"EFFICACY OF HYDROCOLLOID DRESSINGS AND CONVENTIONAL WET SALINE DRESSINGS IN THE HEALING OF CHRONIC DIABETIC ULCER OF LOWER LIMB"- A RANDOMIZED CONTROL TRIAL AT KLES DR. PRABHAKAR KORE HOSPITAL AND MRC, BELGAUM

By

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Dissertation submitted to the
KLE University, Belgaum, Karnataka

In Partial Fulfillment
of the requirements for the degree of

MASTER OF SURGERY (M.S.)
IN
GENERAL SURGERY

Under the Guidance of

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MAY - 2010

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I hereby declare that this dissertation entitled "EFFICACY OF HYDROCOLLOID DRESSINGS AND CONVENTIONAL WET SALINE DRESSINGS IN THE HEALING OF CHRONIC DIABETIC ULCER OF LOWER LIMB"- A RANDOMIZED CONTROL TRIAL AT KLES DR. PRABHAKAR KORE HOSPITAL AND MRC, BELGAUM is a bonafide and genuine research work carried out by me under the guidance of Dr. V. M. UPPIN MS Professor, Department of Surgery, Jawaharlal Nehru Medical College, Nehru Nagar, Belgaum – 590010.

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LIST OF ABBREVIATIONS USED

DF	-	Degree of freedom
DM	-	Diabetes mellitus
ECM	-	Extra cellular matrix
EGF	-	Endothelial derived growth factor
ER	-	Endoplasmic reticulum
HCD	-	Hydrocolloid
MM	-	Millimeter
MMP	-	Matrix metalloproteinase
PDGF	-	Platelet derived growth factor
SD	-	Standard deviation

ABSTRACT

Background and objectives

The management of wound and wound dressing is an important aspect of diabetic ulcer management. Choosing an appropriate dressing can be a complex process. Hydrocolloid dressing is an occlusive dressing which can be used in a variety of wounds. The objective of the study was to assess the hydrocolloid dressings in comparison to conventional wet saline dressings in achieving mean percentage wound reduction in patients with diabetic ulcer of lower extremities more than four weeks duration, using transparency sheet.

Methodology

The present randomized control study from January 2008 to December 2008 was conducted at KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belgaum on 68 patients with chronic diabetic ulcers of lower limb divided into two groups. Study group received hydrocolloid dressings for a period of 14 days while control group received conventional normal saline dressings. Wound measurement was taken on day one and fourteen in both the groups. Percentage area of wound reduction was calculated.

Results

There was no statistical difference in the baseline characteristics like age, sex and initial wound area of the ulcer between the two groups. The percentage of area reduction was 33.1±6.30 in patients treated with Hydrocolloid dressings and 17.8±4.28 in patients treated with Normal saline dressings, which is statistically significant ($p<0.001$).

Conclusion

Hydrocolloid dressing can be used for the healing of chronic diabetic ulcer of lower limb.

Key Words

Diabetic ulcers; Hydrocolloid dressing; Normal saline dressing; Wound area;

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INTRODUCTION

The incidence of diabetes mellitus (DM) and its complications are on a rise, the risk of lower extremity amputations is 15 fold higher in diabetics as compared to non-diabetics. Essential to mention here that chronic diabetic foot ulcer is the leading cause of amputations in these patients, also that 15% of all diabetics develop diabetic ulcer and the commonest site being the foot.[1] Although the fundamental pathophysiologic factors leading to diabetic ulcer remain incompletely understood, the triad of neuropathy, ischemia and infections commonly is considered the most important. These diabetic ulcers are known to be resistant to conventional treatment and may herald severe complications if not treated wisely.

The management of wound and wound dressing is an important aspect of diabetic ulcer management, which is neglected many a time. Care of the wound involves management of the ulcer, care of the exudates and knowledge and rational use of myriad dressing materials.

Basic requirements of the ideal ulcer dressing:[2]

- Maintain high humidity between wound and dressing.
- Absorbent, removes excess exudates.
- Non-adherent, allowing easy removal without trauma at dressing change.
- Safe and acceptable to patient (non-allergic).
- Permit gaseous exchange but impermeable to micro-organism.
- Cost-effective.

Choosing an appropriate dressing can be a complex process, as there are numerous dressings currently available to help optimize the local environment for healing, although the ideal dressing does not exist.

In granulating wounds, the topical application of wet saline gauze has traditionally been used and acknowledged as a standard of care. But, the use of saline soaked gauze dressing as packs often causes patient discomfort as these expand into a hard mass on absorbing fluid. The dressings also shed fibres, which may delay wound healing if not removed at the time of dressing change. When early removal of saline soaked gauze dressing is needed, as in case wound infection, wound epithelialization may slough accompanied by local pain aggravation and wound deeping.[2]

Hydrocolloid (HCD) dressing is an occlusive dressing that contains a HCD matrix (for example, gelatin, pectin and carboxymethylcellulose) with elastomeric and adhesive substances attached to a polymer base. On contact with wound exudates, the HCD matrix absorbs water, swells and liquefies to form a moist gel. This has been claimed to expedite healing by providing a moist and warm environment at wound surface and also by preventing external bacterial colonization. Applying a dressing that is impermeable to bacteria reduces infection rates by 50%.[3] The bacterial content of wounds under occlusive dressings is less than that of similar wounds treated with conventional absorbent materials, possibly because active phagocytic cells are retained at moist wound surface.[4] In vitro studies show that relatively low oxygen tension stimulates angiogenesis and fibroblast and epidermal cell turnover and, therefore, is expected to provide good conditions for wound healing.[5]

Hydrocolloid dressings can be used in a variety of wounds, for example, venous leg ulcer, pressure sores, superficial burn wounds, small donar site wounds and minor abrasions.

It is suggested that HCD dressings may be used on diabetic patients with foot ulcers, although there is much debate on this issue.[6] This has largely stemmed from subjective reports of infection associated with their use.[7] and adverse events, such as contact dermatitis.[8] which has been directly attributed to their use.

There is limited evidence within medical literature to determine whether HCDs are safe to use on the diabetic ulcer. The evidence that does exist is largely contradictory, based on subjective reports of clinical experience or small studies that do not provide conclusive evidence.

In view of inadequate studies the following study is designed to assess the of HCD dressings in comparison to conventional wet saline dressing in achieving mean percentage wound reduction in patients with diabetic ulcer of lower extremities more than four weeks old admitted in KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belgaum.

OBJECTIVES

The objective of the present study was to assess the HCD dressings in comparison to conventional wet saline dressings in achieving mean percentage wound reduction in patients with diabetic ulcer of lower extremities more than four weeks duration, using transparency sheet.

REVIEW OF LITERATURE

DIABETES MELLITUS

Definition

"Diabetes mellitus (DM) is characterized by chronic hyperglycemia with disturbances of carbohydrates, fat, and protein metabolism resulting from defects in insulin secretion, insulin action, or both".[9-15]

Classification[9-15]

Type I

Type	*Pathology*
IA	: Autoimmune beta cell destruction which leads to insulin deficiency.
IB	: Lack of immunologic markers indicative of an autoimmune destructive process of the beta cells..

Type II

It is a heterogeneous group of disorders characterized by:-

- Impaired insulin secretion.
- Variable degree of insulin resistance.
- Increased glucose production

Chronic Complications of Diabetes Mellitus[9-15]

The chronic complications of DM affect many organ systems and are responsible for the majority of morbidity and morality associated with the disease.

Figure 1: Microvascular complications in diabetes mellitus

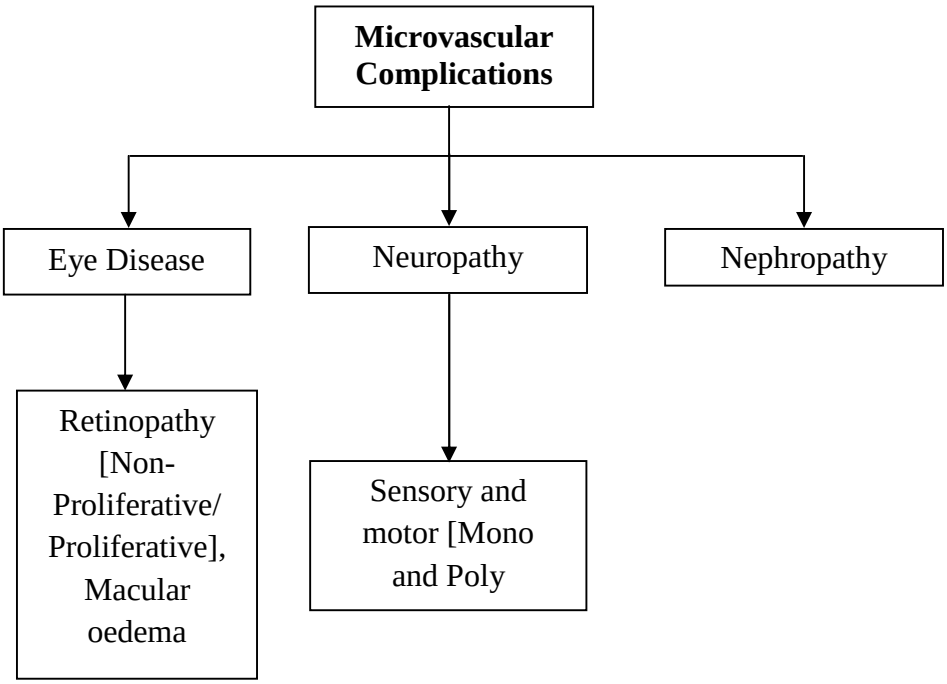
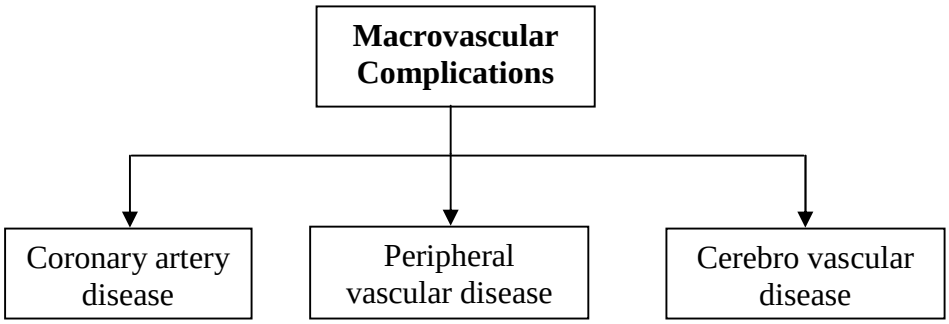


Figure 2: Macrovascular complications in diabetes mellitus



Other complications

- Gastro-intestinal [Gastropareasis, diarrhea]
- Genitor-urinary [Uropathy / Sexual dysfunction]
- Dermatologic
- Infections
- Cataracts and Galucoma

Microvascular complications of both type 1 and type 2 diabetes mellitus results from chronic hyperglycemia.

Lower Extremity Complications and diabetes mellitus[9-15]

- Foot ulcers and infections are a major source of morbidity in individuals with DM.
- The reasons for the increased incidence of these disorders in DM involve the interaction of several pathogenic factors:
    - Neuropathy.
    - Peripheral arterial disease.
    - Abnormal foot biomechanics.

Neuropathy

Neuropathy is present in over 80 percent of patients with foot ulcers.

Peripheral sensory neuropathy

Interferes with normal protective mechanisms and allows the patient to sustain major or repeated minor trauma to the foot, often without knowledge of the injury.

Motor and sensory neuropathy

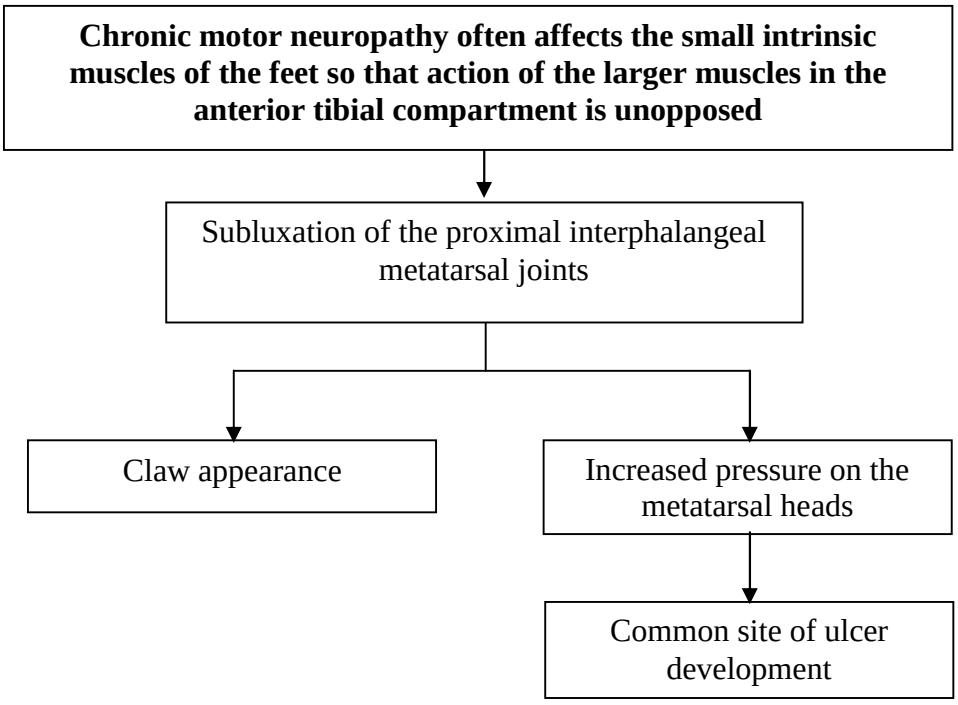
Lead to abnormal foot muscle mechanics and to structural changes in the foot [hammer toe, claw toe deformity, prominent metatarsal heads, Charoct arthropathy].

Charcoat arthropathy (Diabetic neurpathic arthropathy):

It is characterized by collapse of the arch of the mid foot and bony prominences in peculiar places. It is caused by triad of;

a. Small muscle wasting.
b. Decreased sensation.
c. Abnormal distribution of weight when standing.

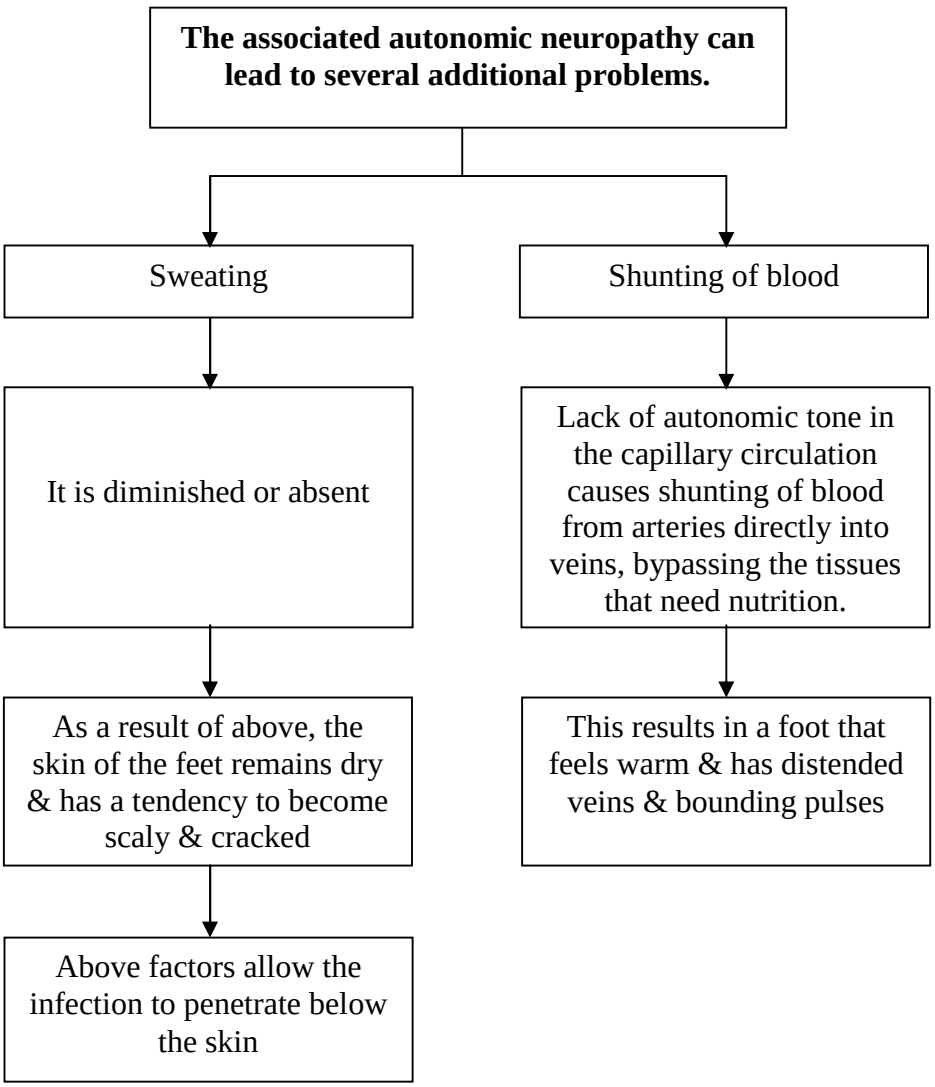
Figure 3: Pathophysiology of Charcoat arthropathy



Autonomic neuropathy

Results in anhidrosis and altered superficial blood flow in the foot, which promotes drying of the skin and fissure formation.

Figure 4: Pathophysiology of Autonomic Neuropathy in Diabetes Mellitus



Peripheral arterial disease and poor wound healing

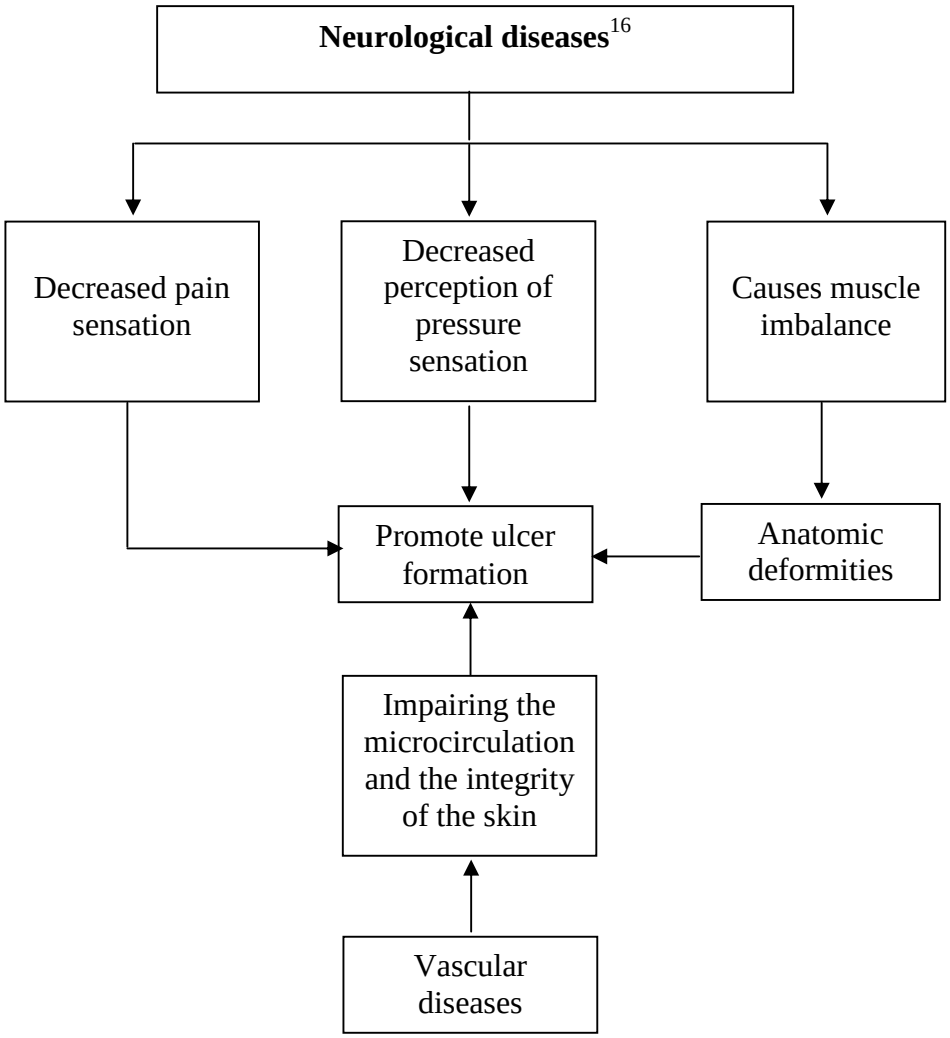
Development of arhtosclerosis is accelerated in DM leading to increased morbidity and mortality. All the large vessels are involved in this process and clinical manifestations are apparent as a result of atherosclerotic narrowing and

thrombosis of coronary, cerebral and leg vessels. It impedes resolution of minor breaks in the skin of the lower limb, allowing them to enlarge and to become infected.

Abnormal foot biomechanics

Disordered proprioception causes abnormal weight bearing while walking and subsequent formation of callus or ulceration.

Figure 5: Pathogenesis of diabetic foot



Changes in foot caused by diabetes

1. Dryness of skin and callus formation due to peripheral neuropathy.
2. High pressure at bony prominences due to;
    - Decrease plantar tissue thickness
    - Weak intrinsic muscles of foot
    - Imbalances of flexors and extensors causing clawing of foot
    - Pulling away fat padding from metatarsal heads.
3. Limited joint mobility due to;
    - Collagen abnormality
    - Thickening of skin tendons and joint capsule
    - Decreased tissue flexibility
    - Increased plantar pressure

Recommendations[17]

- The feet should be examined at least annually in patients with Type-2 diabetes and in those with Type-1 diabetes for more than five years.
- A detailed neurological examination and assessment for Peripheral vascular disease should be performed.
- We recommend using the quantitative foot assessment for neurologic symptoms.
- Patients should be considered at particularly high risk for future plantar ulceration if they have[18]
    - A Previous history of foot ulceration or amputation.

- Neuropathic foot deformities, especially with overlying bunions or calluses.

Prophylactic foot care

It is important that prophylactic advice on foot care be given to any patient whose feet are at high risk. The recommendations for prophylactic foot care are.

Avoid:

- Smoking
- Walking barefoot
- The use of heating pads or hot water bottles
- Stepping into a bath without checking the temperature.

The feet should be:

- Washed daily in tepid water.
- Mild soap should be used and the feet should be dried by gentle patting.
- A moisturizing cream or lotion should then be applied.

Toe Nails:

The toe mails should be:

- Trimmed to the shape of the toe
- Filed to remove sharp edges.

Shoes:

- The patient's shoes should be snug, not tight,

- Patients who have misshapen feet or have had a previous foot ulcer may benefit from the use of special customized shoes.

Socks:

Socks should be

- Cotton
- Loose fitting
- Should be changed every day

Inspection of feet:

- The feet should be inspected daily. Looking between and underneath the toes and at pressure areas for skin breaks, blisters, swelling, or redness. The patient may need to use a mirror or, if vision is impaired, have someone else perform the examination.

Examination of foot by medical person:

- A particularly effective strategy is to make specific recommendations to the patient in the form of a 'contract' and to advise the patient to request that his or her feet be examined at every visit to the doctor or nurse.[19]

Risk factors for foot ulcers or amputation

- Male sex
- Diabetes> 10 years duration
- Peripheral neuropathy [9-15,20,21]
- Abnormal structure of foot [bony abnormalities, callus, thickened nails]

- Peripheral arterial disease
- Smoking
- History of previous ulcer or amputation.[9-15,20,21]
- Poor glycemic control. [9-15,20,21]

ULCER

Definition

An ulcer is defined as break in the continuity of an epithelial surface, characterised by progressive destruction of the surface epithelium.

Acute wound

It is defined as the traumatic loss of normal structure and function to recently uninjured tissue after a noxious insult.[22]

Chronic wound

Wounds more than or equal to four weeks duration, is known as chronic wounds. Disruption in the event of healing regulated by process of cellular, humoral , and molecular events and resulting in a time dependent but predictable and orderly pattern of tissue repair.[23]

Characteristics of chronic wound

Floor is covered with pale granulation tissue, scanty discharge indurated base, edge and surrounding skin.

WOUND HEALING

Historical background

- Wounds were probably earliest problems of human race.
- Early surgeons like Ambrose Pare, John Hunter and Sir James Paget have given some scientific knowledge to their handling of wounds, particularly those resulted from war.[24]
- Halsted was intensely interested in wound healing process.
- In the early 1900's Carrel and his associates made investigation with the scientific approach to wound healing. Later Carrel (1916), Harvey and Howe's (1930), studied incised wounds and contributed to the knowledge of wound healing.[24]

Definition

"Body replacement of destroyed tissue by the living tissue" or "Integrated series of cellular and biochemical events which restores the functional integrity and regains the strength of injured tissue".

Phases of healing

Wound healing and repair are complex processes that involve dynamic series of events.

Coagulation

- Helps in preventing blood loss, covering wound surface and holding the wound edges together and thus contributing to the healing process

- Ross (1980) and Knighton et al (1982) have shown equivocally that fibrin and platelates play an important role in initiating the wound healing.

Granulation phase of wound healing

Granulation tissue[25]

"This is a highly vascular tissue, contains largely of;

1. Fibroblast.
2. Endothelial cells lining capillaries of newly spouting blood vessels.
3. Macrophages.
4. Pleuripotent pericytes.

Above all are embedded in a matrix consisting.

1. Fibronectin
2. Proteoglycans rich in Hyaluronicacid and collagen [This collagen is at first mainly of Type-III, changing later to Type I].

The term granulation tissue derived from it is pink, soft, granular appearance on the surface of wounds.[25]

Functions

- Fill the gap of the wound
- Supports the growing and migrating epithelial cells – The connective tissue matrix of granulation tissue forms nutritive substrate, over which regenerating epidermis can migrate and is gradually replaced by scar tissue.

Imporant factors for granulation tissue formation

- Chemotactic factor.
- Growth factor.
- Structural molecules.
- Proteases [Digests connective tissue matrix].

Angiogenesis or neo-vascularisation

It is a vital part of proliferative phase of wound healing and repair.[26]

Without angiogenesis**,** invasion of the wound bed by macrophages and fibroblasts would cease due to lack of oxygen and nutrients.[26]

In the initial stages, these vessels lack basement membrane and have loose cellular junction and are fragile in nature. Due to this, on slightest touch, the vessels bleed profusely which is a characteristic feature of newly formed capillaries. The leakage facilitates the movement of cells and macromolecules into wound site.[26]

There are four steps in angiogenesis:[25,26]

- *Step-1* Proteolytic degradation of basement membrane of parent vessel is to allow formation of capillary sprout and subsequent cell migration.[26] Angiogenic factors acts on capillary endothelial cells, which releases collagenase. This enzyme degrades the collagen of basement membrane.[25]

- *Step-2* Fragmentation of the collagen of basement membrane, permits the migration of endothelial cells into peri-vascular spaces.[12]

- *Step-3* Endothelial cells migrate into the peri-vascular spaces where they form buds.[26]

- *Step-4* Maturation of endothelial cells and organisation into capillary loops**.**

    - Functional capillary loops: During dermal repair, these buds grow rapidly towards the free surface, where they branch at their tips and unite to form **functional capillary loops**.

    - Superficial capillary plexus: On these loops, new buds develop, so that, a **superficial capillary plexus** rapidly forms in the granulation tissue.

    - Canalization: Proliferation and branching of cords of endothelial cells later become canalized to form growing capillary buds of healing wound.

    - Fusion: Capillaries originating from opposite sides of the wound fuse and establish a complete circulation within the wound.

Remodelling of the vasculature

There is constant remodelling of the vasculature, which involves obliteration of many of the capillaries.

As each capillary loop becomes functional, it brings nutrient and oxygen to nearby cells, enabling the fibroblast to secrete materials for the matrix, through which macrophages and other cells can migrate further.

As the scar maturation proceeds, capillaries gradually regress and the red vascular rich wound tissue transforms into a white, relatively avascular poor scar.

The above proliferative and migratory processes are repeated sequentially, until wound bed is filled with granulation tissue.

Macrophagia[26]

- It is the point at which protecting and clearing functions of inflammatory response are linked to starting of reparatory process

  Macrophagia is;

  1. Migration of Monocyte [from blood] to tissue injury site.
  2. Conversion of monocyte to Macrophage after migration to tissue injury site.
     - They are key cells in dermal repair
     - Wound macrophages, which appear subsequent to the cells, play pivotal role in healing by liberating various factors.

Functions of macrophages[27]

- Take over the function of phagocytes that is debridement.
- Release matrix metalloproteinases (MMP).
- Macrophages secrete numerous cytokines.
- Macrophages also release growth factors that stimulate fibroblast, endothelial cells and keratinocyte proliferation.
- Promote angiogenesis by liberating endothelial growth factor [EGF].

- Macrophage-secreted platelet derived growth factor (PDGF) stimulate collagen and proteoglycan synthesis.

***Fibroplasia*[27]**

After injury, the normally and sparse fibroblasts are chemoattracted to the inflammatory site, where they divide and produce the components of the extra cellular matrix (ECM). After stimulation by macrophage- and platelate-derived cytokines and growth factors, the fibroblast which is normally arrested in G_0 phase, undergoes replication and proliferation.

The primary function of fibroblsts is to synthesize collagen. The rate of collagen synthesis declines after 4 weeks and eventually balances the rate of collagen destruction by collagenase (MMP-1). At this point the wound enters a phase of collagen maturation. The maturation phase continues for months or even years.

Collagen

Structure[27]

The proline- and glycine- rich collagen molecule is a long, stiff, triple-stranded helical structure that consists of three collagen polypeptide α chains twist around one another in a ropelike superhelix. With its ringlike structure, proline provides stability to the helical conformation in each α chain, whereas glycine, because of its small size, allows tight packing of the three α chains to form the final superhelix. There are at least 20 types of collagen, the main

constituents of connective tissue being types I, II, III,V, IX. In early wound healing there is increased expression of type III collagen.

Collagen synthesis[27]

Collagen polypeptide chains are synthesized on membrane-bound ribosomes and enter the endoplasmic reticulum (ER) lumen as pro-α chains. Within the lumen of the ER, some of the prolines and lysines undergo hydroxylation to form hydroxyproline and hydroxylysine. Hydroxylation results in the stable triple-stranded helix through the formation of interchain hydrogen bonds. The pro-α chain then combines with two others to form procollagen, a hydrogen-bonded, triple-stranded helical molecule. After secretion into the ECM, specific proteases cleave the propeptides of procollagen molecule to form collagen monomer. These monomers assemble to form collagen fibrils in the ECM.

Functions

a) Collagen is essentially a product of fibroblast.
b) Collagen is the most abundant proteins of the connective tissue.
c) Supports to the tissues
d) Provides structural framework to other types of tissues.
e) Acts as a medium where blood vessels and nerves are passing.
f) Brings and keeps the wound edges together and provides tensile strength for holding together – this holding strength prevents the breakdown of tissue (organ) at the healed site.
g) Fill the gap caused by the tissue loss.

Ground substance in healing wound[27]

- Connective tissue consists of cellular and non cellular (matrix). Matrix is again composed of fibres and ground substance.
- Ground substance is non-fibrous part of the matrix in which cells and fibres are embedded.
- Consistency: Except in mineralized connective tissue, the ground substance is viscous gel.

Constituents

- Water (High proportion).
- Mucopolysaccharides.
- Fibronectin.
- Chondronectin.
- Mucoproteins.
- Glycoproteins.
- Lamenin.
- Entactin.

Wound contraction[27]

- Definition: "Wound contraction may be defined as a process by which the size of full thickness open wound is diminished by centripetal movement of the thickness of surrounding skin".
- The feature that most clearly differentiates primary from secondary healing is the phenomenon of wound contraction, which occurs in large surface wounds.

- Wound contraction is one function of granulation tissue which is critical for repair.
- The events of wound healing from injury to fibroplasia, occur in all wounds. But certain events like wound contraction occurs characteristically in dermal wound.
- In humans, the wound contraction is less because in most part of the body the skin is somewhat firmly attached to subcutaneous tissue but it can occur in ares like back of neck and buttocks.

Timing

Wound contraction starts from about third or fourth day of healing and continues upto 15^{th} or 16^{th} day and stops thereafter, irrespective of whether the wound is totally closed or not.

Rate

- The rate of wound contraction is about 0.60 - 0.75 mm /day.
- Wound contraction is not materially affected by size or shape of the wound but perhaps by the length of the wound perimeter.

Mechanism

- The mechanism of wound contraction is disputable and debatable. Many theory like Pull theory, Push theory / Picture Frame theory etc. have been proposed but none of them appears to be satisfactory.
- Modified fibroblasts rich in actin filaments are responsible for wound contraction.

- Myofibroblasts are situated just under the advancing edges of the wound.
- In early phases of wound contraction, contractile epidermal cells in wound edges are suggested as a source of force.
- Wound contraction can be both beneficial or detrimental. Wound contraction can lead to distortion, disfigurement and impairment of function.

Epithelization

Definition

- Epithelization is a process of wound healing involving body surface.
- Unlike healing by fibroplasia where lost parenchymal cells are replaced by non-specific connective tissue, in epithelialisation lost epithelial cells are replaced by epithelial cells only. It is an example of healing by regeneration.

Stages

a) Mobilization and loosening of basal cells from their dermal attachment.
b) Migration or movement of cells to a position of cell deficit.
c) Proliferation or replacement of cells to a position of cell deficit and
d) Differentiation or restoration of cellular function.

Epithelization which depends on several factors;

- Size of wound.
- Location of wound.
- Shape of wound.
- Impairment of blood supply.

- Pathological modification of wound

Healing by epithelisation occurs in;

- Dermal wounds.
- Wounds of tracheobronchial surface.
- Surface wounds in gut, urinary bladder, uterus etc.

Timing

First 24 hrs of injury: Changes in epidermis leading to re-epithelization begin within 24 hours of the formation of a cutaneous wound.

Types of wound healing[27]

Healing by first intention

The wounds are sealed immediately with simple suturing, skin graft placement, or flap closure, such as closure of the wound at the end of surgical procedure.

Healing by secondary intention

No active intent to seal the wound. Generally, this type of repair is associated with a highly contaminated wound and will close by re-epithelialization, which results in contraction of the wound.

Heeling by tertiary intention

It is also referred to as delayed primary closure. A contaminated wound in initially treated by repeated debridement, systemic or topical antibiotics, or negative pressure wound therapy for several days to control infection. Once the

wound is assessed as being ready for closure, surgical intervention, such as suturing skin graft placement, or flap design is performed.

MANAGEMENT OF CHRONIC WOUNDS

Wound dressings have been used since antiquity to facilitate the healing process. A material which when applied to the surface of a wound, provides and maintains an environment in which healing can take place at maximum rate; Thomas (1986).[28] The first antiseptic dressing was introduced by Lister in 1867 who soaked the lint and gauze in carbolic acid.[29]

Dressings used in chronic diabetic ulcer

Conventional dressings, such as gauze, impregnated gauze, gauze and cotton, packing strips have been in use for over fifty years.

Action of saline dressing

Normal saline dressing keeps the environment moist for proper healing. Normal saline dressing acts as an osmotic dressing, with time the concentration of the saline increases due to evaporation altering it from isotonic to hypertonic dressing which in turn decreases evaporation of fluid from the wound, keeping it moist.[30]

Moist wound environment that these dressings provide are best for wound regeneration and repair and increasing the velocity of healing. Effective wound management aims to strike a balance that is a moist environment to promote healing, but not so wet as to cause maceration and excoriation.

Two factors are important for natural wound healing. One is wound exudates which is generic term given to liquid produced from wounds. Exudate keeps the wound moist, supplies nutrients, and provides the medium for migration and mitosis of epithelial cells. This in turn, keeps the wound supplied with leucocytes, helping to control micro organism. Second factor is the presence of white cells in the wound. White cells play a major role in wound healing by cleaning the wound, remove potentially pathogenic micro organisms and producing collagen, the building block of new tissue. Excessive exudates can cause maceration and hence the dressing should be able to absorb excessive exudates from the wound.[31]

Basic requirements of the ideal ulcer dressing[2]

- Maintain high humidity between wound and dressing
- Absorbent, removes excess exudates
- Non-adherent, allowing easy removal without trauma at dressing change
- Safe and acceptable to patient(non-allergic)
- Permit gaseous exchange but impermeable to micro-organism
- Cost-effective

Newer dressings available for diabetic ulcer

A wide variety of new dressing materials have been developed. However none of the newer dressing fulfill all the characteristics of an ideal dressings.

- Film dressing.
- Foam dressing.

- Nonadherent dressing (Paraffin-impregnated tulle dressing).
- Hydrogels.
- Hydrocolloids.
- Alginates.

Newer therapies available for diabetic ulcer

- Plate derived growth factors.
- Demagraft.
- Apligraf.
- Granulocyte-colony stimulating factor.
- Hyaff.

Role of hydrocolloid dressing in wound healing

Hydrocolloid dressing is an occlusive dressing that contains a HCD matrix (for example, gelatin, pectin and carboxy methylcellulose dressing) with elastomeric and adhesive substances attached to a polymer base. On contact with wound exudates, the HCD matrix absorbs water, swells and liquefies to form a moist gel. This has been claimed to expedite healing by providing moist and warm environment at the wound surface. In vitro studies show that relatively low oxygen tension stimulates angiogenesis and fibroblast and epidermal cell turnover and, therefore, is expected to provide good conditions for wound healing.[5]

In their intact state, all hydrocolloid dressings are impermeable to moist vapour, however, when they have absorbed wound exudates, their permeability

increases allowing some moisture vapour to pass through the back of dressing. This action enhances the ability of HCD to cope with exudate production.[32]

Hydrocolloid dressings have an outer waterproof layer that excludes atmospheric oxygen, liquids and bacteria, as well as protecting the wound from contaminants. They can also reduce the risk of cross infection if a colonised wound is occluded, by preventing the spread of organisms into the environment. As the dressings are waterproof, this will allow patients to bathe and shower.

A study has found that the occlusive nature of the dressing promotes angiogenesis in wound, which is important as this can promote granulation tissue formation in wounds and healing by secondary intention.[33] Another key function of HCD in maintaining an optimum environment for wound healing to occur, is providing thermal insulation to enable the wound surface to remain near body temperature. This is imperative if cell mitosis is to continue, as cell division will only occur if cells are kept at body temperature. Another study found that a slightly acidic environment (pH 6.4) is maintained under a HCD dressing, which has a bactericidal effect especially against pseudomonas.[34] The occlusive nature of the dressing also provides macrophages with an optimal environment to ingest dead tissue, which is relevant in the debridement of necrotic and sloughy tissue.[32]

As the dressing absorbs fluid, a gel is formed which provides a moist environment at the wound surface. This assists epithelial migration[35] and provides pain relief in superficial wounds as nerve endings are bathed in gel. The moist environment also aids the process of autolysis, by rehydrating and lifting off necrotic or sloughy tissue in the wound bed. It is also suggested that as the hydrocolloid liquefies and swells in the wound space, pressure is increased

approaches that within capillaries so inhibiting loss of further fluid, however, this will only occur if the hydrocolloid dressing maintains an adequate seal over the wound.[36]

Indications for use

Hydrocolloid dressings can be used in a variety of wounds, for example leg ulcers, pressure ulcers, superficial burn wounds, small donar site wounds and minor abrasions.

Role of hydrocolloid dressings in diabetic ulcers of lower extremities

There is limited evidence within medical literature to determine whether hydrocolloids are safe to use on diabetic foot. The evidence that does exist is largely contradictory, based on subjective reports of clinical experience or small studies that do not provide conclusive evidence.

In a study author reviewed the literature on the use of hydrocolloids on the diabetic foot and suggested that concerns regarding the use of hydrocolloids on diabetic foot ulcers has largely centred on subjective reports of adverse events which often arose due to the inappropriate use of these dressings.[6] In the same study author recommended that practitioners should base dressing choice on careful wound assessment and thorough knowledge of dressing properties, in adition to local policy.[6]

Key considerations before selecting a hydrocolloid for a diabetic foot ulcer should include:

- Assessment of the viability of the surrounding skin before application as the skin can be vulnerable to tearing when dressings are removed.
- There is a recognised need for further studies to investigate HCD use and infection rates in diabetic foot ulcers, in the absence of such evidence it is suggested that caution should be exercised in the presence of infection.[6,37,38]
- It is suggested that HCD can be used safely on diabetic foot ulcers, providing that they are used on appropriate wounds after a thorough patient assessment, the wound is superficial with no signs of infection, there is low to moderate exudate and dressings are changed frequently. The best advice would seem to be "use with caution in patients with diabetes".

METHODOLOGY

The present study was carried out at Department of Surgery, Jawaharlal Nehru Medical College and KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belgaum for a period of one year (from January 2008 to December 2008).

Study design

The Randomised clinical trial was conducted on patients with chronic diabetic ulcer of lower extremities.

Source of data

Patients with chronic diabetic ulcers of lower limb extremities admitted at Department of Surgery, KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belgaum during the study period.

Sample size

The present study comprised of 68 patients.

Sampling procedure

The sample size was calculated based on patient data for the last three consecutive years that is 2005, 2006, 2007 of KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belgaum.

Selection Criteria

Inclusion criteria

- Type 2 diabetes mellitus patient.
- Diabetes between 18 to 65 years.
- Ulcer size smaller than (6 x 6) cm^2.
- Duration of ulcer more than four weeks.
- Tunnelling or undermining less than one cm in depth.
- Fasting blood glucose levels measured on two occasions 24 hours apart between 140 mg/dl to 200 mg/dL
- HbA1C levels less than 7.5 mg/dL
- Grade I diabetic wound Wagner's classification.
- Negative wound cultures.

Exclusion criteria

- Pulseless limb.
- Immunocompromised patient.
- Associated osteomyelitis.
- Haemoglobin level less than 10 gm/dL.
- Skin malignancy.
- Cellulitis.
- Diabetic ketoacidosis.
- Diabetic gangrene.
- Connective tissue disorder.

- Uncontrolled hypertension and renal failure.

Procedure

The study was approved by the Ethical and Research Committee of Jawaharlal Nehru Medical College, Belgaum. After finding the suitability as per inclusion and exclusion criteria patients were selected for the study and briefed about the nature of the study, the interventions used and written informed consent was obtained (Annexure–I).

Further patients were randomized with the help of a computer generated randomization chart into two groups namely study group and control group. Out of the 68 participants, 34 were (Control group) treated in the form of conventional wet saline dressings and the remaining 34 were (Interventional group) treated in the form of HCD dressings.

The descriptive data of the participants like name, age, sex, detailed history, were obtained by interviewing the participants and clinical examination and necessary investigations like complete blood count, blood urea and serum creatinine and culture of the ulcer were recorded on predesigned and pretested proforma (Annexure-II).

Initial wound measurement was taken in both the groups before starting their respective treatment that is conventional wet saline dressing in control group and hydrocolloid dressing in study group.

Initial wound area measurement

Ulcer examination was done in all these patients and wound was assessed of its characteristics and photographed. Ulcer was assessed by the investigator at the beginning of the study and at the end of the study (Investigator being the staff and residents in the unit excluding the guide). Wound area measurement was recorded over the transparency sheet on day one in both study groups and control group.

The dressing was changed every third day; similar four dressings were done to all the patients to both the groups. Final wound area was measured on 14^{th} day over the transparency sheet.

Outcome was measured in terms of wound reduction between the two groups. Data was tabulated and the two groups were compared with reference to area and percentage of reduction.

Statistical analysis

The data obtained was tabulated and analysed using students ‘t’ test, chi-square test and a ‘p’ less than 0.05 was taken as significant.

RESULTS

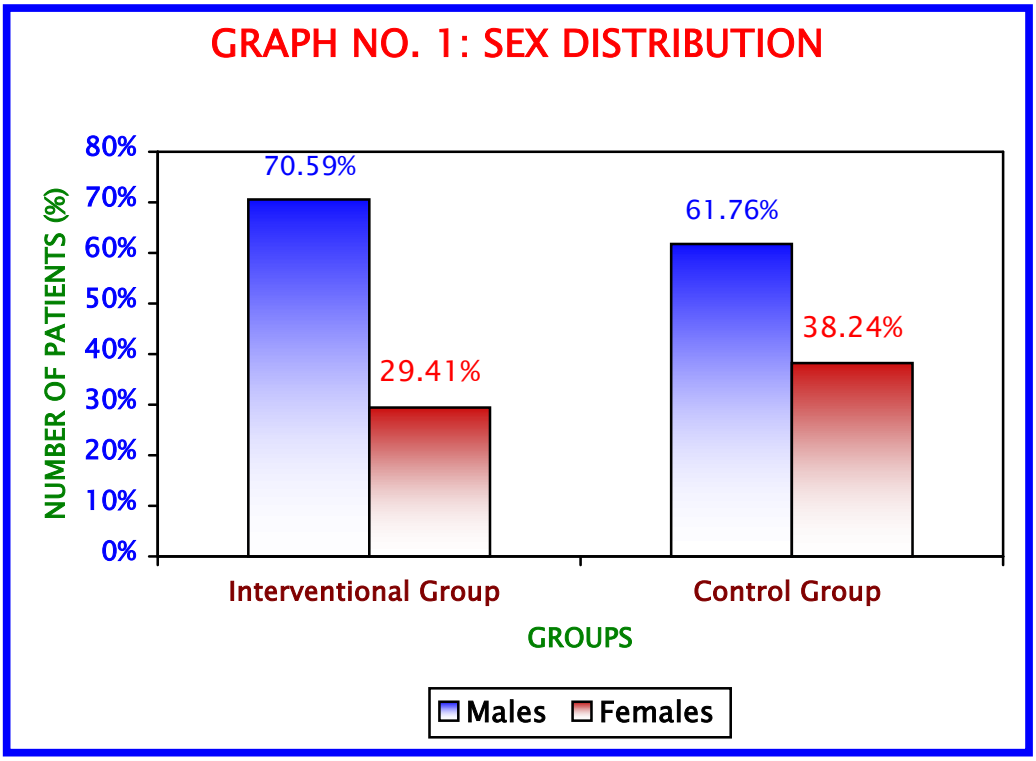
The present study was conducted in KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belgaum and the findings are tabulated as below.

During the study year from January 2008 to December 2008, 68 patients with chronic diabetic ulcers of the lower limb were randomized into study (HCD dressings) and control (Normal saline dressings) group. These groups were studied for the effect of conventional saline dressings versus HCD dressings on wound reduction.

A total of 68 patients satisfied the selection criteria, analysis was done by using students 't' test and chi square test.

Table 1: Sex distribution

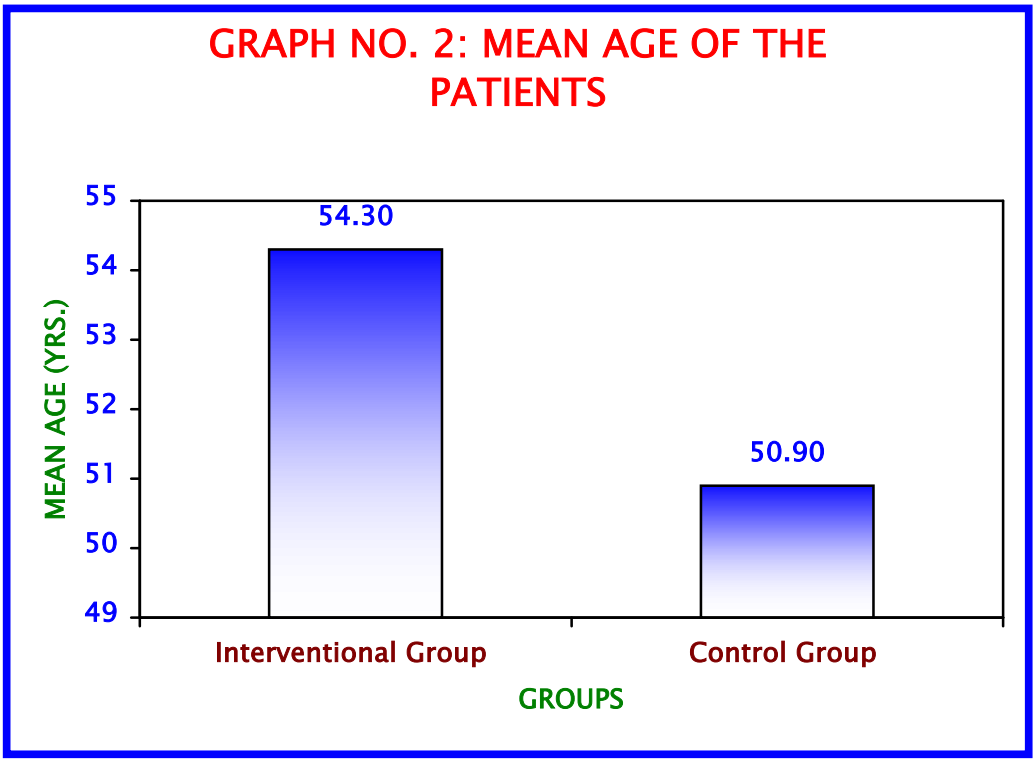
Groups	Interventional Group		Control group	
	Number	**Percentage**	**Number**	**Percentage**
Males	24	70.59%	21	61.76%
Females	10	29.41%	13	38.24%
Total	34	100.00%	34	100.00%



In the interventional group, total number of males and females were 24 (70.59%) and 21 (29.41%) respectively. The male:female ratio was 2.4:1. In control group, total number of male and females were 21 (61.76%) and 13 (38.24%) respectively. The male:female ratio was 1.62:1. Statistically in this study, there was no significant difference in sex distribution between interventional and control group.

Table 2: Mean age of the patients

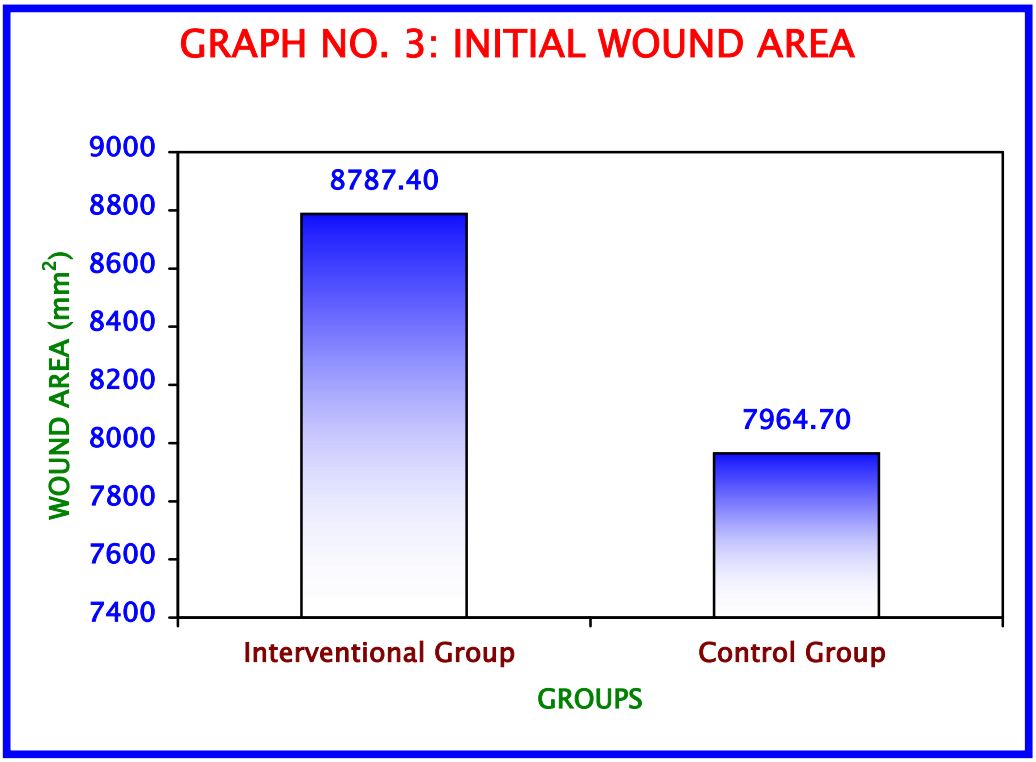
Groups	Mean age (Years)	
	Mean	**S.D.**
Cases	54.3	6.83
Controls	50.9	8.11
t= 1.875	DF = 66	p= 0.065



In this study, the mean age in interventional group and control group were 54.30±6.83 and 50.90±8.11 respectively. Statistically there was no significant difference in mean age between interventional and control groups.

Table 3: Initial wound area

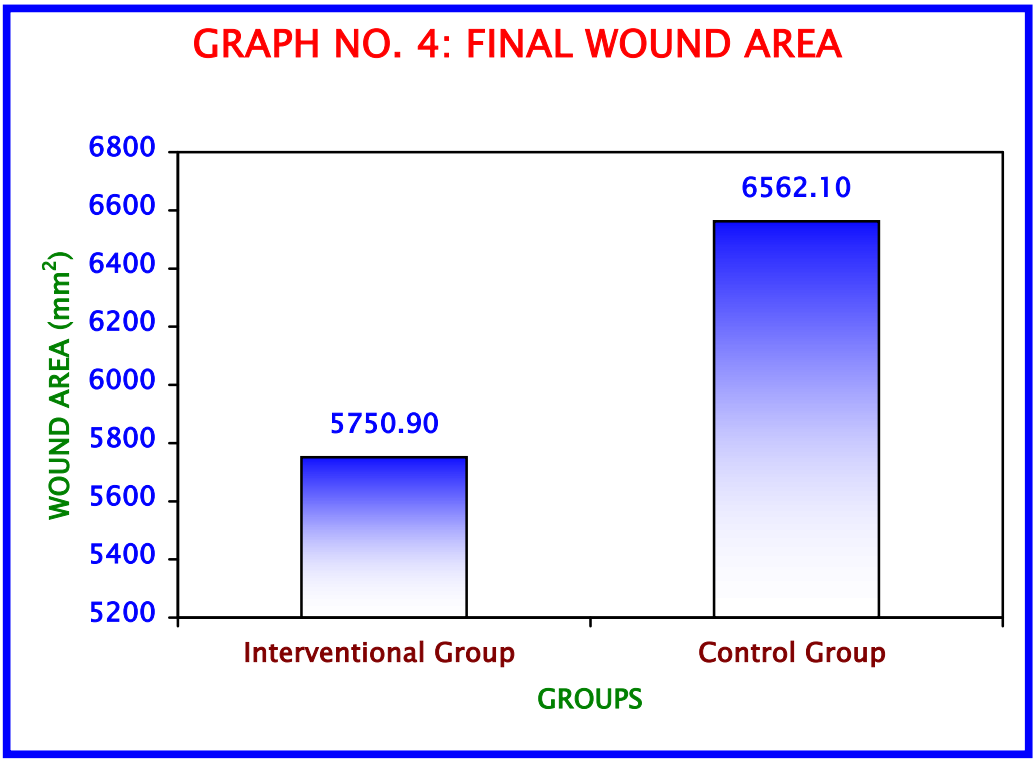
Groups	Initial wound area (mm^2)	
	Mean	**S.D.**
Interventional	8787.40	394.59
Controls	7964.70	341.10
t = 0.920	DF = 66	p= 0.361



The mean area at the beginning of the study was 8787.4±394.59 mm^2 in the HCD and 7964.7±341.10 mm^2 in the normal saline group. There was no significant difference in the mean area between the two groups (p=0.361).

Table 4: Final wound area

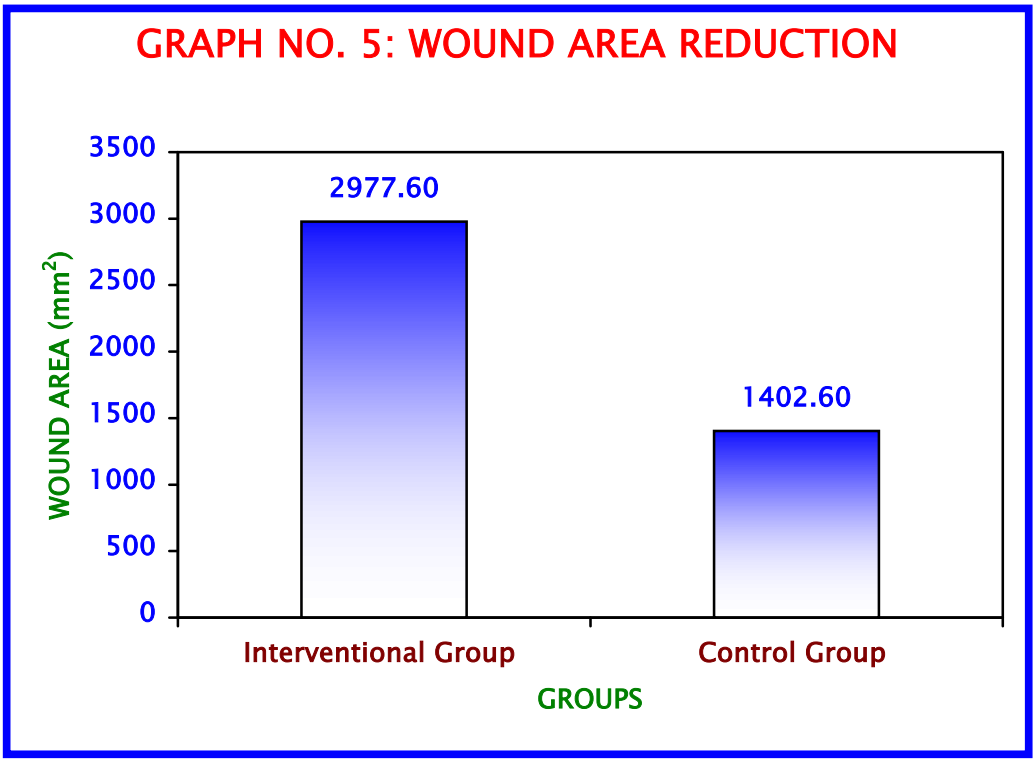
Groups	Final wound area (mm^2)	
	Mean	**S.D.**
Interventional	5750.90	244.79
Controls	6562.10	290.53
t = 1.245	DF = 66	p= 0.218



At the end of the study the mean area were 5750.9 ± 244.79 mm^2 in the group treated with HCD dressings and 6562.1±290.53 mm^2 in the group treated with normal saline dressings.

Table 5: Wound area reduction

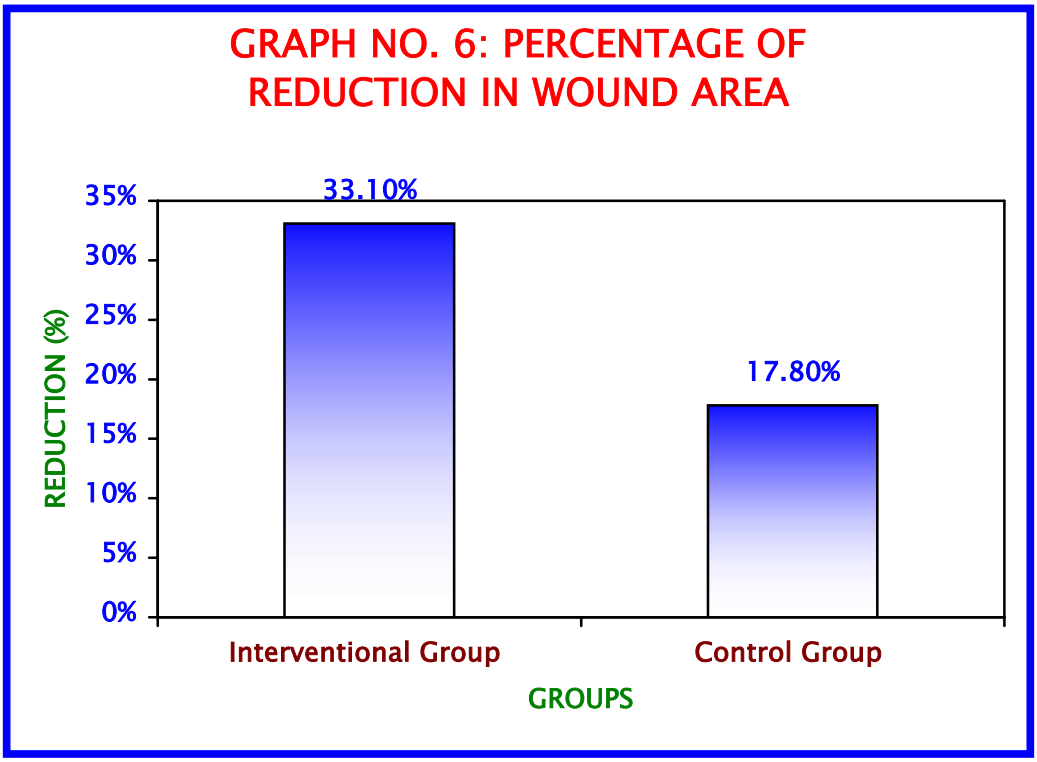
Groups	Reduction wound area (mm^2)	
	Mean	**S.D.**
Interventional	2977.60	158.63
Controls	1402.60	65.56
t = 5.350	DF = 66	p<0.001



The study shows that the final wound reduction achieved between the two groups were 2977.6±158.63 mm^2 in patients treated with HCD dressing and 1402.6±65.56 mm^2 in patients treated with normal saline dressing, which is statistically significant (p<0.001).

Table 6: Percentage of reduction in wound area

Groups	Percentage of reduction	
	Mean	**S.D.**
Interventional	33.10	6.30
Controls	17.80	4.28
t = 11.696	DF = 66	p<0.001



The percentage of area reduction were 33.1±6.30 in patients treated with HCD dressing and 17.8±4.28 in patients treated with Normal saline dressing, which is statistically significant (p<0.001).

DISCUSSION

It is every surgeon's desire that after dressing the wound, it should heal without any complications. Successful wound dressing should keep the wound moist and be devoid of any adverse reactions such as infection, maceration and allergey. Diabetic ulcers of lower limb are chronic wounds, stuck in inflammation phase and shows cessation of epidermal growth or migration over the wound surface.

Hydrocolloid dressing has shown great promise as a procedure for healing of chronic wounds (Venous ulcers, pressure sores, superficial burn wounds, small donar site wounds and minor abrasions).

It is suggested that HCD dressings may be used on diabetic patients with foot ulcers, although there is much debate on this issue.[6] This is largely stemmed from subjective reports of infection associated with their use[7] and adverse events, such as contact dermatitis [8] which has been directly attributed to their use.

In the present study, an attempt has been made to establish better healing rates with use of HCD dressing in chronic diabetic ulcer of lower limb. In this study the base line characteristics such as age, sex and location of the ulcer were similar in the patients who received HCD dressing in the study group and in patients who received normal saline dressing in the control group.

This study was based on a randomized controlled study[39] which aimed to document the safety and performance of HCD dressing in the treatment of established foot ulcers in patients with diabetes. Participants had an ulcer bigger

than one cm^2 and less than eight cm^2 in any direction. The treatment period was six weeks. The mean wound area reduced from 5.4 cm^2 to 2.5 cm^2. Relative wound area reduced from 100% at baseline to 40% at week six. This study demonstrates that treatment of diabetic foot ulcer with hydrocolloid dressing results in considerable wound area reduction and prevents any deterioration in maceration.

Another open randomized controlled study was carried out of 44 diabetic patients with necrotic foot ulcers treated with adhesive zinc oxide tape or with HCD dressing.[40] Fourteen of the 21 patients treated with zinc oxide had their necrotic ulcers improved by at least 50% compared to six out of 21 with the hydrocolloid dressing ($p<0.025$).Fifteen patients showed an increase in the area of necrosis during the course of the five-week study and of these, 10 had been treated with the hydrocolloid dressing. This study was limited to individuals with necrotic foot ulcers, thus excluding other types of diabetic foot wounds. Other clinicians firmly recommend that HCDs, should be used on wound after the removal of necrotic tissue.

This study shows that the final wound reduction achieved between the two groups were 2977.6±158.63 mm^2 in patients treated with hydrocolloid dressing and 1402.6±65.56 mm^2 in patients treated with Normal saline dressings. The percentage of area reduction was 33.1±6.30 in patients treated with Hydrocolloid dressing and 17.8±4.28 in patients treated with Normal saline dressings.

However, the final area of the ulcer (in mm^2) was significantly reduced in patients with HCD dressing group as compared to the patients in Normal Saline group at the end of the study. The percentage reduction in the area of the ulcer was more in the HCD dressing group as compared to the Normal Saline group and this difference was statistically significant.

We have applied the following formula to calculate % reduction in area of wound after two weeks period in both cases and controls.

$$\% \text{ Reduction of wound after two weeks} = \frac{\text{Initial area} - \text{Final area}}{\text{Initial area}} \times 100$$

Overall this study shows that HCD dressing is a safe and effective in treating chronic diabetic ulcers of lower limb. This study was conducted only for two weeks and complete epithelialisation and wound reduction was not awaited.

Limitations of our study

- Not a blinded study
- Follow up is short to derive conclusion on long term healing of the ulcers.
- The cost involved was not analyzed in this study.

Scope for further study

There is further scope of study among infective diabetic wound with respect to anti-infective properties of HCD dressing.

CONCLUSION

With the use of HCD dressing in comparison with the conventional normal saline dressing for the treatment of chronic diabetic ulcers of lower limb, the following conclusions were derived;

- Hydrocolloid dressing showed faster and better healing rates among the study group.
- Area reduction and percentage reduction was better in HCD dressing group.
- There was no adverse effect or reactions seen when HCD dressing was applied over the ulcer.

SUMMARY

The present study was conducted in KLES' Prabhakar Kore Hospital and Medical Research Centre, Belgaum on 68 patients with chronic diabetic ulcers of lower limb.

The objective of the present study was to assess the HCD dressing in comparison to conventional wet saline dressing in achieving mean percentage reduction in patients with diabetic ulcer of lower extremities more than four weeks duration using transparency sheet.

The two groups were randomized into study (Hydrocolloid) and conventional (Normal Saline) group. One group received treatment in the form of HCD dressing and other received conventional wet saline dressing.

There was no statistical difference in the baseline characteristics like age, sex and initial wound area of the ulcer between the two groups.

The final area reduced and percentage of area reduced were statistically significant in the study as compared to the control group.

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ANNEXURE I

CONSENT FORM

Introduction

Mr./Miss./Mrs. _____

Your are invited to participate in our research study that is **"RANDOMIZED CONTROL TRIAL COMPARING THE EFFICACY OF THE HYDROCOLLOID DRESSINGS VS SALINE DRESSINGS IN THE HEALING OF DIABETIC ULCER OF THE LOWER EXTREMITIES".**

Since you are suffering from Diabetes and lower limb 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 HCD dressing and saline soaked gauze dressings on your lower limb ulcer and the result of this research will help in a better treatment of similar participants in the future.

On the 4^{th}, 7^{th}, 10^{th} and 14^{th} day ulcer area will be measured using four setting of dressings (1^{st}, 4^{th}, 7^{th} and 10^{th}).

If you agree to be part of this research, we would ask you some relevant clinical history. You are free to not to answer to which ever questions you think are not relevant. A clinical examination will be done and the culture will be taken from wound. On the 1^{st}, 4^{th}, 7^{th} and 10^{th} day in one group dressing will be done by HCD and in another group will be done by saline soaked gauze dressing.

There are chances you may have a speedy and better recovery with this therapy and it will also help in the treatment if participants with similar complaints in the future. Your decision of whether or not to participate in this study will not effect the quality of treatment you receive. Further you may withdraw from the study at any time.

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

Privacy and Confidentiality

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

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

Authorization to Publish Results

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

Compensation

In the event of injury or complication related to the study, treatment will be made available to you through KLES Dr. Prabhakar Kore Hospital and MRC, Belgaum. There is no compensation or payment for such medical treatment by law. In case of any complication or injury please do feel free to contact Dr. Soumitra Saha. PG M.S. General Surgery, KLES Dr. Prabhakar Kore Hospital and MRC, Belgaum Phone No.9986794823.

Questions

In case you have any questions related to the study, you can contact Dr. Soumitra Saha on Mobile No. 9986794823.

In case you have any question about your rights as a study participant, you can contact Dr. V.D. Patil (0831-2471350).

Consent for participation in research trial

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

Subject Name : _____

Signature or the Left Thumb Print of Subject : _____

Witness Name: _____ **Signature :** _____

Investigators Name: _____ Signature : _____

Date : _____ Place : _____

ANNEXURE II

PROFOMA

I) <u>PATIENT IDENTIFICATION DATA</u> :

NAME IP/OPD NO.

AGE DOA :

SEX DOD:

OCCUPATION

ADDRESS

II) <u>CHIEF COMPLAINTS</u> :

<u>MEDICAL HISTORY</u> :

Peripheral Neuropathy : ( )

Nephropathy ( )

Retinopathy ( )

PVD ( )

CVD ( )

<u>DIABETIC STATUS</u> :

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. Contracture

NERUROLOGICAL EXAMINATION

VASCULAR EXAMINATION	Left	Right
Popliteal a.	( )	( )
Ant . Tibial	( )	( )
Post Tibial	( )	( )
Dorsalis Pedis	( )	( )

ANY FOOT DEFORMITY PRESENT

Toe deformity

Bunion

Charcots foot

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.

INVESTIGATION

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

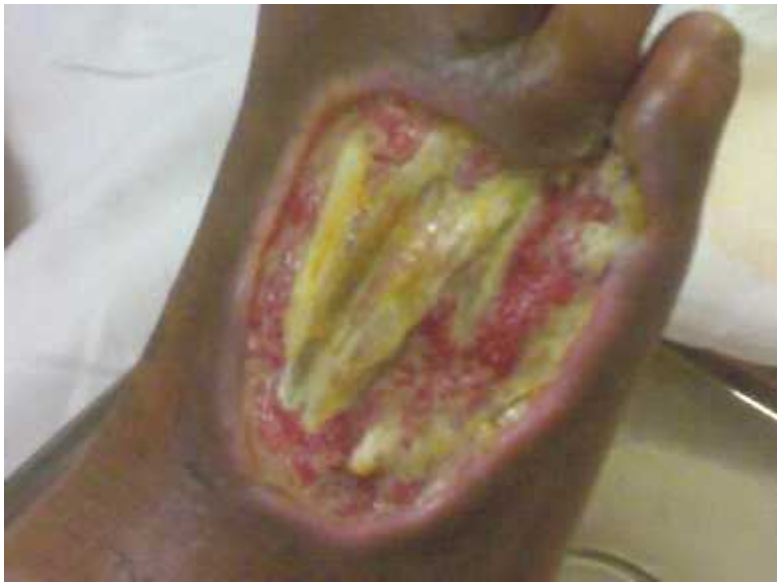
WOUND AREA MEASUREMENT ON D_1 in cm^2

Type of Dressing – Saline gauze ( )

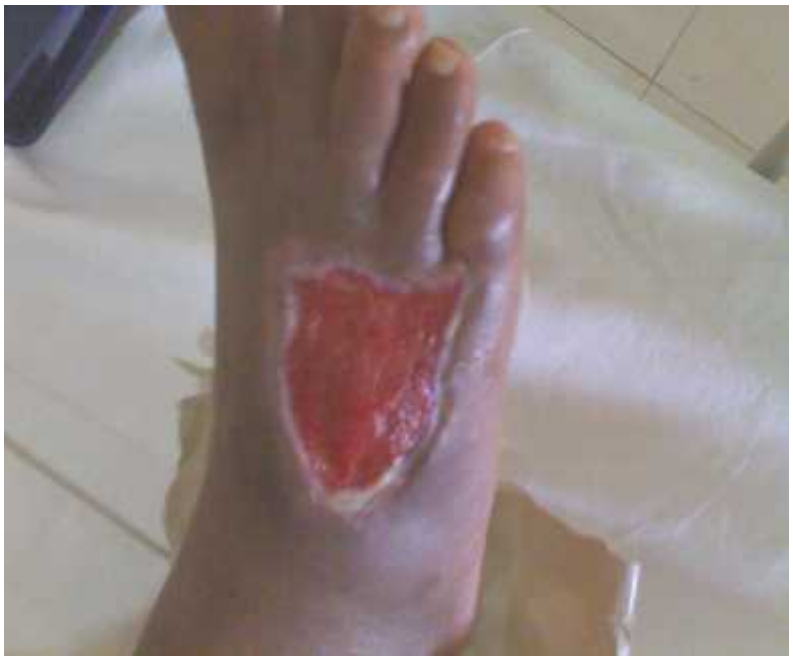
Hydrocolloid ( )

ANNEXURE III

PHOTOGRAPHS



Photographs 1: Ulcer size on day one



Photographs 2: Ulcer size on day 14 using hydrocolloid dressing

ANNEXURE IV

MASTER CHART – CONTROL GROUP

S. No	I.P No	GROUP	AGE /SEX	SITE	SITE	ANTI DM RX	X-Ray	C/S	INTITIAL AREA	FINAL AREA	AREA REDUCED	% AREA REDUCTION
1	206181	CONTROL	60/M	T	SRF	I	N	NOGC	931	750	181	19.44
2	209559	CONTROL	58/M	T	DRF	O	N	NOGC	1024	900	124	12.11
3	210650	CONTROL	55/F	T	DLF	O	N	NOGC	905	735	170	18.78
4	212162	CONTROL	58/M	S	SLF	I	N	NOGC	1493	1324	169	11.32
5	216391	CONTROL	46/M	S	HRF	I	N	NOGC	753	667	86	11.42
6	222426	CONTROL	58/M	T	SRL	0	N	NOGC	1590	1286	304	19.12
7	225342	CONTROL	36/M	S	DLF	I	N	NOGC	1475	1189	286	19.39
8	248861	CONTROL	57/M	T	SLF	O	N	NOGC	714	620	94	13.17
9	249318	CONTROL	45/M	S	LMLL	I	N	NOGC	498	395	103	20.68
10	251652	CONTROL	55/F	T	LMRL	I	N	NOGC	300	240	60	20
11	231785	CONTROL	38/F	S	SLL	O	N	NOGC	695	600	95	13.67
12	249737	CONTROL	56/F	T	DRF	I	N	NOGC	713	570	143	20.06
13	246209	CONTROL	55/F	S	SLF	I	N	NOGC	575	496	79	13.74
14	251254	CONTROL	59/F	T	DLF	O	N	NOGC	512	410	102	19.92
15	265929	CONTROL	51/F	T	SRL	O	N	NOGC	450	400	50	11.11
16	271426	CONTROL	53/F	T	SLL	I	N	NOGC	386	320	66	17.1
17	271474	CONTROL	56/M	S	SRL	O	N	NOGC	430	350	80	18.6
18	275071	CONTROL	45/F	T	LMLL	I	N	NOGC	375	300	75	20
19	275209	CONTROL	56/F	S	LMRL	O	N	NOGC	356	290	66	18.54
20	275226	CONTROL	36/M	T	HRL	O	N	NOGC	410	340	70	17.07
21	278559	CONTROL	52/M	T	SRL	I	N	NOGC	925	720	205	22.16
22	278827	CONTROL	59/M	T	SLF	I	N	NOGC	840	700	140	16.66
23	276298	CONTROL	50/M	S	GLL	O	N	NOGC	575	490	85	14.78
24	278413	CONTROL	60/F	S	DRL	I	N	NOGC	735	625	110	14.96
25	300412	CONTROL	49/F	T	DLF	O	N	NOGC	1225	1080	145	11.84
26	301942	CONTROL	27/M	S	DLF	I	N	NOGC	1385	1205	180	12.99
27	304076	CONTROL	50/M	S	MMRL	I	N	NOGC	975	812	163	16.71
28	306510	CONTROL	45/F	T	ATRL	O	N	NOGC	988	785	203	20.54
29	305124	CONTROL	58/M	S	SLF	O	N	NOGC	798	614	184	23.05
30	311828	CONTROL	56/M	T	DRF	I	N	NOGC	844	700	144	17.06
31	312572	CONTROL	54/M	S	GLL	O	N	NOGC	625	482	143	22.88
32	313497	CONTROL	52/M	T	DLF	I	N	NOGC	988	744	244	24.69
33	314415	CONTROL	39/M	T	ATRL	O	N	NOGC	728	544	184	25.27
34	315250	CONTROL	48/M	S	MMRL	I	N	NOGC	864	628	236	27.31

CASE GROUP

S. No	I.P No	GROUP	AGE /SEX	SITE	SITE	ANTI DM RX	X-Ray	C/S	INTITIAL AREA	FINAL AREA	AREA REDUCED	% AREA REDUCTION
1	271411	CASE	62/M	T	DRF	I	N	NOGC	838	580	258	30.79
2	258292	CASE	45/F	T	GLL	I	N	NOGC	1166	790	376	32.25
3	254416	CASE	65/M	S	DLF	O	N	NOGC	642	420	222	34.58
4	261540	CASE	70/M	T	GRL	I	N	NOGC	1228	890	338	27.52
5	257477	CASE	55/F	T	DRF	O	N	NOGC	696	480	216	31.03
6	25779	CASE	59/F	S	DLF	O	N	NOGC	1548	1090	458	29.59
7	258203	CASE	56/M	T	DRF	I	N	NOGC	874	600	274	31.35
8	262264	CASE	45/F	S	DLF	I	N	NOGC	350	230	120	34.29
9	261878	CASE	59/M	S	GLL	I	N	NOGC	1248	800	448	35.9
10	265487	CASE	51/M	T	DRF	O	N	NOGC	502	322	180	35.86
11	2719108	CASE	50/M	S	DRF	I	N	NOGC	459	303	156	33.99
12	281164	CASE	58/M	T	DLF	O	N	NOGC	550	360	190	34.55
13	281140	CASE	60/M	T	GRL	O	N	NOGC	452	395	57	12.61
14	281111	CASE	39/F	S	GLL	I	N	NOGC	390	260	130	33.33
15	281070	CASE	55/M	S	DLF	I	N	NOGC	335	230	105	31.34
16	281070	CASE	52/M	S	DRF	I	N	NOGC	500	350	150	30
17	283904	CASE	44/M	T	GLL	O	N	NOGC	417	280	137	32.85
18	283813	CASE	57/F	T	DLF	O	N	NOGC	396	350	46	11.62
19	284690	CASE	58/F	T	DRF	I	N	NOGC	320	210	110	34.38
20	284724	CASE	40/M	T	DRF	I	N	NOGC	495	330	165	33.33
21	1944247	CASE	53/F	S	GRL	O	N	NOGC	1250		410	32.8
22	292218	CASE	49/M	S	GLL	O	N	NOGC	950	600	350	36.84
23	296061	CASE	60/F	S	DLF	I	N	NOGC	895	560	335	37.43
24	296170	CASE	57/M	T	DRF	O	N	NOGC	1020	650	270	36.27
25	293747	CASE	56/M	S	SRL	I	N	NOGC	1365	810		40.65
26	296068	CASE	56/M	T	DLF	O	N	NOGC	1425	950	475	33.33
27	295205	CASE	48/M	S	GLL	I	N	NOGC	1628	929	699	42.93
28	294708	CASE	53/M	T	DRF	O	N	NOGC	1224	720	504	41.17
29	291650	CASE	58/F	T	GRL	I	N	NOGC	1320	822	498	37.72
30	299525	CASE	60/M	S	GLL	O	N	NOGC	1082	642	340	31.42
31	299579	CASE	48/M	S	DLF	I	N	NOGC	1114	750	364	32.67
32	300422	CASE	52/M	T	DRF	O	N	NOGC	1125	700	425	37.77
33	300672	CASE	60/M	T	SLL	I	N	NOGC	1093	698	395	36.14
34	301520	CASE	58/M	T	DRF	O	N	NOGC	980	612	368	37.55

KEY TO MASTER CHART

SRF	: Sole of right foot
SLF	: Sole of left foot
DRF	: Dorsum of right foot
DLF	: Dorsum of left foot
GRL	: Gaiter area right limb
GLL	: Gaiter area left Limb
SRL	: Shin right limb
SLL	: Shin left limb
LMLL	: Lateral malleolus left limb
LMRL	: Lateral malleolus right limb
HRF	: Heel right foot
MMRL	: Medial malleolous right limb
ATRL	: achiles tendon right limb
I	: Insulin
O	: Oral hypoglycemic
FBS	: Fasting blood sugar
C/S	: Culture sensitivity
NOGC	: No organism grown