"A RANDOMISED CONTROL TRIAL TO COMPARE THE DESLOUGHING EFFECT OF HYDROGEL VERSUS NORMAL SALINE DRESSING IN LEG ULCERS"

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KARNATAKA

ENDORSEMENT

This is to certify that the dissertation entitled **"A RANDOMISED CONTROL TRIAL TO COMPARE THE DESLOUGHING EFFECT OF HYDROGEL VERSUS NORMAL SALINE DRESSING IN LEG ULCERS"** is a bonafide research work done by **REG NO. BH0111004.**

Dr. V. M. UPPIN **MS**
Professor and Head,
Department of Surgery,
J. N. Medical College,
Nehru Nagar, Belgaum – 10

Date:
Place: Belgaum

Dr. A. S. Godhi **MS,FICS**
Principal,
J. N. Medical College,
Nehru Nagar, Belgaum – 10

Date:
Place: Belgaum

LIST OF ABBREVIATIONS USED

A.D	-Anno Domini
ADP	-Adenosine diphosphate
B.C	-Before Christ
EGF	-Epidermal growth factor
FGF	-Fibroblast derived growth factor
KL	-Klebsiella
MMPs	-Matrix metalloproteinases
MRSA	-Methicillin resistant Staphylococcus aureus
PDGF	-Platelet derived growth factor
PDGF	-Platelet-derived growth factor
PM	-Proteus mirabilis
PMNs	-Polymorphonuclear leukocytes
PS	-Pseudomonas
PTV	-Proteus vulgaris
rhPDGF	-Recombinant human platelet-derived growth factor
STPH	-Staphylococcus aureus
TGF	-Transforming growth factor
TGF-α	-Transforming growth factor alpha
TGF-β	-Transforming growth factor beta

ABSTRACT

Background and objectives

Numerous topical medication and gels are promoted for ulcer care debridement and healing. Relatively few have proved to be more efficacious than saline wet to dry dressings. The present study was aimed to compare the desloughing effect of hydrogel and normal saline dressing in healing of leg ulcers.

Methodology

The present one year hospital based randomized controlled trial was conducted in the Department of General Surgery, KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belgaum from January 2012 to December 2012. A total of 60 patients with leg ulcers were studied. Based on the envelop method, patients were divided into two groups of 30 patients each that is group A (hydrogel) and group B (normal saline).

Results

In this males (76.66% in group A and 86.66% in group B) outnumbered females in both the groups (p=0.317). The mean age in group A was 55.73 ± 9.40 years and in group B it was 54.66 ± 12.73 years (p=0.713). In the course of the study with the hydrogel the proportion of the wound area covered with slough fell from 32.1 ± 13.13% to 11.54 ± 9.9 %, with higher mean percentage reduction in slough (68.86 ± 14 %) whereas in saline group the proportion of the wound area covered with slough fell from 34 ± 13.76 % to 17.1± 11.48 %, with less mean percentage reduction in

slough (54.3 ± 13.02%).The difference between the percentage reduction in slough was statistically significant ($p<0.001$).

Conclusion and interpretation

Overall, hydrogel dressing provided favorable outcome in patients with leg ulcer by significant reduction in slough area when compared to normal saline dressing.

Keywords

Leg ulcers; Normal saline; hydrogel; Wound healing; autolytic debridement

CONTENTS

LIST OF TABLES

LIST OF GRAPHS

LIST OF FIGURES

LIST OF PHOTOGRAPHS

Chapter 1

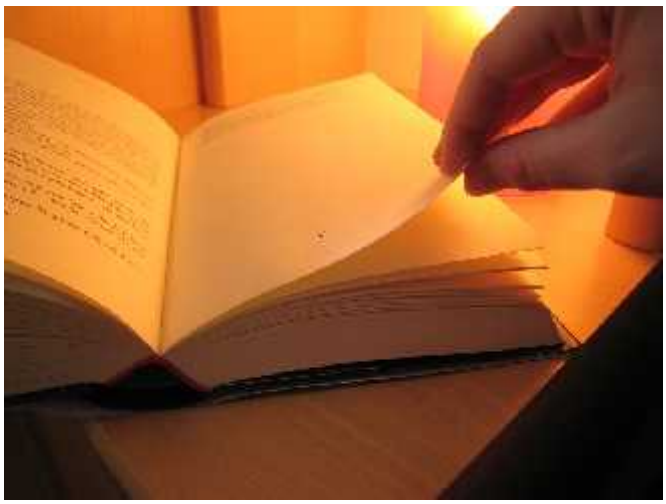
Introduction

Chapter 2

Objectives

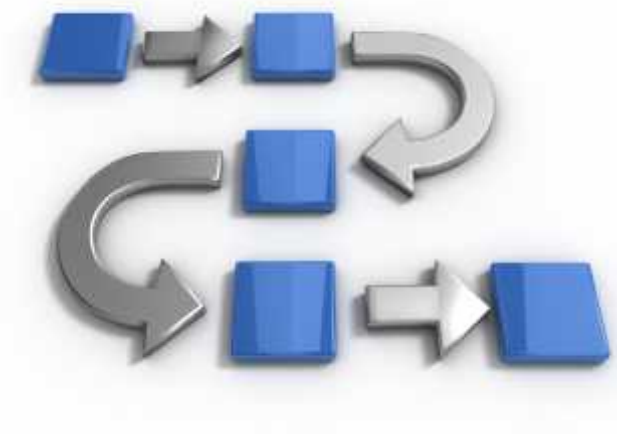
Chapter 3

Review of Literature



Chapter 4

Methodology



Chapter 5

Results

Chapter 6

Discussion

Chapter 7

Conclusion

Chapter 8

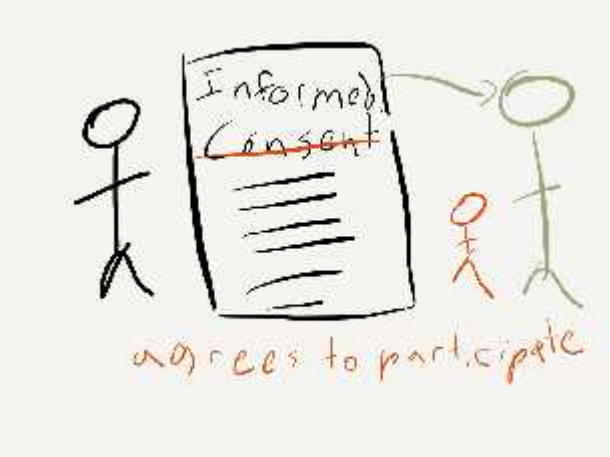
Summary

Chapter 9

Bibliography

Annexures

Annexure I



Annexures

Annexure II

Annexures

Annexure III

Annexures

Annexure IV

INTRODUCTION

Chronic wounds are common conditions and are wide in distribution. They could be associated with a number of surgical and also few medical and dermatological conditions. Slow healing ulcers represent a major health burden and drain on resources, contributing to substantial disability, morbidity, and costs.[1]

The incidence of ulcers is more in elderly population and in people with increased risk factors for atherosclerotic occlusion such as smoking, obesity and diabetics. Chronic wounds are defined as wounds, which have failed to proceed through an orderly and timely reparative process to produce anatomical and functional integrity.

Wound bed preparation continues to be an essential component of chronic wound management. By addressing the relationship between necrotic tissues, exudate, bacterial and cellular dysfunction, an optimal wound healing environment can be achieved. The goal of debridement, integral to the wound bed preparation concept, is to get rid of the necrotic tissue.

Types of debridement include surgical excision, bedside sharp debridement, chemical, enzymatic, biological, mechanical agents and autolytic agents.

Surgical or sharp debridement describes the use of surgical instruments such as scissors, scalpels, dermatomes, or curettes to remove devitalized tissue. This is often the fastest type of debridement; however it is invasive, could need anesthesia and its involved risk and hospitalization, and should be performed by a certified professional and also involves costs.

The choice of debridement methods depends upon the clinician, who considers wound characteristics and other local assessment findings, patient comorbidities, the time frame needed to achieve debridement, and the available skills and resources to safely manage the debridement process in the particular clinical setting.[2]

With the exception of surgical debridement, complete debridement of necrotic tissue in one patient encounter, is a rarity.[3] Numerous studies have previously shown the debridement efficacy of chemical, enzymatic, biological agents when compared to wet-to-dry dressings.[4]

Enzymatic and autolytic debridement is frequently used in the long-term care setting as a simple, safe, and practical method to achieve the removal of nonviable tissue in the population served by these facilities. It can be used as either a short term conservative approach while arranging for surgical or sharp debridement or as a longer term strategy, as long as the wound bed parameters are improving.[5]

Initial debridement, defined as "the removal of necrotic, damaged, or infected tissue"[4] differs from "maintenance debridement," which is a method that not only frees the wound bed from obstacles to healing, but offers a continuous and active debridement of that wound bed. These obstacles include the persistence of cellular debris, uncontrolled matrix metalloproteases, and bacterial bio burden.

While comparison of hydrogel dressings and enzymatic debriding agents on wound healing have been done using swine[6], there are no reports in the clinical area, or, specifically, the long-term care setting, where these agents are extensively used.

Wet to dry dressings are not any longer considered standard of care, and there has been a shift with in the literature that compares topical wound care therapies to moist wound care using hydrogel.

Once the autolytic debridement is done the surgeon can take further actions like skin grafting or flap mobilization and closure of the raw area. Treatment is variable and there are no documented, consistent responses. Rapid healing of chronic wounds could result in decreased hospitalization and an earlier return of function.[7]

The treatment of chronic, open wounds is variable and expensive, demanding lengthy hospital stays or specialized home care requiring skilled nursing and expensive supplies.

This study will aim to assess the desloughing effect of hydrogel dressing particularly in our hospital and our region.

OBJECTIVES

The objective of the present study was to compare the desloughing effect of hydrogel with normal saline dressing in leg ulcers.

REVIEW OF LITERATURE

ANATOMY OF SKIN:

Layers of skin

1. The epidermis
2. The dermis, and
3. The subcutaneous tissue.

Epidermis:

Epidermis is less than a millimeter thick, and consists of three forms of cells.

The most thickly settled of that are the keratinocytes.

1. Keratinocytes migrate to skin surface from the base of the epidermis; loss of water from keratinocytes makes them hard. They form the outermost protective layer of the epidermis until they are eventually sloughed off and replaced.
2. Melanocytes manufacture the pigment melanin which supplies skin its color.
3. Langerhans cells are a part of the immune system that acts as a defense against pathogens.

Dermis:

Dermis is divided into two layers superficial papillary and deep reticular layer is the thickest of the skin's three layers. The first cells at work here are known as fibroblasts that maintain the network of collagen and elastin proteins, forming the

structure of the skin and provide it its elasticity and resilience. It has capillaries and Langerhans- manufacturing lymph nodes; it is additionally home of the sebaceous glands which generate the protective sebum that travels via small hair follicles from the dermis to the epidermis where it lubricates and protects the skin's surface.

Subcutaneous Tissue

Is composed of fat cells, and is liable for providing insulation and cushioning, additionally acts as housing for the sweat glands and a system of small muscles connected to hair follicles. Cutaneous vessels arise from underlying vessels. Cutaneous vessels anastomose with other cutaneous vessels to create a continuous vascular network within the skin.[8]

EPIDEMIOLOGY OF CHRONIC WOUNDS

In Western countries, ten per thousand of the adult population are likely to have a chronic leg ulcer at some time.[9] The point prevalence seems to be between 1.1 and 3.0 per thousand, this range being partly explained by age differences in the populations studied and the inclusion of foot ulcers in those nearer the higher figure. Otherwise there is a remarkable similarity in the findings of a number of large population studies.[10-17] There are many causes of chronic non healing ulcers .These studies found that about 60-80% of chronic leg ulcers had a venous component, 10-30% were associated with arterial insufficiency and that other factors included diabetes mellitus and rheumatoid disease. Arterial and venous insufficiency combined in 10-20% of cases.

Prevalence rises with age and is more common in women. The point prevalence of active ulceration ranges internationally from 1.48/1000 to 3.05/1000. [16]

WOUND HEALING

Wound healing is a mechanism whereby the body attempts to restore the integrity of the injured part. [18]

Wound healing involves a complex and dynamic series of processes, namely chemotaxis, cell division, neovascularization, synthesis of new extracellular matrix components, formation and remodeling of scar tissue.

The knowledge of the physiology of wound healing trajectory through the phases of hemostasis, inflammation, granulation and maturation provides a framework for an understanding of the basic principles of wound healing.

HISTORY OF WOUND HEALING .[19]

The earliest accounts of wound healing dates back to about 2000 B.C., the Sumerians employed two modes of treatment: a spiritual method consisting of incantations and a physical method of applying poultice-like materials to the wound.

The Egyptians differentiated between infected and diseased wounds compared to noninfected wounds.

In 1650 B.C. Edwin Smith Surgical Papyrus, described at least 48 different types of wounds.

A document Ebers Papyrus, 1550 B.C. quotes the use of honey (antibacterial properties), lint (absorbent properties), and grease (barrier) for treating wounds. The Greeks, classified wounds as acute and chronic.

Galen of Pergamum (120–201 A.D) emphasized the importance of maintaining a moist environment to ensure adequate healing.

Ignaz Philipp Semmelweis, a Hungarian obstetrician (1818–1865), noted that the incidence of puerperal fever was much lower if medical students, after cadaver-dissection class and before attending childbirth, washed their hands with soap and hypochlorite.

Louis Pasteur (1822–1895) - theory of spontaneous generation of germs and proving that germs were always introduced into the wound from the environment.

Joseph Lister made significant contributions to wound healing. He discovered that the water from pipes that were dumping waste containing carbolic acid (phenol)

was clear. In 1865, Lister began soaking his instruments in phenol and spraying the operating rooms, reducing the mortality rates from 50 to 15%.

STAGES OF WOUND HEALING

1. Hemostasis
2. Inflammation,
3. Tissue formation, and
4. Tissue remodeling. [20]

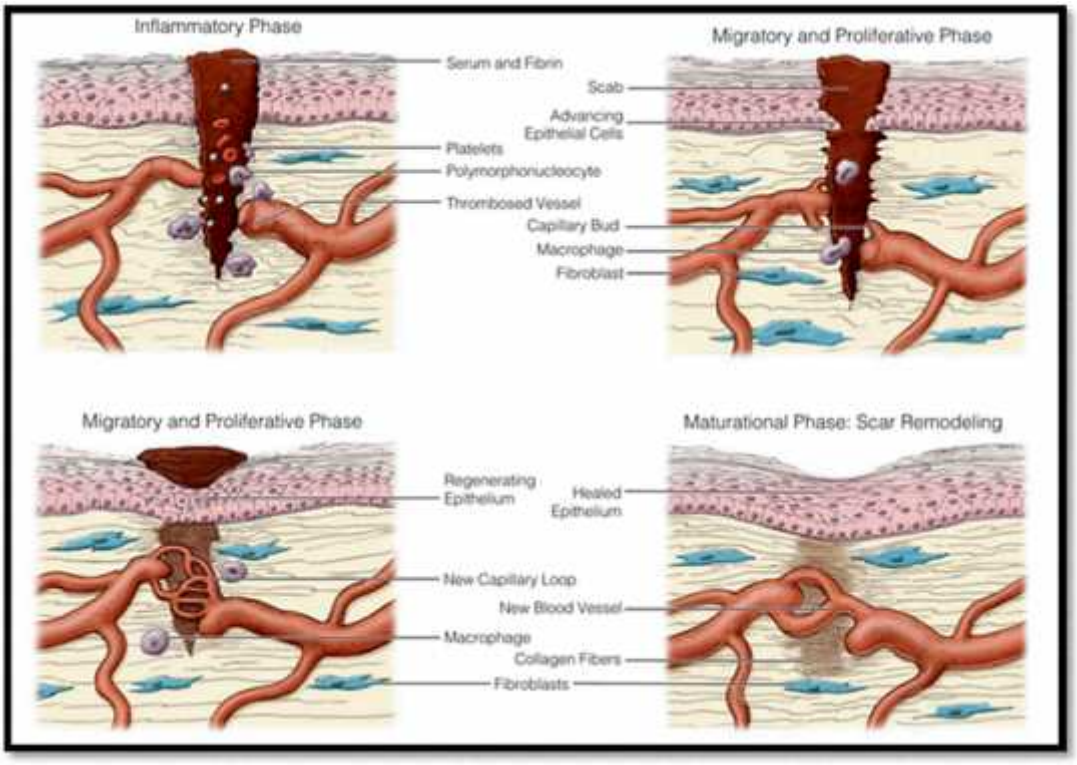


Figure.1 STAGES OF WOUND HEALING

Hemostasis:

Hemostasis is achieved within few minutes of the initial injury unless there are underlying coagulation disorders. Cascade of vasoconstriction and coagulation commences with clotted blood instantly impregnating the wound, resulting in hemostasis.

After vasoconstriction, the recruitment of inflammatory cells into the wound occurs; platelets adhere to damaged endothelium and release ADP that promotes platelet clumping. Release of various cytokines by platelets initiates inflammatory phase. Alpha granules liberate platelet derived growth factor (PDGF), platelet factor-IV, and transforming growth factor beta (TGF-β), whereas vasoactive amines like histamine and serotonin are discharged from dense bodies found in platelets. PDGF a chemotactic agent for fibroblasts and TGF-β is a fibroblastic mitosis modulator. Fibrinogen gets cleaved into fibrin and the framework for completion of the coagulation process is achieved. Fibrin provides the structural support.[20]

Inflammation

Cellular infiltration follows a predetermined sequence. PMNs are the first to enter the wound site, which peak at 24 to 48 hrs. Primary role of neutrophils is phagocytosis of the bacteria and tissue debris. Clinically inflammation presents as erythema, swelling and warmth often associated with pain, the classic “rubor et tumor cum calore et dolore”

Second population of inflammatory cells consists of macrophages which peak at 48 to 96 hrs, which participate in wound debridement through phagocytosis, and

contribute to microbial stasis by production of oxygen radicals and nitric oxide. The most important function is the activation and recruitment of other cells. Macrophages regulate the cell proliferation, matrix synthesis, angiogenesis and also matrix deposition and remodeling. [19] T-lymphocytes peak at about 1 week post injury, whose role in healing not well defined.[19]

Tissue formation and remodeling.

Starts during fibroblastic phase which is characterized by reorganization of already synthesized collagen. Collagen is broken down by matrix metallipoproteinases (MMPs) and the net collagen in the wound is result of a balance between collagen break down and collagen synthesis. There is a shift towards collagen synthesis and reestablishment of extracellular matrix composed of relatively acellular collagen rich scar. [19]

Wound strength and mechanical integrity in a wound are determined by quantity and quality of freshly deposited collagen. Fibronectin and collagen type III constitute early matrix scaffolding; glycosaminoglycans and proteoglycans come next and collagen type I forms the final matrix. Several weeks after injury the quantity of collagen reaches a plateau, but tensile strength continues to increase for still more months. Fibril formation and cross linking results in decreased collagen solubility, increased strength and increased resistance to enzymatic degradation of the collagen matrix. Scar remodeling continues for several (6 to 12) months post injury, gradually resulting in a mature, avascular, and acellular scar**.**[19]

Epithelialization:

The process is characterized by proliferation and migration of epithelial cells adjacent to the wound. Re-epithelialization is complete in 48 hrs. In case of approximated incised wound, but may take substantially longer in case of larger wounds .[19] The keratinocytes are responsible for epithelialization.

Wound contraction:

All wounds undergo some degree of wound contraction and the final area of wound will be decreased. Myofibroblasts have been postulated as being major cells responsible for wound contraction. This cell contains alpha smooth muscle actin in thick bundles called stress fibers .[19]

TYPES OF WOUND HEALING [21]

1. Primary Healing (First Intention)
2. Secondary Healing (Second Intention)
3. Tertiary Healing (Delayed Primary Intention)

Primary Intention

Repair by primary intention is intended for acute, clean surgical wounds. The skin edges are approximated to each other, either by suturing, by staples, or by adhesive plasters. This procedure facilitates a relatively rapid process of wound healing. Leads to a healthy scar.

Secondary Intention.

In the case of chronic ulcers, or in wounds that have a higher probability of developing infection, repair should be achieved by secondary intention. The edges of such wounds should not be approximated. Closure and complete healing is achieved gradually by granulation tissue formation and re-epithelialization.

Tertiary Intention.

Tertiary intention, also called delayed primary closure, is intended for wounds where the surgeon approximates the wound edges only after a few days. The delay allows natural physiological processes to take place, such as drainage of exudates or reduction in the extent of edema.

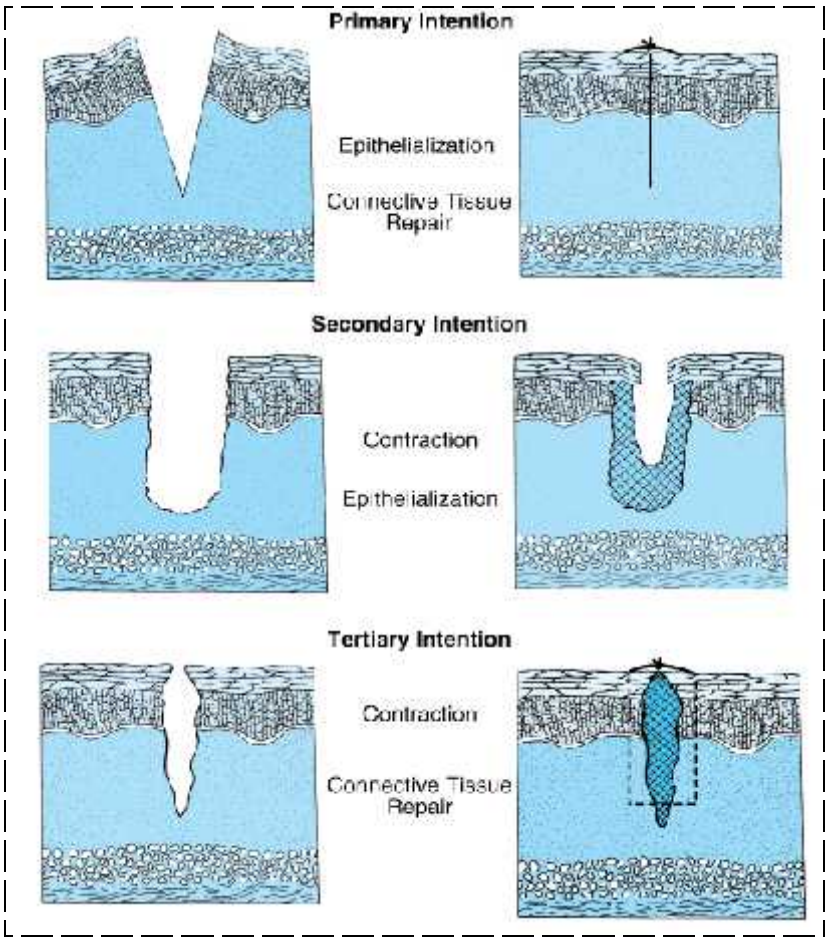


Figure.2 TYPES OF WOUND HEALING [21]

FACTORS AFFECTING WOUND HEALING

Local Factors:

1. **Location of the wound**: A surgical wound in highly vascularized area heals faster, like face than those in poorly vascularized area like the foot or areas where the skin adheres to bony surfaces, as over the tibia.
2. **Vascular supply**: Wounds with impaired blood supply heal slowly, like in patients with varicose veins. Pressure produces ischemia and thus bedsores and then delays healing. Ischemia caused by arterial obstruction, often in the lower extremities prevents healing.
3. **Infection**: Wounds provide a portal of entry for microorganisms. Infection delays or healing. If the bacterial count in the wound exceeds 10^5 organisms per gram of tissue, or if any beta hemolytic Streptococcus is present, the wound will not heal by any means, including flap closure, skin graft placement, or primary sutures.[22]
4. **Foreign Bodies** Foreign bodies are a physical obstacle to wound healing and source of bacteria. Foreign bodies extend the inflammatory phase. Wounds with foreign bodies cannot contract, neovascularize, or completely epithelize. Wounds with necrotic tissue will not heal until all the necrotic tissue is removed**.**[23]
5. **Movement**: Early motion, particularly before tensile strength has been established, subjects a wound to persistent traumas, thereby delays healing.
6. **Edema/Elevated Tissue Pressure** The edema and locally raised pressures associated with ischemic tissue injury can potentially further compromise perfusion. The inflammatory response to wounding may be prolonged as a

result, thus delaying the healing process. This is dramatically demonstrated in the development of compartment syndrome in limb skeletal muscles following ischemia and reperfusion.[24]

7. **Ionizing radiation**: Prior irradiation interferes with blood supply and result in slow wound healing. Acutely, irradiation of a wound blocks cell proliferation, inhibits contraction, and retards the formation of granulation tissue.
8. **Ultraviolet light**: Exposure of wounds to ultraviolet light accelerates the rate of healing.

Systemic Factors:

1. **Regional vascularity**: The vascularity of the area surrounding the wound is important. Impaired perfusion results in poor healing.
2. **Sepsis**: Delays wound healing.
3. **Metabolic status**: Diabetes mellitus is associated with delayed wound healing because of increased wound infection in diabetics and also microangiopathy.
4. **Nutrition**: Malnutrition impedes wound healing. Methionine and Zinc is needed for proper healing. Vitamin C is also required for collagen synthesis and secretion if deficient results in impaired wound healing.
5. **Hormones**: Corticosteroids impair wound healing by inhibition of collagen synthesis, anti-inflammatory actions and depression of protein synthesis. Thyroid hormones, androgens, estrogens, and growth hormone also influence wound healing.

CLASSIFICATION OF WOUNDS: [25]

Two types of classification can be made.

1. Pathological
2. Clinical

PATHOLOGICAL CLASSIFICATION:

NON-SPECIFIC ULCERS: These ulcers can be classified into the following categories

1. Traumatic
2. Venous
3. Arterial
4. Tropical
5. Trophic
6. Certain types – bazin's, martorell's, meleney's

SPECIFIC ULCERS: Ulcers due to specific underlying conditions.

1. Tuberculous ulcers
2. Syphilitic ulcers
3. Actinomycosis

In developed countries, the commonest chronic wounds are leg ulcers.[26]

CLINICAL CLASSIFICATION:

Clinically ulcer can be categorized into three types:

1. Spreading ulcer
2. Healing ulcer
3. Chronic or callous ulcer.

SPREADING ULCER:

A Spreading ulcer has the surrounding space inflamed and the floor is covered with riotous and offensive slough with no proof of granulation tissue. The edges are inflamed, dropsical and ragged.

HEALING ULCER:

In a healing ulcer the floor is covered with pink or red granulation tissue. The edges are red and sloping with granulation tissue and the margins appear bluish with growing epithelium.

CHRONIC ULCER:

Ulcers which have failed to proceed through the orderly process that produces satisfactory anatomic and functional integrity are known as chronic ulcers .[19]

The accepted definition of 'chronic wound or ulcer' relates to any wound that fails to heal within a reasonable period. However, most surgeons would agree that a wound that fails to heal within 3–4 months may be regarded as chronic.[27]

The floor is covered with pale granulation tissue. It shows typical wash leather slough with scanty discharge. The surrounding skin and the edge are indurated. If a wound does not follow the normal trajectory it may become stuck in one of the stages and they become chronic.

Chronic Ulcers and Protracted Inflammation

In contrast to the normal, natural course of wound repair described above, chronic cutaneous ulcers are considered to be arrested and 'trapped' in an ongoing inflammatory phase .[28-30]

A protracted inflammatory process develops in ulcers where normal mechanisms of wound healing are not sufficient to enable the wound to heal completely. This occurs due to bacterial infection or because of foreign material that cannot be removed, solubilized or phagocytized. Clinically, the bed of a chronic cutaneous ulcer appears fibrotic and to contains variable amount of necrotic tissue.

The main features that characterize chronic ulcers are as follows:

1. Increased enzymatic activity of matrix proteases
2. Reduced response to growth factors
3. Cell senescence

Increased Enzymatic Activity of Matrix Proteases

Chronic ulcers have high enzymatic activity of matrix metalloproteases (MMP), which degrade growth factors and extracellular matrix components such as collagen, fibronectin and vitronectin.[29-35]

There is decreased activity of MMP inhibitors, which neutralizes those unwanted effects.[36, 37] The ongoing degradation of a freshly formed matrix by MMP impairs and prevents normal wound healing.

Reduced Responsiveness to Growth Factors

The level of growth factors is not necessarily lower in chronic ulcers than in acute lesions. Numerous studies of growth factor levels in chronic ulcers have reported a wide range of results.[29, 36] General theory is that the growth factors in chronic ulcers are subjected to ongoing degradation due to increased protease activity.

Studies suggest that in chronic ulcers there is decreased expression of growth factor receptors. [41, 42]

Cell Senescence

"Senescence' is derived from the Latin word senescere, which means growing old. As described by Dorland's Medical Dictionary, 'senescence' indicates the process of growing old, especially the condition resulting from the transitions and accumulations of the deleterious aging process. Old cells are characterized by reduced proliferative capacity.[43-46] Studies suggests that each human cell is programmed to have a limited number of cellular divisions, defined by its origin and nature. After a finite number of divisions, the cells reach a state of senescence, with subsequent reduced proliferative capacity. An in-vivo model of neonatal fibroblasts demonstrated that these cells reached growth arrest after 40–60 population doublings.[47] Senescent cells have characteristic morphological features; i.e., they tend to be larger than cells that have not undergone such changes.[48, 49] In addition, they have specific biochemical changes, such as an over-expression of matrix proteins (e.g., cellular fibronectin). Senescent cells have a decreased response to growth factors, Mendez et al[50] and Vande-Berg et al[51] demonstrated that fibroblasts extracted from the margins and beds of chronic cutaneous ulcers appear prematurely senescent. Agren et al.[52] demonstrated that fibroblasts extracted from chronic cutaneous ulcers revealed characteristics of senescence; their invitro growth was significantly slower compared with that of fibroblasts isolated from acute wounds or normal skin.

Possible explanations for the presence of senescent cells in cutaneous ulcers are:

1. Cells within the surface or margin of a cutaneous ulcer are continuously stimulated to proliferate. On the opposite hand, the essential pathologic processes resulting in ulceration (e.g., infection, poor vascularization, external pressure) still exist and prevent healing. Mendez[50]suggests that in these cases, cells undergo numerous unnecessary futile divisions and bit by bit lose their proliferative capability.
2. It is suggested that chronic wound fluid and the ulcer microenvironment contain certain components that lead to cellular senescence. Certain cytokines[53] or bacterial toxins[54] may be involved in this process. Research studies have shown that chronic wound fluid suppresses in-vitro proliferation of fibroblasts, keratinocytes and endothelial cells.[54]

The following are some of the accepted classification system for chronic wounds like diabetic ulcers, venous ulcers and pressure sores.

Classification of Diabetic Foot ulcers: (Meggitt 1976, Wagner 1981) [55]

- Grade 0 - Intact Skin
- Grade 1 - Superficial Ulcer
- Grade 2 - Deep Ulcer to tendon, bone or joint
- Grade 3 - Ulcer with abscess or osteomyelitis
- Grade 4 - Forefoot gangrene
- Grade 5 - Whole foot gangrene

Staging of Venous Stasis Ulcers: [56] (Regional Wound Care Guidelines)

- **Partial thickness**: skin loss involving the epidermis or the dermis or both, a shallow crater, blister, abrasion or skin tear.

- **Full Thickness**: skin loss involving damage to or necrosis of the dermis and the epidermis, it may also affect subcutaneous tissue, muscle, tendon the bone.

Staging for Pressure Sores :(National Pressure Ulcer Advisory Panel-NPUAP) [57]

- Stage I - Non blanch able erythema of intact skin.
- Stage II - Partial thickness skin loss involving the epidermis and/or the dermis.
- Stage III - Full thickness skin loss involving damage or necrosis of subcutaneous tissue that may extend down up to but not through the underlying fascia.
- Stage IV - Full thickness skin loss with extensive obstruction, tissue necrosis or damage to muscle, bone, or supporting structures.

TREATMENT OF CHRONIC WOUNDS[58]

I. Some Treatment Options Available at Present**

General

1. Prevention and control of infection.
2. Treating the associated factors like edema, diabetes, heart disease, ischemia, varicose veins and anemia
3. Relieve the pressure if any.
4. Prevention of malnutrition and vitamin deficiencies.
5. Removal of any necrotic material or foreign body.

Specific

1. Physical modalities:

- Ultrasound.
- Hyperbaric oxygen.
- Vacuum-assisted closure.
- Bio surgery (myiasis).
- Electrical and electromagnetic therapy.
- Intermittent pneumatic compression.
- Pressure relieving mattresses and other appliances (in pressure ulcers).
- Compression garments (to treat lymphedema).
- Various forms of compression bandages and graduated compression hosieries.
- Other forms of mechanical wound debridement.

3. Drugs shown to be beneficial:

The treatment modalities listed here are recommended for direct treatment of the wound or for predisposing or associated factors. They should be chosen on an individual patient basis taking different factors like general condition of the patient, etiology of the ulcer, state of the ulcer bed, infection, etc, into consideration.

Systemic

- Pentoxifylline.
- Prostacyclin analogues (e.g., Iloprost).

Topical

- Phenytoin sodium.
- Nitric oxide donors (e.g., glyceryltrinitrate)
- Calcium channel blockers (e.g., diltiazem) in the treatment of chronic anal fissures.

3. Surgery:

- General: Debridement, skin grafting.
- Pressure ulcers: Ostectomy, excision of callus reconstructive surgeries (e.g., various rotation flaps).
- Venous ulcers: For varicose veins: Ligation, stripping, graded compression stockings, endoscopic subfascial perforator vein ligation, and valvuloplasty.
- Arterial ulcers: Bypass procedures, angioplasty.
- Ulcers due to lymphedema: Lymphangioplasty, shunts (lympho-venous, lympholymphatic), excision of skin and subcutaneous tissue (Charles Procedure).
- Amputation of affected limb

4. Others:

- Tissue-engineered skin substitutes.
- Gene therapy—application of various recombinant growth factors to the wound.

II. Future Treatment Strategies

1. Use of natural or synthetic exogenous protease inhibitors.
2. Protection of protease inhibitors from oxidative damage (including use of free radical scavengers).

3. Functional modulation of neutrophils (blocks or inhibits neutrophil functions, such as chemotaxis, adherence, infiltration, or degranulation).
4. Inhibition of enzymes involved in generating reactive oxygen metabolites.
5. Direct application of bone marrow-derived cells to chronic wounds.
6. Inhibit or neutralize neutrophil elastase activity.

Additionally antibiotics, both oral and topical are used to prevent and control infection in chronic wounds. [58]

CLINICAL ASPECTS OF LOWER LIMB ULCERATION:

The risk for an adult individual in developing a leg ulcer is 2.6%, however could also be high as 20% in the tropics. Chronic ulcers have a decreased frequency of spontaneous healing with healing times varying from 89-140 days. Identification of the underlying etiology of the ulcer is vital, although is the neglected part of the surgical care. Establishment of the etiology of the ulcer helps in correction of primary condition. This can be achieved by team work from doctor nursing staff and podiatrists.

The patient's co-morbidity and the control of cardiac failure, diabetes mellitus, and other causes of leg swelling should be reviewed as routine and appropriate treatment regarding same is to be given. In addition, exercise with graduated compression support, if appropriate, may be of beneficial. Patients are more likely to comply with treatment if they are properly informed about the disease and its management. As with all wounds, patients with chronic leg ulcers should have optimum nutrition.

Diabetic Ulcers:

Diabetics have high risk of developing an ulcer on the foot. Ulceration occurs due to reduced blood supply or due to impaired nerve function. Dermal ischemia is a consequence of narrowing of the large blood vessels of the leg and impaired function of the small vessels.

The effect of neuropathy is complex. Sensory loss in the foot leads to abnormal and prolonged pressure in these areas. Motor neuropathy leads to foot deformity, further increasing pressure and contributing to the ulcer formation. Loss of innervations to the sweat glands results in dry skin due to diminished sweating and leads to dry, cracked skin. These cracks may are portals for infection which complicates diabetic neuropathy. Alteration in the distribution of microcirculatory blood flow due to autonomic dysfunction directs the blood flow through shunts and away from the nutritive skin capillaries. It is the interplay of these factors and foot trauma that results in skin breakdown.

The Neuro-Ischaemic Foot:

Research on the etiology of foot ulceration would suggest only around 10% are vascular, 40% are neuropathic and 40% are neuroischaemic. The combination of both vascular and neuropathic features simultaneously in the same foot has a bad prognosis. The features of the neuro-ischaemic foot are a combination of those of ischemic and neuropathy.

Principles of Management of Ulcers in Diabetic Patients

Management of the Neuropathic Ulcer:

Prevention is vital in the management of the neuropathic foot. Patients are to be educated to minimize the risk of injury to the foot. Patients should be educated and encouraged to inspect their feet daily, to look for early ulceration. Moisturizers are to be used for dry skin to prevent cracking of the keratin. Patients with decreased temperature sensation should be advised to test water temperature with a hand before putting their foot in. Barefoot walking is to be avoided. Ill-fitting shoes are to be discarded and use of socks daily to be advocated .They should be careful when wearing new shoes, checking their feet for any signs of pressure and advised to buy shoes of bigger size.

Callus formation should be attended and callus to be pared away. Reduction in pressure can be achieved by the use of offloading shoes, custom made for each patient's foot.

The ischemic foot may be suitable for revascularization, and should be assessed as described above. The management of the diabetic foot is multidisciplinary involving a surgeon, diabetologist, vascular surgeon, and nurse. Irrespective of the etiology of the ulcer, local wound care is important. The removal of necrotic tissue is to be achieved surgically or by using formulations which encourage debridement. This reduces the risk of infection and will enhance wound healing. Hydrophilic dressings can be used to maintain moist environment at the ulcer surface. Treatment of infection with antibiotic courses according to bacterial cultures is advocated.

Adequate relief of pain must be achieved with analgesia; if this cannot be satisfactorily achieved, intervention may be required.

Ischemic Ulcers

Most patients with peripheral arterial disease are over the age of 70. In contrast to venous ulcers, arterial ulcers are increasing in number. People live longer nowadays, and peripheral arterial disease is becoming more prevalent. Arterial ulcers are estimated to constitute about 10% of leg ulcers.[59] In most of the cases; 'arterial' ulcers develop after physical trauma**.** In rest of the cases, arterial ulcers develop without any trauma, when critical limb ischemia has developed.

Principles of Treatment:

Patients will have rest pain and low ankle blood pressures (< 50 mmHg). The suggested inclusion criteria for critical leg ischemia are absolute ankle pressure below 50–70 mmHg or reduced toe pressure (<30–50 mmHg).[59] Without intervention chance of losing limb is high. Angioplasty or bypass surgery or a combination of both will be necessary. When revascularization is poor patient will require amputation. When ankle pressure is around 80 mmHg and above ulcers may heal with conservative management.

There is experimental evidence that sympathectomy has a positive effect on wound healing and as the sympathetic chain contains some pain fibres it may alter pain perception. There is no strong evidence that sympathectomy benefits in ischemic ulcers.

Venous Ulcers

Principles of Treatment:

Increased venous pressure is the etiology in venous ulcer formation and its treatment is directed towards reduction of this increased pressure. Measures like raising the foot end of the bed and elevating the leg can be tried. Graded compression can also be tried using compression stockings, which exert more pressure at the ankle than the calf, the ulcer at this region makes it difficult to apply and elastic crepe bandages have to be used.

Surgically Ligation of an incompetent sapheno-femoral junction or calf perforator is helpful in fastening the healing of venous ulcers and even reduced risk of recurrence.

THE ULCER DEBRIDEMENT AND CLEANSING

WOUND BED PREPARATION: AN OVERVIEW

Wound bed preparation can be defined as the global management of the wound to accelerate endogenous healing or to facilitate the effectiveness of other therapeutic measures. Difference between wound bed preparation and wound debridement is to be understood for proper wound management. With this knowledge confusion may develop that wound bed preparation is same as wound debridement. In acute wounds, debridement is necessary to remove necrotic tissue and bacteria. But in the case for chronic wounds defining the necrotic material in chronic wounds wouldn’t be easy. In chronic wounds, necrotic burden consists of both necrotic tissue and exudate. Studies on exudates from chronic wounds have been shown to inhibit the

proliferation and function of key resident cells and to contain proteases that breakdown extracellular matrixproteins.[60, 61]

Chronic wounds are intensely inflammatory which produce large amounts of exudate which interfere with wound healing or with the effectiveness of therapeutic products, such as growth factors and bioengineered skin. In wound bed preparation, removal of actual eschars and dead tissue, and also the exudative component is noticed. Moreover, there is increasing realization that the resident cells in chronic wounds, e.g., fibroblasts and keratinocytes, may be phenotypically altered and no longer are responsive to certain signals, including growth factors.[62]

Factors such as infection, necrotic tissue, desiccation, pressure, nutritional status, age, and co-morbid diseases hinder healing. Steps taken to promote wound healing, known as wound bed preparation, is a multistep process which can be defined as "the global management of wound to accelerate endogenous healing or to facilitate the effectiveness of other therapeutic measures." It involves decreasing bacterial load, managing exudates, and removing necrotic or fibrous tissue. [63]

Necrotic Tissue and its Accumulation in Chronic Wounds

Necrotic tissue often at the ulcer centre is called slough. [26] Necrotic tissue is dead tissue, and is because of inadequate local blood supply. It contains dead cells and debris. As dehydration sets in and starts progressing, it changes its color from red to brown or black/purple, finally, it forms eschar which is dry, black, thick, and leathery. This is seen in burns and all types of chronic wounds. Slough is yellow fibrinous tissue which contains pus, proteinaceous material, and fibrin. Slough can be found on

the surface of a previously clean wound bed and it is thought to be associated with bacterial activity.[58]

Necrotic tissue and or slough accumulation in chronic wounds is of clinical significance, as it is thought to promote bacterial colonization and delay wound healing. Lately, the term necrotic burden has been proposed as an all-encompassing term to describe excess exudate, necrotic tissue, and high levels of bacteria present within dead tissue.[64]

Due to the underlying pathogenic abnormalities in chronic wounds and the altered biochemical and cellular environment, necrotic tissue tends to continually accumulate.[64] It is not always feasible to fully remove the underlying pathogenic abnormality, making it even more essential to adequately prepare the wound bed. If the necrotic burden is allowed to accumulate in the chronic wound, it can prolong the inflammatory response, mechanically obstruct the process of wound contraction, and impede re-epithelisation.[65]

Debridement

The term ‘debridement’ was first coined by Desault (1744–1795) from Paris, referring to the surgical removal of necrotic material from open wounds.[66] As defined by Dorland’s Medical Dictionary “the removal of foreign material and devitalized or contaminated tissue from or adjacent to a traumatized or infected lesion, until surrounding healthy tissue is exposed.”

The choice of the method of debridement, during the course of the treatment of an ulcer, changes. Which is decided by the systemic and local factors and also availability of resources along with the experience of the surgeon. Necrotic material

forms in two types which are slough, and eschar or crust. Slough will be yellow, green, and gray/white in colour and is soft, liquefied mass to semi-solid or relatively solid material and is composed of necrotic proteins, devitalized collagen, and fibrin. Necrotic material dries hardens and forming brown or black eschar. Eschar is formed from devitalized proteins and collagen, with cell debris and solidified secretions. Secretions on the ulcer bed dries and forms crust. It does not contain dead tissue, though resemble eschar requires debridement for removal.

Effect of slough on ulcer healing

- Necrotic material over ulcer bed increases bacterial colonization and results in infection.[67-69]
- Necrotic material induces activation of the alternative pathway of the complement system .[70] This may result in ongoing inflammation, destruction of surrounding healthy tissue, and delay of the wound healing process.
- Foreign bodies in ulcer, acts like physical barrier and prevents the conventional course of wound healing and epithelialization.[67,69] Dry crust or eschar over ulcer bed results in slower epithelialization, as compared with a moist wound environment which has faster healing rate.[71]

Objectives of debridement:

- **Removal of a fibrin layer**: A layer of fibrin coats the ulcer bed, forming a physical barrier that prevents penetration of growth factors and prevents take up of a skin graft or transplanted keratinocytes.
- **Achieving a better vascular bed**: debridement results in an ulcer bed which is more vascular, and provides a better substrate for further treatment modalities.

- **Removal of senescent cells**: Senescent cells, whose ability to produce cytokines and to proliferate is reduced, are present in chronic cutaneous ulcers.[72-74] Removal of senescent cells from the ulcer's margin and its surface may improve wound repair.[75]

Benefits of debridement

Removal of bacteria.

Necrotic tissue supports bacterial growth in an ulcer. Microbes in a chronic wound are inevitable and are not necessarily detrimental to healing. Most of chronic wounds heal in a polymicrobial environment. But ulcers with increased bacterial burden (> 10^5 colonies / gram of tissue) show reduced healing compared to wounds containing fewer bacteria.[76]

Infected or heavily colonized wounds feature friable and hemorrhagic granulation tissue and decreased tensile strength. [77] The type of bacterial species will also be important. A single species, such as beta haemolytic streptococci or combinations of species can be harmful to a wound regardless of number.[78] Debridement will be effective in detachment and removal of biofilms from the wound bed. Prolonged exposure to bacteria in chronic wounds leads to an altered and prolonged inflammatory response, resulting in the release of free oxygen radicals and various lytic enzymes that stimulate tissue damage. [79] Decreasing the bacterial burden, debridement ultimately reduces the factors that impede the healing process.

Stimulation of growth factor activity.

Chronic wounds are deficient in availability of important growth factors, such as platelet-derived growth factor (PDGF), fibroblast growth factor (FGF), epidermal

growth factor (EGF), and transforming growth factor beta (TGF-α).[80] Growth factors which are present but are unavailable due to abnormal binding to matrix proteins; explained by 'growth factor trap hypothesis', proposed as a cause for venous ulceration.[81] Here it is proposed that venous hypertension results in the leakage of macromolecules into the dermis that trap growth factors, making them unavailable for wound repair. Even if present, growth factors need to be exposed to properly functioning cells with the appropriate receptors to bind effectively. In chronic wounds, dead tissue is unresponsive to growth factors and act as physical barrier for growth factor-receptor interaction.[82]

Debridement accelerates healing by clearing dead necrotic tissue, thereby, uncovering viable receptors for growth factors to bind. Debridement, leads in bleeding, which stimulates blood borne growth factors production. In practice, debridement commonly precedes topical application of growth factors. The reason for this practice is that patients heal within a greater percentage of the time when rhPDGF is combined with surgically debrided diabetic foot ulcers compared with application of rhPDGF alone**.**[83]

Removal of senescent cells.

Cellular senescence may contribute to the impaired healing in chronic wounds. Senescent or aged cells are cells have decreased proliferation and protein synthesis even though they are viable. These senescent cells and fibroblasts have been found in a variety of chronic wound types.[84] Studies that have found that wounds present for long duration are more difficult to heal.[85] The quantity of senescent fibroblasts increases as wound healing progresses. Debridement removes the senescent fibroblasts leaving younger cells and an overall healthier environment for the wound

to heal. One role of debridement is to remove the callus often surrounding chronic wounds, especially neuropathic or pressure ulcers. In addition to callus, the edge of a chronic wound may be thickened or hyperproliferative. This finding may be accentuated to a degree that the wound edge can take on, histologically, a pseudo carcinomatous appearance. Unfortunately, epithelial proliferation and migration are two distinct biologic phenomena. A hyperproliferative epithelium is nonmigratory and thus slows healing. Debridement can remove this hyperproliferative, nonmigratory edge.[86]

TYPES OF DEBRIDEMENT:

1) Autolytic debridement.

Autolytic debridement is a natural process occurring in ulcers, wherein endogenous enzymes digest and break down dead tissues and help in separating it from healthy tissue. This is more efficient in well-hydrated ulcers. In every occlusive or semi-occlusive dressing there will be some degree of autolytic debridement, as the dressings prevent water from evaporating, and fluids accumulate within the ulcer's environment.

All wounds show some level of autolytic debridement process by which endogenous proteolytic enzymes break down the necrotic tissue. These endogenous enzymes are produced by neutrophils and include elastase, collagenase, myeloperoxidase, acid hydrolase, and lysosomal Enzymes.[87] Autolytic debridement is not fast process and rapid wound healing doesn't take place, use of an occlusive dressing enhances the process, while maintaining a moist wound bed and managing excess exudate.[88] This allows painless, selective debridement and promotes the formation of healthy granulation tissue.[89] Typical practice for autolytic debridement involves the use of a hydrogel to soften and break down necrotic tissue covered with

an absorptive, occlusive dressing to absorb the excess exudates. Autolytic debridement products can be found in many different varieties, including different properties, benefits and limitations. They can be defined in the following groups:

A Hydrogels, or hydrogel-based dressings, are three-dimensional, cross-linked homopolymers or copolymers, saturated with water. The proportion of water in hydrogel dressings can vary from 30% to 90%. Different gel-forming agents, such as carboxymethylcellulose, are incorporated into most hydrogels**.**[90]

B Hydrocolloids are composed of carbomethylcellulose, gelatin, pectin, elastomers and adhesives that turn in to a gel when exudate is absorbed.[91]

C Highly absorptive dressings with autolytic and occlusive properties, such as dressings with a multifunctional polymeric membrane formulation and hydrations techniques (e.g. hydration response technology). These dressings are designed for exudation management, aiming to create a moist and physiological environment for autolytic debridement.[92]

2) Surgical debridement.

Surgical instruments like a scalpel or scissors are used to cut away and remove necrotic tissue. Forceps should be used to grasp necrotic tissue while it is being cut and to remove it from the ulcer.[93] Surgical debridement is the fastest way to remove dead tissue. It causes pain and was restricted to the treatment of neuropathic diabetic ulcers. Although surgical debridement is thought to be selective, there may be some damage to viable tissue, and bleeding is likely. Mild to moderate bleeding could be controlled by the application of pressure and a hemostatic calcium alginate dressing**.**[94] Surgical debridement removes firm, black necrotic material but it may be only

possible to remove yellow/ gray slough, if the slough is relatively solid. Soft, liquefying slough can't be handled efficiently by a surgical knife or forceps. When liquefying slough is present over ulcer bed it's difficult to differentiate between necrotic and vital tissues clearly then other debridement methods need to be used.

Pale and slough-like normal tissue may recover following appropriate treatment. Here surgical procedures may lead to unnecessary loss of healthy and vital tissue.

3) Mechanical debridement.

Mechanical debridement is a nonselective, physical method of removing necrotic tissue and debris from a wound using mechanical force. However, this nonselective method can damage healthy granulation tissue both in the wound bed and at the margins of the wound thus causing significant discomfort to the patient.[58]

Subtypes of mechanical debridement are:

- Hydro debridement,
- Wet-to-moist' dressings,
- 'Wet-to-dry' dressings,
- Irrigation with saline,
- Mechanical scrubbing.

Hydro debridement

Ulcer soaked in a bath of water softens and loosens slough or dry necrotic tissue, separating it from the ulcer bed. In addition, bacteria and residues of topical preparations are washed away.[95, 96]

Wet-to-Moist Technique

Achieved by covering the ulcer gauze soaked with fluid (e.g., saline), with repeated wetting. In the 'wet-to-moist' dressing, the ulcer is not allowed to dry out which is aimed at keeping ulcers moist which results in a debriding effect. Similar to the hydro debridement method, the water may soften and loosen slough or dry necrotic tissue. It is intended to soften and loosen slough or dry necrotic tissue. In contrast to soaking, repeated wetting achieves the opposite effect, as described below. When the added water (either by washing or repeatedly applying a damp cloth or damp gauze) evaporates, the treated area gradually dries out. This is intended for secreting ulcers. Repeated wetting is not considered to be a debridement technique – it is just a cleansing method that can also be used for drying out any other types of inflamed, secreting areas of the skin.

Wet-to-Dry Technique

This is a modification of the Wet-to-MoistTechnique technique, where gauze dressing is left to adhere to surface of the ulcer. It is a useful where necrotic tissue has moderate amounts of exudate. Here a gauze dressing is moistened with saline and applied to the ulcer, over the necrotic material and left to dry. After a few hours, when the gauze is dry and adherent to the ulcer bed, it is pulled firmly, with the necrotic tissue attached to the gauze. This procedure may be repeated several times a day. The main disadvantage of this debridement method is that, being non-selective, newly regenerated epithelium and healthy granulation tissue are removed from the ulcer bed together with necrotic material. It also causes pain to the patient when sensations are intact. In view of this, a 'wet-to-dry' dressing is generally not favored as a debridement procedure.

Irrigation with Saline

Frequent irrigation with saline is a good method for removing seropurulent or purulent secretions and liquefied slough. But it won't remove solid slough necrotic eschar which is firmly adherent to the ulcer bed. The procedure can be performed once or twice daily, while the wound dressing is being changed, with the aim of removing remnants of topical preparations previously used on the ulcer.

Mechanical Scrubbing

Removal of slough and necrotic tissue by scrubbing also has an adverse effect like the 'wet-to-dry' technique causing damage to regenerating epithelium and granulation tissue. It should therefore be avoided.

Absorptive Debridement- A Variant of Mechanical Debridement:

The mechanical effect of absorption may be regarded as an additional method of debridement. Such procedures use the absorptive qualities of hydrophilic dextranomer granules or activated charcoal for removal of tiny pieces of necrotic material and bacteria from the ulcer bed. These preparations, intended for secreting ulcers.

Other topical methods of debridement may be based, atleast in part, on absorptive/osmotic activity. These include preparations such as sugars .[97-101]

4) Enzymatic Debridement

The concept of using proteolytic enzymes to digest necrotic tissue as an adjunct in the treatment of complex wounds is rather old and probably stems from observing the ageless healing techniques of natives in tropical countries. As an

example, for wound debridement, these natives seem to have utilized the papain-rich material obtained by scratching the skin of the green fruit of the papaw tree (Carica papaya). There are commercial enzymatic preparations directed specifically towards certain substances contained in necrotic tissue such as fibrin, collagen, or various other proteins. In order not to damage healthy tissue, enzymatic debridement is used for an ulcer whose entire surface is covered by necrotic material. In addition, there is a basic assumption with this approach that vital cells are capable of producing inhibitors against these enzymatic preparations and remain intact, while necrotic tissue is being dissolved. Enzymes for chemical debridement are classified as proteolytics, fibrinolytics, or collagenases. However, the distinction presented above is not clear-cut. There are no definite data in the literature regarding the preferred enzymatic preparation for any particular type of necrotic material. Enzymatic Preparations Documented in the Literature Collagenase is derived from Clostridium histolyticum. Fibrinolysin is derived from bovine plasmin. A streptokinasepreparation, produced from Streptococcus. Trypsin is derived from an extract of ox pancreas. Krill enzymes are derived from the digestive system of a small shrimp (Antarctic krill – Euphausiasuperba).These products are potentially allergenic.[102]

5) Bio surgery (myiasis).

This also coined as ‘biological debridement’, ‘biotherapy’, or ‘bio surgery’. This procedure based on the theory that few strains of maggots are nourished only by dead tissue and not on healthy living tissues. The type of larvae that are commonly used for this procedure, being safe and therapeutically efficient, are Lucilia sericata

(green bottle fly).[103] Beneficial effect of maggots was documented by. Ambroise Pare[104] a few centuries ago.

Sterile maggots are used in this technique, which eat slough and necrotic material in the wound without damaging the surrounding healthy tissue. A study by Mumcuoglu, et al.[105] showed complete debridement using maggots in 38 of the 43 patients (88%) with chronic leg ulcers and pressure ulcers. Of them, five patients had their limbs salvaged after being referred for amputation of the leg. It remains unclear how maggots debride the wound and promote wound healing. However, it is hypothesized that they might act by ingesting and killing bacteria, by increasing wound pH they exert a bacteriostatic effect, [106] secreting proteolytic enzymes that are important in eschar degradation, and increasing tissue oxygenation.[107]

DRESSINGS

Surgeons experimented with numerous antiseptic solutions and various types of surgical dressing. A principle of wound treatment applied by means of debridement and irrigation eventually evolved. Henry Dakin (1880–1952), an English chemist, and Alexis Carrel (1873–1944), the Nobel prize-winning French American surgeon, were the principal protagonists in the development of this extensive system of wound management and dressings.[108]

Ideal wound dressing:

There is no single dressing is suitable for all types of wounds. Often a number of different types of dressings will be used during the healing process of a single wound. Dressings should perform one or more of the following functions:

- Maintains a moist environment at wound/dressing interface

- Absorbs excess exudates
- Provides insulation and mechanical protection
- Provides bacterial protection
- Allows gaseous and fluid exchange
- Absorbs wound odour.
- Should be non-adherent to the wound and easily removed without trauma**.**
- Provides some debridement
- Be non-toxic, non-allergenic and non-sensitizing (to both patient and medical staff)
- Sterile
- Maintain high humidity
- Provide optimum pH (slightly acidic)
- Discourage infection
- Be easy to apply
- Be cost and resource effective

Classification of Dressings:

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

Dressings can be classified according to:

1. Short term application dressings: need replacement at regular intervals.
2. Long term applications / skin substitutes: They are further subdivided into:

- Temporary – Applied on fresh partial thickness wounds until complete healing

is ensured.

- Semi-Permanent – Applied on full thickness wounds until auto grafting.

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

In each of the category, the dressings can further be classified as:

1. Primary Dressing- in physical contact with the wound bed.
2. Secondary Dressing –that covers the primary dressing.
3. Island Dressing –that is constructed with a central absorbent portion surrounded by an adhesive portion.

The four main classes of dressings, as suggested by the Food and Drug Administration (FDA) on November 4, 1999, are: [109]

1. Non-resorbable gauze/sponge
2. Hydrophilic/absorptive
3. Occlusive
4. Hydrogel

A. Conventional Dressings

These dressings are made of fabric material such as gauze, which allow evaporation of moisture resulting in a dry desiccated wound bed and also allow entry of exogenous bacteria into the wound. This led to the origin of compound dressings like Tulle grass which is wide mesh gauze impregnated with medical grade paraffin. This finally results in a relatively non-adherent dressing. Further developments in 1980 involved incorporation of antibacterial agents like carbolic acid and mercuric chloride, penicillin and polymyxin creams in combination with absorbent dressings. A

recent innovation uses silicone polymer in place of paraffin. These dressings are non-adherent to the wounds making dressing changes less traumatic. The concept of moist wound dressings gained importance during the mid-1980. Based on a study by Atiyeh BS, El-Musa KA, Dham R, full and partial thickness cutaneous wounds, when exposed to wet and moist environment showed improved healing. It has been found that moist environment prevents desiccation of denuded dermis or deeper tissues and allows faster and unimpeded migration of keratinocytes over the wound surface and also facilitates the cytokines to exert their effects on wound contracture and re epithelisation.[110]

As conventional dressings had limitations for application on full thickens wounds, researches into the development of more advanced wound dressings for the treatment of wounds have 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 with an adhesive on one side. The polymers include polyurethane, polyethylene, polytetra-fluoroethylene, dimethylaminoethylmethacrylate, Polycaprolactone. They are well suited for superficial wounds; still they lack absorbing capacity and are impermeable to water vapor and gases. They are not suitable for larger wounds.

2. Foams and sprays - Foam dressings are sheets of foamed solution of polymers such as polyvinyl-alcohol and polyurethane that are manufactured to contain air bubbles, they provide thermal insulation and help to maintain moist environment.

They are non-adherent, light, permeable to gas and comfortable. Examples: silastic foam and lyofoam.Theyare formable insitufor treating irregular cavity wounds.. Spray dressings are more comfortable to the ulcer cavity or surface. Most sprays are copolymers e.g., Aeroplast a copolymer of hydroxy vinyl chloride acetate modified maleic resin ester. Researches have also resulted in the development of dressings composed of spray and foam combinations, like gelatin based spray able foam.

3. Composite dressings - These are made of laminates of two or more layers. The outer layer is for elasticity and durability and may serves as controller for water evaporation, while the inter layer is for maximum adherence and elasticity. Composite dressings may be classified as follows:

A.Hydrocolloid dressings - Hydrocolloid dressings contain hydrophilic particles (mainly sodium carboxy methyl cellulose) that are gel-forming. Other substances may be included such as gelatin or pectin.

When applied, there is interaction between the hydrocolloid substance and the ulcer fluid, which forms a gelatinous mass over the ulcer. This gelatinous mass provides a moist environment, which helps in autolytic debridement, granulation tissue formation, and epithelialization.

B. Hydrogel sheets – These are sheets of 3-D networks of cross linked hydrophilic polymers. They interact with aqueous solutions or the ulcer fluid, which forms a gelatinous mass over the ulcer. Most commonly used polymers are polyethylene oxide, polyacrylamide, and polyvinylpyrrolidine. They also have cooling ability, and may be used in thermal burns.They are slippery to use and difficult to keep in place in a high sheer stress.

C. Hydrogel Amorphous – These are similar in composition to sheet hydrogels except that the polymer has not been cross linked so doesn't form a sheet. They contain alginate, silver, collagen, or complex carbohydrates. They donate moisture to a dry wound eschar and facilitate autolytic debridement. Hydrogel dressings exhibit more rapid rate of closure and re-epithelialization when compared with the hydrocolloid wound dressing.

d. Gels – Several types of gel based dressings have also been developed. For example hydroxyethyl methacrylate (HEMA) based hydrogel or silver impregnated hydrogel which was biocompatible and nontoxic.

e. Super Absorbents – This dressing has an island configuration consisting of an extra thin hydrocolloid as the adhesive portion with a central area of non-woven absorbent covering the superabsorbent particles encased inside, e.g., Combiderm, Conva Tec., etc.

C. Biological Dressings

These dressings are derived from the natural tissues consisting of various formulations and combinations of collagen, elastin, and lipid. They are superior to synthetic dressings in that they:

1. Restore a water vapour barrier and prevent dehydration of the wound.
2. Decrease evaporational heat loss.
3. Decrease protein and electrolyte losses in wound exudate.
4. Prevent bacterial contamination of the wound and hence protect the wound and patient from sepsis.
5. Permit less painful dressing changes.

6. Permit painless movement over joints.
7. Facilitate debridement of wounds.
8. Create good granulation tissue bed for autografting of deep wounds.
9. Can be used to test for successful subsequent autograft.
10. Decrease healing time of partial thickness burns and donor sites.
11. Improve quality of healing, inhibit excessive fibroblasts, and decrease contraction.

Biological dressings range from allograft, heterografts from pigs, dogs and other species, to embryonic membranes, embryofoetus 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.

Allograft:

Is a graft from one person to another of the same species, who do not have identical genetic characteristics. Allograft skin can be obtained from a family member but is most commonly harvested from cadavers. In burns especially for extensive full thickness burns use of fresh frozen lyophilized allograft is effective. Amniotic membranes are also been tried as allograft, but are not effective in the prevention of evaporative water loss.

Xenograft:

A graft taken from an individual of one species and transplanted onto an individual of another species. These are grafts from animal sources and porcine skin is the most commonly use xenograft owing to structural similarity to human skin with respect to its texture, adherence, and collagen content although not similar at the

microscopic level. The major disadvantages of xenograft and allograft are for them to become vascularized, immunosuppressive drugs are to be administered to prevent rejection. Again the excessive use of immunosuppressive drugs decreases the wound healing and increases the risk of wound infection.

Collagen Dressings

The dressings is are developed because of its structural and functional characteristics. The most highly characterized form of collagen aggregate is the fibril or fiber. It provides mechanical support to the connective tissue, and also form as an essential substrate for cellular adhesion and migration. So collagen is considered to be a most important morphogenetic factor in embryonic development and in the regenerative process. The hydrophilic nature of the collagen attributed by its molecular structure provides a surface geometry suitable for cell adhesion.

Engineered Skin Substitutes:

Collagen from bovine origin is used as substratum for culture. Cells grown on collagen are involved in the synthesis of glycosamimnoglycans which play a major role in the fastening ulcer healing. These gels, serve as a dermal like matrix over which keratinocytes can be cultured so as to form a better skin substitute. Keratinocytes grown on permeable base are exposed to air/liquid environment, which creates a good physiological environment and helping them to differentiate to a greater extent and more similar to the parent tissue. The air/liquid culture system can be divided into three categories.

1. Synthetic Membranes – Temporary alive skin replacement made of human neonatal fibroblasts cultured in a biosynthetic dressing material has replaced the cadaveric allograft skin.

2. Dermal Equivalent – A dermal equivalent is developed by culturing keratinocytes over fibroblasts that are derived from contracted collagen gel. This enables firm attachment of human keratinocytes to the skin substrate for graft survival on the ulcer.

3. Dead De-Epidermized dermis – Acellular de-epidermized dermis is less antigenic and provides natural base for engineering artificial skin, so it is proved to be an effective dressing for burns with extensive connective tissue loss. These human skin composites composed of normal human Keratinocytes and fibroblasts on human acellular de-epidermized dermis serves as better skin substitutes on wound**.**[111]

Hydrogel dressings:

Hydrogel is a colloidal gel in which water is the dispersion medium.It is a swellable and hydrophilic material, prepared from synthetic polymers like polymethacrylate or polyvinylpyrrolidine. Hydrogels can be produced in two types, amorphous or solid sheet/films. If hydrogels are applied to the wound as gels, usually they need a secondary dressing. When applied as films to the wound, they can be used both as a primary and secondary dressing.[112]

Hydrogels can be applied and removed with minimal trauma and pain from wound bed. Because of the cooling effect that hydrogels have on wound bed, they can give a relief feeling to patients.[113] However, hydrogels have some disadvantages also. Accumulation of fluid in hydrogels provides a suitable environment for bacterial

growth and can produce infected smell afterward. Therefore, hydrogels should be changed quite often. Hydrogels have low mechanical strength.[112]

Hydrogel dressings may also be considered for softening black, dry necrotic material, due to their water-donating properties. Due to the waterdonating properties of hydrogels, care must be taken that the ulcer and the healthy tissue around it do not become macerated.[114] Hydrogel should be used in conjunction with a secondary dressing. The gel changing interval may be up to 3 days.

Hydrogels fit most criteria for a suitable wound dressing as they:

- Help in the rehydration of dead tissues.
- Are suitable for cleansing of dry, sloughy or necrotic wounds.
- Do not react with biological agents.
- Are permeable to metabolites.
- Are non-irritant.
- Promote moist healing.
- Are non-adherent.
- Cool the surface of the wound.

Hydrogel dressings available in two forms:

1. Flexible sheet form: Have stable structure, swells and increases in volume as they absorb fluid.
2. Amorphous form : there is no fixed structure and becomes more fluidy and less viscous as it absorbs fluid.[115]

The ingredients of hydrogel used in this study are:

1. Purified water
2. Carboxymethyl cellulose
3. Calcium alginate.

More than 90% is purified water, providing a gentle debridement.

It has both hydrating and absorbing properties, which improves the natural autolytic debridement and moist wound healing. The gel will rehydrate and absorb debris and exudate when needed.

Once in contact with an exuding wound, an ion-exchange takes place between the calcium ions in the dressing and sodium ions in serum or wound fluid. When a significant proportion of the calcium ions on the fiber have been replaced by sodium, the fiber swells and partially dissolves forming a gel-like mass

The high cohesion makes Gel stay in place. The gel is easy to apply to the wound. Due to its viscous texture, the gel will not run. Even after absorption of debris and excess exudates, the gel will remain cohesive and counteract leakage, can absorb significant fluid volume without significant increase in size. It is semi permeable to fluids and vapors. It is non adherent to wound base.

Indications

It is indicated for dry and sloughy necrotic wounds as well as wounds with a mix of necrotic and granulated tissue such as:

- Leg ulcers
- Pressure ulcers
- diabetic foot ulcers
- 1st and 2nd degree burns
- Can also be used as a supplement to support moist wound healing in general

METHODOLOGY

The present study was conducted in the Department of General Surgery, KLE's Dr. Prabhakar Kore Hospital and Medical Research Centre, Belgaum over a period, from January 2012 to December 2012.

Study design

The study design was one year hospital based randomized controlled trial.

Study period and duration

The present one year study was conducted during the period of January 2012 to December 2012.

Place

This study was carried out at Department of General Surgery, KLE's Dr. Prabhakar Kore Hospital and Medical Research Centre, Belgaum a teaching hospital attached to Jawaharlal Nehru Medical College, Belgaum.

Source of Data

Patients presenting with leg ulcers at Department of General Surgery, KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belgaum

Sample size

A total of 60 patients were studied. Divided into two groups.

Sampling procedure

As the effect size was not available, by applying thumb rule a total of 60 cases were divided into two groups that is, 30 each in Hydrogel group and Normal saline group were studied.

Randomization

Based on the envelope method patients were randomized divided into two groups that is;

- Group A (n=30) – Patients in this group underwent dressing with Hydrogel.
- Group B (n=30) – Patients in this group underwent dressing with Normal saline.

Selection criteria

Inclusion Criteria

1. Patients having infected leg ulcers measuring more than 1cms,with slough, foul smell and minimal granulation tissue , between the age groups 18-80 years
2. Patients with grade I and grade II ulcers of Wegener's classification
3. Not allergic to hydro gel
4. Not allergic to semi-occlusive secondary dressing
5. Written informed consent

Exclusion Criteria

1. Patients with grade III, grade IV and grade V ulcers of Wegener's classification
2. Patients who are not regular on follow-up.
3. Patients not willing to give consent

Ethical clearance

Prior to the commencement of the study the study was approved from the Ethical and Research Committee, Jawaharlal Nehru Medical College, Belgaum.

Informed Consent

All the patients satisfying selection criteria were explained about the nature of study and a written informed consent was obtained before enrollment (Annexure I).

Method of collection of data

Demographic data such as age and sex were recorded. Patients were interviewed for the history and a thorough physical examination was conducted including vitals and systemic examination. These findings were recorded on a predesigned and pretested proforma (Annexure II).

Investigations

Routine investigations such as complete blood count, fasting blood sugar, serum protein levels, blood urea, serum creatinine and X-ray foot (AP and lateral view) and special investigation such as colour Doppler were done.

Procedure

Wound discharge was sent for culture and sensitivity if present. Empirical antibiotics namely ciprofloxacin and metronidazole were started and changed to sensitive antibiotics after sensitivity report.

Dressing

- In both groups povidine iodine was used as an antiseptic.

Group A

In Group A, Hydrogel was used, 0.5 mm thickness of gel was applied to the wound area; care was taken that gel was not applied to normal skin and secondary dressing was done. Depending upon the amount of exudate, dressing was changed daily or once in two days.

Group B

In group B plain normal saline was used which was one of the standard procedure for ulcer dressings.

Study variables

Ulcer was assessed by the investigator at the beginning of the study and at every 4 days. Ulcer mapping was made and slough area was recorded. Final slough area was measured on 16^{th} day.

Area of the slough was assessed on Day 0 and every 4^{th} day upto 16^{th} day or when the debridement reached 80%. To calculate and compare slough area, wounds were photographed with help of Sony 8 MP camera, after placing a scale next to the

wound. This picture was then analyzed using Image J™ software. The image was linearly calibrated using the 'ruler' and it was possible to derive the exact size of the marked area (slough) over the wound.

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

Statistical analysis

The data obtained was tabulated, categorical data was expressed as rates, ratios and percentages and comparison was done using chi-square test. Continuous data was expressed as mean ± standard deviation and comparison was done using student unpaired 't' test. A 'p' value of less than or equal to 0.05 was considered as statistically significant.

RESULTS

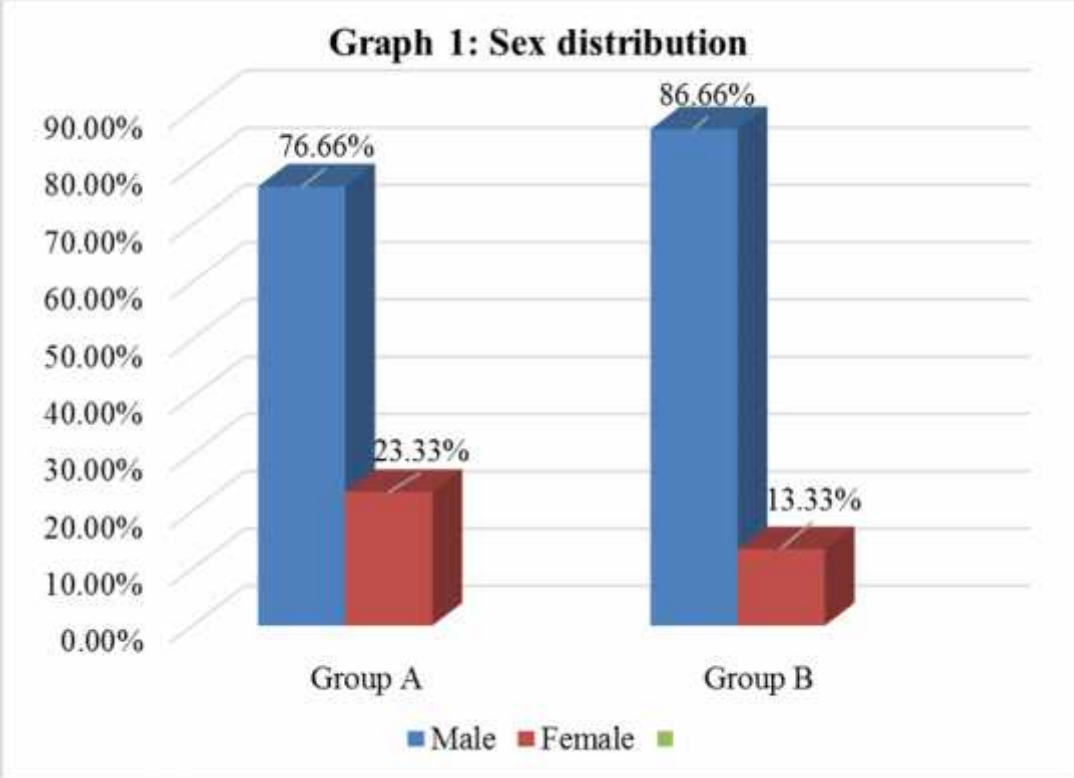
The present one year hospital based randomized controlled trial was conducted in the Department of General Surgery, KLES Dr.PrabhakarKore Hospital and Medical Research Centre, Belgaum from January 2012 to December 2012. A total of 60 patients with leg ulcers were studied. Based on the envelope method patients were randomized into two groups that is;

- Group A (n=30) – Patients in this group underwent dressing with Hydrogel.
- Group B (n=30) – Patients in this group underwent dressing with Normal saline.

Data obtained was tabulated on Microsoft excel spreadsheet and analysis was done. The final results were tabulated as below.

Table 1: Sex distribution

Sex	Group A (n=30)		Group B (n=30)	
	Number	**Percent**	**Number**	**Percent**
Male	23	76.66	26	86.66
Female	7	23.33	4	13.33
Total	**30**	**100.00**	**30**	**100.00**
	x^2 = 1.002			**p = 0.317**



In this study majority of the patients were males in both the groups (76.66 % in group A and 86.66% in group B).

Table 2: Age distribution

Age group (Years)	Group A (n=30)		Group B (n=30)	
	Number	Percent	Number	Percent
< 30	2	6.66	1	3.33
31 to 45	1	3.33	6	20
46 to 60	18	60	15	50
60 to 70	9	30	3	10
>70	0	0	5	16.66
	30	100	30	100

In this study most of the patients were aged between 46 to 60 years in both the groups.

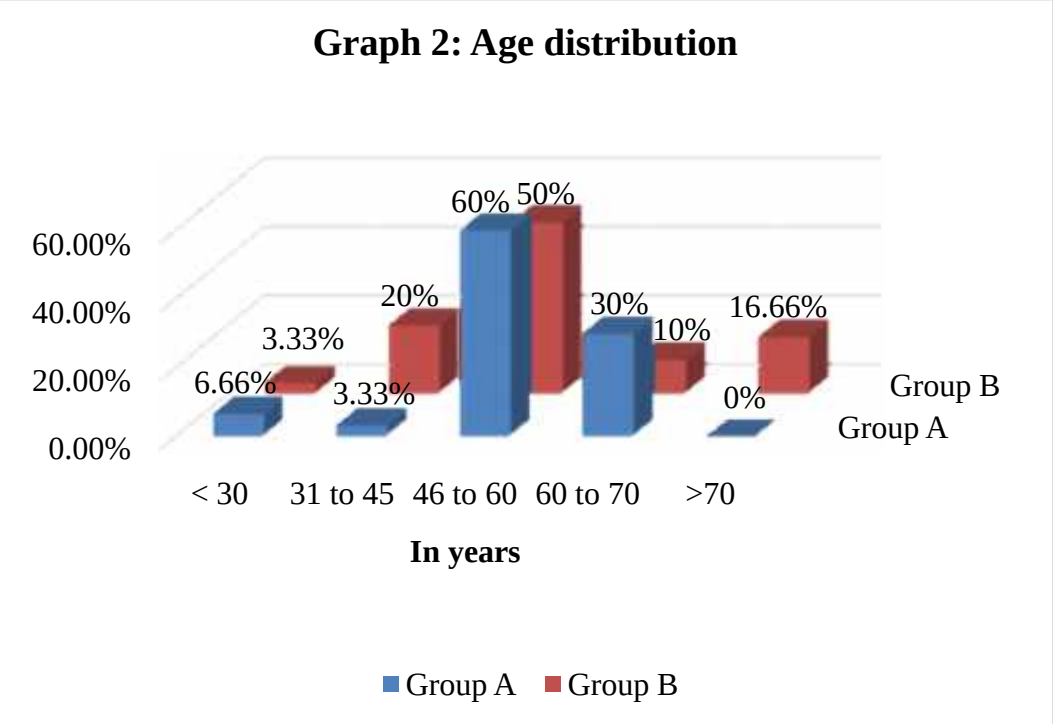
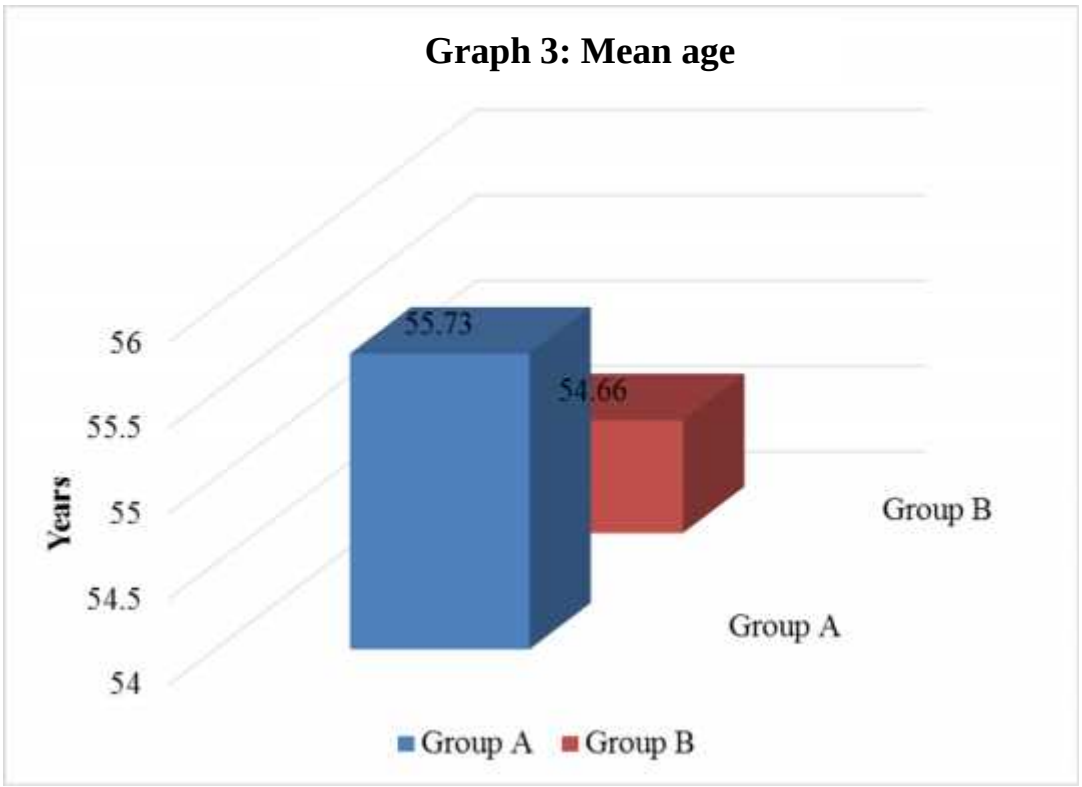


Table 3: Mean age

Variable (Years)	Group A (n=30)	Group B (n=30)
Mean	55.73	54.66
SD	9.40	12.73

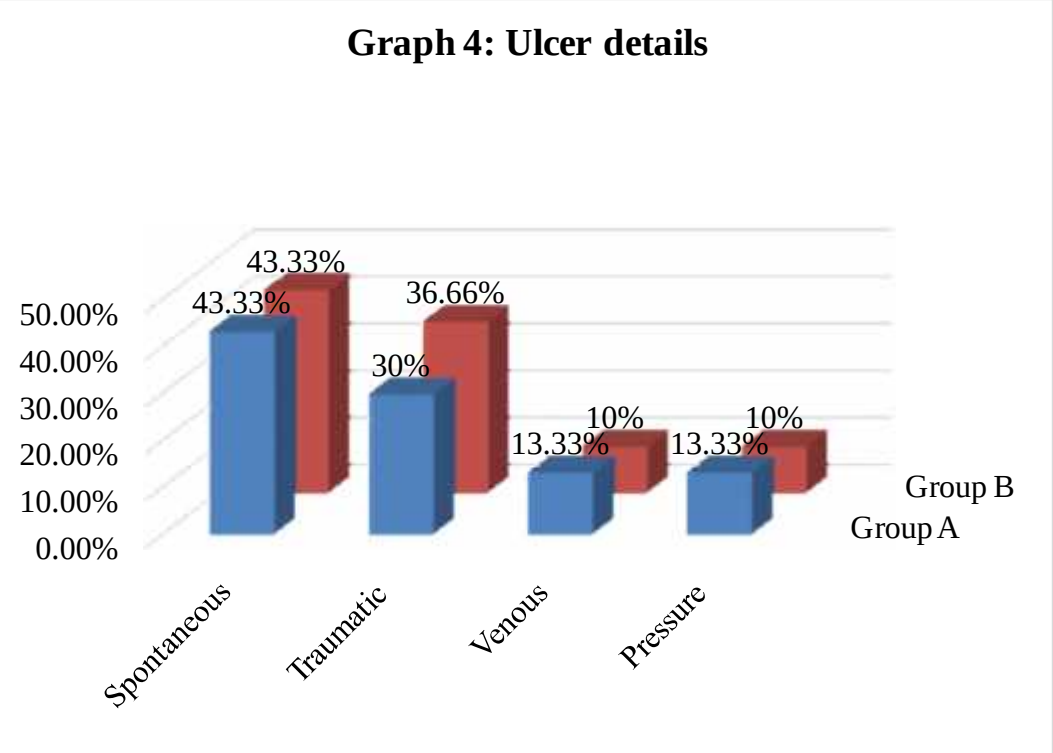
p value = 0.713



The mean age in group A was 55.73 ± 9.40 years and in group B it was 54.66 ±12.73 years. The mean age was comparable in both the groups (p=0.71).

Table 4: Ulcer details – Onset

	Group A		Group B	
	No	**Percentage**	**No**	**Percentage**
Spontaneous	13	43.33	13	43.33
Traumatic	9	30	11	36.66
Venous	4	13.33	3	10
Pressure	4	13.33	3	10
Total	30	100	30	100



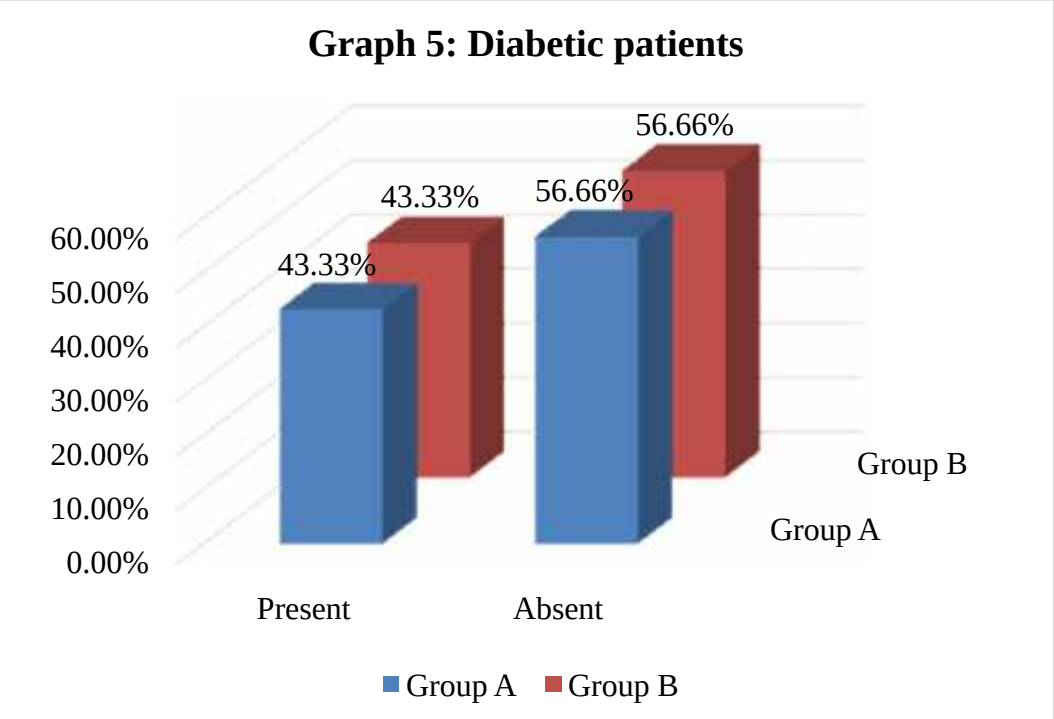
In the present study, 43.33% patients had spontaneous onset of ulcer in both group. However the onset of ulcer was comparable in both the groups.

Co morbid conditions

Table 5: Diabetes mellitus

	Group A		Group B	
	No	**Percentage**	**No**	**Percentage**
Present	13	43.33%	13	43.33%
Absent	17	56.66%	17	56.66%
total	30	100%	30	100%

p value = 1

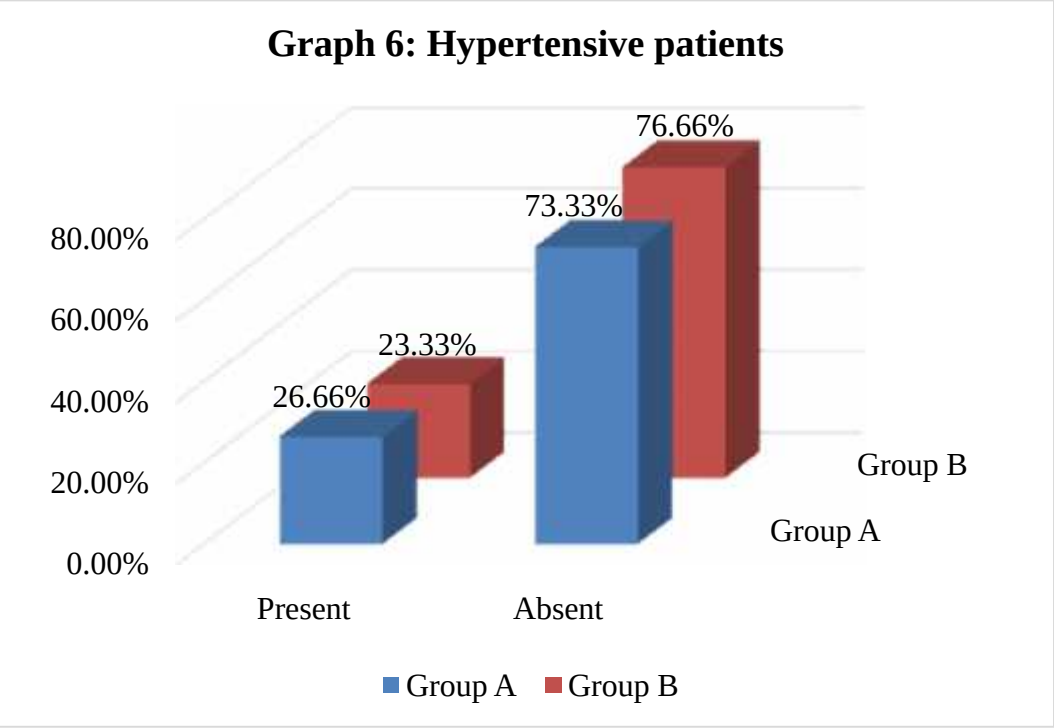


In both the groups 43.33% were diabetics and were comparable. (p=1)

Table 6: Hypertension

	Group A		Group B	
	No	**Percentage**	**No**	**Percentage**
Present	8	26.66%	7	23.33%
Absent	22	73.33%	23	76.66%
total	30	100%	30	100%

p value = 0.765



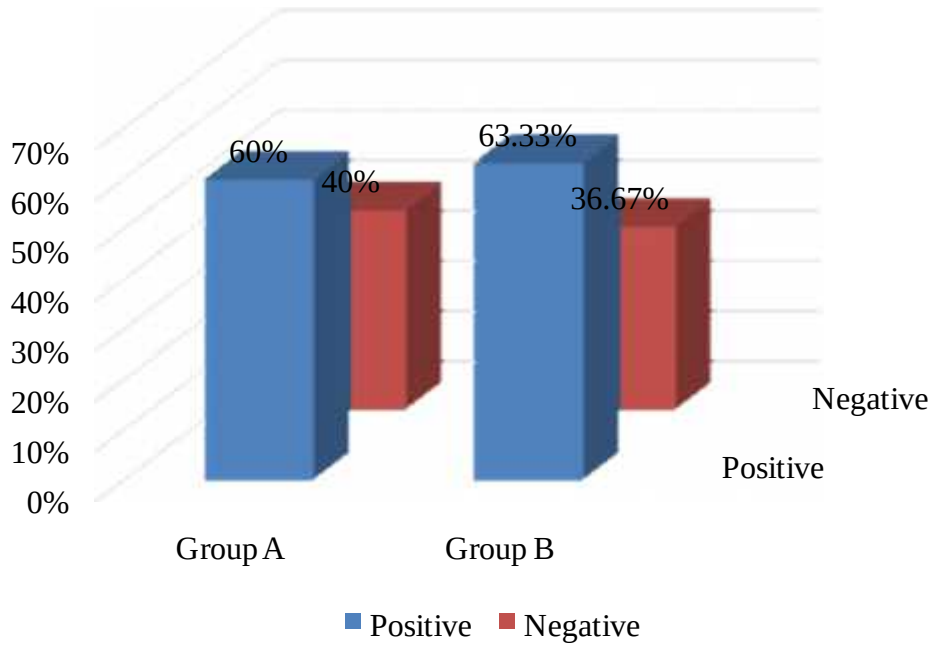
In group A 26.66% and in group B 23.33% were Hypertensives and were comparable in both the groups. (p =0.765)

Table 7: Initial wound culture

Culture	Group A		Group B	
	Number	**Percent**	**Number**	**Percent**
Positive	18	60	19	63.33
Negative	12	40	11	36.67
Total	30	100.00	30	100.00

p value = 0.791

Graph 7: Initial wound culture

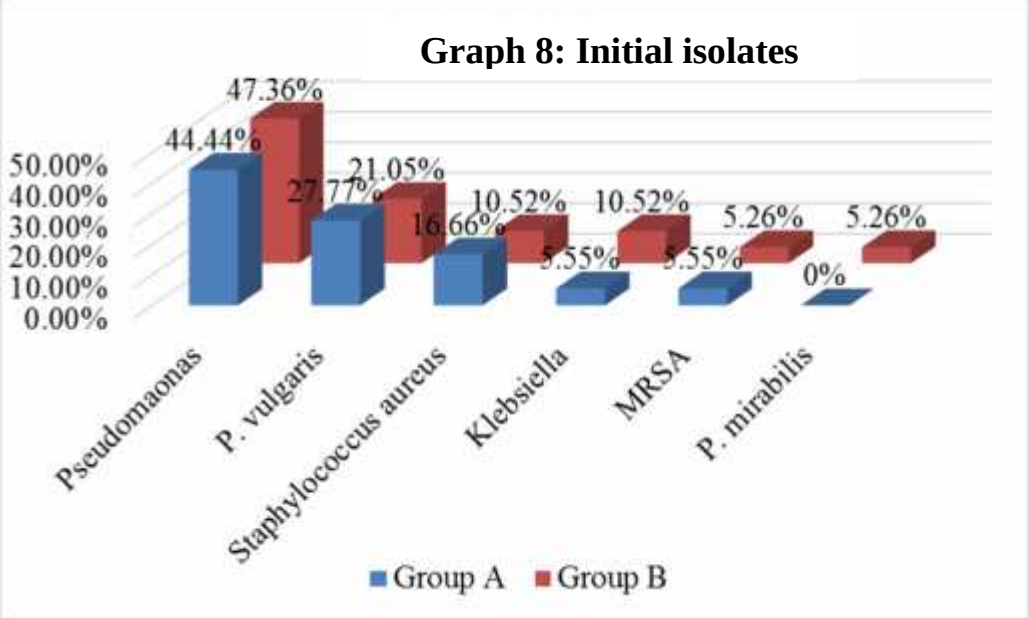


The wound culture was positive in 60% of patients in group A and 63.33% in group B. However no statistically significant difference was observed between the two groups. (P value-0.791)

Table 8: Initial isolates

Isolates	Group A		Group B	
	Number	Percent	Number	Percent
Pseudomonas	8	44.44	9	47.36
P. vulgaris	5	27.77	4	21.05
Staph. Aureus	3	16.66	2	10.52
Klebsiella	1	5.55	2	10.52
MRSA	1	5.55	1	5.26
P. mirabilis	0	0	1	5.26
Total	18	100.00	19	100.00

P value-0.791

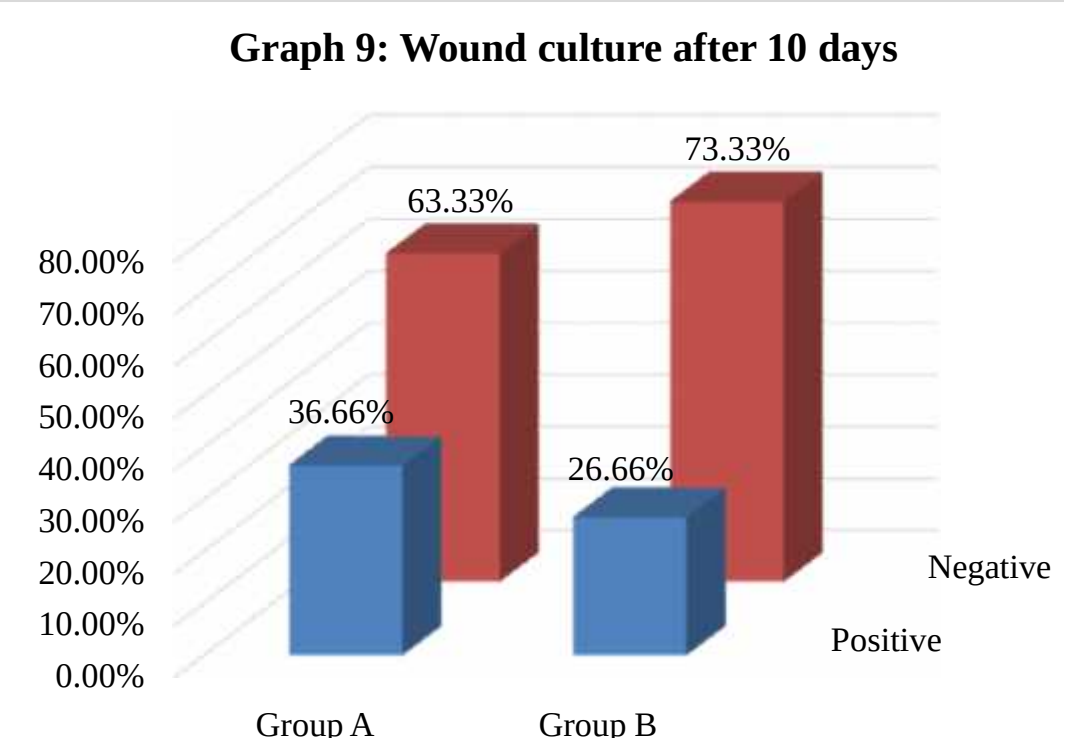


In this study the most common organism was Pseudomonas in both the groups (44.44% in group A and 47.36% in group B).

Table 9: Wound culture after 10 days

Culture	Group A		Group B	
	Number	Percent	Number	Percent
Positive	11	36.66	8	26.66
Negative	19	63.33	22	73.33
Total	30	100.00	30	100.00

p value – 0.405



In the present study, the wound culture on day 10 was negative in 63.33% patients in group A compared 73.33% in group B. However no statistically significant difference was observed between the two groups (p=0.405).

Table 10: Isolates

Isolates	Group A		Group B	
	Number	Percent	Number	Percent
Pseudomonas	2	18.18	2	25
P. vulgaris	5	45.45	2	25
Staph. Aureus	2	18.18	1	12.5
Klebsiella	1	9.09	2	25
MRSA	1	9.09	1	12.5
P. mirabilis	0	0	0	0
Total	11	100.00	8	100.00

Graph 10:Isolates on day 10

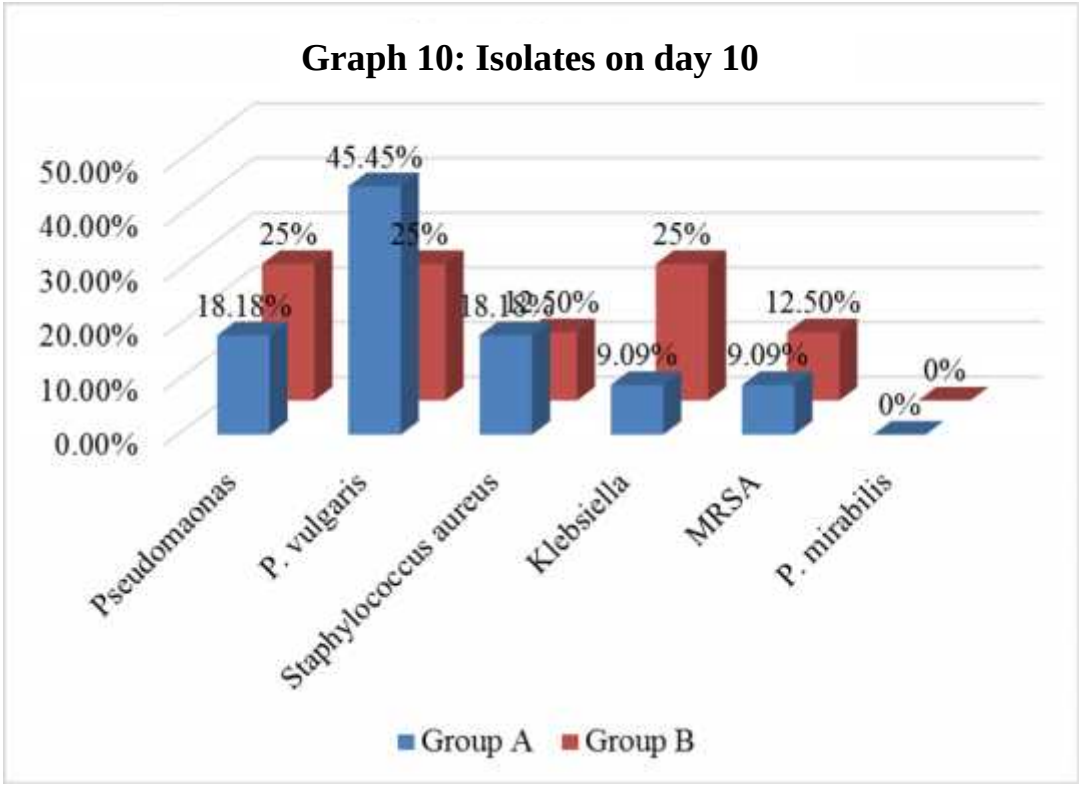
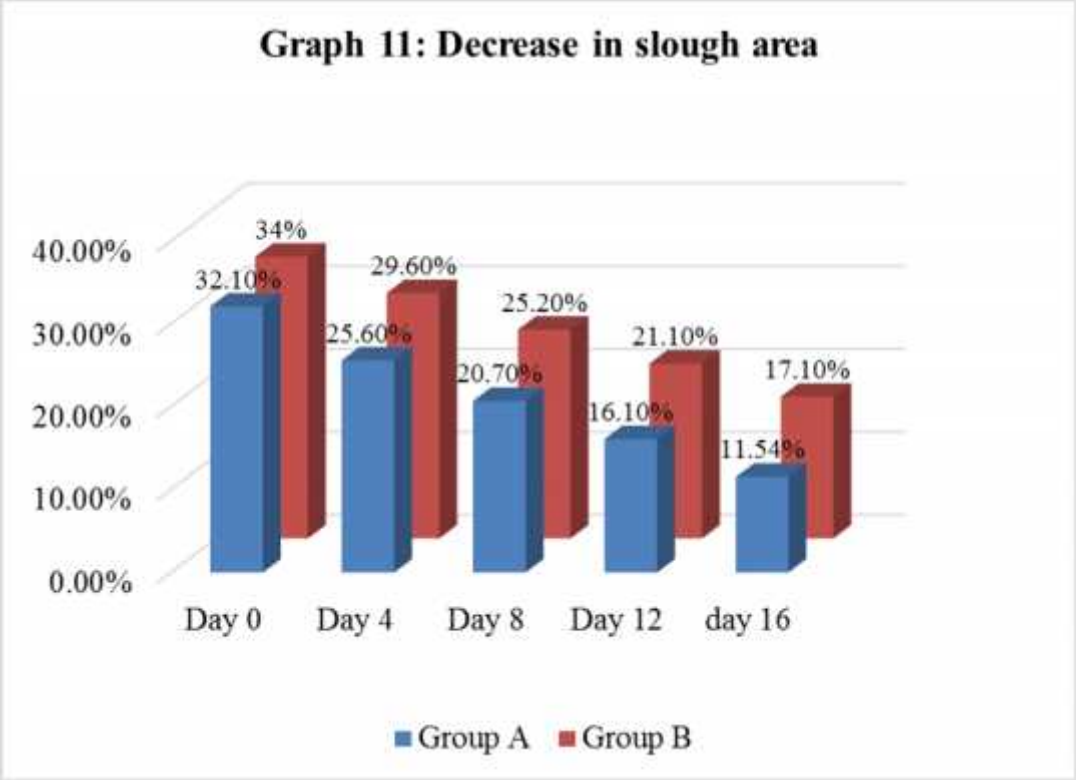


Table 11: Assessment of slough debridement

% of slough on	Group A	Group B	t	P value
Day 0 / Initial	32.1± 13.13	34± 13.76	0.55	0.580
Day 4	25.6± 12.13	29.6± 13.22	1.20	0.232
Day 8	20.7± 11.86	25.2± 12.67	1.432	0.150
Day 12	16.1± 10.68	21.1± 11.94	1.69	0.096
Day 16 / Final	11.54± 9.9	17.1± 11.48	2.00	**0.049**
Percentage change in slough on day 16	68.86± 14.00	54.3 ± 13.02	4.150242	**<0.001**



Reduction of slough

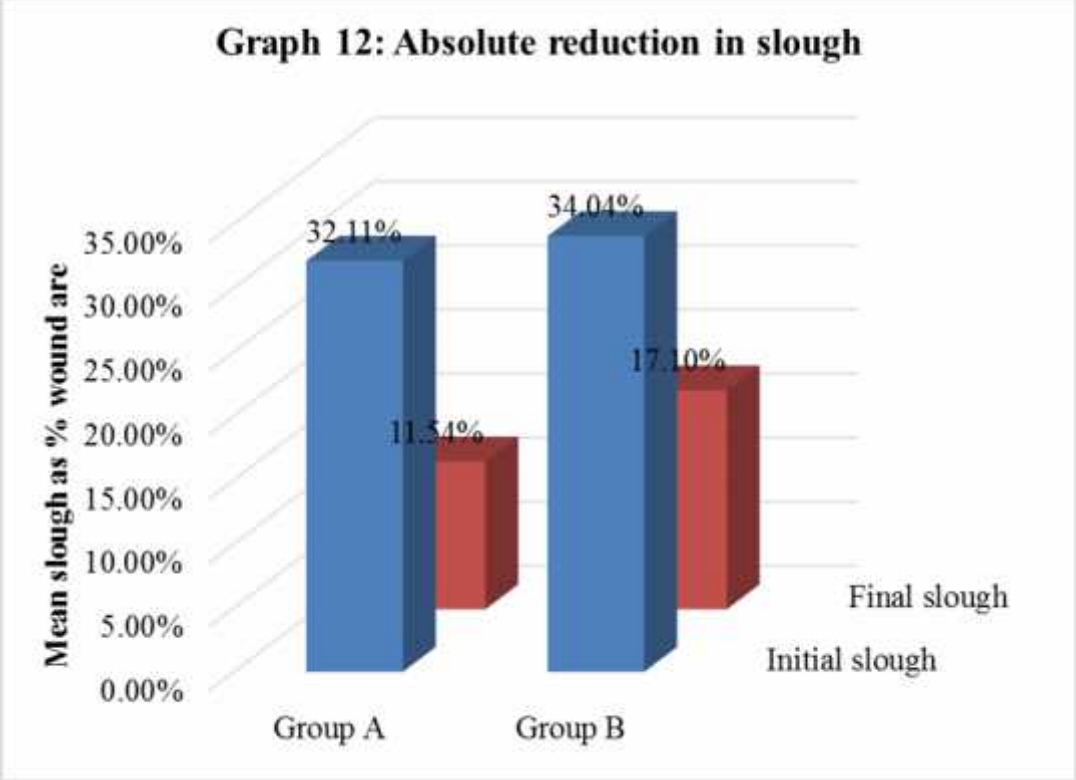
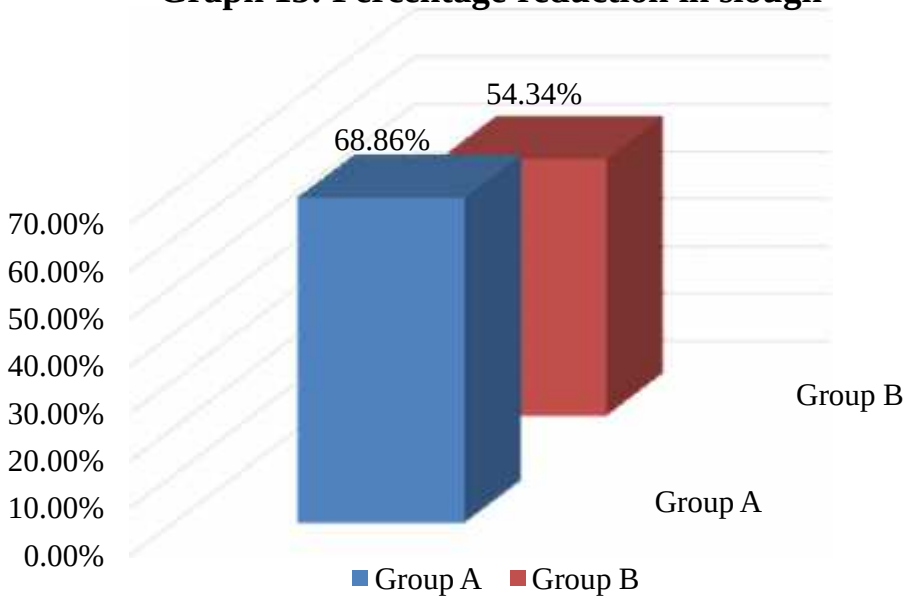


Table 12: Percentage reduction in slough

Assessment	Group A (n=30)		Group B (n=30)		p' value
	Mean	SD	Mean	SD	
Final percentage decrease in slough (%)	68.86	14.00	54.3	13.02	<0.001

Graph 13: Percentage reduction in slough



In this study among patients with group A the proportion of the wound area covered with slough fell from **32.1 ± 13.13%** to **11.54 ± 9.9%,** with higher mean percentage reduction in slough (68.86± 14.00%) whereas in group B the proportion of the wound area covered with slough fell from **34** ± **13.76 %** to **17.1** ± **11.48%,** with less mean percentage reduction in slough (54.3 ± 13.02%). The difference between the percentage reduction in slough was statistically significant ($p<0.001$).

DISCUSSION

Artificial dressings can be classified as hydrocolloids, calcium alginates, hydrofibres and hydrogels. Each of them differ in their composition, hydrating ability, absorbent capacity, vapor permeability, flexibility, conformability, adhesive properties. Indication and handling as well as in their distinctive benefits and limitations.[116]

Amorphous hydrogels are three-dimensional in there structure. They are cross-linked structures containing carboxy-methylcellulose, calcium alginates and a high percentage of water, some of which may be transferred from the gel to the wound surface to facilitate hydration. [116]

The principles of wound care are cleansing, debridement and protection. Presence of moist healing environment during the course of healing is very important. Wound debridement is essential for the successful wound healing. Proper and effective debridement in the early stages of healing shortens the entire wound healing period.

The present study was undertaken to compare the desloughing effect of hydrogel and normal saline dressing in leg ulcers.

The present one year hospital based randomized controlled trial was conducted in the Department of General Surgery, KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belgaum from January 2012 to December 2012. A total of 60 patients with leg ulcers were studied. Based on the envelop method, patients were divided into two groups of 30 patients each that is group A (Patients in this group

underwent dressing with Hydrogel) and group B (Patients in this group underwent dressing with Normal saline).

In this study males (76.66 % in group A and 86.66% in group B) outnumbered females in both the groups. The mean age in group A was 55.73 ± 9.40 years and in group B it was 54.66 ± 12.73 years (p=0.713) suggesting the demographic characteristics of the study population were comparable in both the groups.

In the present study, 43.33% patients had spontaneous onset of ulcer in both group. However the onset of ulcer was comparable in both the groups. The wound culture was positive in 60% of patients in group A and 63.33% in group B. The most common organism was Pseudomonas in both the groups (44.44% in group A and 47.36% in group B) suggesting the equal distribution of patients with regard to ulcer characteristics. The wound culture on day 10 was negative in 63.33% patients in group A compared 73.33% in group B, however no statistically significant difference was observed between the two groups (p=0.405).

In the present study the mean proportion of the wound area covered with slough in group A was 32.1 ± 13.13% and in group B was 34 ± 13.76%. The mean slough area at the beginning in both the groups was comparable. (p=0.580)

In the course of the study with the hydrogel the proportion of the wound area covered with slough fell from 32.1 ± 13.13% to 11.54 ± 9.9 %, with higher mean percentage reduction in slough (68.86± 14.00%) whereas in saline group the proportion of the wound area covered with slough fell from 34 ± 13.76 % to 17.1 ± 11.48 %, with less mean percentage reduction in slough (54.3 ± 13.02%). The difference between the mean percentage reduction in slough area was statistically

significant ($p<0.001$) showing significantly favorable outcome in patients who underwent hydrogel dressing.

In a prospective, multicenter, ambulant application study by Holger Kapp, 81 patients (average age 67 years) were studied, Venous and arterial ulcers were the most common wound types with 40%. They were treated with the Hydrogel for an average of twelve days and the dressing was changed every 4 days. In their course of the study with the Hydrogel, the proportion of the wound area covered with slough fell from 62.6 to 23.1%. At the same time, the area covered with granulation and epithelial tissue markedly increased**.** The patients also reported markedly less pain. Whereas 29.6% of the patients reported no pain at the beginning of the study, this proportion increased to 56.3% at the final assessment. [117]

In a prospective study 27 spinal-cord injury patients with a total of 49 pressure ulcers. Wounds were randomized into a treatment group (n=25) treated with hydrogel and control group (n=24) with normal saline. 84% of the wounds in the treatment group and 54% in the control group epithelialized, which was statistically significant ($p=0.04$). Hydrogel dressings facilitate healing by promoting more rapid autolytic debridement and epithelialization of pressure ulcers, when compared with conservative wound care.[118]

A systematic review by Smith[119] reported that, in patients with diabetic foot ulcers, debridement using hydrogel seems to be more effective than standard wound care for wound healing and is associated with fewer complications. The review suggests that hydrogels increased the proportion of foot ulcers completely healed within 3–5 months compared with standard care or gauze dressings. The extent to which this increased healing is a consequence of debridement is not clear. Hydrogels

provide a moist healing environment, and the effect may be caused by debridement, moisture, or an interaction between the two. It is worth noting that a statistically significant benefit was found with the use of hydrogel preparations, suggesting that routine use may be reasonable.[119]

Overall, in this study, hydrogel dressing provided favorable outcome in patients with leg ulcers by significant reduction in slough area when compared to normal saline dressing; however there was no role of Hydrogel in reducing the infection when compared to normal saline dressing.

Limitations of the study:

The properties such as granulation tissue formation has not been taken as a parameter because larger ulcers were taken for study which required split skin grafting to avoid prolonged morbidity of the patient. Reduced pain after the use of hydrogel that is the cooling effect has not been addressed in the study.

Another important limitation of the present study is its sample size. Although a sample size of 60 patients is sufficient for statistical analysis, a randomized controlled comparative study with a much larger population may help to further substantiate the findings or reveal variations which were not observed in the present study.

CONCLUSION

Based on the results of the present study it may be concluded that, hydrogel dressing provides favorable outcome in patients with leg ulcers by significant reduction in slough area when compared to normal saline dressing.

SUMMARY

Numerous topical medication and gels have emerged for ulcer dressing, to achieve debridement and promote healing. Relatively few have proved to be more efficacious in debridement and wound bed preparation than saline wet to dry dressings. The present study was aimed to compare the effect of hydrogel dressing and normal saline dressing in debridement of leg ulcers.

The present one year hospital based randomized controlled trial was conducted in the Department of General Surgery, KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belgaum from January 2012 to December 2012. A total of 60 patients with leg ulcers were studied. Based on the envelop method, patients were divided into two groups of 30 patients each that is group A (Patients in this group underwent dressing with Hydrogel) and group B (Patients in this group underwent dressing with Normal saline).

In this study males (76.66% in group A and 86.66% in group B) outnumbered females in both the groups (p=0.317). The mean age in group A was 55.73 ± 9.40 years and in group B it was 54.66 ± 12.73 years (p=0.713).

In the present study, 43.33% patients had spontaneous onset of ulcer in both group A and in group B. The wound culture was positive in 60 % of patients in group A and 63.33% in group B. The most common organism was Pseudomonas in both the groups (44.44 % in group A and 47.36 % in group B). The wound culture on day 10 was negative in 63.33% patients in group A compared 73.33% in group B, however no statistically significant difference was observed between the two groups (p=0.405).

In the course of the study with the hydrogel the proportion of the wound area covered with slough fell from 32.1 ± 13.13% to 11.54 ± 9.9 %, with higher mean percentage reduction in slough (68.86 ± 14.00 %) whereas in saline group the proportion of the wound area covered with slough fell from 34 ± 13.76 % to 17.1± 11.48 %, with less mean percentage reduction in slough (54.3 ± 13.02%). The difference between the percentage reduction in slough was statistically significant ($p<0.001$).

Overall, Hydrogel dressing provided favorable outcome in patients with leg ulcers by significant reduction in slough area, and had no role in reducing the infection when compared to normal saline dressing.

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ANNEXURE-II

PROFORMA

PATIENT IDENFICIATION DATA

GROUP : CASE NO. :

I.P/ O.P.D NO.: D.O.A:

NAME : D.O.S:

SEX : D.O.D:

OCCUPATION:

MEDICAL HISTORY

	Yes	**No**
Peripheral neuropathy		
Nephropathy		
PVD		
CVD		

DIABETIC STATUS

Type :

Medication :

Drug	Dose	Duration

Complication:

	Yes	No
Neuropathy		
Vasculopathy		

ULCER DETAIL

1. Mode of onset

Traumatic	
Spontaneous	
Pressure	
Others	

2. Duration

WOUND OBSERVATION

1. Site
2. Size
3. Shape
4. Edge
5. Margin
6. Floor
7. Base
8. Discharge
9. Surrounding Skin
10. Slough /necrotic tissue

NEUROLOGICAL EXAMINATION

VASCULAR EXAMINATION

	Right	Left
Femoral a.		
Popliteal a.		
Ant. Tibial a.		
Post Tibial a.		
Dorsalis Pedis a.		

ANY FOOT DEFORMITY PRESENT

	Yes	No
Bunion		
Charcots foot		
Toe deformity		
Foot drop		

IF DEBRIDEMENT HAS BEEN DONE

Specify, Date :

Side/anesthesia :

INVESTIGATIONS

CBC

FBS Date Time

Blood Urea

Sr. Creatinine

Urine :

Routine

Microscopy

X-ray Foot

AP view

Lat. View

Tissue culture/ sensitivity

ANALYSIS PLAN

Patients are divided into Group A and Group B

Group A – Study group – Dressed with hydrogel

Group B – Control group – Dressed with normal saline

Group:

Name: Age:

Sex: Ethnic background:

Site of ulcer: ***Size of ulcer:***

Etiology:

Predominant tissue type in ulcer:

Slough	
granulation tissue	
necrotic eschar	
Epithelium	

Type of exudate :

Purulent	
Serosanguinous	
Bloody	
Serous	
None	

Wound inspection:

Week	Date	Measurement of slough area in %
Initial slough		
End of 4th day		
End of 8th day		
End of 12th day		
End of 16th day		
End of 20th day		

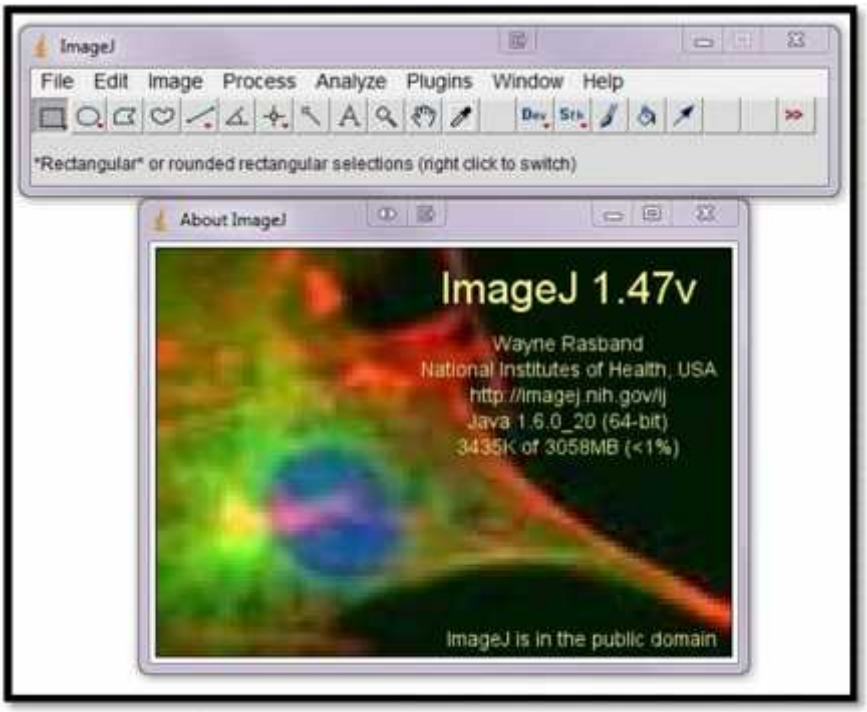
For 30 records of desloughing in each group mean and standard deviation will be calculated. To find out the significance between these two means student's unpaired 't' test will be used. Level of significance will be taken as 0.05.

ANNEXURE -III

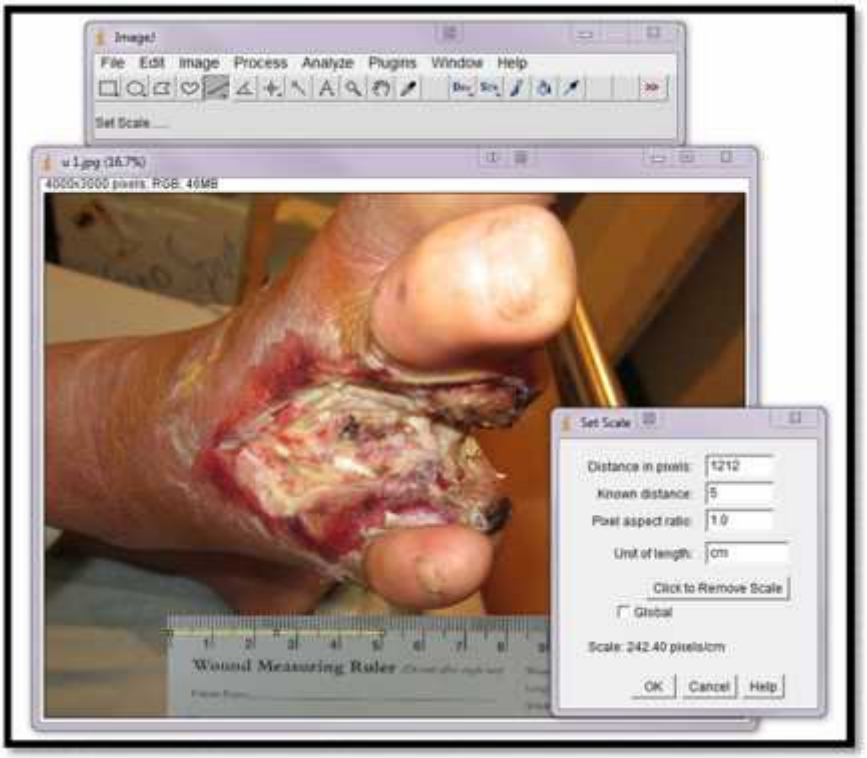
PHOTOGRAPHS

Photograph 1: Normal saline

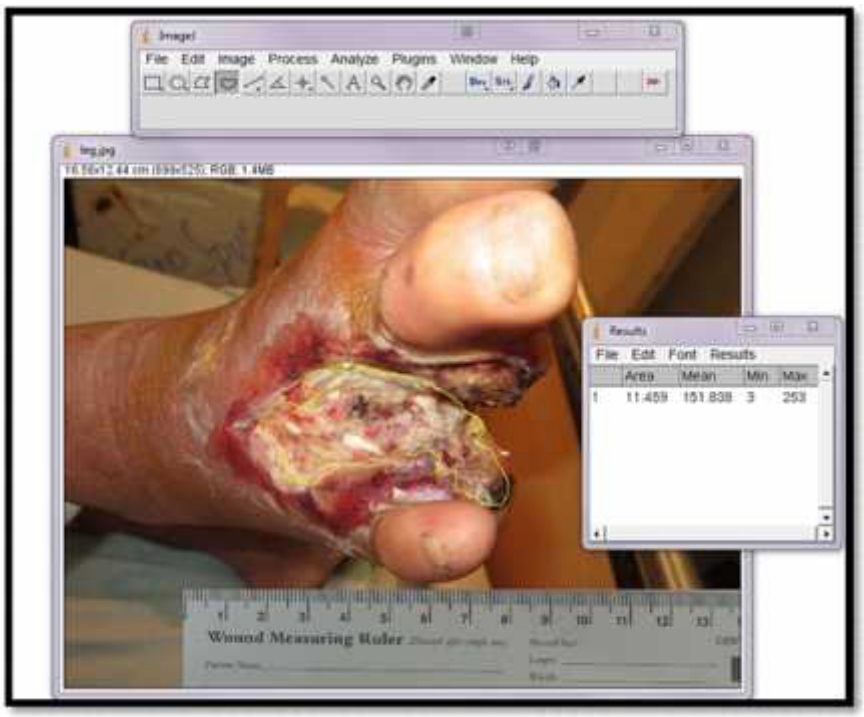
Photograph 2: Hydrogel



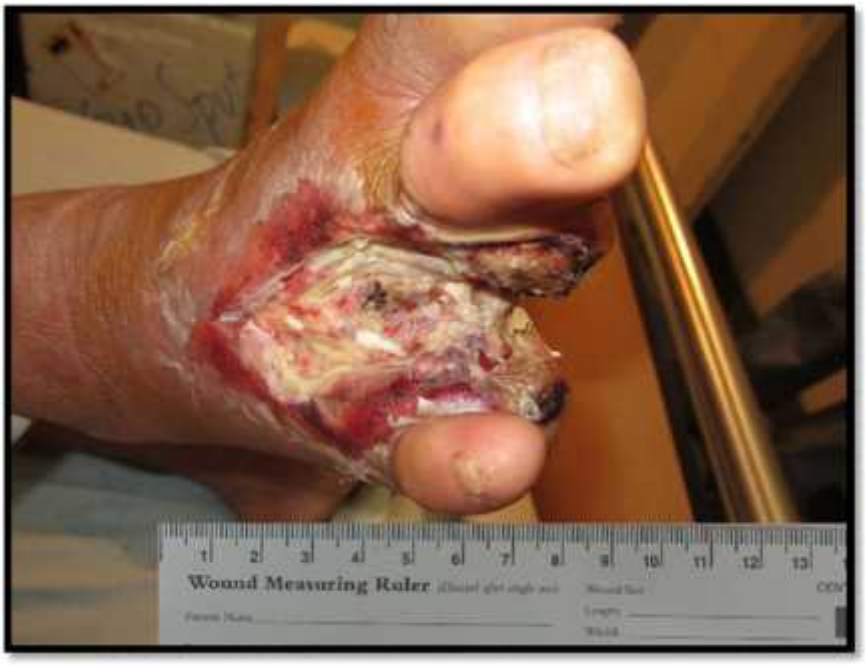
Photograph 3: ImajeJ software



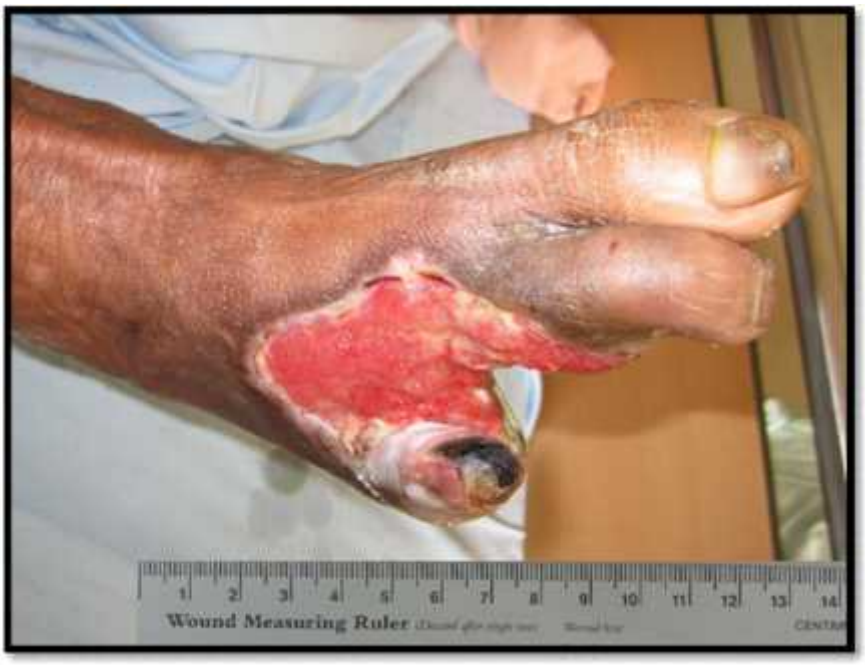
Photograph 4: Photo calibration



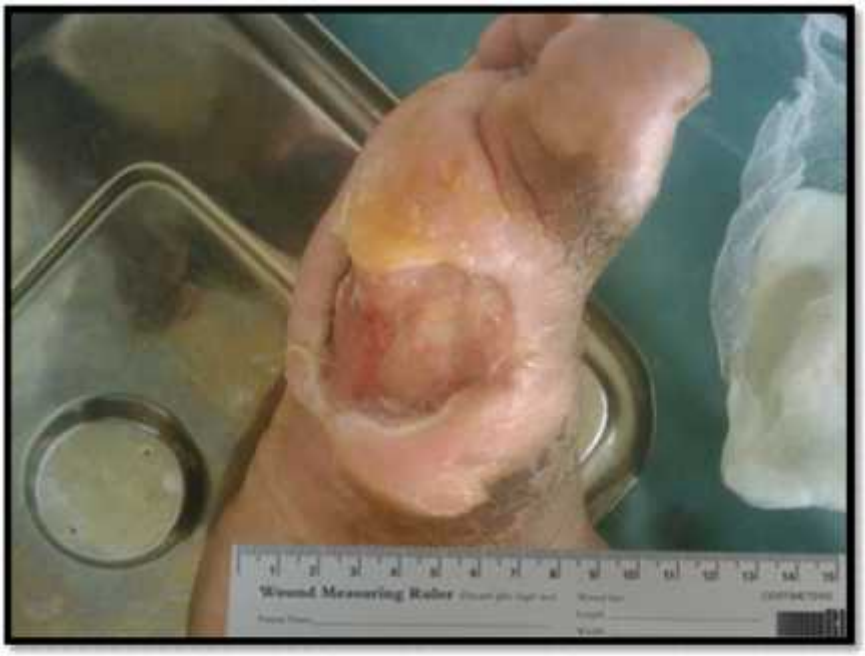
Photograph 5: Measuring slough area



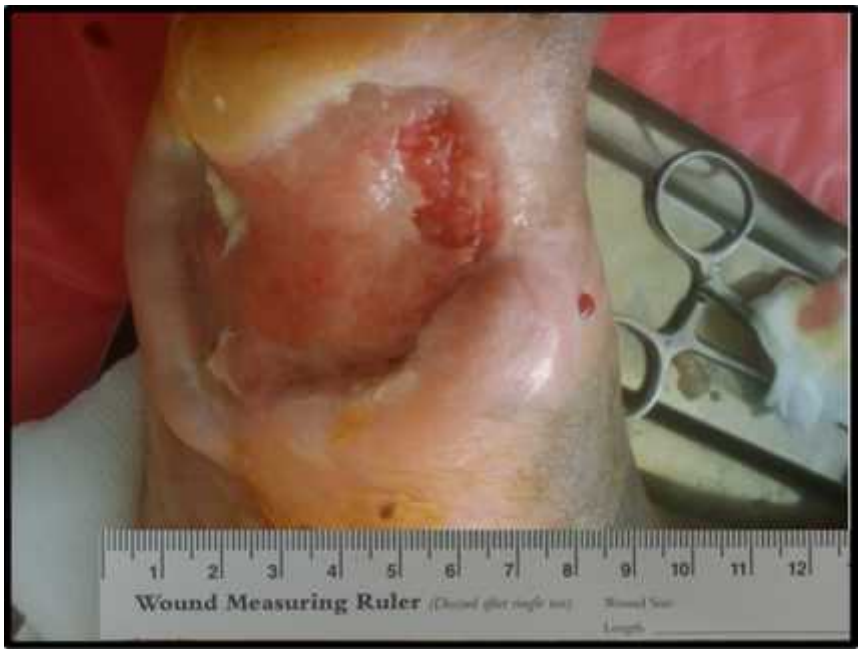
Photograph 6: Slough on day one in group A



Photograph 7: Slough on day 16 in group A



Photograph 8: Slough on day one in group B



Photograph 9: Slough on day 16 in group B

GROUP A

S. No	I.P No	AGE	sex	onset	Co- morbid conditions		Pus Culture Before		Pus Culture After		Percentage of slough					Absolute Change in slough	Percentage reduction
					DM	HTN	Sensitivity	Organism	Sensitivity	Organism	day 0	day 4	day 8	day 12	day 16		(day 0-day16/day 0)x100
1	455692	50	m	TR	-	-	-	-	-	-	62	54.4	50	44.6	39.8	22.2	35.80645161
2	453631	55	m	TR	-	-	-	-	-	-	52	42	37	32	25.6	26.4	50.76923077
3	456909	63	m	PR	Y	-	+	STPH	-	-	52	44.4	39.8	34.2	29.6	22.4	43.07692308
4	456118	60	m	TR	-	Y	-	-	-	-	25	19.2	15	10.2	7	18	72
5	464830	65	m	SP	Y	Y	+	PS	-	-	32	24	19.6	15.6	11.4	20.6	64.375
6	464620	61	m	TR	-	-	-	-	-	-	62	50.4	48	40	35.8	26.2	42.25806452
7	466116	61	m	SP	-	Y	+	PTV	+	PTV	22	18.2	12.8	8.8	4.6	17.4	79.09090909
8	466979	58	m	PR	Y	-	+	PTV	+	PTV	42	36.2	31	26.8	20.6	21.4	50.95238095
9	456753	60	m	SP	-	-	-	-	-	-	32.2	24.4	19.8	15.8	11.6	20.6	63.97515528
10	467005	58	m	SP	-	-	-	-	-	-	37	29.2	24.6	20.6	16.4	20.6	55.67567568
11	463843	55	f	SP	-	-	+	PS	+	PS	36.2	28.4	22.8	18.8	14.6	21.6	59.66850829
12	461940	60	m	SP	-	-	-	-	-	-	16	10.8	7	5	1.6	14.4	90
13	457204	58	m	VN	-	-	+	PS	-	-	38	32.2	27	22.8	16.6	21.4	56.31578947
14	464746	30	f	TR	-	Y	+	PS	-	-	26	20.8	15.8	11.8	7.2	18.8	72.30769231
15	468203	37	f	PR	Y	-	-	-	-	-	26	20	15	11	6.6	19.4	74.61538462
16	468020	56	m	SP	Y	-	+	PS	-	-	22	14	9	7	2.6	19.4	88.18181818
17	472737	55	m	PR	Y	Y	+	MRSA	+	MRSA	26.6	22.4	17.4	13.4	8.8	17.8	66.91729323

18	472455	28	m	SP	-	-	-	-	-	-	32	27.2	19	14	7.8	24.2	75.625
19	474112	52	m	VN	-	-	+	KL	+	KL	26	18	13	9	5.6	20.4	78.46153846
20	479738	62	m	TR	Y	-	+	PTV	+	PTV	30	22	17	12	6.6	23.4	78
21	497463	58	m	SP	Y	-	-	-	-	-	52	46	37	26	16.6	35.4	68.07692308
22	506011	58	m	SP	Y	Y	+	STPH	+	STPH	16.4	12.2	8.2	4.4	3	13.4	81.70731707
23	506205	52	m	VN	Y	-	+	PTV	+	PTV	27	18	12.2	8	3.6	23.4	86.66666667
24	472163	61	f	TR	-	-	+	PTV	+	PTV	13	7.8	5.2	4	2.6	10.4	80
25	460656	58	f	SP	Y	-	-	-	-	-	42	37	30	22	11.6	30.4	72.38095238
26	469379	60	m	SP	-	-	+	PS	+	PS	17	12.8	8.8	5	3.6	13.4	78.82352941
27	478340	68	f	TR	-	-	+	PS	-	-	22	16	12	8	4.6	17.4	79.09090909
28	470036	69	m	TR	Y	-	+	PS	-	-	26	22	16	12	6	20	76.92307692
29	474902	52	m	VN	-	Y	+	STPH	+	STPH	25	19.2	15	10.2	7	18	72
30	475766	52	f	SP	Y	Y	-	-	-	-	26	20.8	15.8	11.8	7.2	18.8	72.30769231

GROUP B

S. No	I.P No	AGE	sex	onset	Co- morbid conditions		Pus Culture Before		Pus Culture After		Percentage of slough					Absolute Change in slough	Percentage reduction
					DM	HTN	Sensitivity	Organism	Sensitivity	Organism	day 0	day 4	day 8	day 12	day 16		(day 0-day16/day 0)x100
1	464830	60	m	SP	Y	_	-	-	-	-	66	60	55.2	50	47	19	28.78787879
2	464918	62	m	PR	Y	_	+	PTV	-	-	52	48	42	37	31	21	40.38461538
3	467414	38	m	SP	_	_	+	PTV	+	PTV	56	50	45.2	40	37	19	33.92857143
4	467623	31	m	TR	_	_	+	KL	+	KL	26.6	22	18.2	14	11.4	15.2	57.14285714
5	465805	40	m	SP	_	_	+	PS	-	-	32	27.8	23.1	19.8	15.4	16.6	51.875
6	468596	39	m	SP	_	_	-	-	-	-	62	56	51.2	46	43	19	30.64516129
7	468209	48	m	TR	_	_	+	STPH	-	-	23	20	16.2	12.6	8.4	14.6	63.47826087
8	467601	45	m	TR	_	_	+	PS	-	-	46.2	42	36.8	32	27.4	18.8	40.69264069
9	467632	60	m	PR	Y	_	+	PS	-	-	32.2	28	23.8	20	15.6	16.6	51.55279503
10	468106	55	m	VN	_	Y	+	PM	-	-	37	32.8	28.6	24.8	20.4	16.6	44.86486486
11	464830	55	f	SP	_	_	-	-	-	-	36.2	32	27.8	24	19.6	16.6	45.85635359
12	408981	60	m	SP	_	Y	+	PS	-	-	16.2	12.2	9.2	6	3	13.2	81.48148148
13	470515	28	m	VN	_	_	-	-	-	-	42.2	38	32.8	28	23.4	18.8	44.54976303
14	468224	48	m	SP	_	Y	+	PTV	-	-	30	26	21.6	17.8	13.4	16.6	55.33333333
15	467623	62	m	TR	Y	_	-	-	-	-	26	22	18	15	12	14	53.84615385
16	469572	85	m	PR	Y	_	-	-	-	-	22	18	13	9	5	17	77.27272727
17	408981	65	m	SP	Y	Y	+	KL	+	KL	31.6	27.6	23.2	19.6	15	16.6	52.53164557
18	468093	58	m	TR	_	_	+	PS	-	-	33.8	28	23.6	19	14.6	19.2	56.80473373

19	466837	52	m	VN	_	_	+	PS	+	PS	26	22	18	14	9	17	65.38461538
20	478097	48	m	SP	Y	_	-	-	-	-	30	26	22	18	13	17	56.66666667
21	442181	75	m	SP	Y	_	+	STPH	+	STPH	56	52	46	40	33	23	41.07142857
22	475044	53	m	TR	Y	Y	-	-	-	-	18.4	14.4	10.6	7.6	5.4	13	70.65217391
23	475473	70	m	SP	Y	_	+	PS	-	-	28.8	23.4	18.4	13.2	8.8	20	69.44444444
24	456242	58	f	TR	_	_	+	PS	-	-	13	9.8	7.6	5.4	4	9	69.23076923
25	471519	56	f	TR	Y	_	+	PS	+	PS	49	42	37	31	24	25	51.02040816
26	478264	60	m	TR	_	_	-	-	-	-	19	15	11.2	8.2	6	13	68.42105263
27	481860	47	m	TR	_	_	-	-	-	-	23.6	19.6	15.2	12	9.6	14	59.3220339
28	481245	69	m	SP	Y	_	+	PTV	+	PTV	30	26	21.6	17.4	13	17	56.66666667
29	478194	68	m	TR	_	Y	-	-	-	-	26.6	22	18.2	14	11.4	15.2	57.14285714
30	441010	45	f	SP	Y	Y	+	MRSA	+	MRSA	30	26	21.6	17.8	13.4	16.6	55.33333333

ANNEXURE IV – MASTER CHART

-	- Negative
+	- Positive
F	- Female
KL	- Klebsiella
M	- Male
MRSA	- Methicillin-resistant Staphylococcus aureus
PM	- Proteus Mirabilis
PR	- Pressure
PS	- Pseudomonas
PTV	- Proteus vulgaris
SP	- Spontaneous
STPH	- Staphylococcus aureus
TR	- Traumatic
VN	- venous
Y	- Yes

ANNEXURE-I

INFORMED CONSENT

***Title of Research Study*: A RANDOMISED CONTROL TRIAL TO COMPARE THE DESLOUGHING EFFECT OF HYDROGEL VERSUS NORMAL SALINE DRESSING IN LEG ULCERS**

You are requested to participate in a study that is an attempt to find out the effectiveness of hydro gel dressing with wet to dry dressing normal saline dressing in desloughing of leg ulcers.

The clinical use of amorphous hydrogel dressings as an alternative to wet-to-dry normal saline gauze dressings is limited by lack of scientific evidence of debriding necrotic ulcers. The purpose of this experimental research, therefore, is to compare the effect of desloughing with wet-to-dry normal saline gauze dressings to the effect of desloughing with amorphous hydrogel dressings.

You need to be eligible, meeting all the selection criteria to participate in this study. You should be willing to provide information about yourself. 60 subjects will be enrolled in this study who will then be randomised in either of 2 groups (details given below).

If you agree to participate in this study, you will be randomly allotted into a group (A or B) and accordingly receive either the standard treatment (saline dressing) or the newer treatment (hydrogel). Periodically, your wound will be examined and assessed and discharge will be sent for culture and sensitivity. The further treatment will then be initiated depending on the sensitivity report.

The new dressing (hydrogel) might be allergic in few participants. There is no additional risk compared to the standard method of dressing

Taking part in the study will not affect the cost of treatment i.e. it will be similar to the cost of standard procedure. In the event that you become injured as a result of taking part in this study, treatment will be offered to you or you will be given information about where to receive medical care: but you/your insurance company will be responsible for the costs. However, no reimbursement, compensation or free medical care will be given.

Every effort will be made to protect the confidentiality of the information you provide. This means that the researchers will not let anyone, not a part of the study, see the information you provide.

Taking part in this study is voluntary; you may choose not to enroll in this study. Your decision will not change the present or future health care services offered to you at KLES Dr. Prabhakar Kore Hospital, Belgaum. The alternative that you have is to undergo standard treatment that is carried out in KLES Hospital.

You may also contact at Department of General Surgery, KLES Dr. Prabhakar Kore Hospital, Phone No. 0831-2473777.

CONSENT TO PARTICIPATE IN THE STUDY

I Mr./Ms./Mrs_____ have been explained about the research study, the need of the study, the intervention, their risks, benefits and alternatives available in my own vernacular language.

I voluntarily agree to participate in this study by signing up this form below. I understand that I may withdraw at any time from this study. I have been given adequate time to clarify my doubts about the study and my rights as a study participant. My signature / thumb impression below indicates that I have read or information in the consent been read and explained to me including the risks and benefits and have cleared my doubts. I will be given a copy of this consent form.

Name of participant: Signature/LTI:

Name of legally authorized Signature/LTI:

Representative (if applicable):

Relationship with participant:

Name of witness: Signature:

Name of investigator: Signature:

Date:

Place: