ground, Tirunelveli, Tamilnadu.

Signature of the participant or legally authorized representative:
Place : _____
Participant's name _____
Signature :_____
Experimenter/witness's Name :_____
Signature : _____Date **:**_____

ANNEXURE-III

PROFORMA

I) PATIENT IDENTIFICATION DATA :

NAME IP/OPD NO.

AGE DOA :

SEX DOD:

OCCUPATION

ADDRESS

II) CHIEF COMPLAINTS :

MEDICAL HISTORY :

Peripheral Neuropathy : ( )

Nephropathy ( )

Retinopathy ( )

PVD ( )

CVD ( )

DIABETIC STATUS :

TYPE :

DURATION :

MEDICATION : Oral Hypoglycemics Insulin

( ) ( )

COMPLICATION Neuropathy ( )Vasculopathy ( )

ULCER DETAIL :

1. **Mode of onset**

Traumatic ( )

Spontaneous ( )

Pressure ( )

Others ( )

2. Duration

3. Progress

WOUND OBSERVATION:

1. Site

2. Size

3. Shape

4. Edge

5. Margin

6. Floor

7. Base

8. Discharge

9. Surrounding Skin

10. Contractor

NERUROLOGICAL EXAMINATION:

VASCULAR EXAMINATION	Left	Right
Popliteal a.	( )	( )
Ant . Tibial	( )	( )

Post Tibial ( ) ( )

Dorsalis Pedis ( ) ( )

ANY FOOT DEFORMITY PRESENT:

Toe deformity

Bunion

Charcots joint

Foot drop

IF AMPUTATION HAS BEEN DONE

SPECIFY : Date

: Side

: Level

: Cause for amputation

FOOT WEAR ASSESSMENT:

Does patient wear appropriate shoes

Does patient require contact cast immobilization.

INVESTIGATIONS.

CBC

FBS 1st _____ Date :_____ Time :_____

2nd (24 hr apart) _____ Date :_____ Time : _____

Sr. Creatinine

UKB

Urine : Routine

Microscopy

X-ray Foot

AP View

Lat. View

Wound C/s

WOUND AREA MEASUREMENT ON D1 in mm2

Type of Dressing – saline dressing ( )

- Sucralfate dressing ( )

Key for using the master sheet:

Sl.no	:	Serial number
M	:	Male
F	:	Female
FBS	:	Fasting Blood Sugar
C/s (A)	:	Culture sensitivity report After sucralfate dressing
C/s (B)	:	Culture sensitivity report Before sucralfate dressing
mm2	:	millimetre square
T	:	Traumatic
S	:	Spontaneous
D	:	Dorsal
P	:	Plantar
MM	:	Medial Malleoli
LM	:	Lateral malleoli
NOGC	:	No Organisms Grown in Culture
SA	:	Staphylococcus Aureus
KP	:	Klebsiella Pneumonia
PM	:	Proteus Mirabilis
PA	:	Pseudomonas Aeruginosa
EC	:	Eischericia Coli

MASTER CHART

Control Group

SL NO	NAME	AGE	SEX	ONSET	SITE	FBS	XRAY	C/S before	C/S after	INITIAL AREA in mm2	FINAL AREA in mm2	IA-FA = CA (controlled area) mm2	AREA OF REDUCTION in %	WEEKS FOR COMPLETE HEALING
1	ANDI	60	M	S	P	104	N	PM	POS	3187.09	2780.73	406.36	12.75	4
2	SOOSAIYAMMAL	63	F	S	P	109	N	SA	POS	1925.77	1561.63	364.14	18.9	5
3	KOVILPILLAI	48	M	S	P	206	N	SA	POS	1948.87	1662.90	285.96	14.6	5
4	MAHAKALI	82	M	S	P	302	N	NOGC	NEG	3117.32	1766.06	1551.33	46.7	5
5	KUMARAPPAN	78	M	T	P	108	N	NOGC	NEG	3536.87	2991.73	545.14	15.4	5
6	SUBBAIAH	64	M	T	P	124	N	NOGC	NEG	3147.49	2283.48	864.01	27.4	5
7	SHAHUL HAMEED	63	M	S	P	126	N	PM	POS	3428.93	2887.52	541.40	15.7	6
8	LAKSHMI	78	F	S	D	132	N	PA	POS	3473.83	2961.90	511.92	14.7	6
9	AGNIMUTHU	64	M	S	D	108	N	PA	POS	2763.83	2402.70	361.12	13.06	6
10	MADASAMY	55	M	S	MM	204	N	SA	POS	2265.74	1939.14	326.60	14.4	5
11	KALIMUTHU	58	M	S	LL	111	N	PM	POS	3456.65	3132.76	332.88	9.63	6
12	RAMALAKSHMI	62	F	S	LL	132	N	NOGC	POS	3226.56	2895.51	331.04	10.2	6
13	RAJENDRAN	66	M	S	MM	104	N	NOGC	POS	2436.79	2163.10	273.69	11.2	5
14	CHELLADURAI	62	M	S	D	96	N	NOGC	NEG	2663.75	2228.39	435.36	16.3	4
15	GANESH	63	M	T	P	98	N	NOGC	NEG	2893.00	2619.32	273.67	9.4	5
16	KANAGAVALLI	59	F	T	P	93	N	PM	POS	2693.85	2402.70	291.14	10.8	6
17	THANGALAKSHMI	62	F	T	P	88	N	SA	POS	3365.95	2875.51	490.43	14.57	6
18	RAJAKANI	66	F	S	P	116	N	SA	POS	3564.98	3217.03	347.94	9.76	5
19	SHEEBAMUTHAMA	60	F	S	P	128	N	SA	POS	1344.99	1153.01	191.98	14.2	5
20	BHASKAR	68	M	S	P	122	N	SA	POS	2535.55	2271.88	263.67	10.3	4
21	ISAKKI	80	M	S	P	204	N	SA	POS	3547.20	3078.67	468.52	13.2	6
22	MUTHU	80	M	S	P	222	N	EC	POS	2673.99	2360.06	313.90	11.7	5
23	ARUMUGHAN	54	M	S	D	168	N	PM	POS	3134.45	2845.14	289.30	9.23	6

SL NO	NAME	AGE	SEX	ONSET	SITE	FBS	XRAY	C/S before	C/S after	INITIAL AREA in mm2	FINAL AREA in mm2	IA-FA = CA (controlled area) mm2	AREA OF REDUCTION in %	WEEKS FOR COMPLETE HEALING
24	THAIYAMMAL	68	F	T	D	108	N	PM	POS	1425.78	1201.91	223.80	15.7	5
25	PITCHAIMUTHU	60	M	S	D	137	N	PM	POS	2918.10	2590.72	327.29	11.2	6
26	MALATHI	58	F	S	P	126	N	KP	POS	2973.09	2281.36	691.73	23.26	6
27	ANDINACHI	54	F	S	D	131	N	SA	POS	1783.06	683.04	1100.02	61.69	6
28	PALANIGURU	62	M	S	P	144	N	KP	POS	1436.08	736.04	700.04	49.08	6
29	VELLAIYAPPAN	70	M	S	P	156	N	PM	POS	1536.21	526.02	1010.01	65.74	6
30	SOMASUNDARAM	49	M	S	MM	142	N	PM	POS	3426.77	3086.11	340.66	9.94	6
31	NAGARAJAN	53	M	S	P	160	N	PM	POS	3034.45	2645.15	389.30	12.8	5
32	NATARAJ	56	M	S	D	172	N	SA	POS	2473.43	2261.11	222.00	8.57	6
33	MARIYAMMAL	62	F	S	D	208	N	NOGC	NEG	3427.26	2991.67	435.58	12.7	5
34	ANNAPUSHPAM	66	F	S	D	214	N	EC	POS	2439.11	2071.82	367.28	15.05	5
35	ARUMUGADEVAR	68	M	T	P	164	N	SA	POS	922.09	723.01	199.08	21.5	5
36	VELAYUDHAM	72	M	T	P	206	N	SA	POS	3555.91	3017.11	538.79	15.1	5
37	MARICHAMI	48	M	S	P	168	N	PA	POS	2592.82	2202.72	390.09	15.0	5
38	ISMAIL	70	M	S	P	102	N	KP	POS	2881.99	2419.32	462.66	16.05	6
39	AKHILA	64	F	S	D	134	N	PA	POS	2552.75	2221.49	331.26	12.9	5
40	AMBUJAM	63	F	S	D	138	N	SA	POS	2310.73	2078.66	232.06	10.0	5
41	NELLAIYAPPAN	54	M	S	S	144	N	PM	POS	2236.72	2063.99	172.73	7.7	6
42	PATTIVEERAN	55	M	S	MM	128	N	PM	POS	3026.36	2691.31	335.04	11.07	5
43	SAROJA	59	F	S	MM	110	N	KP	POS	3056.69	2811.71	244.98	8.0	6
44	PALAVESAM	62	F	S	LL	107	N	SA	POS	2199.72	1722.60	477.11	21.6	6
45	CHINNATHAI	53	F	S	P	68	N	SA	POS	2211.83	1902.11	309.71	14.0	5
46	NAFISABEEVI	64	F	T	D	96	N	SA	POS	2999.11	2561.9	437.21	14.5	5
47	SOORIYAN	62	M	T	P	102	N	NOGC	POS	3011.91	2675.52	336.38	11.1	5
48	PETCHIKUTTY	72	F	S	D	88	N	KP	POS	3083.40	2140.11	943.29	30.5	5
49	CHANDRAN	65	M	S	P	95	N	SA	POS	5336.81	3741.73	1595.07	29.8	5
50	ABRAHAM	60	M	S	D	100	N	KP	POS	3015.70	1504.01	1511.69	50.1	6

Study Group

SL NO	NAME	AGE	SEX	ONSET	SITE	FBS (mg/dl)	XRAY	C/S (before)	C/S (after)	INITIAL AREA in mm2	FINAL AREA in mm2	IA-FA = CA (controlled area) mm2	AREA OF REDUCTION in %	WEEKS FOR COMPLETE HEALING
1	palanisamy	50	m	S	D	132	N	NOGC	NEG	1496.05	436.02	1060.03	70.8	2
2	MARIYAMMAL	60	F	S	D	108	N	NOGC	NEG	1386.60	642.40	744.20	53.6	2
3	PARVATHY	55	F	S	P	164	N	SA	NEG	2292.05	1122.89	1169.15	51.0	2
4	MURUGAN	49	M	S	P	134	N	PM	NEG	3181.93	2060.76	1121.17	35.2	2
5	MARIYAPPAN	46	M	T	P	150	N	SA	NEG	2192.00	1322.87	869.12	39.6	3
6	VELU	70	M	S	MM	120	N	NOGC	NEG	1090.55	432.22	658.33	60.3	3
7	SHANMUGAM	65	M	S	D	137	N	NOGC	NEG	2735.65	1656.43	1079.22	39.4	3
8	SANKARAYYAH	55	M	S	D	162	N	SA	NEG	1563.08	942.36	620.71	39.7	3
9	PETCHIAPPAN	62	M	S	MM	155	N	SA	NEG	2536.97	1625.69	911.28	35.9	3
10	MUTHAMMAL	70	F	T	MM	150	N	SA	NEG	1535.73	989.14	546.59	35.5	3
11	NATHAN	48	M	T	D	140	N	SA	NEG	1432.38	881.68	550.70	38.4	3
12	KUMARASAMY	49	M	T	D	160	N	NOGC	NEG	2293.00	1122.82	1170.18	51.0	2
13	MURUGATHAL	55	F	S	D	142	N	PM	NEG	3191.83	2067.93	1123.90	35.2	3
14	SAMY	56	M	S	MM	128	N	NOGC	NEG	2392.00	1422.87	969.12	40.5	3
15	PAPATHI	62	F	T	MM	108	N	NOGC	NEG	1191.66	532.22	659.44	55.3	3
16	YAGAMMAL	48	F	S	P	239	N	PM	NEG	2835.65	1756.43	1079.21	38.05	3
17	MUNIYAPPAN	82	M	S	P	202	N	PM	NEG	1633.08	1042.36	620.71	37.32	3
18	PAPPA	77	F	S	P	196	N	SA	NEG	2537.9	1725.69	812.28	32.0	3
19	MARIMUTHU	60	M	T	LL	132	N	SA	NEG	3233.13	1923.00	1310.13	40.5	3
20	SATYAMOORTHI	48	M	T	MM	204	N	SA	NEG	3563.00	2110.00	1453.00	40.7	3
21	KALIYAMMAL	38	F	S	D	302	N	SA	NEG	3543.93	2293.45	1250.40	35.28	3
22	MUTHUMARI	46	F	S	P	226	N	NOGC	NEG	2369.83	1372.29	997.53	42.09	3
23	CHITRA	44	F	T	P	138	N	PM	NEG	3487.83	2108.77	1379.08	39.54	3
24	CHELAMMAL	49	F	T	P	104	N	PA	NEG	3425.44	1971.57	1453.87	42.4	2
25	MUTHUKANI	60	F	S	D	324	N	EC	NEG	2194.93	1241.01	953.91	43.4	3

SL NO	NAME	AGE	SEX	ONSET	SITE	FBS	XRAY	C/S (before)	C/S (after)	INITIAL AREA in mm2	FINAL AREA in mm2	IA-FA = CA (controlled area) mm2	AREA OF REDUCTION in %	WEEKS FOR COMPLETE HEALING
26	MANI	72	M	S	P	168	N	PA	NEG	2425.62	1393.67	1031.95	42.5	2
27	PALPANDI	86	M	S	P	148	N	SA	NEG	3333.13	2023.00	1310.13	39.3	2
28	BOOPALAN	80	M	S	P	136	N	NOGC	NEG	3663.10	2110.10	1553.0	42.3	3
29	VELDURAI	76	M	S	MM	222	N	NOGC	NEG	3698.93	2373.45	1325.47	35.8	3
30	KANNIYAPPAN	62	M	S	LL	201	N	NOGC	NEG	2399.83	1432.27	967.53	40.3	3
31	KANAGA	40	F	S	D	186	N	PM	NEG	3587.83	2208.74	1379.08	38.4	3
32	DURAICHI	39	F	S	D	232	N	PA	NEG	3429.82	1871.59	1558.22	45.4	3
33	SANTHAPERUMAL	64	M	T	P	106	N	PA	NEG	2174.93	1242.01	932.92	42.8	3
34	ANTONY	49	M	T	P	96	N	PA	NEG	2392.63	1293.67	1098.96	45.9	3
35	SAKUNTHALA	63	F	S	P	108	N	PA	NEG	3546.63	1493.67	2052.96	57.88	2
36	KAMALAM	64	F	S	P	92	N	SA	NEG	2533.94	1598.00	935.94	36.93	3
37	MUTHUVELPANDIYAN	74	M	T	P	106	N	NOGC	NEG	2533.76	1617.93	915.83	36.14	3
38	KARUPAPILLAI	62	M	T	P	108	N	SA	NEG	3316.07	1829.88	1486.18	44.81	3
39	NAGAMMAL	69	F	S	P	138	N	NOGC	NEG	2532.84	1440.97	1091.87	43.1	3
40	SELVIMARY	59	F	S	P	142	N	SA	NEG	3331.93	2101.91	1230.02	36.9	2
41	FATHIMA	60	F	T	P	165	N	NOGC	NEG	2636.84	1692.39	944.44	35.8	2
42	NOORNIZA	62	F	S	P	138	N	NOGC	NEG	1635.73	990.23	645.49	39.4	3
43	SAMYMUTHU	48	M	S	MM	154	N	NOGC	NEG	3231.93	2010.91	1221.02	37.7	2
43	PECHITHAI	64	F	S	LL	172	N	NOGC	NEG	2535.83	1350.19	1185.63	46.7	3
44	KANNIYAMMAL	65	F	T	P	229	N	PM	NEG	3216.07	1739.28	1476.79	45.91	3
45	KESAVAN	61	M	T	D	109	N	SA	NEG	2423.95	1498.09	925.86	38.1	3
46	AMUDA	70	F	T	D	136	N	SA	NEG	2425.62	1393.67	1031.95	42.5	2
47	ULAGATHAL	68	F	S	D	201	N	SA	NEG	3487.83	2108.74	1379.08	39.5	2
48	THIRUPATHI	55	M	S	P	106	N	SA	NEG	2550.02	1550.00	1000.02	39.2	2
49	PALANI	60	M	S	P	92	N	NOGC	NEG	1896.04	1294.04	602.0	32.7	2
50	ARUCHAMI	55	M	S	D	104	N	NOGC	NEG	2290.05	1120.89	1169.15	51.0	3