"A COMPARATIVE STUDY OF HYDROCOLLOID GEL AND CONVENTIONAL NORMAL SALINE DRESSINGS IN THE MANAGEMENT OF DIABETIC FOOT ULCERS"

By

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IN

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Under the guidance of

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Dr. PALREDDY AVINASH REDDY

LIST OF ABBREVATIONS

DM	Diabetic Mellitus
ECM	Extra Cellular Matrix
EGF	Endothelial Growth Factor
HCD	Hydrocolloid
PDGF	Platelet Derived Growth Factor
MRSA	Methicillin Resistant Staphalococus aureus
TGF β	Transforming Growth Factor Beta
CAM's	Cell Adhesion Molecules
TNF	Tumor Necrosis Factor
VEGF	Vascular Endothelial Cell Growth Factor
bFGF	basic Fibroblasts Growth Factor
MMP's	Matrix Metallo Proteinases

ABSTRACT

BACKGROUND AND OBJECTIVES

India is the diabetic capital of the world. It is one of the oldest known condition and having a wide spectrum of manifestations. One of the chronic manifestations are the diabetic foot ulcers. A wound care revolution is currently in the making. Many techniques have been tried over the centuries to heal chronic leg ulcers. Although wound dressings have been used for atleast two millennia, there exist no ideal dressing. During the last two decades a wide variety of innovative dressings have been introduced. Conventional dressings are cumbersome and painful. Hydrocolloids are among the most widely used modern dressings. These are easy to use, require changing only every 3-5 days and do not cause trauma on removal. This study primarily compares Hydrocolloid dressings with conventional normal saline dressings in the wound reduction, appearance of granulation tissue and infections.

METHODOLOGY

The present study is a randomized control trial. All patients admitted and treated at RLJH and teaching hospitals attached to SDUMC were included in the study. Study includes 40 study subjects and 40 controls. Study group received hydrocolloid dressings while control group received conventional normal saline dressings. Wound assessment was done on day one, second, third and after fourth week in both the groups. Percentage area of wound reduction was calculated, loss of necrotic tissue in the wound, granulation tissue filling the wound and infections noted. Statistical analysis was done using Chi square test and student t test. p value of less than 0.05 was considered significant.

RESULTS

In the last several decades, there has been a tremendous increase in the development of new active wound coverings. Years of research have shown that moist wound healing creates a more optimal healing environment. Base line characteristics like age, sex, initial wound surface area, necrotic tissue or slough covering ulcer and

granulation tissue filling wound were matched and statistically similar. The wound surface area considerably decreased in both the groups. The mean final wound area in the HCD group was 35.94 sq cm in HCD group and 40.97 sq cm in normal saline group (P=.384). Mean percentage reduction of wound areas were 43.57 in HCD group and 29.40 in normal saline group and showed statistically significant reduction of wound area (p=.000). After receiving the dressings in both groups there was considerable decrease in the necrotic tissue covering the wound by end of the study. No necrotic tissue was seen in twelve patients (40%) in HCD group and seven patients (17.5%) in Normal saline group (p=0.050). During the study there was very good progression of granulation tissue in the study group when compared to the controls. There was high statistical significance (p=0.003).Speeding up of granulation tissue thus provides faster healing and faster wound bed preparation which was shown in the study. Infections were minimal compared to the normal saline group.

CONCLUSION

- ✓ After a study on eighty patients, Hydrocolloid dressing showed faster and better healing rates.
- ✓ Considerable amount of necrotic tissue was reduced in HCD group.
- ✓ Granulation and epithelialisation appeared to occur early in ulcers treated with HCD dressings than with normal saline dressings, thus preparing the wound bed and facilitating early cover of raw area by split skin grafting.

KEY WORDS: Diabetic foot ulcers; Hydrocolloid dressing; Normal saline dressing; Wound area reduction; Necrotic tissue; Granulation tissue; Infection.

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1 INTRODUCTION

Diabetes mellitus (DM) is a chronic disorder of glucose metabolism with multisystem involvement and serious long term complications. Diabetes has been with us for long time, as far back as 1500 years B.C. and more. The symptoms of diabetes like constant thirst and urination were being written about by doctors of their time. Globally there is a notable increase in the incidence of DM. By 2030, it is estimated that the number of diabetic people will increase from 336 million currently to 552 million (8.3% of adult population).[1] Foot sepsis is a complication leading to hospitalization of a diabetic patient and often leads in amputation. Globally, over a million people lose a leg to diabetes related complications every year. In simple terms this equates to one lower limb every 30 seconds and this figure is increasing.[2] 15 to 25 % of all diabetics suffer from foot ulceration during their lifetime.[3] These ulcers take several weeks to months to heal and in some cases may never heal at all. Up to 80% of diabetic individuals with previous foot ulcerations have recurrent ulcerations over next 5 years.[4] Risk of lower limb amputation is 10-16 times greater in people with diabetes.[5] A wound care revolution is currently in the making. Many techniques have been tried over the centuries to heal chronic leg ulcers. Although wound dressings have been used for atleast two millennia, there exists no ideal dressing. During the last two decades a wide variety of innovative dressings have been introduced.[6] Conventional normal saline dressings are cumbersome and painful. Hydrocolloids are among the most widely used modern dressings. Hydrocolloids are adhesive, pliable, absorbent and waterproof wound dressings that can provide an effective barrier to micro organisms. They create a moist wound environment, facilitate autolysis and promote granulation tissue formation (Fletcher et al, 2011).

These are easy to use, require changing only every 3-5 days and do not cause trauma on removal. They have been used to prevent the spread of methycillin resistant staphylococcus aureus (MRSA).[7] Hydrocolloid dressings(HCD) can be used in a variety of wounds for example venous leg ulcers, pressure sores, superficial burn wounds, donor 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.[8] There is limited evidence within medical literature to determine whether HCDs are safe to use on the diabetic ulcer. In view of inadequate studies the following study is designed to assess the use of HCD dressings in comparison to conventional wet saline dressing in terms of duration of healing.

2 OBJECTIVES OF STUDY

1. To study the efficacy of hydrocolloid gel in the management of diabetic foot ulcers.

2. To compare the efficacy of hydrocolloid gel with conventional normal saline dressings in the management of diabetic foot ulcers in terms of duration of healing and infection.

3 REVIEW OF LITERATURE

WOUND HEALING

"Wound healing consists of a complex but very orderly array of overlapping phases in which highly specialized cells interact with an extracellular matrix to lay down a new framework for tissue growth and repair".[9]

HISTORY OF WOUND HEALING

The treatment and healing of wounds are some of the oldest subjects discussed in the medical literature and probably earliest problems of human race.[10] Empirically the ancient physicians of Egypt, Greece, India and Europe developed gentle methods of treating wounds by removing foreign bodies, suturing, covering wounds with clean material and protecting injured tissue from corrosive agents.[11]

The theory of the *"three healing gestures"* was formed more than 4000 years ago, with earliest writing recorded on a clay tablet from 2200 BC The tablet described the three gestures as *washing the wound*, *making plasters and bandaging the wound.*[12] The modern era of gentle wound care started in the mid sixteenth century when Ambrose 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 stump with dramatic results.

One of the early writings relating the concept to wounds was by James Carrick Moore, a member of the Surgeon's Company of London in 1789[13]. In his dissertation Moore states: "When any accident or disease injures the human frame, it was early observed, that the body possessed within itself, a power of alleviating or remedying the evil. John Hunter, William Steward Halsted, Alexis Carrel and 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 processes will produce the best possible result. In 1893, Lister[14] extended the earlier studies of Koch and Pasteur and demonstrated the evidence for bacterial growth in wounds and the ability of this growth to lead to abscess formation or invasive infection, sepsis and gangrene. Later Semmelweis, Ehrlich, Fleming and Florey also realized that bacteria by asepsis, antiseptics and antimicrobials heralded a new era in wound management. World War I resulted in rapid discoveries surrounding the care of wounds, the foremost among those being the use of extensive debridement.[11]

James Paget has given some scientific knowledge to their handling of wounds, particularly those resulted from war. In the early 1900's Carrel and his associates made investigations 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.[15]It was not until the 1960' s that the concept of moist wound environment for healing was considered. In addition to protecting the wound from infection, the moist environment would help to facilitate debridement, minimize inflammation, reduce pain and diminish scarring.[16]

TABLE:1 Historical aspects of topical wound agents

Agents Used	**Surgeons**
Olive oil, honeycomb, gum Arabic, incense	Johannes DeKetham, 1491
Zinc ointments, alum, sal ammoniac, turpentine	John Bell, 1810
Oil or wax, honey, sulfate, mercurial salts	Dominique Jean Larrey, 1814
Wine, lead acetate	Asley Cooper, 1825
Zinc sulfate, lead acetate, copper acetate, mercuric chloride	James Syme, 1832
Mercuric chloride, ammonium chloride, mercury and zinc cyanide, antiseptic treatment	Joseph Lister, 1884-1889
Sodium hypochlorite, epicutaneous treatment	Alexis Carrell, 1910
Silver foil, mercuric chloride, sodium hypochlorite	William Halsed,1883-1917
Sulfonamide, penicillin, sodium hypochlorite, allantoin	Hamilton Bailey, 1947
Sulfonamide, penicillin, acetic acid	George Crile, 1947

TYPES OF WOUND HEALING[17]

HEALING BY PRIMARY INTENTION

The wounds are closed after the surgery with simple suturing, skin graft or flaps.

HEALING BY SECONDARY INTENTION

There is no active intent to seal the wound. This type of repair is associated with a highly contaminated wound and will close by re epithelialization which results in contraction of the wound.

HEALING BY TERTIARY INTENTION

Also referred to as delayed primary closure. A contaminated wound is 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 is performed.

PHASES OF WOUND HEALING

There are four distinct but overlapping phases of wound healing.They are hemostasis, inflammation, proliferation and remodeling .**(Fig.** 1) The phases are influenced by the various cellular interactions and are regulated by the local release of chemical signals such as cytokines, chemokines, growth factors and inhibitors.[18,19]

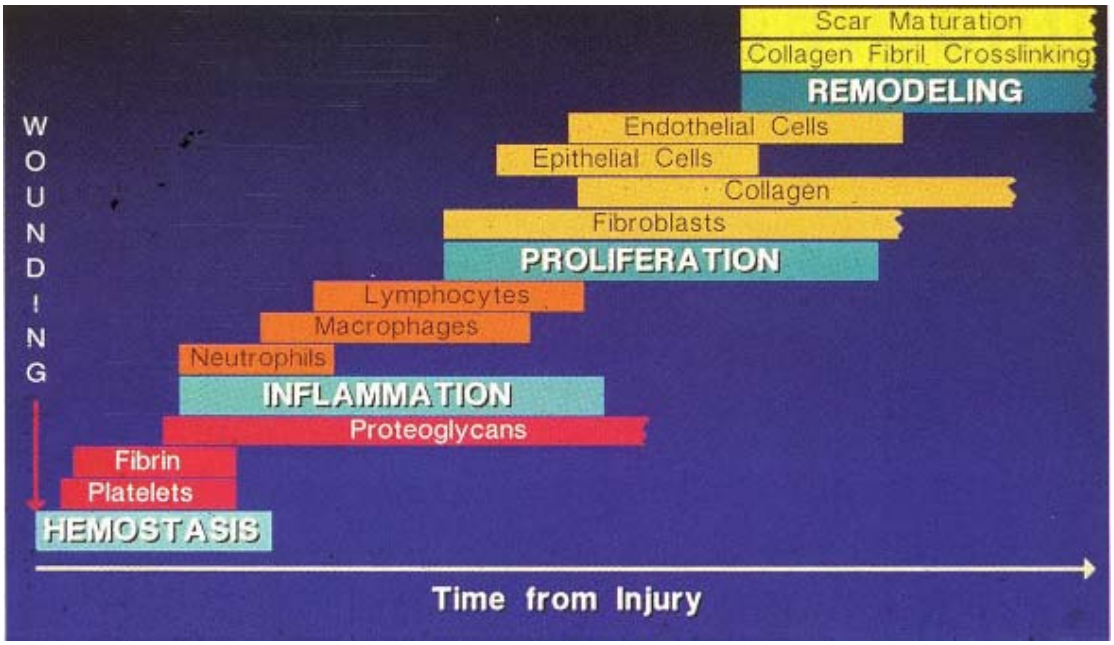


Fig. 1. Phases of normal wound healing. Cellular and molecular events during normal wound healing progress through four major integrated phases: hemostasis, inflammation, proliferation and remodeling.

HEMOSTASIS PHASE

Immediately after tissue injury hemostasis occurs to control hemorrhage. While the blood vessels constrict platelets are activated by binding to the exposed collagen in the extracellular matrix. The platelets then release fibronectin,

thrombospondin, sphingosine-1 phosphate and Von Willebrand factor which promote further platelet activation and aggregation.[20]

As these activation and other clotting factors are released a fibrin matrix is deposited in the wound which functions as a provisional matrix to stabilize the wound site. The aggregated platelets then become trapped in the fibrin matrix, thus forming a stable clot within the provisional matrix (Fig. 2).[21]

Several important mediators that are released by platelets are responsible for the initiation and progression of wounds through the subsequent phases of wound healing. These mediators include platelet-derived growth factor (PDGF) and transforming growth factor beta (TGF-B).TGF-B and PDGF recruit additional cells such as neutrophils and macrophages to enter the wound. PDGF also recruits fibroblasts to the wound and activates the production of collagen and glycosaminoglycans by fibroblasts, which are important for the repair of the extracellular matrix.[18,19,22] Excessive levels of these growth factors have been indicated in conditions of abnormal wound healing.

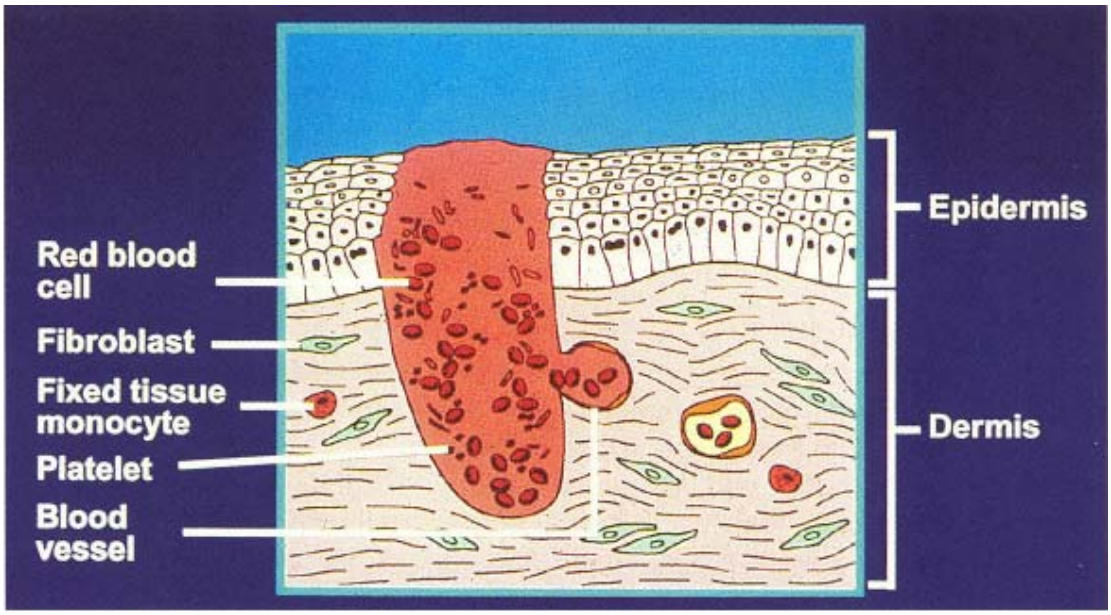


Fig. 2. Hemostasis phase. At the time of injury, the fibrin clot forms the provisional wound matrix and platelets release multiple growth factors that initiate the repair process.

INFLAMMATORY PHASE

The next phase of wound healing is inflammation which begins within the first 24 hours after an injury.

The stage can last up to 2 weeks in patients whose wounds are healing appropriately but can last longer in those patients with chronic non healing wounds. The mast cell mediators cause surrounding vessels to become leaky and thus allow the movement of neutrophils from the vasculature to the site of injury. In addition to mast cells neutrophils and macrophages play key roles in the inflammatory phase (Fig. 3).

As various chemical signals are released from the wound site the endothelial cells in the nearby vessels are activated and begin to express specialized cell adhesion molecules (CAMs) called selectins.

These CAMs function as molecular hooks to grab circulating neutrophils to bind to the endothelial cell surface by a process called pavementing. The adherent neutrophils begin to roll along the endothelial cell lining and then by a process called diapedesis they squeeze through the cell junctions that have been made leaky by the mast cell mediators.[23,24] The neutrophils are attracted to the site of injury by a process called chemotaxis and are drawn there by soluble mediators, such as a breakdown product of a complement called C5a and the potent chemokine interleukin (IL)-8.[25-27] On their arrival at the wound site the neutrophils begin to aggressively phagocytize any foreign materials and kill bacteria by the powerful battery of enzymes and reactive oxygen species which they can generate. The neutrophils actually initiate the first stages of the proliferative phase by releasing IL-1 and tumor necrosis factor (TNF)-a to begin the activation of fibroblasts and epithelial cells. During the inflammatory phase activated wound macrophages also play a key role in the regulation and progression of wound healing.

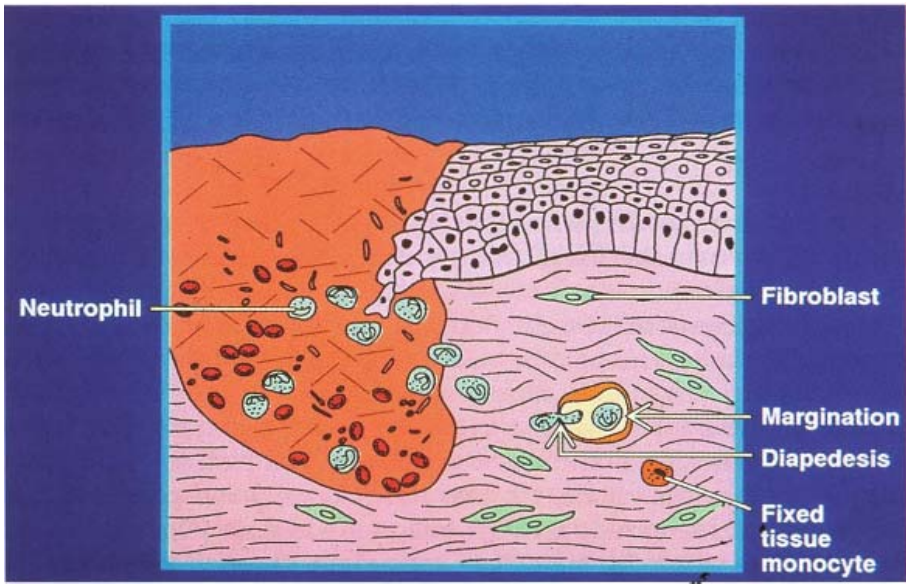


Fig.3. Inflammatory phase. Within a day after injury the inflammatory phase is initiated by Neutrophils that attach to endothelial cells in the vessel walls surrounding the wound(margination) change shape and move through the cell junctions (diapedesis) and migrate to the wound site.

PROLIFERATIVE PHASE[28]

The proliferative phase is characterized by fibroblast proliferation and collagen deposition to replace the provisional fibrin matrix and to provide a stable extracellular matrix at the wound site. The new matrix consists of collagen, proteoglycans and fibronectins. In addition angiogenesis occurs such that new blood Vessels replace the previously damaged capillaries and provide nourishment for the matrix. Granulation tissue formation and the process of epithelization also occur. Once the fibroblasts have entered the wound they produce collagen, proteoglycans and other components. Fibroblast activity is predominately regulated by PDGF and TGF-B. PDGF secreted by platelets and macrophages stimulates fibroblast proliferation, chemotaxis and collagenase expression.(Fig.4)

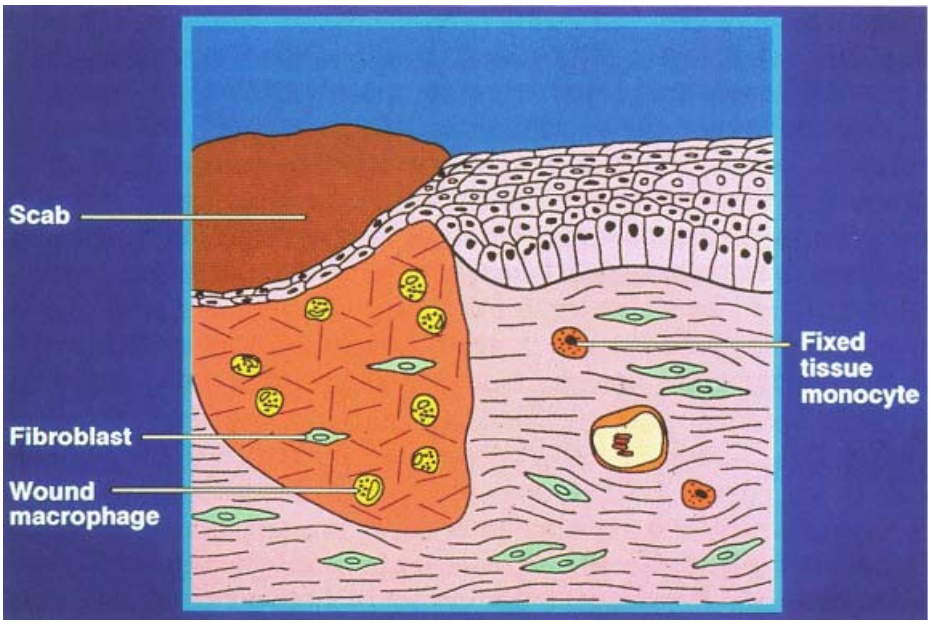


Fig.4. Proliferation phase. Fixed tissue monocytes become activated move into the site of injury, transform into activated wound macrophages that kill bacteria, release proteases that remove denatured extracellular matrix and secrete growth factors that stimulate fibroblast, epidermal cells and endothelial cells to proliferate and produce scar tissue.

Endothelial cells are activated by TNF-a and basic FGF (bFGF) to initiate angiogenesis such that new blood vessels are initiated to promote blood flow to support the high metabolic activity in the newly deposited tissue. Angiogenesis is regulated by a combination of local stimulatory factors such as vascular endothelial cell growth factor (VEGF) and antiangiogenic factors such as angiostatin, endostatin, thrombospondin and pigment epithelium-derived growth factor. As the wound continues to heal, the granulation tissue forms to provide the transitional replacement for normal dermis and ultimately evolves into a scar.

REMODELING PHASE

The last phase of wound healing is the remodeling phase in which granulation tissue matures into a scar **(**Fig. 5). Small capillaries aggregate into larger blood vessels and there is an overall decrease in the water content of the wound. Similarly cell density and overall metabolic activity of the wound decrease. Initially there is increased deposition of type III collagen also referred to as reticular collagen that is gradually replaced by type I collagen, the dominant fibrillar collagen in skin.[29] As the wound continues to remodel, changes in collagen organization increases the tensile strength to a maximum of about 80% of normal tissue. Matrix metallo proteinases(MMPs) control the degradation of extra cellular matrix components to facilitate epithelial cell migration into the wound, angiogenesis and overall tissue remodeling.

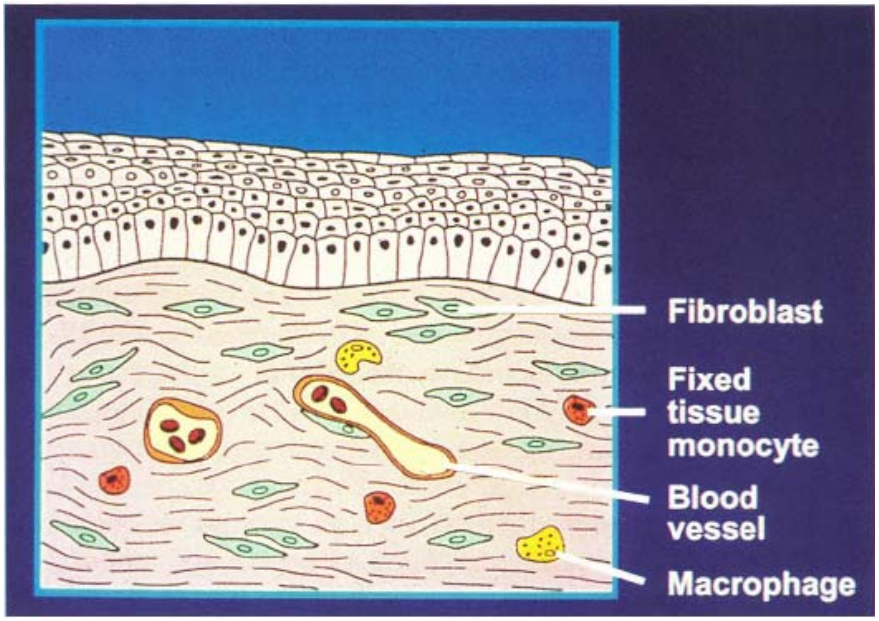


Fig. 5. Remodeling phase. The initial disorganized scar tissue is slowly replaced by a matrix that more closely resembles the organized extracellular matrix of normal skin.

FACTORS AFFECTING WOUND HEALING

LOCAL FACTORS:

- **Type, size and location of the wound:** A surgical wound heals faster. Injuries in richly vascularised areas (e.g., the face) heal faster than those in poorly vascularised ones (e.g., the foot).
- **Vascular supply:** Wounds with impaired blood supply heal slowly. For example, the healing of venous ulcers is prolonged. Ischemia caused by arterial obstruction in the lower extremities of diabetics also prevents healing.
- **Infection:** Wounds provide a portal of entry for microorganisms. Infection delays or prevents healing, promotes the formation of excessive granulation tissue and may result in scars.
- **Movement:** Early mobility particularly before tensile strength prevents or retards healing.
- **Ionizing radiation:** Previous irradiation interferes with blood supply and result in delayed wound healing. Acute irradiation of a wound blocks cell proliferation, inhibits contraction and retards the formation of granulation tissue.

SYSTEMIC FACTORS:

- **Regional vascularity:** The vascularity of the area surrounding the wound is important. Impaired perfusion results in poor healing.
- **Infections:** Delay wound healing.
- **Metabolic status:** Diabetes mellitus is associated with delayed wound healing because of increased risk of wound infection and angiopathy.

- **Nutrition:** Malnutrition impairs wound healing process. Methionine and Zinc are needed for proper healing. Vitamin C, required for collagen synthesis and secretion, if deficient results in impaired wound healing.
- **Hormones:** Corticosteroids delay wound healing by inhibition of collagen synthesis, anti- inflammatory actions and depression of protein synthesis. Thyroid hormones, androgens also influence healing.

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".[30]

CLASSIFICATION[30]

Type 1

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 2

Heterogeneous group of disorders characterized by

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

GESTATIONAL DIABETES

DIABETIC FOOT

Foot ulcers and infections are commonest among diabetic patients. Foot ulceration is the most common single precursor to lower extremity amputations among persons with diabetes[31-33]

PATHOPHYSIOLOGY OF DIABETIC FOOT

There are 4 main causes for development of foot lesion in a diabetic.

1) Peripheral Neuropathy. 2) Peripheral Vascular disease (PVD).

3) Charcot foot. 4) Infection.

1) NEUROPATHY34: The commonest Diabetic polyneuropathy is distal symmetrical neuropathy, the main initiating factor for foot ulceration, affecting about 30% of all diabetic people.[35,36]

Age, duration of diabetes and poor glycemic control are major determinants.[37]

HYPOTHESES FOR THE PATHOGENESIS OF DIABETIC PERIPHERAL NERVE DAMAGE:

1)Chronic hyperglycemia 2) Oxidative stress.

3) Increased polyol pathway flux. 4) Nonenzymatic glycation.

5) Neurotrophic factors. 6) Protein c kinase activation.

7)Vascular factors.

2) DIABETIC ANGIOPATHY[38,39]

Disease of blood vessels is a major cause of complications in diabetics.The vascular lesions are:

I. Macro vascular disease.

a) Atherosclerosis

b) Medial calcification

c) Diffuse intimal fibrosis

II. Micro vascular disease.

a) Arteriosclerosis

b) Specific Diabetic microangiopathy

c) Diabetic Fibrosis

3) CHARCOT FOOT [40, 41]

Bone and joint damage in the tarsometatarsal joints and mid-tarsal joints leads to two classical deformities.

a) Rocker bottom deformity: displacement and subluxation of the tarsus downwards.

b) Medial convexity: results from displacement of the talonavicular joint or from tars metatarsal dislocation.

Both are often associated with a bony prominence which is very prone to ulceration and healing difficult.

4) INFECTION

Infection of the plantar space accounts for majority of diabetic foot infections. Majority of infections start with infected ulcers on the plantar aspect of the foot or interdigital infections and nail bed infections. In the initial stage there is usually an area of localised cellulitis, then infection progresses and once deep space infection is established ascending cellulitis, lymphangitis and lymphadenopathy also develop.

Table:2 Risk factors for ulceration[42-44]

SL NO	SYSTEMIC FACTORS	LOCAL FACTORS
1	Uncontrolled hyperglycemia	Peripheral neuropathy
2	Duration of diabetes	Structural foot deformity
3	Peripheral vascular disease	Trauma
4	Blindness or visual loss	Improper foot wear
5	Chronic renal disease	callous
6	Older age	Limited joint mobility

CLASSIFICATION OF ULCERS

Although no single system has been universally adopted,the classification system most often used was described and popularized by Wagner.[45]

Table 3: WAGNER Grading of ulcers

GRADE	LESION
0	No open lesions,may have deformity or cellulitis
1	Superficial ulcer
2	Deep ulcer to tendon or joint capsule
3	Deep ulcer with abscess,osteomyelitis or joint sepsis
4	Local gangrene-fore foot or heel
5	Gangrene of entire foot

TISSUE MANAGEMENT AND WOUND BED PREPARATION

The concept of wound bed preparation originated from classical principles of Guy de charliac and Pare.[46]

It got organized during 1980's.[47] Wound bed preparation includes debridement of non viable tissue and denatured extracellular matrix,control of bacterial burden and inflammation, establishment of optimal moisture balance and stimulation of epidermal cell migration of wound edge. It became popular under acronym "TIME".[48,49]

T-removal of non viable tissue.

I-control of infection.

M-maintaining moisture balance.

E- deals with advanced wound edge.

In acute wounds, debridement is done to remove devitalized tissue and bacteria.Later it is followed by treating underlying comorbidities and thus clean acute wound heals readily.

DEBRIDEMENT AND CLEANSING

Debridement is removal of all dead (devascularised, necrotic, infected tissue or foreign material) from the wound. Many ulcers present with a clean base but some may require debridement when there is adherent slough and tissue that impairs epithelisation, granulation and wound contraction .

TYPES OF DEBRIDEMENT INCLUDE

1. SURGICAL DEBRIDEMENT: The removal of nonviable, contaminated and infected tissue using surgical technique under anaesthesia. Surgical debridement converts indolent wounds into acute wounds and restores circulation and allows oxygen delivery to the wounds. Used in wounds with large amount of necrotic or infected tissue. It is performed by skilled medical personal.

2. ENZYMATIC DEBRIDEMENT

Specific proteolytic enzymes are applied to the wound to remove and digest necrotic tissue and dissolve the devitalized tissue. It can be used in patients receiving anticoagulants and contraindicated for surgery.

Advantage is it can be done as outpatient dressing. Most common types are: Collagenase (Santyl) and Papain-Urea.Collagenases are enzymes that are isolated from *Clostridium histolyticum*. These have high predisposition for the major collagen types (I and II), but they not active against keratin, fat or fibrin.[50-52]

Papain, obtained from the papaya plant, is effective in the breakdown of fibrinous material and necrotic tissue. When combined with urea, it denatures nonviable protein matter.

3. MECHANICAL DEBRIDEMENT

It is a nonselective, physical method of removing necrotic tissue.It includes wet-to-dry dressings and high- pressure irrigation or pulsed lavage and hydrotherapy.[53,54] Wet-to-dry is one of the most commonly prescribed and overused methods of debridement in acute care settings.

Hydrotherapy in the form of whirlpool may remove surface skin, bacteria, wound exudates and debris.

This modality is good for larger wounds with a significant amount of devitalized tissue. Used when the local bacterial burden is more of a concern than stimulation of healing in a non-healing wounds.

Disadvantage: Painful and non-selective (removes both healthy and necrotic tissue).

4. AUTOLYTIC DEBRIDEMENT

Seen with the use of dressings such as hydrocolloids and hydrogels. Occurs naturally in a healthy, moist wound environment when arterial perfusion and venous drainage are maintained.

5. CHEMICAL DEBRIDEMENT

Specific chemicals are used to remove necrotic tissue

- Povidone iodine-Broad spectrum antimicrobial activity.
- Acetic acid(O.5%-5%)- Effective against Pseudomonas species.
- Chlorhexidine- Active against gram-positive and gram-negative organisms.
- Hydrogen peroxide- Desloughing agent with some bactericidal effect.
- Sodium hypochlorite solution-High pH causes irritation to skin.

6. BIOLOGICAL (LARVAL) THERAPY

Larval therapy utilizes the sterile form of the *Lucilia sericata* blowfly for the debridement of necrotic and infected wounds. Maggots secrete a powerful proteolytic enzyme that liquefies necrotic tissue.[55,56] It has been noted that wound odor and bacterial count, including methicillin-resistant Staphylococcus aureus, diminish significantly with larval therapy. Larval therapy seems to be beneficial, but there is paucity of controlled studies to support its routine use in the diabetic foot wounds.

WOUND DRESSINGS AND WOUND CARE PRODUCTS

For centuries,wound healing was considered as a series of mysterious mechanisms and numerous topical treatments have been applied to wounds like honey, diluted wine, flowers etc. Till many years commonly used dressings were pads or strips of linen. In 1962,Winter's work on moist wound healing was the major achievement in wound care dressings.

Wound dressings are categorized into two :

1)Primary dressings: Also known as contact layer dressings. These directly interact with the wound surface and promote healing.

2)Secondary dressings(Passive dressings): Dressings covering the primary dressing.

Today many wound care products are present in the market which need to be tailored for our use.

The principal characteristics of modern dressings:

1) Maintain moist environment at the wound surface and improve cell migration.
2) Absorb excess exudates
3) Allow painless removals
4) Being impermeable to bacteria.

SELECTION OF WOUND DRESSINGS

Before choosing a wound dressing a thorough wound assessment should be done and it should be tailored to the patients wound to influence wound healing.

The main criteria before choosing a wound dressing are:

1) Clinical appearance of the wound, healing phase, etiology and size of wound.
2) Amount of exudates.
3) Presence of infection.
4) Depth of wound
5) Condition of surrounding skin
6) Dressing availability and cost
7) Patient acceptability.

WOUND CARE DRESSINGS

Based on the type of material used for the preparation of dressing they may be classified as conventional, synthetic and biological dressings.

A. CONVENTIONAL DRESSINGS

These dressing materials are made up of fabric material such as gauze.They evaporate moisture resulting in a dry desiccated wound bed and allow entry of exogenous bacteria into the wound. This led to the origin of compound dressings such as Tulle grass which is a wide mesh gauze impregnated with medical grade paraffin. They are relatively non-adherent.

ACTIONS OF NORMAL SALINE

Normal saline dressing keeps the environment moist for proper healing. They act 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.[57]

Since the conventional dressings had limitations for application on full thickness wounds, research into the development of more advanced wound dressings for the treatment of wounds has resulted in the development of synthetic and biological dressings.

B. SYNTHETIC DRESSINGS

THESE DRESSINGS CAN BE CLASSIFIED INTO

1. **Films -** They are homogeneous dressings composed of a polymer sheet coated on one side with an adhesive. The most commonly used Polymers include poIyurethane, poIyethyIene, Polycapro Iactone, poIytetra -fluoroethy Iene, dimethyl aminoethyl methacrylate. Film dressings are well suited for superficial wounds.

2. **Foams and sprays -** Foam dressings are sheets of foamed solution of polymers such as polyvinyl-alcohol and polyurethane which are superior to film dressings in that they provide thermal insulation and help to maintain a moist environment at the surface of the wound. They are gas permeable, non-adherent, light and comfortable. Examples are silastic foam and lyofoam. However,these dressings are difficult to use in certain anatomical areas. Spray dressings are more comfortable to the wounds surface and they are totally portable .Most sprays are copolymers e.g., Aeroplast is a copolymer of hydroxy vinyl chloride acetate modified maleic resin ester. Now a days dressings composed of spray and foam combinations are available e.g., gelatin based sprayable foam.

3. **Composite Dressings -** These are composed of laminates of two or more layers.

   The outer layer is designed for durability and elasticity and may serve as a rate controller for water evaporation, while the inner layer is designed for maximum adherence and elasticity.

COMPOSITE DRESSINGS MAY BE CLASSIFIED AS FOLLOWS:

HYDROCOLLOID DRESSINGS - Hydrocolloids are adhesive, pliable, absorbent and waterproof wound dressings that can provide an effective barrier to microorganisms. These dressings are compound formulations containing a cocktail of elastomeric adhesive and gelling agents. Carboxy methyl cellulose is the most common absorptive ingredient acting as absorbent for wound fluid.Other substances used are gelatin and

pectin. They create a moist wound environment,facilitate autolysis and promote granulation tissue formation[58]. The dressings are capable of absorbing low to moderate levels of exudate and can be used to promote autolytic debridement of dry, sloughy, or necrotic wounds.[59]Thinner versions are generally used on wounds that are dry or have low levels of exudates . Additionally hydrocolloids are suitable for promoting granulation tissue.[58] There have been reports of hypergranulation with prolonged use of hydro colloids in moderate to highly exudating wounds so wound tissue assessment is paramount when applying hydrocolloids for long periods.[60]

Key properties of Hydrocolloids[61]

1. Provide an occlusive bacterial and viral barrier – reduce the risk of cross infection.
2. Lower the wound PH – reduce the ability of bacteria to proliferate.
3. Maintain moisture at the wound bed – allow for faster epithelialisation and lower levels of pain.
4. Prevent desiccation of the wound bed – provide a moist wound healing environment.
5. Hydrocolloids are easy to remove and may reduce pain while removing dressing.

6. Hydrocolloids may help to reduce costs as they can be left in situ for up to seven days providing there is no excessive exudate or infection present. On average they stay in place for 3–5 days.

Moisture under occlusive dressings such as hydrocolloids can help to promote angiogenesis, increase the number of dermal fibroblasts, stimulate the production of granulation tissue, and increase the amount of collagen synthesised . In the presence of wound exudate the hydrocolloid forms a hydrophilic gel that facilitates autolytic debridement of the wound by gently softening and rehydrating necrotic tissue and slough .[62] As the gelling process takes place, the dressing becomes progressively more permeable resulting in the loss of water vapour through the dressing and increases the ability of the product to absorb exudate .

HYDROGEL DRESSINGS - Hydro gels are designed to hydrate wounds, rehydrate eschar and aid in autolytic debridement. Hydrogels are insoluble polymers that expand in water and are available in sheet, amorphous gel or sheet hydrogel-impregnated dressings. These are sheets of 3-D networks of cross linked hydrophilic polymers. They interact with aqueous solutions They provide a moist environment for cell migration and absorb some exudate. Autolytic debridement without harm to granulation or epithelial cells is another advantage of hydrogel dressings.Amorphous hydrogels are applied liberally on to or in to a wound and covered with a secondary dressing such as foam or film. Hydrogels can remain in situ for up to three days.

Owing to their unique cooling ability, they may be of great benefit for use as a first aid measure for thermal burns.

ALGINATE DRESSINGS

Alginates (calcium or calcium/ sodium) are highly absorbent, biodegradable dressings derived from Seaweed[63]. An active ion exchange of calcium ions for sodium ions at the wound surface forms soluble sodium alginate gel that provides a moist wound environment. Calcium dressings need moisture/ exudate from the wound to function, therefore they are not suitable for dry wounds or wounds with hardened eschar. They may precipitate an inflammatory reaction as it stimulates a foreign body response. Caution is also needed when using alginate rope dressings in very deep or narrow sinuses, as complete removal can be difficult. Alginate dressings are available in sheet, ribbon or rope form in various sizes and require a secondary dressing.

HYDROFIBRE DRESSINGS

Hydro fibre dressings are non-woven sodium carboxy methyl cellulose spun into fibres and manufactured into sheet or ribbon packing dressings. Aquacel, a hydro fibre dressing, maintains a moist wound healing environment as fibres convert to a gel on contact with exudate. The vertical wicking of exudates reduces maceration of surrounding skin. The dressings are more absorbent than alginates and to promote non-traumatic dressing removal.

SILICONE DRESSINGS

Silicones are polymers with a structure that consists of alternate atoms of silicone and oxygen with organic groups attached to the silicone atoms. The degree of polymerisation determines the various physical forms of the silicone. A soft silicone dressing is coated with soft silicone as an adhesive or a wound contact layer. The intrinsic properties of soft silicone provide gentle adhesion and minimises wound and

surrounding skin trauma at a dressing change.[64] Soft silicone is not intrinsically absorbent but it can be applied as a facing layer to dressings containing absorbent components that are used for the management of exuding wounds.[65,66] Soft silicone dressings have been shown to reduce wound pain. Soft silicones also have a role in scar management and are used in the treatment of hypertrophic and keloid scars.An international advisory group of scar management experts have recently published evidence based clinical recommendations that support the use of silicone gel sheeting as a first-line therapy on immature, linear and widespread burn hypertrophic scars and minor keloids[64].

C. BIOLOGICAL DRESSINGS

These are derived from natural tissues usually consisting of various formulations and combinations of collagen, elastin and lipid. They are far superior to synthetic dressings. Biological dressings range from allograft, heterografts from pigs, dogs and other species, to embryonic membranes, embryo foetus and neonatal skins.Films of reconstituted collagen from bovine and other sources, fibrin, cultured epidermal grafts, dermal matrix grafts and cultured dermal matrix composite grafts are also included.

1) ALLOGRAFT:

Allograft skin normally can be obtained from a family member but is most commonly harvested from cadavers. The use of fresh frozen lyophilized allograft is most effective for thermal injuries especially for extensive full thickness burns. Amniotic membranes have also been used as allograft, but are ineffective as they lack prevention of evaporative water loss leading to wound dehydration.

2) XENOGRAFT:

These are grafts from animal sources and porcine skin is the most commonly used xenograft owing to structural similarity to human skin. The major disadvantages of xenograft and allograft are they need immunosuppression to prevent rejection.Further, the excessive use of immunosuppressive drugs increases the risk of wound infection.

Major disadvantages of using these include cost factors,short half life and potentially antigenic.

3) COLLAGEN DRESSINGS

Collagen is the unique, triple helix protein molecule, which forms the major part of the extracellular dermal matrix (ECM), together with the glycosaminoglycans, proteoglycans, laminin, fibronectin, elastin and cellular components.[67,68] Collagen dressings developed due to structural and functional characteristics.

The packing arrangement for molecules within collagen fibrils imposes several structural requirements on the molecule participating in the construction of such aggregates. In addition to providing mechanical support to the connective tissue, the collagens also form an essential substrate for cellular adhesion and migration.

Therefore, collagen is considered to be an important morphogenetic factor in embryonic development and in the regenerative process.

4) ENGINEERED SKIN SUBSTITUTES

Bioengineered tissues have been shown to significantly increase complete wound closure in venous and diabetic foot ulcers.[69,70] Currently, two bioengineered tissues have been approved to treat diabetic foot ulcers Apligraf™ and Dermagraft™

Both have demonstrated efficacy in randomized controlled trials. Tissue-engineered skin substitutes can provide the cellular substrate and molecular components necessary to accelerate wound healing and angiogenesis. They function both as biologic dressings and as delivery systems for growth factors and extracellular matrix components .[71,72]

Extracellular matrices (nonliving) are generally derived from devitalized tissue to produce an immunologically inert acellular dermal matrix.

1) Dermal regeneration template (Integra™)
2) Allogenic dermal matrix (AlloDerm™)
3) Matrix of human dermal fibroblasts(TransCyte™)
4) Porcine small intestine submucosa (Oasis™)

They are composed of structural cellular components and growth factors utilized to promote natural tissue remodeling .[73,74] Integra™ dermal regeneration template, a collagen-chondroitin sponge overlaid with silicone originally developed for burns, has been shown to be ideally suited to chronic and pathologic wounds .

OTHER TOPICAL AGENTS

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.

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 high-molecular weight polysaccharide synthesized in the plasma membrane of fibroblasts and other cells.

Beta Glucan: It is a major cell-wall carbohydrate extracted from grains as oats and barley. Beta glucan is thought to increase macrophage infiltration, speeding the onset of fibroplasias and fibrogenesis, stimulation of increased tissue granulation, and enhanced re epithelialisation.

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.

L-LYSINE HYDROCHLORIDE[75]

Lysine (abbreviated as Lys or K) is an essential amino acid. L-Lysine Hydrochloride has shown improvement in both the rate and quality of wound healing. Another feature of this molecule is its ability to support healing process in long standing wounds. On histopathology, lysine treated wounds showed a thickening of the dermo-epidermal layer, with increased cell proliferation from the basal keratinocytes.L- lysine monohydrochloride (L-Iysine) has been shown to promote therapeutic angiogenesis in wound healing.

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 then with nutritiona interventions alone.[76]

HONEY DRESSINGS

Medical role of honey were known since ancient past. Dressings promote moist wound healing, autolytic and osmotic debridement and have antimicrobial activity.It is due to the slow release of low levels of hydrogen peroxide.

Table 4: Summarising wound care products

Dressing material	**Trade name**	**Remarks/Uses**	**Contraindications**
Semi-permeable films	Aqua protect film, Bioclusive, Cutifilm, Hydrofilm, Opsite (Flexigrid, Flexifix, Post-Op), Polyskin, Tegaderm.	Non-absorbent; superficial burns,grazes,closed surgical incisions, small skin tears.	Infection,significant drainage,over prominence or friction
Foams	Allevyn,Cavi-care,Curafoam, Hydrosorb,Lyofoam,PermaFoam, Tegafoam, Truefoam.	Moderate to heavily exudating, superficial and cavity wounds, venousulcers,infected ulcers, skin tears, pressure ulcers, skin grafts or donor site.	Dry wounds.
Alginates	AlgisiteM,Algoderm,Comfeel SeaSorb,Curasorb,Kaltostat, Melgisorb, Sorbsan	Requires exudate to function. Heavily exudating leg ulcers, pressure ulcers.	Minimal drainage or dry wounds
Hydrocolloids	Comfeel (ulcer dressing, transparent, contour dressing), CombiDERM, **DuoDERM** (extra thin, CGF, paste), Hydrocoll, RepliCare, Tegasorb	Light to moderately exudating wounds.Causes autolytic debridement. Leg ulcers, pressure ulcers, burns and donor sites.	Heavy drainage and sinus tracts.

Hydrogels	Aquaclear, Purilon Gel (amorphous), DuoDERM Gel (amorphous unpreserved), Hypergel (hypertonic saline, amorphous), Intrasite Conformable (gauze impregnated), Intrasite Gel (amorphous unpreserved), Nu-gel, Second skin, SoloSite Gel.	Minimally exudating or dehydrated wounds such as minor burns, grazes, lacerations, donor sites and pressure ulcers. Used for rehydrating eschar prior to debridement. The thinner viscosity products are useful for soothing burns and acute lesions such as chicken pox.	Moderate or heavy drainage.
Hydrofibre	Aquacel, Aquacel Ag	Heavily exudating wounds such as dehisced abdominal or pelvic wounds,chronic leg ulcers and infected wounds.	Dry wounds
Silicone	Mepitel (non-adherent), Mepilex (non-adherent, thin, absorbent, border, transfer), Mepitac (fixation tape).	Painful wounds, skin tears, difficult wound. Mepitel can be reused, and is usually used under another dressing to reduce pain on dressing changes.They soften and flatten scar tissue and can be washed and reused. Large sizes are also useful under secondary dressings for cancer wounds.	
Silver	Acticoat. Absorbent (calcium -alginate), Actisorb 220 (charcoal impregnated), Aquacel Ag (hydrofibre), Atrauman Ag (wound contact tulle),Avance, Contreet (hydroactive), Contreet-H (hydrocolloid), PolyMem Silver	Wounds having high microbial burden and moderate to high exudate. Useful in partial and full thickness wounds (burns, donor sites).	Healthy granulating Wounds.

ADVANCED WOUND CARE MODALITIES

1. Growth Factors

Chronic ulcers have demonstrated benefit from autologous platelet releasates or geneticallyengineered products such as recombinant DNA plateletderived growth factor becaplermin gel (Regranex™)[77]

2. 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 canister.[78]

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 tension which improve healing mechanisms•[79]

TOPICAL HYPERBARIC OXYGEN THERAPY:

The therapy is based on achieving an atmospheric pressure of 1.02 to 1.03 atm pressures which stimulates fibroblast growth, collagen formation and neo-angiogenesis.It also provides a lethal environment for anaerobes .Topical hyperbaric oxygen is administered using a sealed polyethylene bag over the affected area and administering 100 percent oxygen to a pressure between 20 and 30 mmHg.[80]

4 MATERIALS AND METHODS

The present study was carried out at Department of Surgery, Sri Devaraj Urs medical college, R.L.Jalappa Hospital and Research centre and teaching hospitals attached to SDUMC. Period of study was from February 2012 to September 2013.

STUDY DESIGN

The prospective randomized clinical trial was conducted on patients with diabetic ulcers of lower extremities.

SOURCE OF DATA

Diabetic patients with ulcers of lower limb extremities admitted at Department of Surgery, Sri Devaraj Urs medical college and R.L.Jalappa Hospital and Research centre,Tamaka,Kolar and teaching hospitals attached to SDUMC.

SAMPLE SIZE

The study comprised of 80 patients.40 were subjects receiving hydrocolloid dressings and 40 controls receiving conventional normal saline dressings.

SELECTION CRITERIA

INCLUSION CRITERIA.

1. All patients with type 2 diabetes mellitus with diabetic foot ulcers of Wagners grade 1 and 2.

EXCLUSION CRITERIA

1. Patients with chronic venous insufficiency of lower limbs with dermal changes and lymphedema.
2. Patients with uncontrolled diabetes with severe co morbid medical conditions.

METHODOLOGY

- The study was approved by the Ethical and Research Committee of Sri Devaraj Urs Medical College,Kolar.
- 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.
- The selected patients underwent appropriate treatment for a period of one to two weeks.

  This was to stabilize the wound and institute appropriate medical and surgical treatment.

  It included diabetic control, control of infection by appropriate antibiotics which were based on culture and sensitivity reports.
- Also surgical debridement and correction of other medical illness were considered.
- After the initial treatment period the eligible patients were divided randomly into test group and controls.
- GROUP 1:All patients with odd numbers categorized into group 1 who received hydrocolloid gels.
- GROUP 2:All patients with even numbers categorized into group 2 who formed control group.
- 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,random blood sugar, blood urea and serum creatinine and culture of the ulcer were recorded on predesigned and pretested proforma (Annexure-I).

- 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 ASSESSMENT

- 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).
- The dressing was changed every third day or early if mandated.
- Wounds were inspected for :
    1. Presence of slough as percentage of total ulcer surface area.
    2. Progress of granulation tissue as percentage of total ulcer surface area.
    3. Ulcer size as change in surface area and reduction percentages.
    4. Wound bed preparation for skin grafting.
    5. Infection of wounds.
- The amount of nonviable tissue, degree of wound granulation and overall wound response was evaluated on baseline, one week, two weeks, three weeks and four weeks.
- The visual scores for the percentage of wound covered with necrotic tissues are-
    1. 76-100% of wound covered by necrotic tissue.
    2. 51-75 of wound covered by necrotic tissue.
    3. 26-50% of wound covered by necrotic tissue.
    4. 11-25% of wound covered by necrotic tissue.
    5. 0-10% of wound covered by necrotic tissue.
    6. No necrotic tissue covering the ulcer.

- The visual scores for the percentage of wound filled by granulation tissues are-

    1) No granulation tissue covering the ulcer.
    2) Pink/dull <25% wound filled.
    3) Bright beefy 25-74% wound filled.
    4) Bright beefy red 75-100% wound filled.

- The reduction of wound size area was measured in sq cm .
- We have applied the following formula to calculate % reduction in area of wound after four weeks period in both cases and controls.

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

- The results obtained were statistically evaluated and the main parameters were analyzed by Chi square and student t test. p value of <0.05 was considered significant. SPSS software version 16 and Open Epi info software version 2.3 were used.

5 RESULTS AND OBSERVATIONS

The present study was conducted in Sri Devaraj Urs Medical College, R.L.Jalappa Hospital and Research centre and teaching hospitals attached to SDUMC and the findings are tabulated as below. During the study year from February 2012 to September 2013, 80 diabetic patients with ulcers of the lower limb were randomized into study (HCD dressings) and control (normal saline dressings) groups. These groups were studied for the effect of HCD dressings versus normal saline dressings.

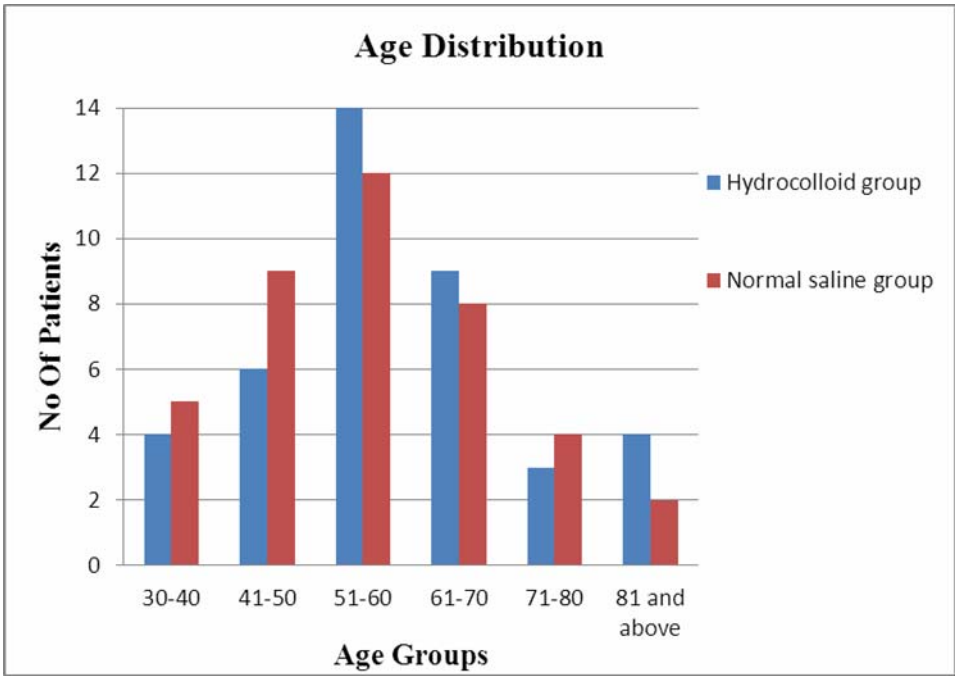
AGE DISTRIBUTION

Out of total of 80 patients, 40 patients received HCD dressings and the other 40 received normal saline dressings. The patients falling into respective age groups are as follows:

Table: 5 Age distribution in hydrocolloid and normal saline group

Age Groups	Hydrocolloid group	Normal saline group
30-40	4	5
41-50	6	9
51-60	14	12
61-70	9	8
71-80	3	4
81 and above	4	2

Graph1: Age distribution in hydrocolloid and normal saline groups



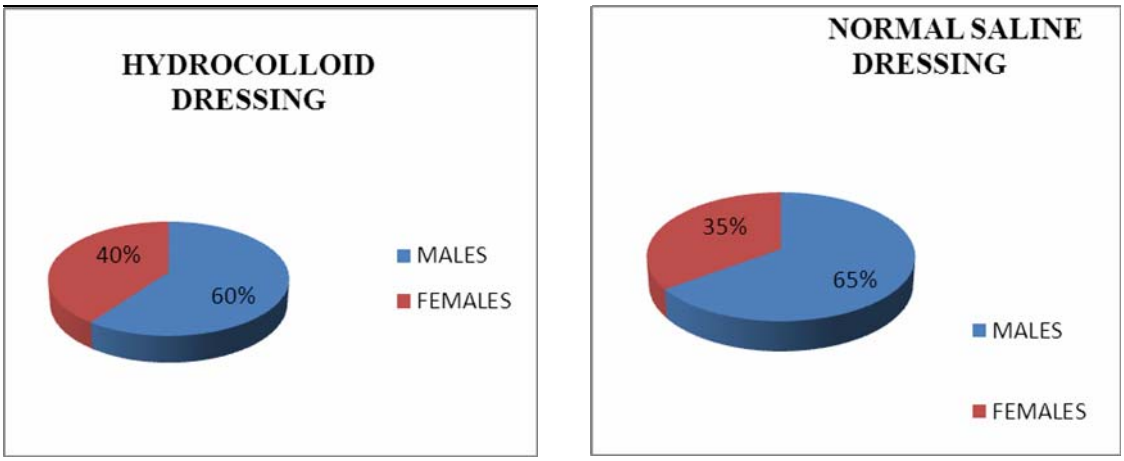
SEX DISTRIBUTION

Out of total of 40 patients receiving HCD dressings, 24 were males and 16 were females and of the 40 patients receiving normal saline dressings 26 were males and 14 patients were females . The male and female ratio of the HCD group is 1.5:1 and the normal saline group is 1.8:1.

TABLE 6 : Sex distribution in case and control groups

SEX DISTRIBUTION	**HYDROCOLLOID GROUP**		**NORMAL SALINE GROUP**	
	No.	%	No.	%
MALE	24	60	26	65
FEMALE	16	40	14	35
TOTAL	**40**	**100**	**40**	**100**
INFERENCE	**Sex distribution was statistically insignificant in the two groups.(p=.206 in HCD), (p= .058 in normal saline group)**			

Graph 2: Sex distribution in hydrocolloid and normal saline groups.



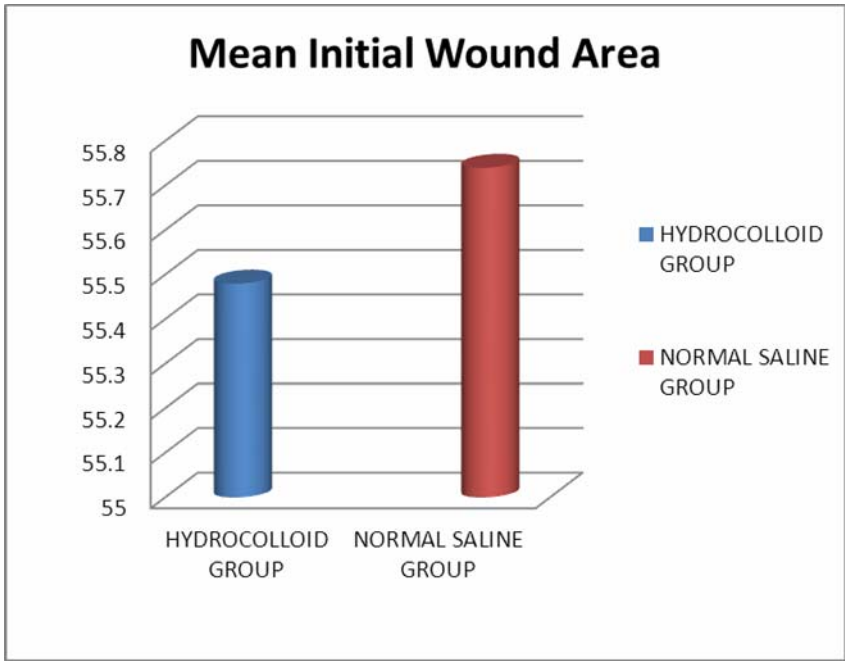
MEAN INITIAL WOUND AREA

The mean area at the beginning of the study was 55.48 sq cm in the HCD and 55.74 sq cm in the normal saline group. The baseline wound surface areas were statistically similar between two groups.

TABLE 7: Mean initial area in hydrocolloid and normal saline groups.

Groups	Mean initial wound area in sq cm.
Hydrocolloid group	55.48
Normal saline group	55.74
Inference	**There was no statistical difference between the initial wound areas (p =.967)**

Graph 3: Mean initial wound area in hydrocolloid and normal saline groups.



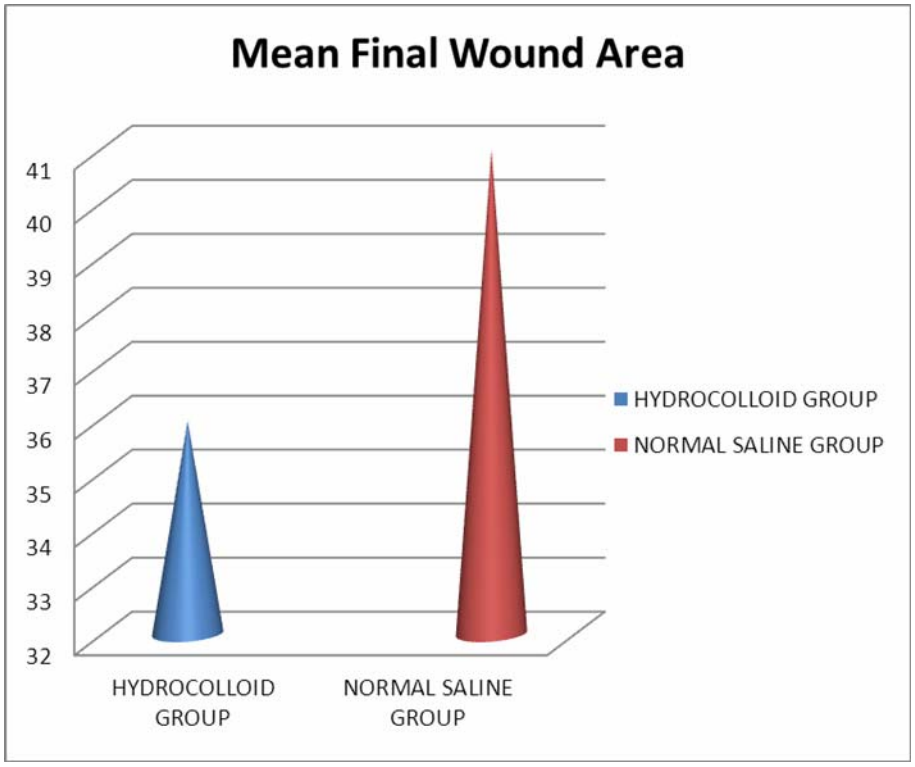
MEAN FINAL WOUND AREA

At the end of the study the mean area were 35.94 sq cm in the group treated with HCD dressings and 40.97 sq cm in the group treated with normal saline dressings. (p=.384)

Table 8:Mean final wound area in hydrocolloid and normal saline groups groups P value being .384 had no statistical significance, however showed considerable difference in final wound Areas.

Groups	Mean Final wound area in sq cm.
Hydrocolloid group	35.94
Normal saline group	40.97

Graph 4: Mean final wound area in hydrocolloid and normal saline groups.



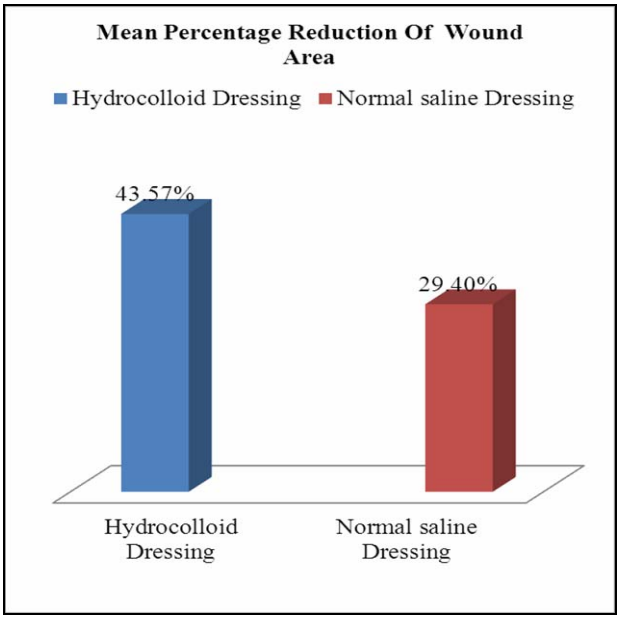
MEAN PERCENTAGE REDUCTION OF WOUND SURFACE

The study shows that the final wound reduction achieved between the two groups were 43.57% in patients treated with HCD dressing and 29.4% in patients treated with normal saline dressing. Reduction percentage was considerably high in HCD group than normal saline group and showed statistical significance with p value of 0.000

Table 9: Mean Percentage reduction of wound area in both cases and controls.

Groups	Mean Percentage Reduction Of Wound Area.
Hydrocolloid group	35.94
Normal saline group	40.97
p value	0.000(<0.05) showing statistical significance.

Graph 5: Percentage reduction in wound area in both groups.



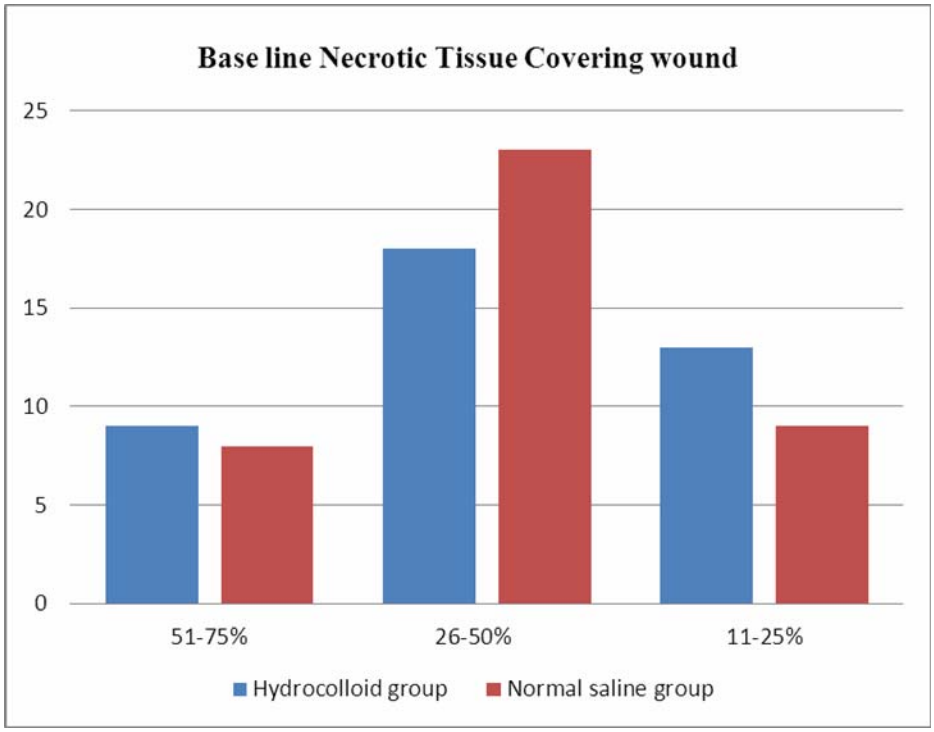
PRESENCE OF NECROTIC TISSUE OR SLOUGH

The study comprised of a total of 80 patients. 40 patients received Hydrocolloid and the other 40 received normal saline dressings for the healing of the ulcer. Both the groups had considerable amount of necrotic tissue at the time of admission which has been compared.

TABLE 10 : Total number of patients with base line necrotic tissue covering wound surface in both groups.

Surface area covered with slough	Hydrocolloid group	Normal saline group
51-75%	9(22.5%)	8(20%)
26-50%	18(45%)	23(57.5%)
11-25%	13(32.5%)	9(22.5%)
***p = .628**		

Graph 6: Base line necrotic tissue in cases and controls.

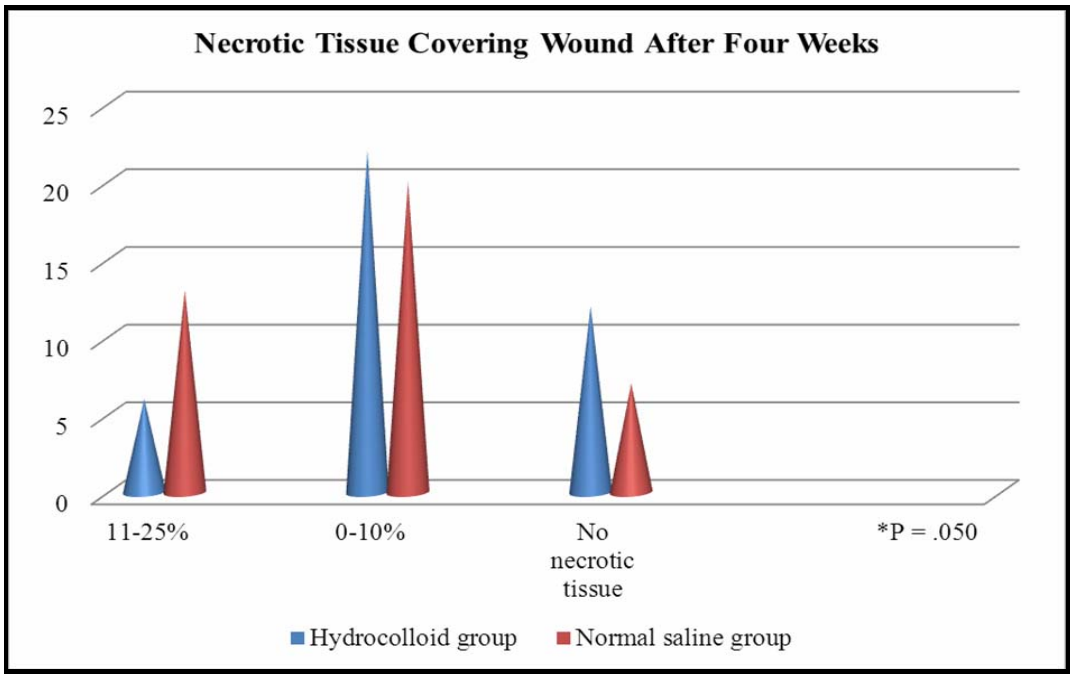


- 9 patients had 51-75% of area covered with the slough in Hydrocolloid group and 8 patients in normal saline group.
- 18 patients had 26-50% of the area covered with the necrotic tissue in Hydrocolloid group and 23 patients in normal saline group.
- 13 patients had 11-25% of area covered with necrotic tissue in Hydrocolloid group and 9 patients in normal saline group.
- Both the groups had considerable amount of necrotic tissue at the time of admission which has been compared.(p=.628)
- Necrotic tissues compared after 4 weeks in both groups showed gradual decrease in the amount of necrotic tissue covering the wound surface area.
- Comparing two groups:

TABLE 11: Total number of patients with necrotic tissue covering wound surface after four weeks in both groups

Surface area covered with slough	Hydrocolloid group	Normal saline group
11-25%	6(15%)	13(32.5%)
0-10%	22(55%)	20(50%)
No necrotic tissue	12(40%)	7(17.5%)
***p = .050**		

Graph 7: Necrotic tissue covering wound surface after four weeks in both groups.



- In Hydrocolloid group 6 Six patients had slough covering 11-25% wound area , 22 patients with 0-10% wound area covered with slough and twelve patients with no necrotic tissue.
- In normal saline group thirteen patients showed 11-25% wound with slough,20 patients with 0-10% slough and only seven patients with no necrotic tissue over the wound.
- Both groups were compared which showed significant reduction of necrotic tissue in HCD group at the end of study.(p=0.05)

PRESENCE OF GRANULATION TISSUE

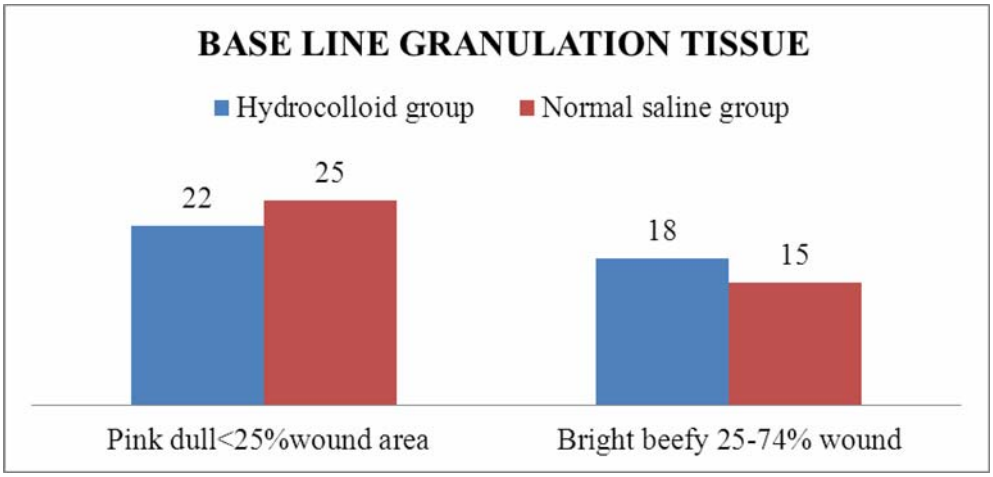
- A total of 80 patients were studied for healing of ulcers. 40 patients received hydrocolloid dressings and 40 patients received normal saline dressings.

COMPARING BOTH GROUPS :

- At the time of admission the number of patients in both the groups were comparable regarding the presence of granulation tissue and baseline granulation tissue was statistically similar..(p=.520)
- Twenty two patients had less than 25% wound filled with pink dull granulation tissue baseline in Hydrocolloid group and eighteen patients with bright beefy granulation tissue filling 25-74% of wound.
- In the control group twenty five patients had less than 25% wound filled with pink dull granulation tissue and fifteen patients with bright beefy granulation tissue filling 25- 74% of wound.

Table 12 : Showing total no. of patients with baseline distribution of granulation tissue in both groups

Wound filled with granulation tissue	Hydrocolloid group	Normal saline group
Pink dull <25% wound	22(55%)	25(62.5%)
Bright beefy 25-74% wound	18 (45%)	15(37.5%)
p=.520		



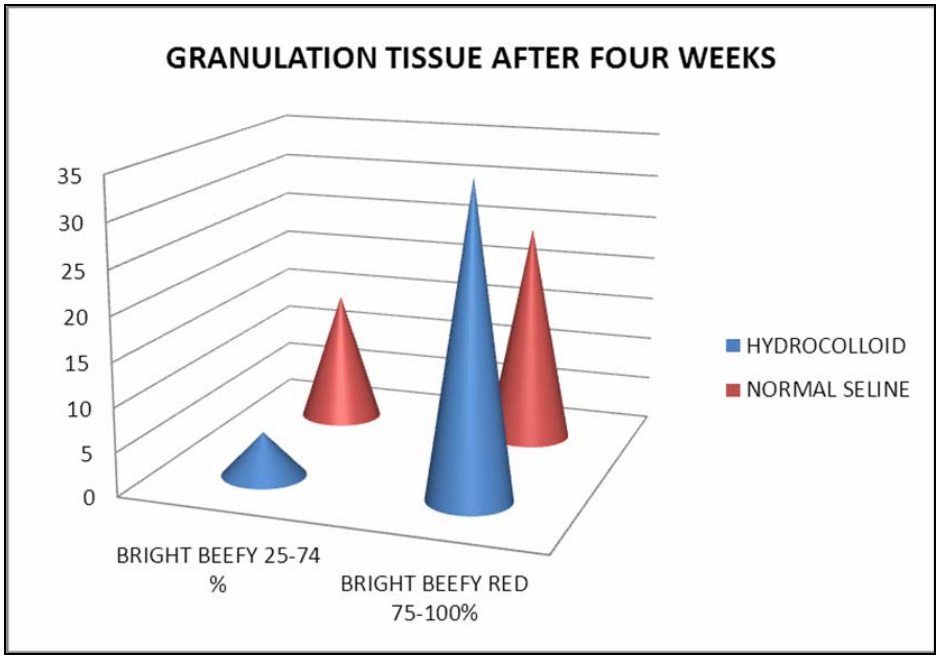
Graph 8: Base line granulation tissue filling wound in both groups.

- Both groups were compared for granulation tissue filling the wound after four weeks.
- Five patients had bright beefy granulation tissue 25-74% filling the wound and thirty five patients had bright beefy red granulation tissue filling the wound in the Hydrocolloid group after four weeks.
- In the control group fifteen patients had bright beefy granulation tissue 25-74% filling the wound and twenty five patients with bright beefy red granulation tissue filling the wound after four weeks.
- Both groups were compared which showed significant granulation tissue covering the wound in Hydrocolloid group than normal saline group at the end of the study .($p=0.003$)

TABLE 13 : Showing total number of patients with granulation tissue filling the wound in both groups after four weeks.

Wound filled with granulation tissue	Hydrocolloid group	Normal saline group
Bright beefy 25-74% wound	5(12.5%)	15(37.5%)
Bright beefy red 75-100% wound	35(87.5%)	25(62.5%)
p=0.003		

Graph 9: Granulation tissue filling wound after four weeks in both groups.



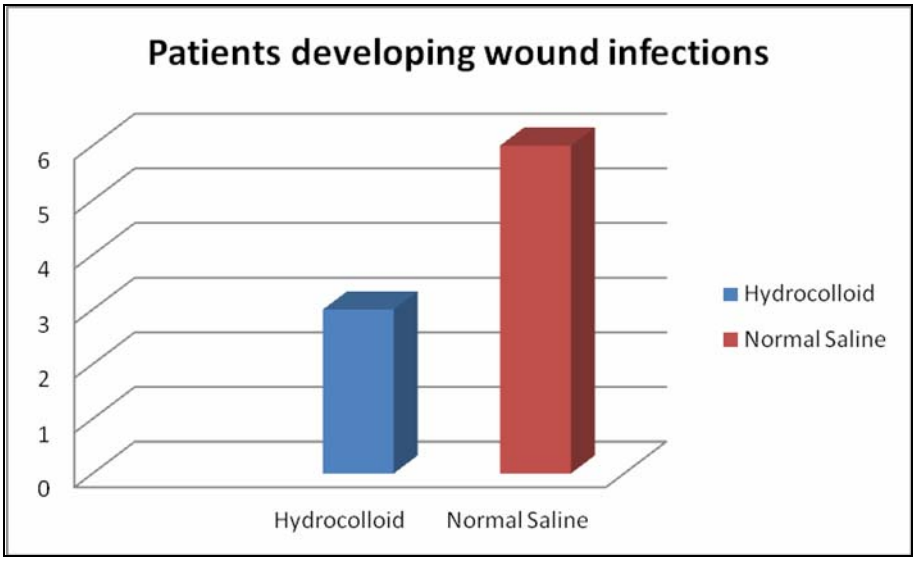
INFECTIONS

- In the study on forty study subjects and forty controls 11.25 % developed infections.
- Three patients in HCD group and six patients in Normal saline group developed infections during the course of the study which were managed accordingly with appropriate antibiotics and any procedure if required.

TABLE 14: Patients who developed wound infections.

Hydrocolloid group	Normal saline group
3 patients (7.5%)	6 patients(15%)

Graph10: Showing patients developing wound infections



6 DISCUSSION

In the last several decades, there has been a tremendous increase in the development of new active wound coverings. Increased understandings of wound healing and factors have brought new materials that optimized wound healing. Years of research have shown that moist wound healing creates a more optimal healing environment .The new synthetic polymer dressings have improved the outcome and replaced the centuries old standard of gauze type dressings. In my study on eighty subjects, forty received Hydrocolloid dressings and forty received conventional normal saline dressings. Base line characteristics like age, sex, wound surface area, necrotic tissue or slough and granulation tissue were matched and statistically similar. Majority of patients in the study fell in the age group of 51-60 years showing that diabetic foot ulcers being common in the elderly age group. Sex distribution was similar in both groups with male : female ratio of 1.5 : 1 in HCD group and 1.8 : 1 in normal saline group.

Mean initial wound area at the beginning of the study was 55.48 sq cm in HCD group. There was no statistical difference between the initial wound surface areas with (p=.967). After receiving the treatment for a period of four weeks in both the groups. The wound surface area considerably decreased in both the groups. The mean final wound area in the HCD group was 35.94 sq cm in HCD group and 40.97 sq cm in normal saline group (P=.384).Mean percentage reduction of wound area was calculated in both the groups. Hydrocolloid group showed considerable result in having better reduction percentage with 43.57% and 29.4% in normal saline group and was statistically significant.(p=0.000)

Base line necrotic tissue in both the groups were statistically similar.

After receiving the dressings in both group there was considerable decrease in the necrotic tissue covering the wound by end of the study and was statistically significant. No necrotic tissue was seen in twelve patients(40%) in HCD group and seven patients in normal saline group.(p=0.05) This shows that the Hydrocolloid dressing has good autolytic properties and aids in removal of necrotic tissue than normal saline dressing. Amount of good granulation tissue is a major indicator of healthy healing. During the study there was very good progression of granulation tissue in the study group when compared to the controls. There was high statistical significance(p=0.003).Speeding up of granulation tissue thus provides faster healing and faster wound bed preparation which was shown in the study. In the study on eighty patients total of nine patients developed infections. Three patients(7.5%) in HCD and 6 patients(15%) in controls. Normal saline group had twice the number of patients who developed infections than in HCD group, showing higher infection rates. Significant group of patients in HCD group(90%) had painless removal of dressing(p=0.0006). On contrary only seventeen patients(42.5%) in controls had painless removal of dressing. Overall this study showed that HCD dressing is safe and effective in treating diabetic ulcers of lower limb.

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.

7 CONCLUSION

With the use of HCD dressing in comparison with the conventional normal saline dressing for the treatment of 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.
- ✓ Considerable amount of necrotic tissue was reduced with hydrocolloid tissue thus having better autolytic debridement properties
- ✓ Granulation and epithelialisation appeared to occur early in ulcers treated with HCD dressings than with normal saline dressings, thus preparing the wound bed and facilitating early cover of raw area by split skin grafting.
- ✓ Infections were minimal with hydrocolloid dressings than normal saline dressings.

8 SUMMARY

The present study was conducted in R.L.J.H, Kolar and attached hospitals to SDUMC.

Total of 80 diabetic patients with lower limb ulcers were included in the study. The objective of the present study was to assess the efficacy of HCD dressing and in comparison with conventional normal saline dressings in terms of duration of healing and infections.

The two groups were randomized into study (Hydrocolloid) and conventional (Normal Saline) groups. There was no statistical difference in the baseline characteristics like age,sex,intial wound area of ulcer, baseline necrotic tissue covering ulcer and granulation tissue filling wound between the two groups.

There was significant removal of necrotic tissue covering the wound during the treatment in HCD group. There was good progression of granulation tissue during treatment in the HCD group and was statistically significant. The final wound area reduced was considerably more in the HCD group and percentage reduction of wound area was high in HCD group and statistically significant when compared with normal saline group. Infections were also higher in the patients who received normal saline dressings.

Photographs showing wounds before and after treatment in the test group:

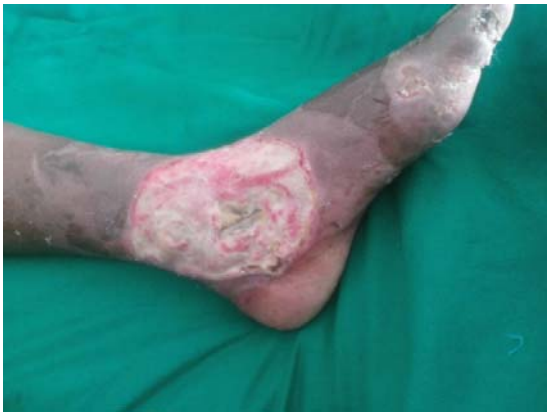


Fig:6 Baseline wound in HCD group

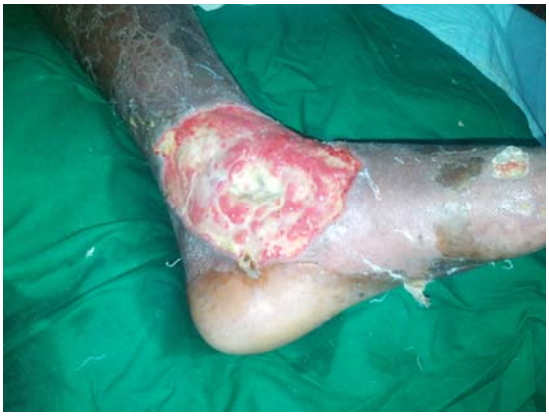


Fig:7 Wound after two weeks in HCD group

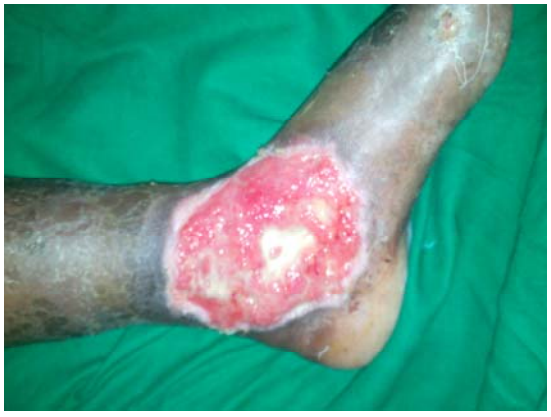


Fig 8: Wound after three weeks in HCD group

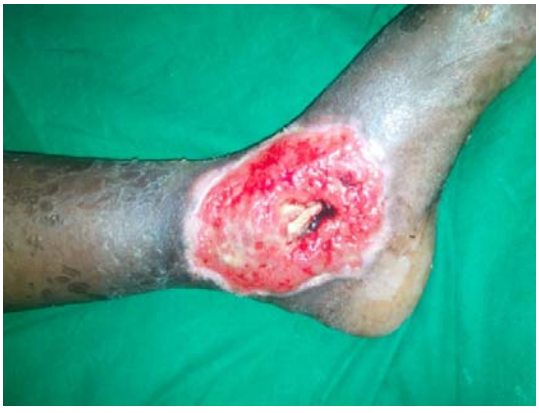


Fig 9 : Wound after four weeks in HCD group

9 BIBLIOGRAPHY

1. Shaw JE ,Sicree RA ,Zimmet PZ .Global estimates of the prevalence of diabetes for 2010 and 2030.Diabetes Res clin Pract.2009;87(1):4-14.
2. Boulton AJ ,Vileikyte L ,Ragnarson – Tennvall G ,et all.The global burden of diabetic foot disease .Lancet 2005;366(9498):1719-24.
3. Singh N ,Armstrong DG ,Lipsky BA.Preventing foot ulcers in patients with diabetes. JAMA 2005;293(2):217-28.
4. Peters EJ ,Armstong DG ,Lavery CA.Risk fators for recurrent diabetic foot ulcers:site matters.Diabetic care.2007;30(8):2077-9.
5. Al-Delaimy WK ,Merchant AT ,Rimm EB , et all.Effect of type 2 diabetes and its duration on the risk of peripheral arterial disease among men.Am J Med .2004;116(4):236-40.
6. Frykberg RG ,Zgonis T ,Armstrong DG ,Driver VR ,Giurini JM ,Kravitz SR ,et all.Suppl JFAS, Diabetic foot disorders ,A clinical pratical guideline Vol 45(5) ,sep/oct – 2006,23-24.
7. Thomas S .Hydrocolloid Journal of wound care 1992:1;2: 27-30.
8. Gill D .The use of hydrocolloids in the treatment of diabetic foot.J wound care 1999;8(4):204 -6.
9. Diegelmann RF, Evans MC. Wound healing: an overview of acute, fibrotic and delayed healing. Front Biosci 2004;9:283-9.
10. Sabiston textbook of surgery, 17th edn vol.(1): 183
11. Madden JW. Textbook of surgery, The biological basis of modern surgical science. 11 th Ed. Philadelphia:WB Saunders and company;1977.p.271
12. Muldner GD, Haberer PA, Jeter KF. Clinician’s pocket guide to chronic wound repair.4 th ed. Springhouse: Springhouse Corporation;1998: p 85

13. JC Moore. A dissertation on the process of nature in the filling up of cavities,healing of wounds and restoring parts which have been destroyed in the human body. John Richardson printer to the Lyceum medicum Londinense ; 1789
14. Lista J.An address on the aseptic management of wounds. Br.Med J 1893; 161(1):277,377
15. Maiya GA; Kumar P; Rao L; Photo Medicine and Laser Surgery; Effect of Low Intensity Helium-Neon (He-Ne) Laser Irradiation on Diabetic Wound Healing Dynamics; April 2005, Vol 23(2) : 187-190.
16. Winter GD.Formation of the scab and the rate of epithelialisation of superficial wounds on the skin of young domestic pig.Nature 1962;193:293-4
17. Townsend CM, Beauchamp RD, Evers BM, Mattox KL. Sabiston Textbook of Surgery; The biologic basis of modern surgical practice. 18th Ed. Philadelphia: Saunders; 2008.
18. Bennett NT, Schultz GS. Growth factors and wound healing: part II. Role in normal and chronic wound healing. Am J Surg 1993;166(1):74-81.
19. Bennett NT, Schultz GS. Growth factors and wound healing biochemical properties of growth factors and their receptors. Am J Surg 1993;165(6):728-37.
20. Cho J, Mosher OF. Role of fibronectin assembly in platelet thrombus formation. J Thromb Haemost 2006;4(7):1461-9.
21. Gailit J, Clark RA. Wound repair in the context of extracellular matrix. Curr Opin Cell Bioi 1994;6(5)717-25.
22. Rumalla VK, Borah GL. Cytokines, growth factors, and plastic surgery. Plast Reconstr Surg 2001;108(3)719-33.
23. Frenette PS, Wagner DO. Adhesion molecules-part 1. N Engl J Med 1996; 334(23): 1526-9.

24. Frenette PS, Wagner DO. Adhesion molecules-part II: blood vessels and blood cells. N Engl J Med 1996;335(1):43-5.
25. Guo RF, Ward PA. Role of C5a in inflammatory responses. Annu Rev Immunol 2005;23:821-52.
26. Tschaikowsky K, Sittl R, Braun GG, et al. Increased fMet-Leu-Phe receptor expression and altered superoxide production of neutrophil granulocytes in septic and posttraumatic patients. Clin Investig 1993; 72(1): 18-25.
27. Roupe KM, Nybo M, Sjobring U, et al. Injury is a major inducer of epidermal innate immune responses during wound healing. J Invest Dermatol 2010; 130(4):1167-77.
28. Greenfield,LJ,editor.Surgery:scientific principles and practice. Philadelphia; J.B.Lippincott,1993.
29. Clore IN, Cohen IK, Diegelmann RF. Quantitation of collagen types I and III during wound healing in rat skin. Proc Soc Exp Bioi Med 1979;161(3)337-40
30. American diabetes association: Clinical practice recommendations 2002. Diabetes Care 2004; 27: 51.
31. Pecoraro RE, Reiber GE, Burgess EM. Pathways to diabetic limb amputation: basis for prevention. Diabetes Care 13:513-521, 1990.
32. Larsson J, Agardh CD, Apelqvist J, Stenstrom A. Long-term prognosis after healed amputation in patients with diabetes.Clin Orthop (350):149-158, 1998.
33. American Diabetes Association. Consensus Development Conference on Diabetic Foot Wound Care. Diabetes Care 22:1354, 1999.
34. Tesfaye S. Diabetic Polyneuropathy. In. The Diabetic foot medical and surgical management. Ist ed.Newjersy:Humana press;2002;75-96.

35. Young MJ, Boulton AJM, Macleod AF, Williams DRR, Sonksen PH. A multicentre study of the prevalence of diabetic peripheral neuropathy in the United Kingdom hospital clinic population. Diabetologia 1993 ; 36: 150-154.
36. Maser RE, Steenkiste AR, Dorman JS, et al. Epidemiological correlates of diabetic neuropathy. RepoI1 from Pittsburgh Epidemiology of Diabetes Complications Study. Diabetes $1989;38: 1456-1461.
37. Tesfaye S, Chaturvedi N, Eaton SEM, Ward JD, Fuller J. Cardiovascular risk Factors predict the development of diabetic neuropathy. Diabetic Med 2000; 17(Suppll):I53.
38. Akbari CM, Logerofo W. Microvascular Changes in the Diabetic Foot. In The Diabetic foot medical and surgical management (Veves A, Giurini JM, LoGerfo FW. eds). Ist ed.Newjersy:Humana press;2002;99-111.
39. Nikhil K, Hamdan A. Clinical Features and Diagnosis of Macrovacular Disease. In The Diabetic foot medical and surgical management (Veves A, Giurini JM, LoGerfo FW. eds). Ist ed.Newjersy:Humana press;2002;113-124.
40. Sanders LJ, Frykberg RG. Diabetic neropathic osteoarthropathy: Charcot Foot, in the High Risk Foot in diabetes Mellitus, Churchill Livingstone, New York: 1991; 297-338.
41. James WB. The Diabetic Foot. In Surgery of the foot and ankle (Mann RA, Couglin MJ.) 6th ed Mosby, londan: 1999; 2: 877-953.
42. Frykberg RG. Diabetic foot ulcers: pathogenesis and management. Am Fam Physician 66:1655-1662, 2002.
43. Boulton AJ, Kirsner RS, Vileikyte L. Clinical practice. Neuropathic diabetic foot ulcers. N Engl J Med 351:48-55, 2004.
44. Boulton AJ. The diabetic foot: from art to science. The 18th Camillo Golgi lecture. Diabetologia, 2004.

45. Wagner FW. The dysvascular foot: a system for diagnosis and treatment. Foot and Ankle 2:64-122, 1981.

46. Prevdorj-Gage B, Costerton WJ, StoodleyP. Phenotypic differentiation and seeding dispersal in nonmucoid and mucoid *Pseudomonas aeroginosa* biofilms.Microbiology 2005;151(Pt 5):1569-76.

47. Doucette MM,Fylling C,Knighton DR,.Amputation prevention in a high – risk population through comprehensive wound healing protocol.Arch Phys Med Rehabil 1989;70(10):780-5.

48. Schultz GS,Sibbald RG, Falanga V et al. Wound bed preparation: a systematic approach to wound management. Wound Repair Regen 11(Suppl 1):S1-S28, 2003

49. Schultz GS,Mozingo D,Romanelli M , et al .Wound healing and TIME ;new concepts and scientific applications. Wound repair Regen 2005;13(Suppl 4).S1-11

50. Schultz GS, Sibbald RG, Falanga V, Ayello EA, Dowsett C, Harding K, Romanelli M, Stacey MC, Teot L, Vanscheidt W. Wound bed preparation: a systematic approach to wound management. Wound Repair Regen 11(Suppl 1):S1-S28, 2003.

51. Jung W, Winter H. Considerations for the use of Clostridial collagenase in clinical practice. Clin Drug Invest 15:245-252, 1998.

52. Drager E, Winter H. Surgical debridement versus enzymatic debridement. In: The Clinical Relevance of Debridement, pp 59-71, edited by M Baharestani, F Gottrup, P Holstein, and W Vanscheidt, Springer- Verlag, New York, 1999.

53. Eaglstein WH, Falanga V. Chronic wounds. Surg Clin North America 77:689-700, 1997.

54. Scott RG, Loehne HB. 5 questions—and answers—about pulsed lavage. Adv Skin Wound Care 13:133-134, 2000.

55. Sherman RA. Maggot therapy for foot and leg wounds. Int J Low Extrem Wounds 1:135-142, 2002.
56. Sherman RA. Maggot therapy for treating diabetic foot ulcers unresponsive to conventional therapy. Diabetes Care 26:446-451, 2003.
57. Lim JK, Saliba L, Smith MJ, Tavish, Raine C. Normal saline dressing: Is it really Normal Br. J Plast Surg 2000; 53(1): 42-5.
58. Fletcher J, Moore Z, Anderson I, Matsuzaki K (2011). Pressure ulcers and hydrocolloids Wounds Made Easy.
59. British National Formulary No. 62 (September 2011)A5.2 Advanced wound dressings A5.2.4 Hydrocolloid dressings.
60. Thomas S, Loveless PA. A comparative study of the properties of twelve hydrocolloid dressings. World Wide Wounds; July 1997. Accessed Nov 2005.
61. Queen D. Technology update: Understanding hydrocolloids. Wounds International 2009; 1(1).
62. Fletcher J (2005). Understanding wound dressings: Hydrocolloids Nursing Times 101 (46);51
63. White R J, Cutting KF. Maceration of the skin and wound bed by indication. In: White R J, editor. Trends in wound care III. London: Quay Books; 2004. p.23-39.
64. Mustoe T A, Cooter R D, Gold MH, Hobbs F D, Ramelet A A, Shakespeare P G, et al. International clinical recommendations on scar management. Plast Reconstr Surg 2002; 110: 560-71.
65. Gotschall C S, Morrison MI, Eichelberger MR. Prospective, randomized study of the efficacy of ‘Mepitel’ on children with partial-thickness scalds. J Burn Care Rehabil 1998; 19: 279-83.

66. Platt A J, Phipps A, Judkins K. A comparative study of silicone net dressing and paraffin gauze dressing in skin-grafted sites. Burns 1996; 22: 543-45.
67. Berry D P, Harding KG, Stanton MR, et al (1998) Human wound contraction: collagen organisation, fibroblasts and myofibroblasts.Plast Reconstr Surg102:124–31.
68. Enoch S, Leaper D (2008) Basic science of wound healing. Surgery 26: 31–7
69. Marston WA, Hanft J, Norwood P, Pollak R. The efficacy and safety of Dermagraft in improving the healing of chronic diabetic foot ulcers: results of a prospective randomized trial. Diabetes Care 26:1701-1705, 2003.
70. Brem H, Balledux J, Bloom T, Kerstein MD, Hollier L. Healing of diabetic foot ulcers and pressure ulcers with human skin equivalent: a new paradigm in wound healing. Arch Surg 135:627-634, 2000.
71. Bello YM, Falabella AF, Eaglstein WH. Tissue-engineered skin. Current status in wound healing. Am J Clin Dermatol 2:305-313,2001.
72. Edmonds M, Bates M, Doxford M, Gough A, Foster A. New treatments in ulcer healing and wound infection. Diabetes Metab Res Rev 16 (Suppl 1):S51-S54, 2000.
73. Donohue K, Falanga V. Skin substitutes in acute and chronic wounds. In: The Wound Management Manual, pp 298-308, edited by B Lee, McGraw-Hill, New York, 2005.
74. Frykberg RG, Hodde JP. Biomaterial wound matrix from small intestine submucosa: review and efficacy in diabetic wound healing.In: The Wound Management Manual, pp 290-297, edited by B Lee,McGraw-Hill, New York, 2005.
75. Datta D,BhingeA,ChandranV.Lysine: is it worth more?Cytotechnology 2001;36:3-32.
76. Demling RH, De Santi L. Involuntary weight loss and the non-healing wound: The role of Anabolic agents. Adv Wound Care 1999; 12(suppl 1): 1-15.
77. Wieman TJ. Clinical efficacy of becaplermin (rhPDGF-BB) gel. Becaplermin Gel Studies Group. Am J Surg 176(2A Suppl):74S-79S, 1998.

78. Argenta LC, Morykwas MJ. Vacuum-assisted vlosure: A new method for Wound control and treatment: Clinical experience. Ann Plast Surg 1997 Jun; 38(6): 563-77
79. Santilli SM, Valusek PA, Robinson C. Use of non-contact Radiant heat bandage for the treatment of chronic venous stasis ulcers. Adv Wound Care 1999; 12(2): 89-93
80. Landau Z. Topical Hyperbaric Oxygen and low energy Laser for the treatment of Diabetic foot ulcers. Arch Orthop Trauma Surg 1998; 117: 156-58

10 ANNEXURE I

PROFORMA

To compare hydrocolloid dressings with normal saline dressings in the management of diabetic foot ulcer

Hospital no:

Name: DOA:

Age: DOD:

Sex:

Address: Religion:

Occupation: Socioeconomic class:

History

Onset of ulcer: Trauma ☐ Spontaneous ☐

Site of ulcer: Plantar ☐ Dorsal ☐

Shoe bite: Massage ☐ Fomentation ☐

Duration of Ulcer:

Progress: gradual ☐ Rapidly ☐

Pain: Yes ☐ No ☐

Discharge: Pus ☐ Serous Sero-sanguinous ☐

Treatment: Received Not Received ☐

Diabetic History:

History of diabetes Mellitus:

Type of diabetes Mellitus:

Duration of diabetes Mellitus:

Type of Treatment at Admission: Oral ☐ Parenteral ☐

Treatment for Diabetes: Regular ☐ Irregular ☐

History Of Any Other Illness:

History Of Alcohol & Tobacco:

Foot Examination:

Lying symmetrical: Yes ☐ No ☐ Skin dry: Yes ☐ No ☐

Corns and calluses: (site and side)

Edema: (site)

Erythema: (site)

Heel fissures:

Scratch marks:

Turgid veins:

Signs of infection:

Any Deformities: Yes ☐ No ☐

Examination Of Foot Ulcer:

Side:	**Right**	**Left**
Site		
Size		
Shape		

Margin

Edge

Floor

Depth

Duration

Granulation

Peri ulcer edema

Slough

Exposed tendons

Bones Keratosis

Tenosynovitis

Necrotising fasciitis

Pulse: Normal (N), Week (W), Absent (A)

Right	Left	
Femoral	☐	☐
Popliteal	☐	☐
Post. tibial	☐	☐
D. pedis	☐	☐

Investigations:

Hametological

Hb%- PCV- T.C- D.C-

R.B.C-

Platlets- E.S.R- B.T- C.T-

Blood grouping-

Bl. Urea- S.Creatinine- FBS- PPBS- Hb1AC-

Urine examination:

Albumin- Sugar- Ketones-

Doppler studies (if needed):

X-Ray of foot:

Pus for Culture Sensitivity (if needed)

Follow-up:

Clinical features	**baseline**	**Follow-up**					
		1st week	**2nd week**	**3rd week**	**4th week**		
Presence of necrotic tissue by clinical evaluation							
Visual scores: 1=76-100%, 2=51-75%, 3=26-50%, 4=11-25%, 5=0-10%, 6= No necrotic tissue covering the ulcer							
Presence of Granulation tissue by clinical evaluation							
Visual scores: 4= bright beefy red 75-100% wound filled 3=bright beefy 25-74% wound filled							

2= pink/dull <25% wound filled ulcer					1= No granulation tissue covering the		
Wound debridement done							
Wound surface area (cm2)	baseline	After 4 weeks					

Any Other Findings:

Comments by the patient:

ANNEXURE II

KEY TO MASTER CHART

RD	Dorsal aspect of right foot
LD	Dorsal aspect of left foot
RP	Plantar aspect of right foot
LP	Plantar aspect of left foot
RA	Right ankle
LA	Left ankle
RL	Right leg
LL	Left leg

MASTER CHART : HYDROCOLLOID GROUP

Sl no	Name	Age/Sex	Hosp no	Site	Initial wound area in cm2	Presence of Necrotic tissue					Presence of Granulation tissue					Final wound area in cm2	% reduction	Painless Removal of Dressing
						Baseline	1st week	2nd week	3rd week	4th week	baseline	1st week	2nd week	3rd week	4th week			
1	Mrs.Ch	38/F	787358	LA	18	4	5	6	6	6	3	4	4	4	4	0	100%	y
2	Mr.Se	60/M	919863	LD	84	2	3	4	4	4	2	3	3	3	4	54.4	35.23%	Y
3	Mr.Na	81/M	640264	LA	21	4	5	6	6	6	3	4	4	4	4	10	52.38%	Y
4	Mrs,Si	85/F	778651	RD	77.28	2	3	4	4	5	2	3	3	3	4	49	36.59%	Y
5	Mrs.Fa	54/F	782428	LA	38.5	3	4	4	5	5	2	3	3	4	4	21	45.45%	Y
6	Mrs.Ma	40/F	793361	RP	22	4	5	5	6	6	3	3	4	4	4	8.8	60%	N
7	Mr.Ma	40/M	797256	LD	35	3	3	4	5	5	3	3	4	4	4	16	54.28%	Y
8	Mr.Mu	45/M	836683	RD	53.2	3	3	4	5	5	3	3	4	4	4	30.16	43.41%	Y
9	Mrs.Ra	45/F	900127	RD	27.5	3	4	4	5	6	3	3	4	4	4	14	49.09%	Y
10	Mr.Ra	55/M	902002	LP	82.5	3	3	4	4	5	2	2	3	3	4	51	38.18%	Y
11	Mr.An	61/M	890447	RP	30	4	4	4	5	5	3	3	3	4	4	16	46.66%	Y
12	Mrs.Ba	70/F	886252	LD	142.5	2	2	3	3	4	2	2	3	3	3	100.8	29.26%	N
13	Mr.La	55/M	799528	LD	113.4	2	3	3	4	4	2	2	3	3	3	77	32.09%	N
14	Mr.Na	82/M	827160	LA	80.75	3	3	4	5	5	2	2	3	3	4	53.25	34.05%	Y
15	Mr.Ya	60/M	840971	LD	104.92	3	3	4	4	4	2	2	3	3	3	169.36	33.89%	N
16	Mrs.Sh	65/F	890792	RA	80.75	3	3	4	5	5	2	3	3	4	4	47.6	41.05%	Y
17	Mr.Na	85/M	922435	RL	94.6	2	3	3	4	4	2	2	3	3	4	60.52	36.02%	Y
18	Mr.Se	58/M	930406	LL	43.2	4	4	4	5	6	3	3	4	4	4	24.75	42.70%	Y
19	Mrs.Mu	68/F	944661	RD	48	3	4	4	5	5	3	3	3	3	4	29.25	39.06%	Y
20	Mr.Th	72/M	835784	LL	35.75	3	4	5	5	5	2	2	3	3	3	22.08	38.23%	Y
21	Mr.Ka	70/M	950167	LD	32.5	4	4	5	5	6	3	3	4	4	4	16.8	48.31%	Y
22	Mr.Ra	72/M	926201	RD	60	3	4	4	5	5	2	2	2	3	4	37.7	37.17%	Y
23	Mr.Ha	75/M	715625	RP	24	4	5	5	6	6	3	3	4	4	4	7.98	66.75%	Y
24	Mrs.Pr	60/F	815695	RA	41	4	4	4	5	5	2	2	3	4	4	23.8	41.95%	Y
25	Mr.Da	60/M	858965	LD	40	3	4	4	5	6	2	2	3	4	4	23.7	40.75%	Y
26	Mr.Ve	58/M	816281	LD	52	2	4	4	5	5	2	2	3	3	4	32.24	38%	Y
27	Mr.Mu	60/M	810169	RA	44.2	3	3	4	5	5	3	3	3	4	4	26	41.17%	Y
28	Mrs.Mu	65/F	783322	RL	36	4	4	5	5	6	3	3	3	4	4	19.2	46.66%	Y
29	Mr.Kr	50/M	869003	RA	88	2	3	4	5	5	2	2	3	3	3	55.18	37.29%	Y
30	Mr.Er	58/M	816030	LA	40.5	3	3	4	5	6	3	3	3	3	4	24.5	39.50%	Y
31	Mrs.La	60/F	857208	RA	60.5	3	3	4	5	5	2	2	3	3	4	37.8	37.52%	Y
32	Mr.Mu	65/M	796635	RL	75.6	4	4	5	5	5	2	2	3	3	4	45.5	39.81%	Y
33	Mr.Sy	48/M	876300	RD	24.5	4	5	5	5	6	3	3	3	4	4	12.5	48.97%	Y
34	Mrs.Ma	65/F	940831	LD	48	4	4	5	5	5	3	3	3	4	4	27	43.75%	Y
35	Mr.Par	65/M	814617	RD	22	4	5	5	5	6	3	3	3	4	4	9.8	55.45%	Y
36	Mrs.Le	40/F	847773	RA	56	3	3	4	5	5	2	2	3	3	4	33	41.07%	Y
37	Mrs.Mu	55/F	13112	LA	72	2	2	4	4	5	2	2	3	3	4	43.56	39.50%	Y
38	Mrs.Bi	52/F	11023	RP	55	3	3	4	5	5	3	3	3	3	4	33.6	38.90%	Y
39	Mrs.Ru	50/F	10114	LP	60.5	2	2	3	4	5	2	2	3	3	4	36.8	39.17%	Y
40	Mr.Sa	39/M	824005	RA	54.4	3	3	4	4	4	2	2	3	3	4	36	33.82%	N

MASTER CHART : NORMAL SALINE GROUP

Sl no	Name	Age/Sex	Hosp no	Site	initial wound area in cm2	Presence of necrotic tissue					Presence of Granulation tissue					final wound area in cm2	% reduction	Painless Removal of dressing
						Baseline	1st week	2nd week	3rd week	4th week	Baseline	1st week	2nd week	3rd week	4th week			
1	Mr.Sa	70/M	787439	RL	37.5	3	3	4	4	5	3	3	3	3	4	24.8	33.86%	Y
2	Mrs. Th	65/F	776254	RH	42.25	3	3	4	4	5	3	3	3	3	3	30.25	28.40%	Y
3	Mr. Cho	50/M	759042	LL	80.36	4	5	5	5	5	2	3	3	3	3	61.2	23.84%	N
4	Mrs. Sul	75/F	926116	LD	70.2	4	4	5	5	5	2	2	3	3	3	54.6	22.22%	N
5	Mr. Km	38/M	830519	RD	88	3	3	4	4	4	2	3	3	3	3	68.6	22.04%	N
6	Mr. Sy	50/M	896303	LD	125.4	4	4	4	5	5	3	3	3	4	4	94.5	24.64%	N
7	Mr. Na	40/M	845986	RA	10	4	5	5	5	6	3	4	4	4	4	9.5	52.50%	N
8	Mr. Ve	45/M	808917	RL	81.7	3	3	4	4	5	2	2	3	3	3	62.4	23.62%	Y
9	Mrs.Me	60/f	777833	LL	110.08	3	3	4	4	5	2	2	3	3	3	84	23.69%	N
10	Mr. Do	85/M	791540	LL	77.9	3	4	4	4	5	3	3	4	4	4	56	28.11%	N
11	Mrs.So	65/F	799535	RA	61.2	2	2	3	4	4	2	3	3	3	4	45	26.47%	N
12	Mrs.Ja	31/F	805375	LL	97.9	2	2	3	3	4	2	2	3	3	3	75.24	23.14%	N
13	Mr. Go	50/m	882502	RA	49.6	3	3	3	4	4	2	2	3	3	3	35.75	27.92%	Y
14	Mr. Ve	65/M	888719	LD	135	3	3	3	4	4	2	2	3	3	4	74.75	29.28%	N
15	Mr. S Kr	35/M	912652	LD	42	3	3	4	4	4	2	2	3	3	4	30.5	27.38%	Y
16	Mr. Pa	62/M	921874	RA	48	4	4	4	4	5	2	2	3	3	3	36.3	24.38%	Y
17	Mr. Do	58/M	918856	LP	16	3	4	4	5	6	3	3	3	4	4	7.5	53.12%	Y
18	Mr. Ra	72/M	926201	RL	32.5	3	3	3	4	4	2	2	3	3	4	23.4	28%	Y
19	Mrs. Ru	42/F	932623	RP	64	2	3	3	3	4	2	2	2	3	3	48.75	23.82%	N
20	Mr. Ra	54/M	917541	RD	36	3	3	4	4	5	2	2	3	3	4	26.4	26.66%	Y
21	Mr. Na	60/m	931652	RD	42	3	3	4	4	5	2	2	2	3	4	31	26.19%	Y
22	Mrs.La	45/F	892825	LD	44	3	3	3	4	5	3	3	3	3	4	32.5	26.13%	N
23	Mrs.Su	84/f	949117	RA	50	3	3	4	4	5	2	2	2	3	3	38.25	23.50%	N
24	Mrs.Ra	80/F	921903	LA	18	4	4	5	5	6	3	3	3	4	4	9	50%	Y
25	Mr. Kr	55/M	911026	LP	40.5	3	3	4	4	5	2	2	3	3	4	29.64	26.81%	N
26	Mr. Ka	60/M	886938	RP	27	4	4	4	5	6	3	3	3	4	4	18	33.33%	Y
27	Mrs.Ra	35/F	846486	RA	86.25	2	3	3	4	4	2	2	3	3	3	68	21.15%	N
28	Mrs.La	61/F	829064	RD	45	3	3	3	4	5	2	3	3	3	4	33.6	25.33%	N
29	Mr. Th	75/M	828232	RH	72	2	2	3	3	4	2	2	2	3	3	57.75	19.79%	N
30	Mr. As	65/M	807628	LD	76.5	3	3	3	4	4	2	2	3	3	3	60	21.56%	N
31	Mrs.Mu	60/F	796066	LD	28	4	4	4	5	6	3	3	3	4	4	17.4	37.85%	N
32	Mr. pra	65/M	778962	LD	41.25	2	2	3	4	5	3	3	3	3	4	31.2	24.36%	Y
33	Mr. Ba	42/M	776959	RD	28	2	3	4	5	5	3	3	3	3	4	20.3	27.50%	N
34	Mrs.Mu	45/F	822865	RP	36	3	3	4	4	5	3	3	3	3	4	26.4	26.60%	Y
35	Mrs. Sy	45/F	812472	LA	20	3	4	4	5	6	3	3	3	4	4	12.16	39.20%	Y
36	Mr. pr	50/M	13025	LA	40	3	3	4	4	5	2	2	3	3	4	29.25	26.87%	N
37	Mr. Go	58/M	11075	LD	69.6	2	3	3	4	4	2	2	3	3	3	54	22.41%	N
38	Mr. Ra	55/M	11036	RP	60	3	3	4	4	5	2	2	3	3	4	45.76	23.73%	N
39	Mr. Mu	60/M	11038	RD	70	3	3	4	4	4	2	2	3	3	3	55.25	21.07%	Y
40	Mr. Ve	58/M	12056	RL	30	4	4	4	5	6	3	3	3	4	4	20.25	32.50%	Y