# "A RANDOMIZED CONTROL TRIAL TO ASSESS THE EFFICACY OF CALCIUM ALGINATE DRESSING VERSUS CONVENTIONAL GAUZE DRESSING ON BACTERIAL LOAD IN INFECTED DIABETIC FOOT ULCER"


**By**

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Dissertation submitted to the
KLE University, Belgaum, Karnataka

In Partial Fulfillment
of the requirements for the degree of

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

**Under the Guidance of**

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**MAY - 2010**

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I hereby declare that this dissertation entitled **"A RANDOMIZED CONTROL TRIAL TO ASSESS THE EFFICACY OF CALCIUM ALGINATE DRESSING VERSUS CONVENTIONAL GAUZE DRESSING ON BACTERIAL LOAD IN INFECTED DIABETIC FOOT ULCER"** is a bonafide and genuine research work carried out by me under the guidance of **Dr. S. C. METGUD** MS Professor, Department of Surgery, Jawaharlal Nehru Medical College, Belgaum-590010.

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Place: Belgaum **Dr. RAKESH KUMAR PANDEY**

# LIST OF ABBREVIATIONS USED

| | | |
|---|---|---|
| ADA | - | American Diabetes Association |
| AGE | - | Advanced Glycosylation End products |
| Approx. | - | Approximately |
| CBC | - | Complete blood count |
| CFU | - | Colony forming unit. |
| CVD | - | Cardiovascular disease |
| DM | - | Diabetes Mellitus |
| DOA | - | Date of admission |
| DOD | - | Date of discharge |
| E. Coli | - | Escherichiae coli |
| FBS | - | Fasting blood sugar |
| GAD | - | Glutamic Acid Decarboxylase |
| HbA1c | - | Glycosylated haemoglobin |
| HDL | - | High Density Lipoproteins |
| HIV | - | Human immunodeficiency virus |
| HLA | - | Human Leukocyte Antigen |
| IDDM | - | Insulin Dependent Diabetes Mellitus |
| IP No | - | Inpatient number |
| Kleb oxytoca | - | Klebsiella oxytoca |
| Kleb Pneumonia | - | Klebsiella pneumonia |
| LDL | - | Low Density Lipoproteins |
| MODY | - | Maturity Onset Diabetes of the Young |
| MRI | - | Magnetic Resonance Imaging |
| MSRA | - | Methicilin Resistant Staphylococcus Aureus |

| | | |
|---|---|---|
| NIDDM | - | Non- Insulin Dependent Diabetes Mellitus |
| P. Aeroginosa | - | Pseudomonas aeroginosa |
| PVD | - | Peripheral vascular disease |
| Sr. creatinine | - | Serum creatinine |
| Staph Aureus | - | Staphylococcus Aureus |
| T. cell | - | Thymus maturing cell |
| TB | - | Tuberculosis |
| UKB | - | Urine ketone body |
| UTI | - | Urinary Tract Infection |
| VRE | - | Vancomycin Resistant Enterococci |

# ABSTRACT

**Background and objectives**

Diabetic foot ulcers are common and estimated to affect 15% of all diabetic individual during their lifetime. One of the major causes of non-healing of ulcer in diabetes is infection. The objectives of the present study was to measure the effect of calcium alginate dressing on bacterial load in infected diabetic foot ulcer in comparison to conventional gauze dressing.

**Methodology**

The present one year randomized controlled trial was conducted in the Department of Surgery, KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belgaum on 60 patients with infected diabetic foot ulcer during the period of January 2008 to December 2008. The patients were divided into two different groups by number randomization (Group 1 Calcium alginate and Group 2 conventional gauze). Bacterial load was determined per gram of the tissue before first and after third dressing in both the groups.

**Results**

Among 60 cases majority of the patients were males. The duration of DM was 6 to 10 years in majority of the patients. The mean bacterial load (X $10^5$ CFU/gm tissue) before the first dressing in calcium alginate group was 513.3±122.4 while in conventional gauze group it was 516.7±117.7 and after the third dressing was 526.7±138.8 and 536.7±121.7 respectively. The mean bacterial load after the third dressing was higher in conventional gauze group as compared to calcium alginate. However this difference was statistically not significant

(p=0.768). There was increase in bacterial load after the three dressings over the diabetic foot ulcer in both the groups. However this increase was not statistically significant (p=0.787).

**Conclusions**

The present study has shown that dressing with calcium alginate is ineffective in reducing the bacterial load of the infected diabetic foot ulcers.

**Key word**

Bacterial load; Calcium alginate dressing; Conventional gauze dressing; Diabetic foot ulcer; Diabetes mellitus.

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# INTRODUCTION

Diabetes mellitus is a metabolic disease characterized by hyperglycemia resulting from defects in insulin secretion, insulin action or both. The chronic hyperglycemia of diabetes is associated with long term damage, dysfunction and failure of various organs, especially the eyes, kidneys, nerves, heart and blood vessels.

The vast majority of cases of the diabetes fall into two broad categories: those having little or no endogenous insulin secretory capacity (IDDM or type 1 DM) and those who retain endogenous insulin secretory capacity but have a combination of resistance to insulin action and an inadequate compensatory insulin secretory response (NIDDM, or Type 2 DM).[1,2] Long term complications of diabetes include retinopathy with potential loss of vision, nephropathy leading to renal failure, peripheral neuropathy with risk of foot ulcers, amputations and Charcot joints, and autonomic neuropathy causing gastro intestinal, genitourinary and cardiovascular symptoms and sexual dysfunction.

Diabetic foot ulcers are common and estimated to affect 15% of all diabetic individual during their lifetime. Patient suffering from diabetic ulcer often require hospitalization. One of the major causes of non-healing of ulcer in diabetes is infection. It is caused by a variety of micro-organism. Most common are *Staphylococcus aureus* and *Pseudomonas aeroginosa* which invade the wound and multiply, producing harmful toxic substances, causing destruction of tissue and disturbance in wound healing.

Treatment plan for diabetic foot includes surgical debridement of wound, improvement of circulation through surgery or therapy, special dressing and antibiotics. Numerous topical medication and gels are promoted for ulcer care and healing. Relatively few have proved to be more efficacious than saline wet to dry dressings. Topical antiseptic, such as povidine-iodine are usually considered to be toxic to healing wounds. Generally a warm moist environment that is protected from external contamination is most conducive to wound healing. This can be provided by commercially available special dressings like calcium alginate.[3,4]

Alginates have been used in various forms for fifty years and yet they remain a poorly understood and probably underused dressing. It consists of naturally occurring polysaccharides, derived from the cell walls of brown seaweed. They are manufactured as non woven, fibrous sheet or rope like packing. It can hold upto 20 times its weight in fluid. Calcium alginate accelerates wound healing by absorbing the exudates and keeping the wound surface in a moist environment. Bacteria on the wound surface moves into the dressing as wound exudates is absorbed. With high level of fluid absorption and bacteria retaining property calcium alginate provide a passive mechanism for reducing the bacterial load of the wounds.[5,6,7]

Experimental studies[5,6,7] over bacteria retaining ability of calcium alginate dressing are available. But there are no adequate studies to evaluate the effect of calcium alginate on bacterial load, in infected, diabetic foot ulcer. In view of the above the present study is undertaken to measure its effect in comparison to conventional gauze dressing.

# OBJECTIVES

The objectives of the present study was to measure the effect of calcium alginate dressing on bacterial load in infected diabetic foot ulcer in comparison to conventional gauze dressing.

# REVIEW OF LITERATURE

## ANATOMY OF THE FOOT[8,9]

The human foot is a marvel of mechanical construction. It acts as a pliable platform to support the body weight in the upright posture as a lever to propel the body forwards in walking, running or jumping. It has 26 bones, 29 joints, 42 intrinsic muscles, various ligaments, 4 mm thick skin, exquisite nerve supply and abundant vascularity with good collaterals. These component works together to provide the body with support, balance with mobility.

### Parts

Structurally the foot has three main parts;

1. *The fore foot:* It is composed of phalanges and metatarsals. They are connected together by metatarsal phalangeal joint at the balls of the foot. The fore foot bears the half of the body weight and balance pressure on the balls of the foot.
2. *The mid foot:* It is composed of five tarsals bones. It forms the foot's arch and serves as a shock absorber.
3. *The hind foot:* It links the mid foot to ankle. It is composed of two long bones of the lower leg, the tibia and the fibula which forms ankle joint with talus. This subtalar joint is formed between talus and calcaneum which is cushioned inferiorly by a fat layer.

**Arches**

The foot consists of three arches.

1. Medial longitudinal arch

    - It is the highest and the most important arch of the foot.
    - It is composed of calcaneum, talus, navicular, cuneiforms and first three metatarsal bones. The submit of the arch is formed by talus.
    - It acts as a shock absorber.

2. Lateral longitudinal arch

    - It is characteristically low arch.
    - It is composed of calcaneum, cuboid, fourth and fifth metatarsal bones. The submit of the arch is formed by calcaneum.
    - It transmits the body weight and thrust to the ground.

3. Transverse arch

    - It is a continuous structure formed by cuboid, three cuneiforms and the bases of the metatarsal bones.

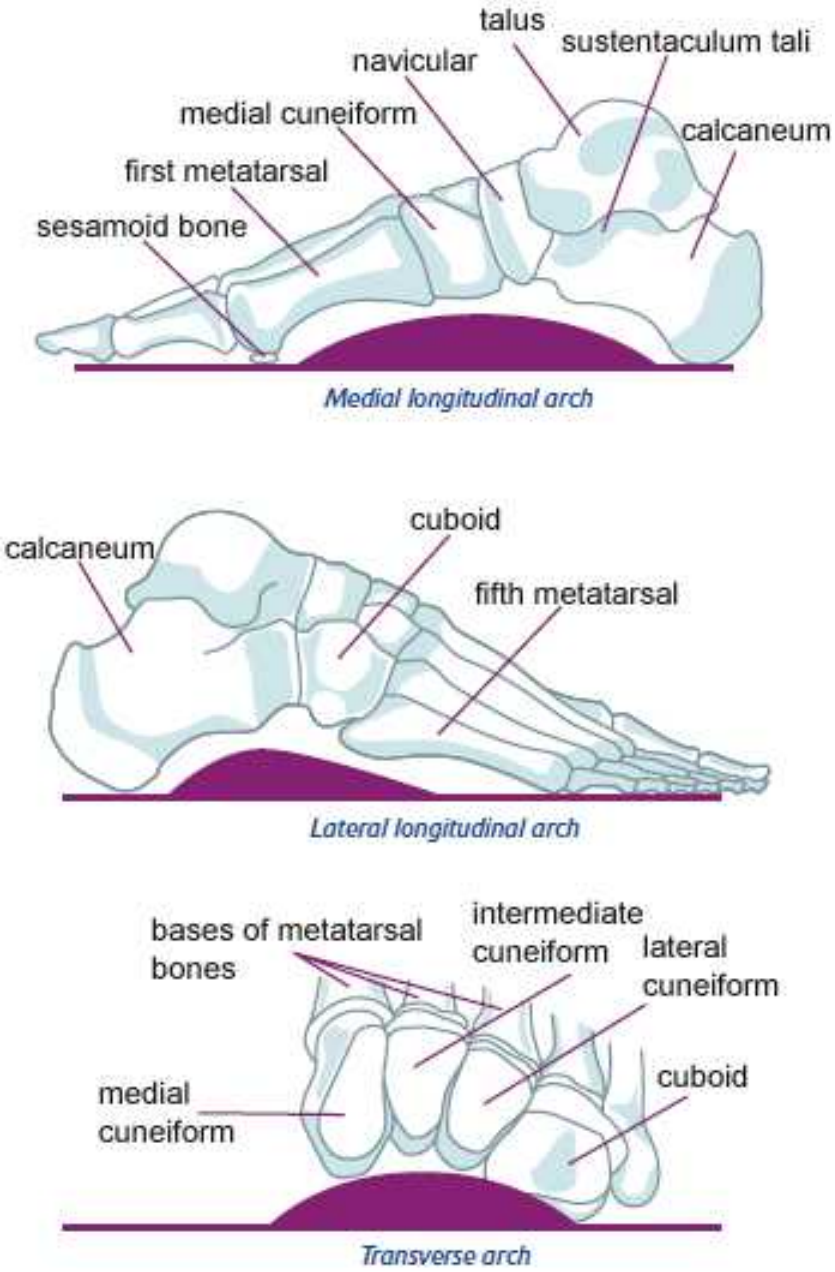



**Figure 1: Arches of the foot**

***Factors responsible for the maintenance of the arches***

1. Ligaments and plantar aponeurosis.
2. Action of extrinsic and intrinsic muscles of the foot.
3. Structure of the bones.

Functions of the arches of the foot

1. They distribute body weight to the weight bearing areas of the sole mainly heel and the base of the toes (first and fifth).
2. They act as a springs chiefly the medial longitudinal arch which helps in walking and running.
3. They also act as a shock absorbers in stepping and jumping.
4. The concavity of the arches protects the soft tissue of the sole against pressure.

**Sole**

The skin of the sole is about 4 mm thick. It is adapted for weight bearing. There are subcutaneous concentrations of the fat over the weight bearing areas such as heel, lateral margin of the sole and across the plantar aspect of the metatarsal heads. Numerous fibrous bands between the skin and the plantar aponeurosis prevent undue movement of sole during walking.

**Muscles**

***Intrinsic***

- Origin and insertion are located within the foot.
- They include plantar flexors, dorsiflexors, abductors and adductors of the toes.
- They also support the arches of the foot.

***Extrinsic***

- Origin of these muscles are in the lower leg.
- They have long tendon that crosses the ankle to insert on the bones of foot except the talus.
- They are responsible for the movement at the ankle, foot and toes.
- They also support the arches of the foot.

**Major joints and movements**

- Ankle joint – Dorsiflexion and plantar flexion.
- Subtalar joint – Inversion and aversion.
- Midtarsal joint – Abduction and adduction.

**Blood supply**

Anterior tibial artery continues as a dorsalis pedis artery in the foot. Dorsalis pedis artery gives off a arcuate artery that along with its branches supplies the outer four toes. The dorsalis pedis artery continues down to supply the great toe.

Posterior tibial artery in the sole of the foot divides into two branches, the lateral and medial plantar arteries that supplies the sole of the foot.

The peroneal artery descends down and supply posterior and the outer aspect of the heel.

**Nerve supply**

***Sensory nerve supply***

*Dorsum*

- The saphenous nerve: It supplies the medial border of the foot upto the ball of the great toe.
- The superficial peroneal nerve: It supplies entire dorsum of the foot except the lateral border, medial border and the cleft between the first and second toe.
- The sural nerve: It supplies the lateral border of the foot upto the tip of the little toe.
- The deep peroneal nerve: It supplies the cleft between the first and the second toes.
- The digital branch of the medial and lateral plantar nerve supplies the distal part of the dorsum of the toes.

*Sole*

- Medial calcanean branch of tibial nerve: It supplies posterior and medial portion of the sole.
- Medial plantar nerve: It supplies the anteromedial portion of the sole and medial three and half digits.
- Lateral plantar nerve: It supplies anterolateral portion of the sole and lateral one and half digits.

***Motor nerve supply***

- Deep peroneal nerve.
- Superficial peroneal nerve.
- Tibial nerve.
    - Medial plantar nerve.
    - Lateral plantar nerve.

## DIABETES MELLITUS

Diabetes mellitus comprises a group of common metabolic disorders that share the phenotype of hyperglycemia. Several distinct types of diabetes mellitus exist and are caused by a complex interaction of genetics, environmental factors, and life style choices. Depending on the etiology of diabetes mellitus, factors contributing to hyperglycemia may include reduced insulin secretion, decreased glucose utilization, and increased glucose production. The metabolic dysregulation associated with diabetes mellitus causes secondary pathophysiologic changes in multiple organ systems. Diabetes mellitus is the leading cause of end-stage renal disease, non-traumatic lower extremity amputation and adult blindness. With an increasing incidence worldwide, diabetes mellitus will be a leading cause of morbidity and mortality in the future.[1]

### Epidemiology

The worldwide prevalence of diabetes mellitus has risen dramatically over past two decades. Although the prevalence of both type 1 and type 2 diabetes mellitus is increasing, rise in type 2 diabetes mellitus is more rapid.[2] In 2000, the

prevalence of diabetes mellitus was estimated to be 8.6% in people > 20 years of age. There is a considerable geographic variation in the incidence of both type 1 and type 2 diabetes mellitus. Scandinivia has the highest incidence of Type 1 diabetes mellitus (35/1,00,000 per year). The prevalence of type 2 diabetes mellitus is highest in certain Pacific islands, intermediate in India and US and relatively low in Russia and China. According to WHO statistics (2000) 31705000 people in India are affected with diabetes and the number is expected to grow upto 79445000 by 2030.[10]

**Classification of diabetes and other categories of glucose regulation**

Diabetes mellitus is classified on the basis of the pathogenic process that leads to hyperglycemia, as opposed to earlier criteria such as age of onset or type of therapy.[1]

**Etiologic classification of diabetes mellitus**

I. Type 1 Diabetes (β-cell destruction, usually leading to absolute insulin deficiency)
II. Type 2 diabetes (may range from predominantly insulin resistance with relative insulin deficiency to a predominantly insulin secretory defect with insulin resistance)
III. Other specific types of diabetes
    A. Genetic defects of β-cell function characterized by mutations like MODY
    B. Genetic defects in insulin action.
    C. Diseases of the exocrine pancreas – pancreatitis, pancreatectomy.

D. Endocrinopathies – Acromegaly, Cushing's syndrome

E. Drug or chemical induced – Glucocorticoids, thyroid hormone, diazoxide, beta-adrenergic agonists, thiazides.

F. Infections – congenital rubella, cytomegalovirus, coxsackie.

IV. Gestational diabetes mellitus

**Type 2 diabetes mellitus**

This form of diabetes, which accounts for ~90-95% of those with diabetes, encompasses individuals who have insulin resistance and usually have relative (rather than absolute) insulin deficiency. The risk of developing this form of diabetes increases with age, obesity, and lack of physical activity. Occurs more frequently in women with prior Gestational diabetes mellitus, and individuals with hypertension or dyslipidemia. It is often associated with a strong genetic predisposition, more so than type 1 diabetes mellitus.

***Figure 2: Pathophysiology***

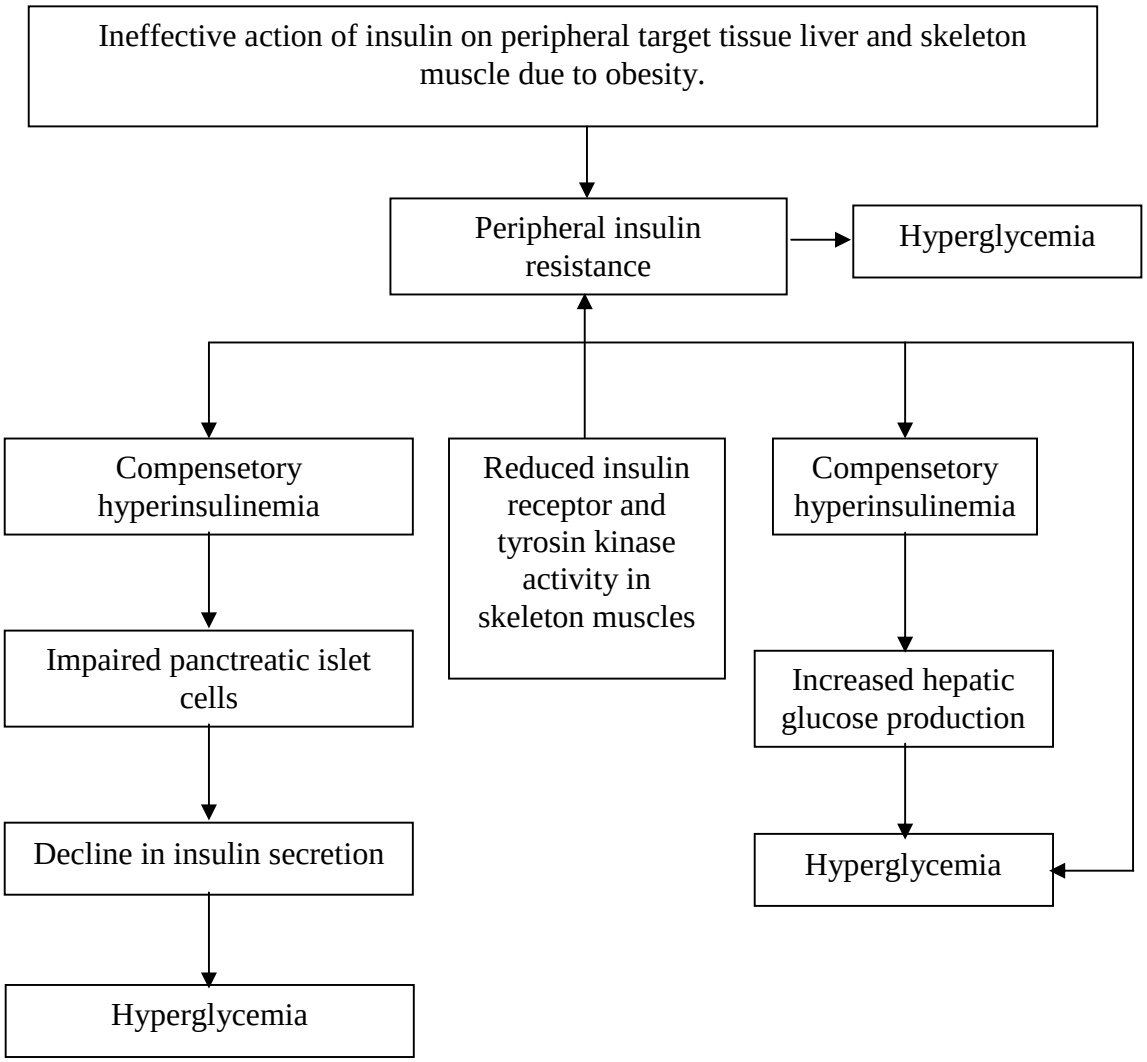


**Table 1: Diagnostic criteria for diabetes mellitus**

The criteria for diagnosis of diabetes are shown in table.[1,2]

| Criteria for the Diagnosis of Diabetes Mellitus |
|---|
| • Symptoms of diabetes plus random blood glucose concentration ≥11.1 mmol/L (200 mg/dL)[a] or<br>• Fasting plasma glucose ≥ 7.0 mol/L (126 mg/dL)[b] or<br>• Two-hour plasma glucose ≥ 11.1 mmol/L (200 mg/dL) during an oral glucose tolerance test[c] |

[a] Random is defined as without regard to time since the last meal.
[b] Fasting is defined as no caloric intake for atleast 8 h.
[c] The test should be performed using a glucose load containing the equivalent of 75 g anhydrous glucose dissolved in water.
(source:Adapted from ADA,2004)

Three ways to diagnose diabetes are possible and each in the absence of unequivocal hyperglycemia, must be confirmed, on a subsequent day by any one of the 3 methods, given in the table.

## DIABETIC FOOT ULCER

Foot disorders such as ulceration, infection, and gangrene are the leading causes of hospitalization in patients with diabetes mellitus.[11] Approximately 15 to 20 percent of the estimated 16 million persons in the United States with diabetes mellitus will be hospitalized with a foot complication at some time during the course of their disease. In India a study has reported 5 to 9% incidence of active / healed diabetic ulcer and 1% incidence of amputation.[12]

Unfortunately, many of these patients will require amputation within the foot or above the ankle as a consequence of severe infection or peripheral ischemia. Neuropathy is often a predisposing factor to ulceration and amputation.

The diabetic foot and its sequelae account for billions of rupees in direct medical expenditures, as well as lengthy hospital stays and periods of disability.[13] The most characteristic lesion of the diabetic foot is a mal perforans ulceration, which consequently is one of the major risk factors for amputation. Approximately 85 percent of all diabetes-related lower-extremity amputations are preceded by foot ulcers.[14,15]

The principal pathogenic mechanism of diabetic foot disease are;

a. Neuropathy
b. Ischaemia
c. Infection

**Diabetic Neuropathy**

Diabetic neuropahy occurs in approximately 50% of individuals with long standing type 1 or type 2 diabetes mellitus. As with other complications of diabetes mellitus, the development of neuropathy correlates with the duration of diabetes and glycemic control.

***Classification***

*a. Symmetric neuropathies*

1. Distal symmetric sensorimotor polyneuropathy.

2. Autonomic neuropathy.
3. Acute painful neuropathy.
4. Hyperglycemic neuropathy.
5. Treatment induced neuropathy.
6. Symmetric proximal lower extremity neuropathy.

*b. Focal and multifocal neuropathy*

1. Cranial neuropathy.
2. Thoracoabdominal neuropathy.
3. Focal limb neuropathy.
4. Diabetic amyotrophy.

The most common form of diabetic neuropathy is distal symmetric polyneuropathy. It frequently presents with distal sensory loss, hyperesthesia, parasthesia and dysesthesia.

Painful neuropathies may develop in these patients. Both acute (lasting < 12 months) and chronic forms of painful diabetic neuropathy have been described. Individuals with long standing type 1 or type 2 diabetes mellitus may develop autonomic neuropathy. This can involve multiple systems, including the cardiovascular, gastrointestinal, genitourinary and metabolic systems. Resting tachycardia, orthostatic hypotension, gastroparesis, bladder emptying abnormalities, hyperhidrosis of upper extremities and anhidrosis of lower extremities are features of diabetic autonomic neuropathy. Anhydrosis of the feet can promote dry skin with cracking which increases the risk of ulceration.

**Peripheral neuropathy**

***Pathophysiology***

The two major theories are;[16,17]

1. Metabolic effect of chronic hyperglycemia.
2. Ischaemic effect on peripheral nerves.
3. Others
    a. Neurotrophic factors.
    b. Immunologic mechanism.

*1. Metabolic theory* -

a. Polyol pathway[18]

Chronic hyperglycemia may result in activation of polyol pathway where glucose get converted into polyol sugars like, sorbitol and fructose catalysed by aldose reductase and sorbitol dehydrogenase. The accumulation of sorbitol may lead to reduced $Na^+/K^+$ ATPase activity which inturn causes abnormal decrease in nodal $Na^+$ membrane potential, increase in intraaxonal $Na^+$, nodal swelling and non specific structural changes of peripheral nerve leading to slowing of nerve conduction velocity.

b. Myo-inositol pathway[19]

In diabetes there may be decrease in intracellular concentration of myoinositol in peripheral neurons which inturn impair protein kinase C

activation. This leads to decreased $Na^+/K^+$ ATPase activity causing impaired nerve conduction.

c. Abnormal lipid metabolism[20]

Enzyme 8-6 desaturase normally converts dietary linoleic acid to δ linolenic acid. In diabetes there is inhibition of enzyme 8-6 desaturase which inturn leads to deficiency of δ linolenic acid causing impaired nerve blood flow, altered nerve membrane structure and nerve conduction.

d. Oxidative stress

In diabetes, there is compromised antioxidant defense mechanism and reduction in oxygen free radicals scavenges like superoxide dismutase, catalase, ascorbic acid, α-tocopherol and glutathione with enhancement of oxygen free radicals activity like, lipid hyperperoxide..

e. Advanced Glycosylation End (AGE) products[21]

It causes structural and functional abnormalities of neural and vascular tissues.

*2. Ischaemic effect on peripheral nerve*[22]

Occlusion of small arteries, arterioles and vasa nervosum of the peripheral nerves may result in structural and functional abnormalities of peripheral nerves causing peripheral neuropathy.

**Features of small and large fibre neuropathy**

***Small fiber dysfunction***

- Burning or lancinating pain.
- Hyperesthesia
- Paresthesia
- Loss of pain and temperature sensation
- Dysautonomia
- Foot ulceration
- Loss of visceral pain

***Large fibre dysfunction***

- Loss of position and vibration sensation.
- Areflexia
- Nerve conduction abnormalities
- Involvement of sensory nerve fiber leads to loss of the protective pain sensation.

Destruction of motor fibres results in small muscle atrophy in the foot causing, abnormal foot muscle mechanics and structural changes in the foot. The various deformities described are;

1. Hammer toe
2. Claw toe deformity
3. Prominent metatarsal heads.
4. Loss of plantar arches – Flattening of foot.
5. Charcots joint (Also called bag of loose bones).

The features of charcots foot are;

- Most common location is tarsometatarsal joint.
- Painless process due to profound sensory neuropathy.
- Unexplained swelling and erythema of the foot.
- No pain or mild discomfort not in proportion to the degree of bone and joint destruction.
- Severely deformed foot (Rocker bottom foot) with bony prominence.
- Susceptible to ulceration due to abnormal pressure points.

**Autonomic neuropathy**

***Pathophysiology***

- Paravertebral sympathetic ganglion dysfunction due to;[23,24]
    - Deposition of lipid rich material.
    - Dilation of endoplasmic reticulum causing vacular degeneration of neurons.
    - Mononuclear cell infiltration.
- Loss of myelinated nerve fibres like;[25,26,27]
    - Sympathetic communicating rami.
    - Vagus nerve.
    - Splanchnic nerves.
    - Nerves to the bladder walls.

Autonomic neuropathy in the foot causes loss of sympathetic effect leading to;[28]

- Denervation of oil and sweat gland – Cracking of dry foot skin.

- Arteriovenous shunting – Inefficient nutrient flow.

**Vascular complications**

Type 2 diabetes is associated with several form of dyslipidemia. The most common pattern of dyslipidemia is hypertriglyceridemia and reduced HDL cholesterol level. LDL particles found in type 2 DM are more atherogenic because they are easily glycated and susceptible to the oxidation. This leads to increased risk of atherosclerosis in DM. Diabetes is the single most important risk factor for the development of critical leg ischaemia and limb loss. Unless ischaemia is recognized and corrected, limb salvage efforts with the diabetic foot will fail even if infection and neuropathy have been appropriately treated. Vascular complications are divided into two groups.

- Macrovascular
- Microvascular

***Macrovascular complications***

Lower extremity arterial disease is more common among patients with diabetes. The presence of diabetes is associated with two to three fold excess risk of intermittent claudication due to accelerated atherosclerosis. The infrapopliteal (tibial) arteries are more commonly affected in diabetes due to atherosclerosis and the arteries of the foot are relatively spared. This allows for successful arterial reconstruction of these distal vessels.[29] Conversely the superficial femoral and popliteal artery is less likely to be affected by the occlusive process allowing these vessels to serve as a possible inflow source for bypass grafting.

***Microvascular complications***

In diabetes due to thickening of basement membrane there is microcirculatory impairment involving the capillaries and arterioles leading to microangiopathy. This leads to;

- Impairment in migration of the leukocytes.[30]
- Impaired hyperemic response following injury.[31]
- Impaired neurogenic vasodilatory response following injury.[32]
- Reduced capillary blood flow to skin extremities leading to functional ischaemia of the skin.[33]
- Increased susceptibility of the diabetic foot to infection.

**DIABETIC FOOT INFECTION**

Foot is the most common site of infection in patients with diabetes. It is responsible for more days of hospitalization than any other diabetic complications.[34] Among diabetic patients, foot infection is the leading cause of lower extremity limb loss. In the United States, it is estimated that 82,000 limb amputations are performed each in diabetic patients.[35] The rate of lower extremity amputation among these patients is 17 to 40 times greater than the nondiabetic patients.

A diabetic foot infection is most simply defined as any inframalleolar infection in a person with diabetes mellitus. These include paronychia, cellulitis, myositis, abscesses, necrotizing fascitis, septic arthritis, tendonitis, and osteomyelitis. The most common and classic lesion, however, is the infected

diabetic "mal perforans" foot ulcer.[4] Wound infection is the deposition and multiplication of bacteria in tissue with colony count of more than $10^5$ bacteria per gram of tissue with an associated host reaction.[3,37]

**Microbiologic features of diabetic foot**

Aerobic Gram-positive cocci are the predominant bacteria that colonize and acutely infect breaks in the skin. *Staph aureus* and the hemolytic streptococci (groups A, C, and G, but especially group B) are the most commonly isolated pathogens.[38] Chronic wounds develop a more complex colonizing flora, including enterococci various Enterobacteriaceae, obligate anaerobes, *Pseudomonas aeruginosa*, and nonfermentative Gram-negative rods.[39] Hospitalization, surgical procedures, and, especially, prolonged or broad-spectrum antibiotic therapy may predispose patients to colonization and/or infection with antibiotic-resistant organisms (e.g., MRSA or vancomycin-resistant enterococci [VRE]).[40] Although MRSA strains have previously been isolated mainly from hospitalized patients, community associated cases are now becoming common and are associated with poor outcomes in patients with diabetic foot infections.[41]

The impaired host defenses around necrotic soft tissue or bone may allow low-virulence colonizers, such as coagulase-negative staphylococci and *Corynebacterium* species ("diphtheroids"), to assume a pathogenic role. Acute infections in patients who have not recently received antimicrobials are often monomicrobial (almost always with an aerobic Gram-positive coccus), whereas chronic infections are often polymicrobial. Cultures of specimens obtained from patients with such mixed infections generally yield 35 isolates, including Gram-

positive and Gram-negative aerobes and anaerobes.[42,43] The pathogenic role of each isolate in a polymicrobial infection is often unclear.

**Table 2: Pathogens associated with various clinical foot-infection syndromes**[34]

| Foot- infection syndrome | Pathogens |
|---|---|
| Cellulitis without an open skin wound. | b-Hemolytic streptococcus* and Staph aureus |
| Infected ulcer and antibiotic naïve (X). | Staph aureus and b-hemolytic streptococcus* |
| Infected ulcer that is chronic or was previously treated with antibiotic therapy (Y). | Staph aureus, b-hemolytic streptococcus, and Enterobacteriaceae |
| Ulcer that is macerated because of soaking (Y). | Pseudomonas aeruginosa (often in combination with other organisms) |
| Long-duration nonhealing wounds with (Y, Z) prolonged broad-spectrum antibiotic therapy | Aerobic gram-positive cocci (Staph aureus, coagulase-negative staphylococci, and enterococci), diphtheroids, Enterobacteriaceae, Pseudomonas species, nonfermentative gram-negative rods, and, possibly, fungi |
| "Fetid foot": extensive necrosis or gangrene or malodorous (Z) | Mixed aerobic gram-positive cocci, including enterococci, gangrene, malodorous Enterobacteriaceae, nonfermentative gram-negative rods, and obligate anaerobes |

*Groups A, B, C, and G; X Often monomicrobial; Y Usually polymicrobial; Z Antibiotic-resistant species (eg, MRSA, vancomycin-resistant enterococci, or extended-spectrum b-lactamase–producing gram-negative rods) are common

**Table 3: Risk Factors for Foot Ulceration and Infection**[34]

| Risk Factor | Mechanism of Injury or Impairment |
|---|---|
| Peripheral motor neuropathy | Abnormal foot anatomy and biomechanics, with clawing of toes, high arch, and subluxed metatarsophalangeal joints, leading to excess pressure, callus formation, and ulcers. |
| Peripheral sensory neuropathy | Lack of protective sensation, leading to unattended minor injuries caused by excess pressure or mechanical or thermal injury. |
| Peripheral autonomic neuropathy | Deficient sweating leading to dry, cracking skin. |
| Neuro-osteoarthropathic deformities (i.e., Charcot disease) or limited joint mobility | Abnormal anatomy and biomechanics, leading to excess pressure, especially in the midplantar area. |
| Vascular (arterial) insufficiency | Impaired tissue viability, wound healing, and delivery of neutrophils. |
| Hyperglycemia and other metabolic derangements | Impaired immunological (especially neutrophil) function and wound healing and excess collagen cross-linking. |
| Patients disabilities | Patient Reduced vision, limited mobility, and previous amputation(s). |
| Maladaptive patient behaviors | Inadequate adherence to precautionary measures and foot inspection and hygiene procedures, poor compliance with medical care, inappropriate activities, excessive weight-bearing, and poor footwear. |
| Health care system failures | Inadequate patient education and monitoring of glycemic control and foot care. |

**Pathophysiology**

Diabetes mellitus is a disorder that primarily affects the microvascular circulation. Most diabetic foot infections occur in the setting of good dorsalis pedis pulses. This finding indicates that the primary problem in diabetic foot infections is microvascular compromise. Impaired microvascular circulation hinders white cell migration into the area of infection and limits the ability of antibiotics to reach the site of infection and achieve effective concentration. Immunological responses are affected in patients with diabetes. This increase the risk and severity of foot infection.[44]

Peripheral neuropathy and angiopathy predisposes the foot to infections. Neuropathy is responsible for the breakdown of the first line of defense, the intact skin. Autonomic neuropathy causes dry skin and predisposes the skin to crack. The foot with sensory neuropathy tends to have repeated trauma resulting in loss of skin integrity and providing a nidus for microbial invasion.Motor neuropathy results in intrinsic foot muscle atrophy, thus altering the foot architecture and change the pressure points of the involved foot as it strikes the ground during walking and thus increases the risk of foot ulceration and infection.

**Table 4: Pathogenic effects of virulent micro-organisms over wound**

| | |
|---|---|
| Toxin production | Vigorous stimulation of immune cells |
| Superantigen release within the blood stream that initiates an uncontrolled proliferation of T cells | Stimulation of T (thymus maturing) cell subsets allowing the release of cytokines that initiate cell and tissue damage |
| Superantigen production | Some species of micro-organisms such as the exotoxins of Staphylococcus and Streptococcus produce superantigens |
| Presence of biofilms | A microbial colony encased in an adhesive polysaccharide matrix that is usually attached to a wound surface. Biofilms present in the form of a transparent sticky film covering the wound surface. Cells in biofilms exhibit a decreased sensitivity to host immunological defence mechanisms, decreased susceptibility to antimicrobial agents and increased virulence. They have also been implicated in persistent infections.[45] |

**Recognition of wound infection**[46]

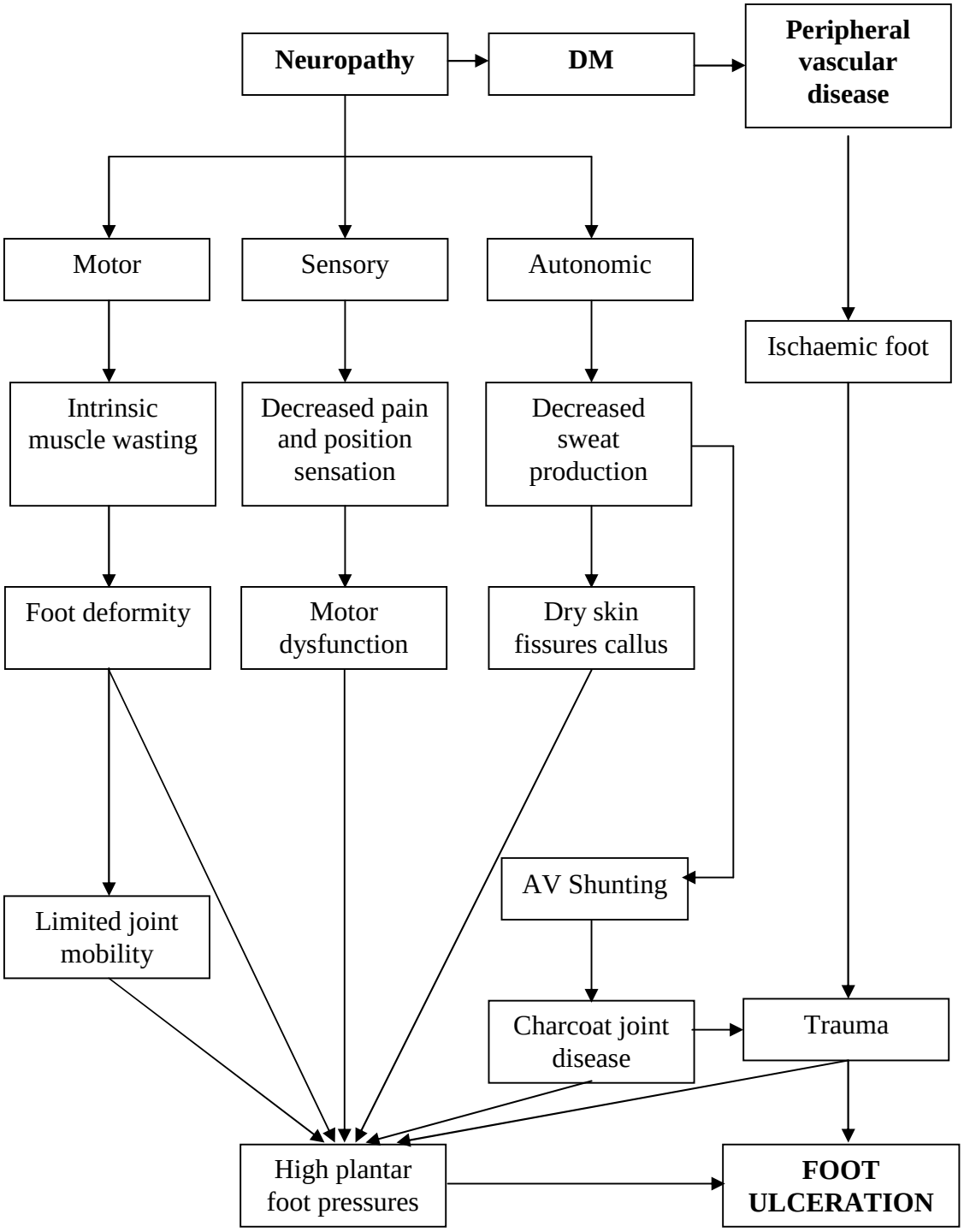
The inflammatory response is a protective mechanism that aims to neutralise and destroy any toxic agents at the site of an injury and restore tissue homeostasis. The classic signs of infection include:

- Localised erythema.
- Localised pain.
- Localised heat.
- Oedema.

Further criteria include:

- Abscess.
- Discharge which may be viscous in nature, discoloured and purulent.
- Delayed healing not previously anticipated.
- Discolouration of tissues both within and at the wound margins.
- Unhealthy granulation tissue.
- Abnormal smell.
- Wound breakdown associated with wound pocketing/bridging at base of wound.

***Figure 3: Clinical pathways leading to foot ulceration***[28,47]



**Evaluation**

- Characteristics: Size, depth, appearance, discharge and location.
- Etiological assessment: Neuropathic, ischemic, or neuro-ischemic.
- Screening for neuropathy.
    - Pressure of a 5.07 (10-g) Semmes Weinstein monofilament.
    - Vibration sensation with the use of standard tuning fork (128 cycles per second)
    - Neurologic reflex hammer.
- Probing of ulcer for underlying osteomyelitis.
- Culture sensitivity of the discharge.
- Radiograph for underlying osteomyelitis.
- Colour Doppler study for vascular pathology.
- MRI for Charcoats neuropathy.

**Classification**

The Wagner system has been widely used for 25 years for grading of diabetic foot ulcer.[48,49]

***Table 5: Wagner Ulcer Classification System***

| Grade | Lesion |
|---|---|
| 0 | No open lesions; may have deformity or cellulites. |
| 1 | Superficial diabetic ulcer (partial or full thickness). |
| 2 | Ulcer extension to ligament, tendon, joint capsule, or deep fascia without abscess or osteomyelitis. |
| 3 | Deep ulcer with abscess, osteomyelitis, or joint sepsis. |
| 4 | Gangrene localized to portion of forefoot or heel. |
| 5 | Extensive gangrenous involvement of the entire foot. |

Wagner ulcer classification system was developed for the "dysvascular" foot. It was skewed toward severe disease and contains all infections within a single grade.

Consensus is developing that the key issues in classifying a diabetic foot wound are its depth (in particular, which tissues are involved) and whether the wound is complicated by either ischemia or infection. The International Consensus on the Diabetic Foot recently published a preliminary progress report on a diabetic foot ulcer classification system for research purposes.[48,50,51] The key elements are summarized by the acronym PEDIS (perfusion, extent/size, depth/tissue loss, infection, and sensation).

**Table 6: PEDIS Classification**

| Clinical Manifestations of Infection | Infection Severity | PEDIS Grade* |
|---|---|---|
| Wound lacking purulence or any manifestations of inflammation | Uninfected | 1 |
| Presence of more than or equal to 2 manifestations of inflammation (purulence, or erythema, pain, tenderness, warmth, or induration), but any cellulitis/erythema extends less than or equal 2 cm around the ulcer, and infection is limited to the skin or superficial subcutaneous tissues; no other local complications or systemic illness | Mild | 2 |
| Infection (as above) in a patient who is systemically well and metabolically stable but who has one of the following characteristics: cellulitis extending more than two cm, lymphangitic streaking, spread beneath the superficial fascia, deep-tissue abscess, gangrene, and involvement of muscle, tendon, joint, or bone | Moderate | 3 |
| Infection in a patient with systemic toxicity or metabolic instability (for example, fever, chills, tachycardia, hypotension, confusion, vomiting, leukocytosis, acidosis, severe hyperglycemia, or azotemia) | Severe | 4 |

*** PEDIS indicates perfusion, extent/size, depth/tissue loss, infection, and sensation.**

**Treatment**

***Figure 4: Part 1: Approach to treating a patient with diabetic foot wound***[34]

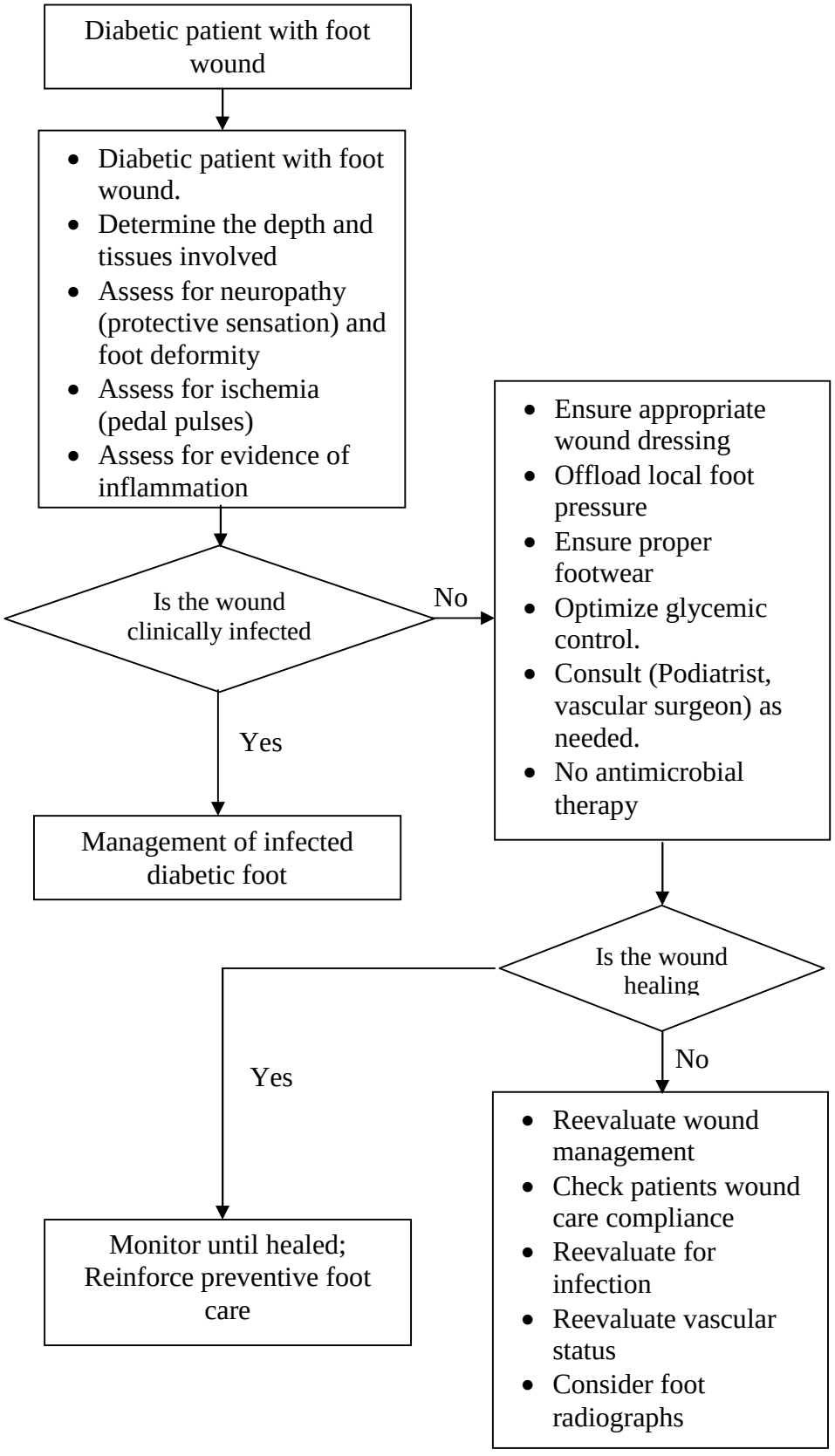


***Figure 5: Part 2: Approach to the management of infected diabetic foot***[34]

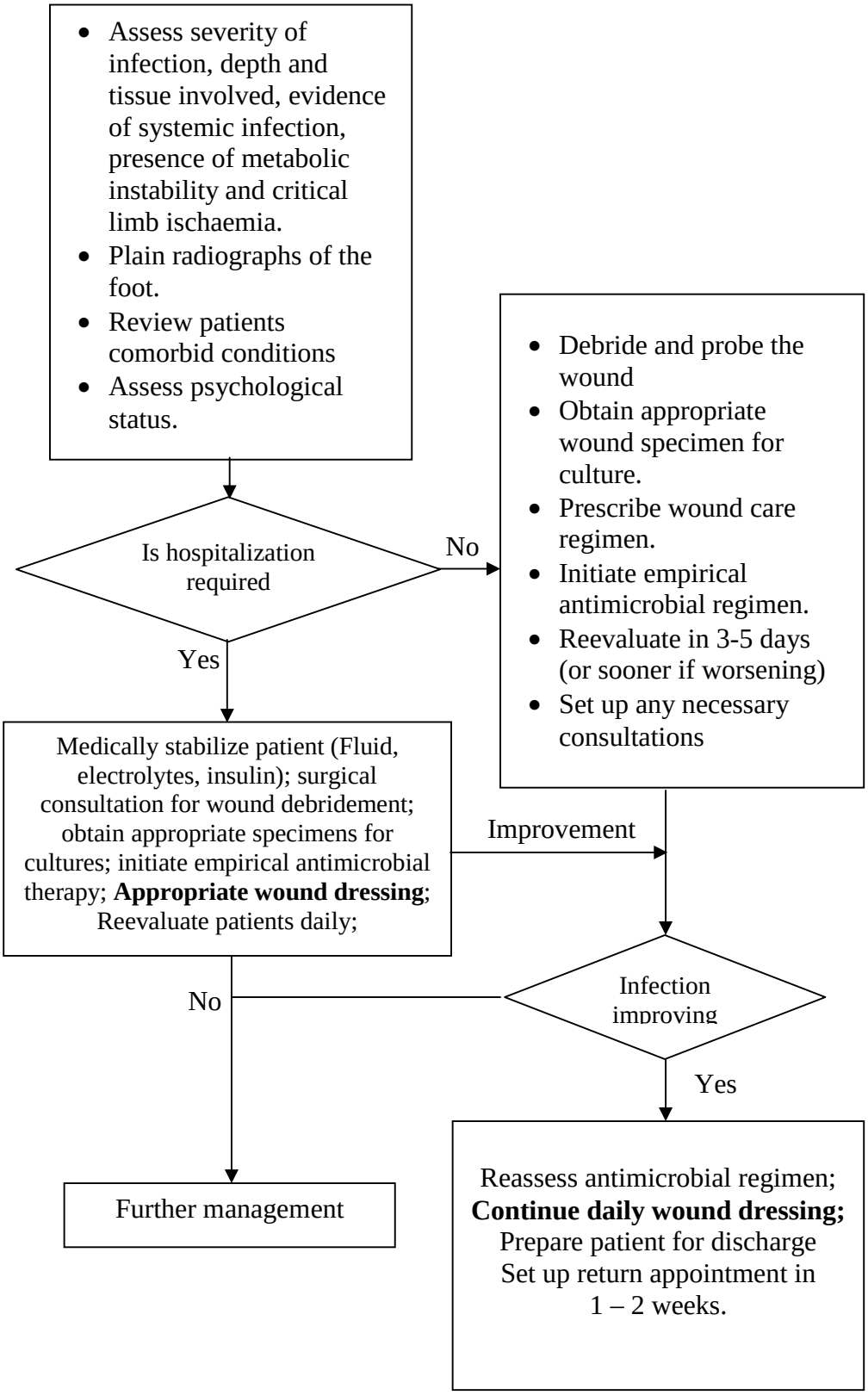
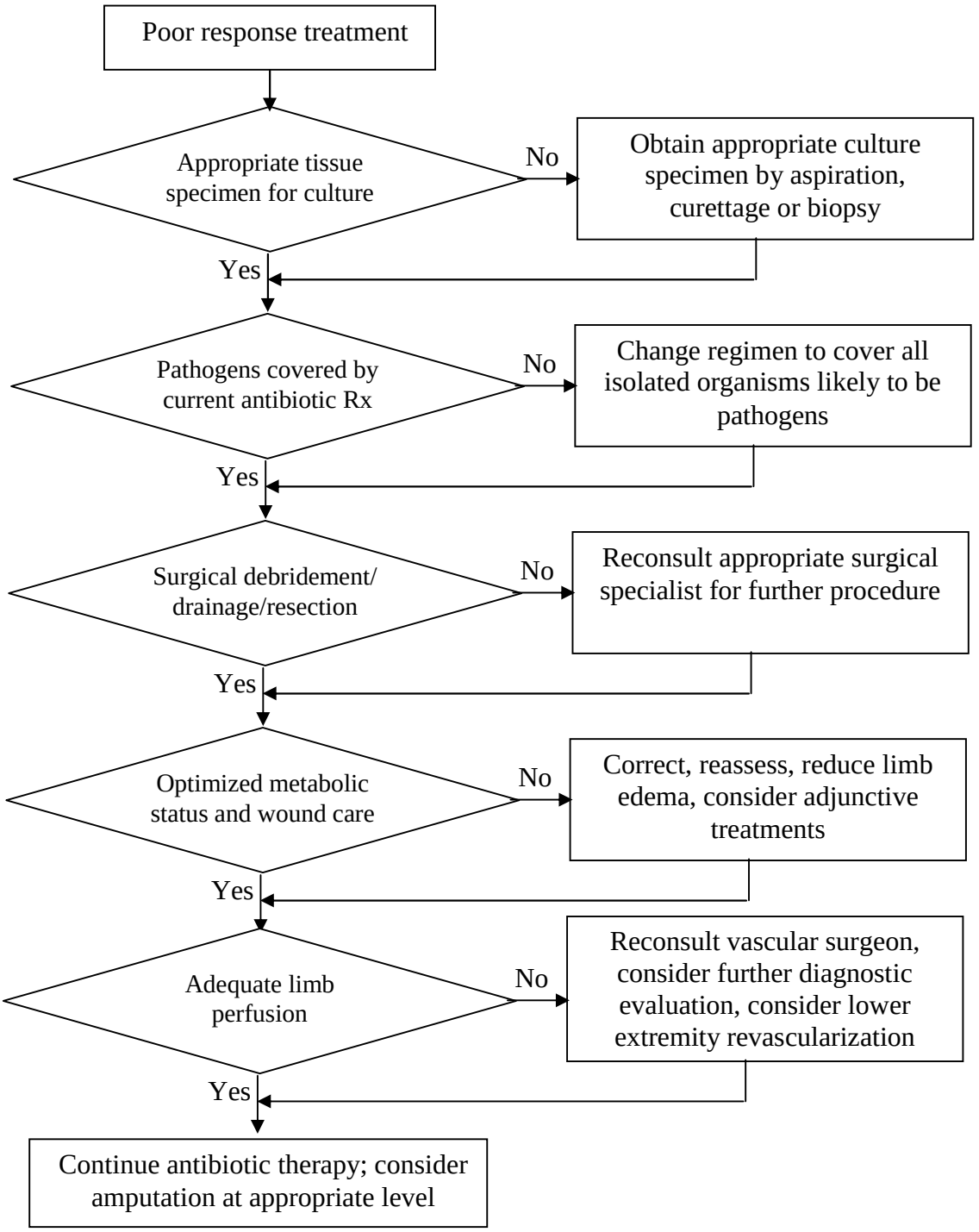


***Figure 6: Part 3: Approach to the patient of infected diabetic foot not responding to the treatment***[34]



**Wound dressings**

Patients suffering from diabetic foot ulcers need special care. Infection of the diabetic ulcer can have a serious consequences. The challenges in treating diabetic foot ulcers includes prolonged hospital stay, high morbidities, medical expenses and sometime leads to lower limb amputation. Dressing is one of the important part of the treatment of the diabetic ulcer.

The types of wound dressing used in diabetic foot ulcer are;

1. Traditional dressing
    a. Gauze dressing
2. Modern wound dressing (Occlusive / moist wound dressing)
    a. Alginate Dressings
    b. Amorphous hydrogels
    c. Hydrogel Dressings
    d. Hydrocolloid Dressings
    e. Composite Dressings
    f. Transparent Films

*Traditional dressing*

In last few centuries gauze dressing was widely used in local wounds care mainly because of its low price and simplicity. The rationale behind this conventional wound management is to absorb exudation from the wound to keep it dry and clean enough to avoid bacterial contamination (Wet to dry approach). Newer studies and concepts have challenged the effectiveness of gauze as a ideal

dressing material. This is because of its poor fluid absorption and retention ability from the wound and associated pain during change of the dressing.[52,53] It is also known that betadine solution and topical gel used along with gauze dressing are toxic to granulation tissue apart from being painful during the change of the dressing.

*Modern wound dressing (Occlusive/moist wound dressing)*

In 1950 a new concept of wound management was introduced. This method was aimed at occluding the wound to protect against bacteria while keeping it moist to supply growth factor and prevent crust formation over the wound – Occlusive wound dressing.

Calcium Alginate

Alginate has been used in various forms for fifty years and yet they remain a poorly understood and probably underused material for wound dressing. It is highly absorbent, biodegradable dressing, derived from cell wall of marine brown algae.

Alginic acid is a polymer of d-mannuronic acid. It was discovered in 1982, (D-mannuronate and L-galuranicocid) Scotland. Its formula bears a striking resemblance to that of cellulose. The main difference is that, the alcoholic group is replaced by the carboxyl group ($C_6H_{10}O_7$).

The sodium salt of alginic acid, sodium alginate (NaAlg), when acted upon by ionic calcium, undergoes instantaneous coagulation;

$$2NaAlg + CaCl_2 \longrightarrow CaAlg_2 + 2NaCl$$

This reaction is the basis of preparing film in calcium alginate dressing. Calcium alginate has so far been found to be most useful, as;

1. The lowest degree of contraction on drying.
2. Its excellent film forming properties.
3. Filaments of calcium alginate can be easily prepared by extruding a viscous solution of sodium alginate through jets passing through a bath of coagulant, such as calcium choride.
4. It absorbs its own weight of water or even more under favourable conditions. It has capacity to absorb and retain about 10 ml of exudates per gm of dressing. Other salts like zinc, chromium and beryllium alginates are less susceptible to swelling.
5. It has low allergic potentials and good tolerance.
6. Its permeability to gas and hemostatic effect.

The calcium alginate promotes the wound healing in various ways;

1. It forms a hydrophilic gel layer on the secreting wound thus, acts like a moist dressing which prevents the wound from drying. A microclimate favourable for wound healing is produced which promotes the formation granulation tissue, limits wound secretion and minimizes bacterial contamination.[54]
2. It increases the proliferation of fibroblasts thus helps in filling of large tissue defect by proliferation or granulation tissue.[55]

3. It has property of absorbing large volumes of exudates and ability to lock the exudates which results in bacterial retention within the dressing matrix. Thus reduces the bacterial load from the wound surface.[5,6,7,56]

It has been demonstrated that following hydration of the calcium alginate, it forms a cohesive gel which is effective in encompassing large population of potentially pathogenic bacteria such as pseudomonas aeruginosa and staphylococcus aureus under the gelled surface, as well as being immobilized within the swollen fibres.[5]

The individual calcium alginate fibres swells after absorbing fluid and compress against each other by capillary action. Consequently the swollen fibres converge together resulting in the formation of a cohesive gel, a compound of the glucoronic acid with calcium.[5]

Calcium alginate is not bactericidal. However it prevents the bacterial adhesion to the plasma membranes of epithelial cells.

It has been demonstrated that calcium alginate has hemostatic action due to the formation of prothrombin and platelet activation induced by Ca2+ ion.[57] In addition calcium has a stabilizing effect on cellular membrane and acts against increased capillary permeability during an allergic reaction. The other advantages of calcium alginate includes reduction in pain during change of dressing.[58]

The disadvantages of calcium alginate include;

a. Limited absorptive capacity.
b. Splitting of fibrous material.

c. Subjectively: Gelatinous material appears like infectious foreign body.
d. Unsuitable for chronic and non reactive wound without exudation.
e. Disturbance of granulation if fibres are removed after incomplete gelatinous transformation.

The alginate dressing is indicated in sloughy wound with exudates. It absorb exudates and forms a moist covering all over the slough preventing it from drying out. For shallow, heavily exudating diabetic foot ulcer, fibrous sheet dressings of alginate fibre is useful, whereas in a wound with cavity, ribbon or rope forms of alginate fiber can be used for dressing.

Deep wound with little secretion can be packed with alginate moistened with ringers solution. The frequency of dressing change depends on the individual wound. Any fibre remaining in the wound may be washed with ringers solution, otherwise gel plug can be removed from the wound with forceps. During the wound cleaning phase, dressing once or twice daily may be required depending on the degree of exudation. Later as granulation tissue commences, a dressing change every two to three days may be sufficient.

**Literature review**

Randomized control trial studies have documented the faster healing rate of ulcers, decrease in ulcer size, amount of exudates and cost effectiveness by using calcium alginate dressing.[52,59]

Study on bacterial retaining ability of calcium alginate has documented 7 to 12% bacteria retaining ability in cases of *Staphylococcus aureus* and 30 to 40% retaining ability in case of *Pseudomonas aeruginosa.*[5]

Another study done by creating artificial infected wound has documented 37% bacteria retaining ability of calcium alginate in case of *Staphylococcus aureus* and 29% in case of *Pseudomonas aeroginosa.*[6]

# METHODOLOGY

The present study was conducted in the Department of Surgery, KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belgaum on patients with infected diabetic foot during the period of January 2008 to December 2008.

**Study design**

One year randomized controlled trial.

**Study period**

The present study was conducted during January 2008 to December 2008.

**Method of collection of data**

**Source of Data**

Patients infected with diabetes foot admitted at KLES, Dr. Prabhakar Kore Hospital and Medical Research Centre, Belgaum.

**Sample size**

A sample size of 60 cases divided into two groups.

**Sampling procedure**

As no data was available regarding the bacterial load over the ulcer after the two dressings, the sample size of 30, in each group has been estimated considering last three years hospital statistics of inpatient diabetic foot ulcer.

**Selection criteria**

***Inclusion Criteria***

- Patients aged between 35 to 65 years.
- Patients with DM (HbA1c < 8.0)
- Patients with infected diabetic foot ulcer with bacterial count of more than 1 X $10^5$ CFU per gm wound tissue.
- Ulcer size < 10 X 10 cms.

***Exclusion Criteria***

- Immuncompromised state.
    - Suffering from HIV or TB.
    - On chronic steroid therapy.
    - Severely malnourished.
- Underlying osteomyelitis.
- Vasculopathy.
- Cellulitis.
- Diabetic ketoacidosis.

**Procedure**

The study was conducted in Department of Surgery at KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belgaum during one year duration. The study was approved by the Ethical and Research Committee of Jawaharlal Nehru Medical College, Belgaum.

After finding the suitability as per inclusion and exclusion criteria patients were selected for the study and briefed about the nature of the study, the interventions used and written informed consent was obtained (Annexure–I ). Further, descriptive data of the participants like name, age, sex, detailed history, were obtained by interviewing the participants and clinical examination and necessary investigations were recorded on predesigned and pretested proforma (Annexure-II).

In all suitable enrollees, bacterial load was determined and infected diabetic foot was confirmed. After that they were divided into two different groups (Group 1 and Group 2) by number randomization. Both the groups were administered with similar sets of antibiotics till result of culture and antibiotic sensitivity. Later based on culture sensitivity, specific antibiotics were started. Calcium alginate dressing was done to every odd number enrollee (Group 1) and conventional gauze dressing to every even number enrollee (Group 2). Dressing was changed after every 24 hours for three days.

Tissue sample from the centre of the ulcer was taken before the first and after the third dressing in both the groups and sample was sent to Department of Microbiology laboratory in the transport medium immediately. The tissue was weighed and one gm of tissue was homogenized, serially diluted in 1:5 dilution in glucose broth, incubated and bacteria was sub cultured on to blood agar, chocolate agar and MacConkey agar under aerobic conditions using standard loop (4 mm internal diameter carrying 0.001 ml) and identified as per the standard protocol and the total viable bacterial count was determined.

**Statistical Methods**

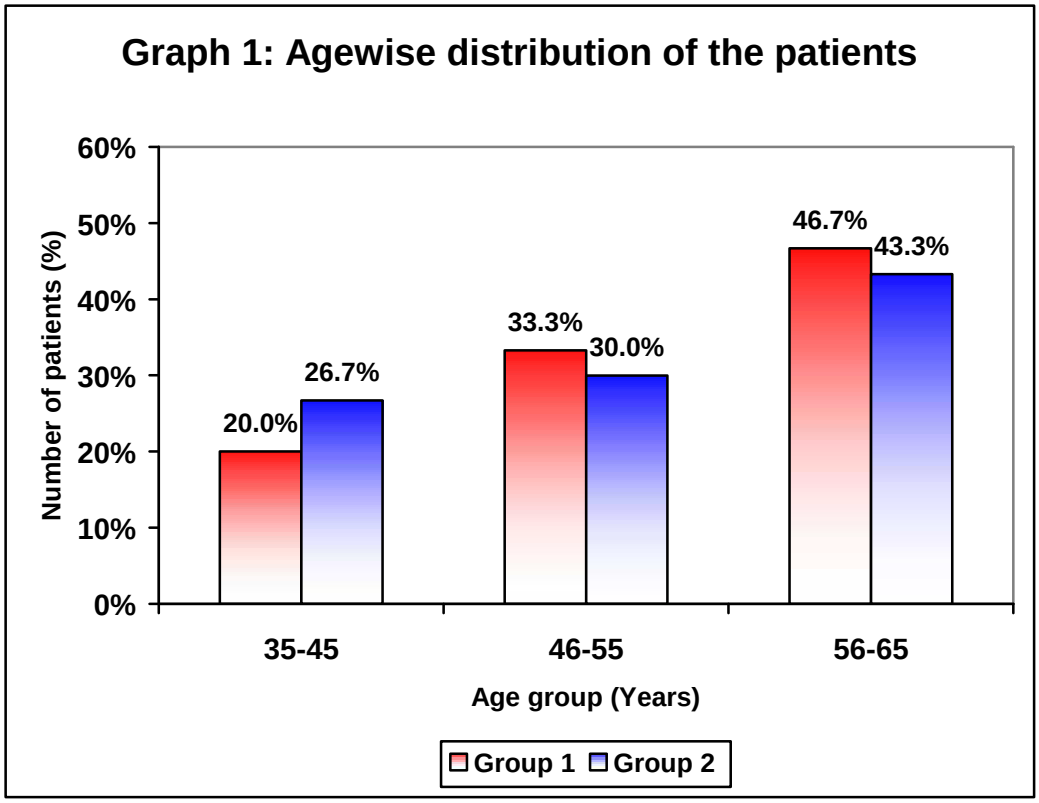
At the end of the study mean bacterial load in the wound of the both groups was determined before the first and after the third dressing. Data was compared by using unpaired ‘t’ test and a ‘p’ value of $< 0.05$ was considered significant.

# RESULTS

The present study was conducted in the Department of Surgery, KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belgaum on patients with infected diabetic ulcer during the period of January 2008 to December 2008. A sample size of 60 cases with infected diabetic ulcer was divided into two equal groups Group 1 (Patients with calcium alginate dressing) and group 2 (Patients with conventional gauze dressing) and studied. The findings and observations were noted, analysed and tabulated as below.

**Table 7: Agewise distribution of the patients**

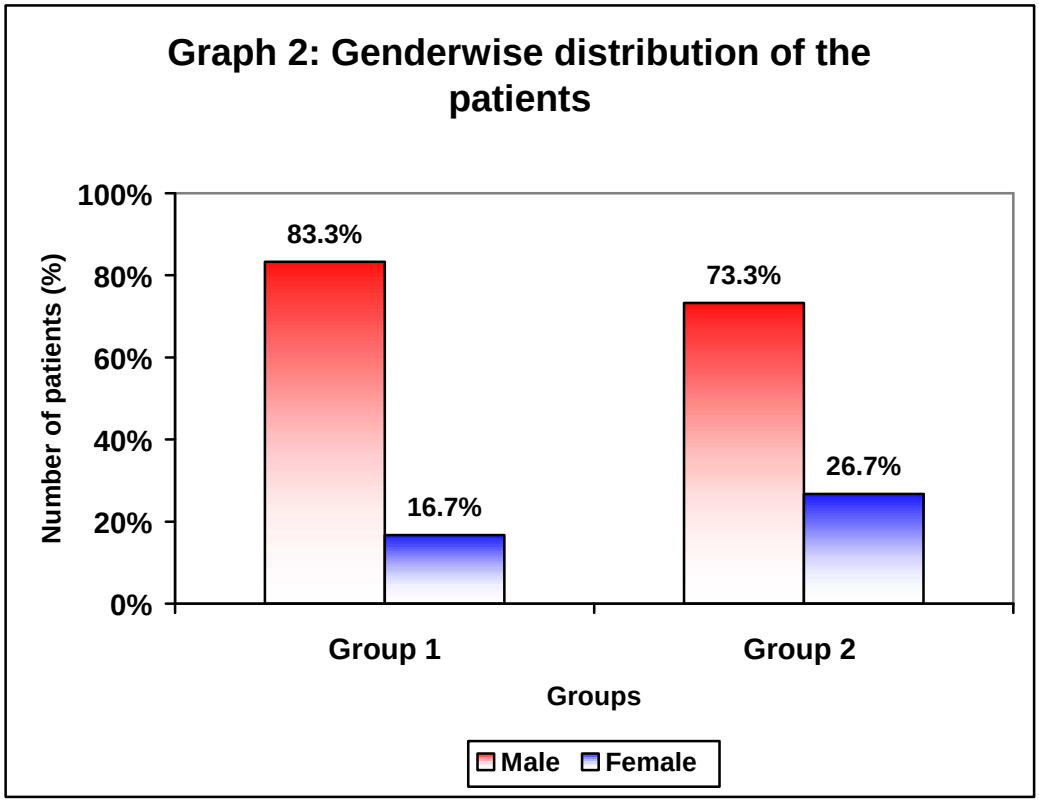
| Age (Years) | Group 1 | | Group 2 | |
|---|---|---|---|---|
| | Number | Percentage | Number | Percentage |
| **35 – 45** | 6 | 20.0% | 08 | 26.7% |
| **46 – 55** | 10 | 33.3% | 09 | 30.0% |
| **56 – 65** | 14 | 46.7% | 13 | 43.3% |
| **Total** | **30** | **100%** | **30** | **100%** |




In group 1 majority (46.7%) of the patients were aged between 56 to 65 years followed by 33.3% between 46 to 55 years and 20.0% were aged between 35 to 45 years. In group 2 majority (43.3%) of the patients were aged between 56 to 65 years followed by 30.0% between 46 to 55 years and 26.7% were aged between 35 to 45 years.

**Table 8: Genderwise distribution of the patients**

| Gender | Group 1 | | Group 2 | |
|---|---|---|---|---|
| | **Number** | **Percentage** | **Number** | **Percentage** |
| **Male** | 25 | 83.3% | 22 | 73.3% |
| **Female** | 05 | 16.7% | 08 | 26.7% |
| **Total** | **30** | **100%** | **30** | **100%** |




Among 60 cases majority of the patients were males (group 1, males 83.3% and females 16.7%; group 2, males 73.3% and females 26.7%). In Group 1 the male to female ratio was 5:1 and in group 2 it was 2.75:1.

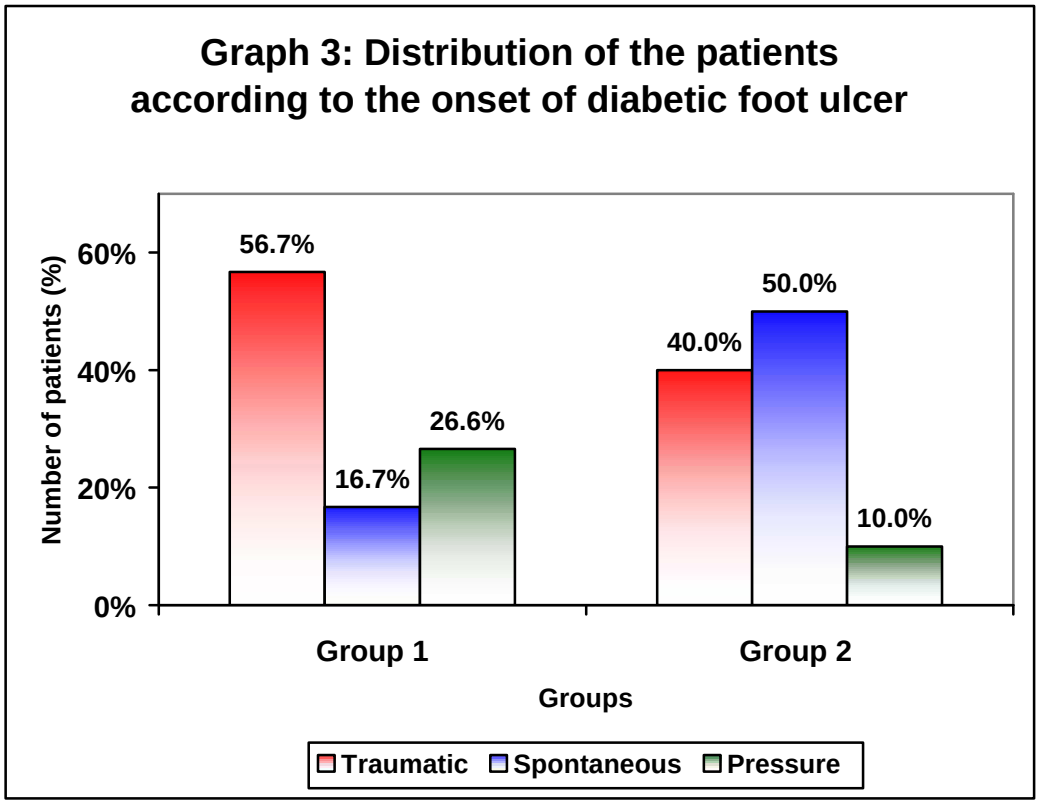
**Table 9: Distribution of the patients according duration of diabetes mellitus**

| Duration (Years) | Group 1 | | Group 2 | |
|---|---|---|---|---|
| | **Number** | **Percentage** | **Number** | **Percentage** |
| **Upto 5** | 08 | 26.6% | 07 | 23.3% |
| **6 – 10** | 12 | 40.0% | 11 | 36.7% |
| **11 – 15** | 04 | 13.4% | 08 | 26.7% |
| **16 – 20** | 06 | 20.0% | 04 | 13.3% |
| **Total** | **30** | **100%** | **30** | **100%** |

In group 1 the percentage of patients with duration of DM less than 5 years was 26.6%, 6 to 10 year was 40%, 11 to 15 years was 13.4% and 16 to 20 years was 20%. In group 2, 23.3% patients had duration of DM less than 5 years, 36.7% had 6 to 10 years, 26.7% had 11 to 15 years and 13.3% of patients had 16 to 20 years.

**Table 10: Distribution of the patients according to the onset of diabetic foot ulcer**

| Onset | Group 1 | | Group 2 | |
|---|---|---|---|---|
| | **Number** | **Percentage** | **Number** | **Percentage** |
| **Traumatic** | 17 | 56.7% | 12 | 40% |
| **Spontaneous** | 05 | 16.7% | 15 | 50% |
| **Pressure** | 08 | 26.6% | 03 | 10% |
| **Total** | **30** | **100%** | **30** | **100%** |




In group 1 trauma was the most common (56.7%) cause for the onset of diabetic foot ulcer. In group 2, diabetic foot ulcers were commonly (50%) spontaneous in onset.

**Table 11: Distribution of the patients according to site of the ulcer**

| Site of ulcer | Group 1 | | Group 2 | | Total | |
|---|---|---|---|---|---|---|
| | No. | % | No. | % | No. | % |
| **Dorsum** | 21 | 70.0% | 20 | 66.7% | 41 | 68.3% |
| **Plantar** | 09 | 30.0% | 10 | 33.3% | 19 | 31.7% |
| **Total** | **30** | **100%** | **30** | **100%** | **60** | **100%** |

Among 60 patients, diabetic foot ulcers were commonly (68.3%) located over the dorsal aspect of the foot. In remaining 31.7% of the patients the ulcers were located over the plantar aspect of the foot (Group 1, dorsum 70% and plantar 30%; Group 2, dorsum 66.7% and plantar 33.3%).

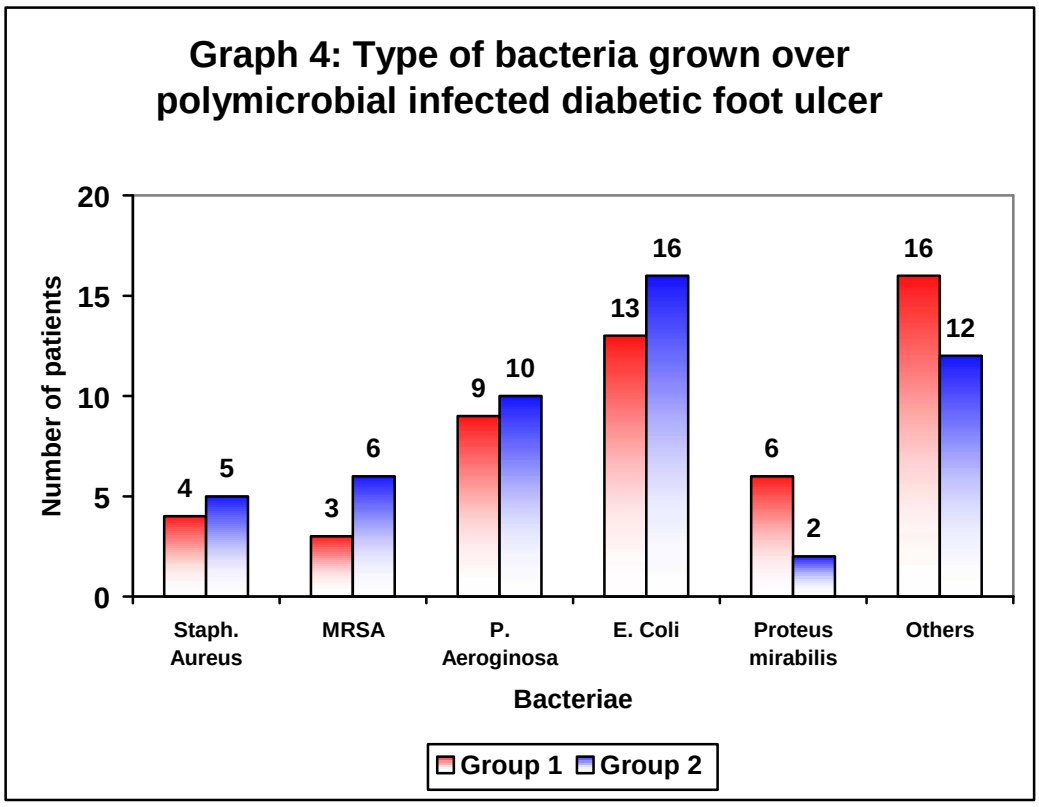
**Table 12: Distribution of the patients according diabetic foot complications**

| Complications | Group 1 (n=30) | | Group 2 (n=30) | |
|---|---|---|---|---|
| | **Number** | **Percentage** | **Number** | **Percentage** |
| Peripheral Neuropathy | 10 | 33.3% | 08 | 26.7% |
| Foot deformity | 06 | 20.0% | 06 | 23.3% |
| Amputation | 04 | 13.3% | 03 | 10% |

It was observed that peripheral neuropathy was the most common (33.3%) complication associated with diabetic foot ulcer in group 1 followed by foot deformity in 20% of the patients and amputation in 13.3% of the patients. In group 2 the most common complication was peripheral neuropathy in 26.7% of the patients followed by foot deformity in 23.3% and amputations in 10% of the patients.

**Table 13: Type of bacteria grown over polymicrobial infected diabetic foot ulcer**

| Bacteria | Group 1 (n=30) | Group 2 (n=30) | Total (n=60) |
|---|---|---|---|
| Staph. aureus | 04 | 05 | 09 |
| MRSA | 03 | 06 | 09 |
| P. aeroginosa | 09 | 10 | 19 |
| E. coli | 13 | 16 | 29 |
| Proteus mirabilis | 06 | 02 | 08 |
| Others | 16 | 12 | 28 |

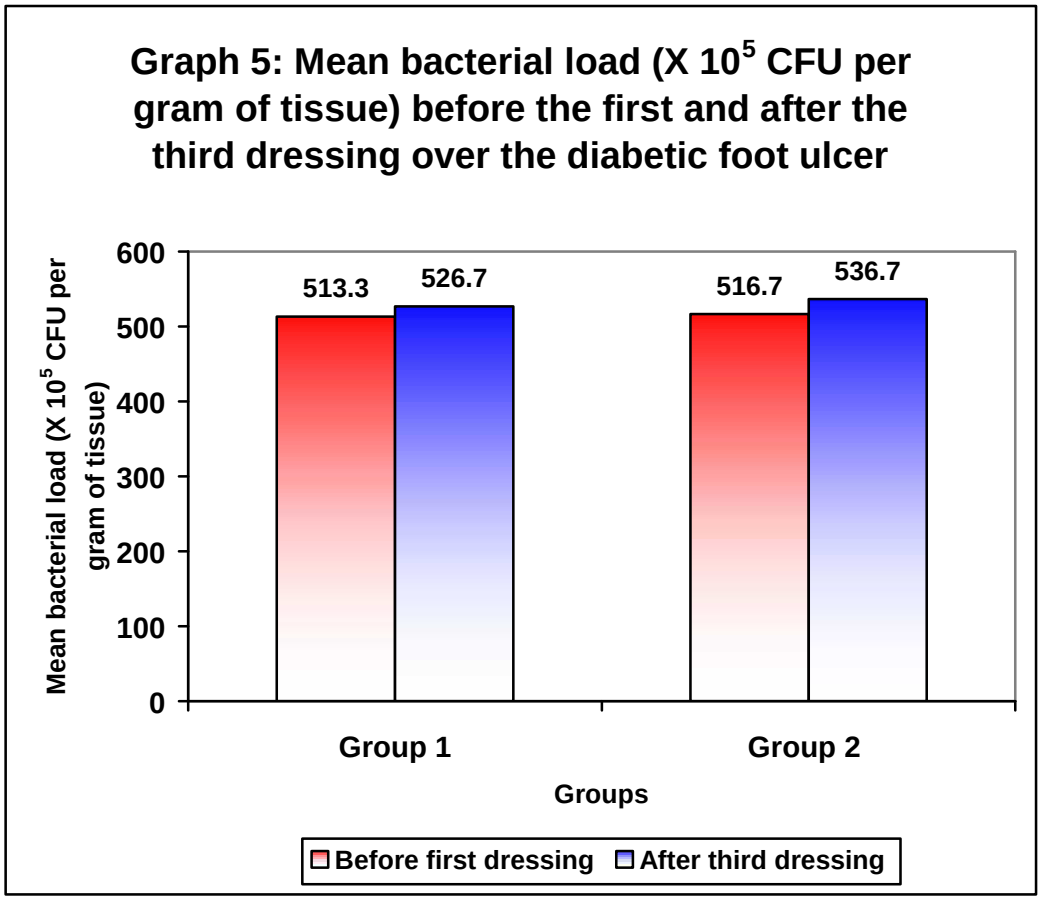


The infection of diabetic ulcer were polymicrobial in nature. In group 1, E. coli (13 cases), pseudomonas aeroginosa (9 cases) staph aureus (4 cases), proteus mirabilis (6 cases) and MRSA (3 cases) were cultured. In group 2, E. coli (16 cases), pseudomonas aeroginosa (10 cases) staph. aureus (5 cases), proteus mirabilis (2 cases) and MRSA (6 cases) were cultured.

Other bacterial growth over diabetic foot ulcer in group 1 was Kleb pneumoniae (9 cases), Kleb oxytoca (2 cases), Citrobacter freundi (2 cases), Citrobacter diverses (2 cases) and Proteus vulgaris (1 case). In group 2, Kleb pneumoniae (6 cases), Kleb oxytoca (4 cases) and Citrobacter freundi (2 cases) were cultured.

**Table 14: Mean bacterial load (X $10^5$ CFU per gram of tissue) before the first and after the third dressing over the diabetic foot ulcer**

| Dressing | Group 1 (n=30) | | Group 2 (n=30) | | p value |
|---|---|---|---|---|---|
| | Mean | S.D. | Mean | S.D. | |
| Before first dressing | 513.3 | 122.4 | 516.7 | 117.7 | 0.915 |
| After third dressing | 526.7 | 138.8 | 536.7 | 121.7 | 0.768 |
| p value | 0.221 | | 0.132 | | |

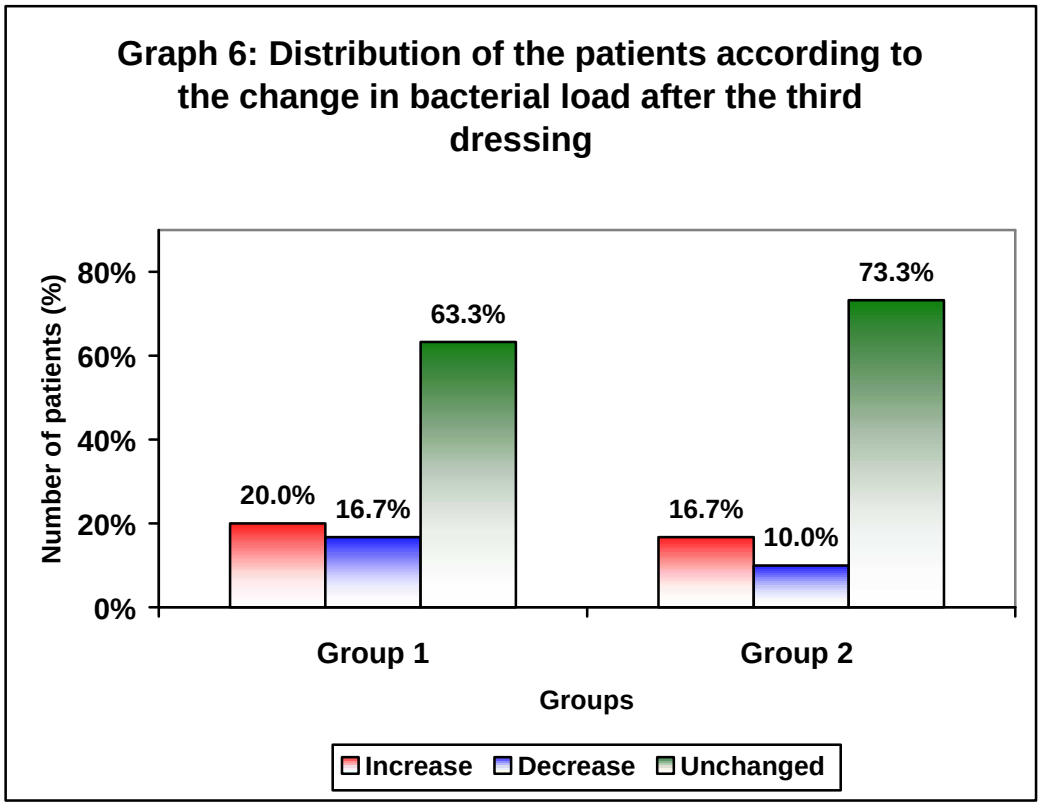



The mean bacterial load (X $10^5$ CFU per gram of tissue) over the diabetic ulcer in group 1 before the first dressing was 513.3 ± 122.4. The bacterial load after the third dressing was 526.7 ± 138.8. There was increase in bacterial load over the diabetic ulcer after the third dressing. However this increase in bacterial load was statistically not significant (p=0.221).

The mean bacterial load (X $10^5$ CFU per gram of tissue) over the diabetic ulcer in group 2 before the first dressing was 516.7 ± 117.7. The bacterial load after the third dressing was 536.7 ± 121.7. There was increase in bacterial load over the ulcer after the third dressing. However this increase was statistically not significant (p=0.132).

**Table 15: Distribution of the patients according to the change in bacterial load after the third dressing**

| Bacterial load | Group 1 (n=30) | | Group 2 (n=30) | |
|---|---|---|---|---|
| | Number | Percentage | Number | Percentage |
| Increase | 06 | 20.0% | 05 | 16.7% |
| Decrease | 05 | 16.7% | 03 | 10.0% |
| Unchanged | 19 | 63.3% | 22 | 73.3% |

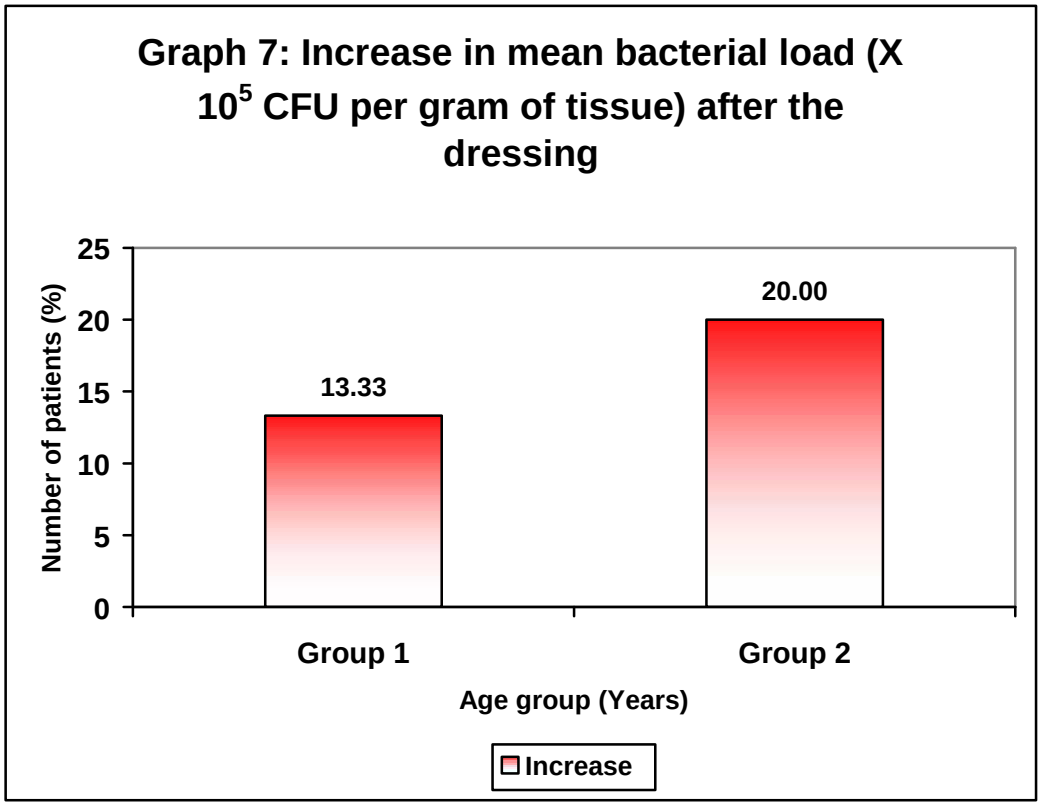



It was observed that after the third dressing in group 1 there was no change in bacterial load in 63.3% of the patients. The bacterial load was increased 20% of the patients and decrease in 16.7%. In group 2 there was no change in bacterial load in 73.3% of the patients. The bacterial load was increased in 16.7% of the patients and decreased in 10%.

**Table 16: Increase in mean bacterial load (X $10^5$ CFU per gram of tissue) after the third dressing**

| | Group 1 | | Group 2 | |
|---|---|---|---|---|
| | **Mean** | **S.D.** | **Mean** | **S.D.** |
| Bacterial load | 13.33 | 93.71 | 20.00 | 96.13 |

p=0.787




After the third dressing in group 1 patients the net increase in mean bacterial load was 13.33 ± 93.71 X $10^5$ CFU per gram of tissue. In group 2 the net increase in mean bacterial load was 20.00 ± 96.13 X $10^5$ CFU per gram of tissue. However the mean increase of bacterial load in both the groups was statistically not significant (p=0.787).

# DISCUSSION

The ulcer dressing is an important aspect of diabetic foot management. The basic function of any dressing is to protect the ulcer from mechanical trauma, to create a moist environment and prevent exposure to infections. The occlusive wound dressing reduces the bacterial load by absorption of the exudates and by preventing the bacterial contamination of the ulcer. This reduces the requirement for phagocytic and autolytic debridement and reduces the source for microbial growth.

The present study was conducted at Department of Surgery, KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belgaum to assess the efficacy of calcium alginate dressing versus conventional gauze dressing on bacterial load over the infected diabetic foot ulcer.

In the present study majority of the patients in both the groups were aged between 56 to 65 years (Calcium alginate group, 46.7%; conventional gauze group, 43.3%).

Among 60 cases majority of the patients were males (Calcium alginate group, males 83.3% and females 16.7%; conventional gauze group, males 73.3% and females 26.7%). In Calcium alginate group the male to female ratio was 5:1 and in conventional gauze group it was 2.75:1.

The duration of DM was 6 to 10 years in majority of the patients in both the groups (Calcium alginate group 40% and conventional gauze 36.7%). Trauma was the main cause of diabetic foot ulcer (56.7%) in Calcium alginate group

while in conventional gauze group diabetic foot ulcers were most commonly spontaneous in onset (50%).

In this study it was observed that out of 60 patients, the diabetic foot ulcer was more commonly (68.3%) occurred over the dorsum of the foot as compared to plantar aspect (31.7%).

In the present study peripheral neuropathy was the most common complication associated with diabetic foot ulcer (Calcium alginate group 33.3% and in conventional gauze group 26.7%).

Infections in diabetic foot ulcer were polymicrobial in nature. In this study, most common organism isolated was E coli (29 cases), followed by pseudomonas aeroginosa (19 cases), staph aureus (9 cases), MRSA (9 cases) proteus mirabilis (8 cases) and others (28 cases) including Klebsiella pneumonia, klebsiella oxytoca, proteus vulgaris, citrobacter freundi and citrobacter diversus.

In this study the mean bacterial load (X $10^5$ CFU per gram of tissue) over diabetic foot ulcer before the first dressing in calcium alginate group was 513.3 ± 122.4 while in conventional gauze group it was 516.7 ± 117.7. The mean bacterial load diabetic foot ulcer was higher in conventional gauze group as compared to calcium alginate group before the dressing. However this difference was statistical not significance between the two groups (p=0.915). The mean bacterial load (X $10^5$ CFU per gram of tissue) over diabetic foot ulcer after the three consecutive dressings in calcium alginate group was 526.7 ± 138.8 while, in conventional gauze group it was 536.7 ± 121.7. The mean bacterial load after the

third dressing was higher in conventional gauze group as compared to calcium alginate. However this difference was statistically not significant (p=0.768).

The mean bacterial load (X $10^5$ CFU per gram of tissue) in calcium alginate group before the first dressing was 513.3 ± 122.4 and after the third dressing it was 526.7 ± 138.8. There was increase in bacterial load after the third dressing over the diabetic foot ulcer. However this increase in bacterial load was statistically not significant (p=0.221).

The mean bacterial load (X $10^5$ CFU per gram of tissue) in conventional gauze group before the first dressing was 516.7 ± 117.7 and after the third dressing it was 536.7 ± 121.7. There was increase in bacterial load after the third dressing over the diabetic foot ulcer. However this increase was not statistically significant (p=0.132).

In this study on comparing both the groups, increase in mean bacterial load (X $10^5$ CFU per gram of tissue) after the third dressing with conventional gauze was 20.00 ± 96.13 as compared to increase in mean bacterial load (X $10^5$ CFU per gram of tissue) 13.33 ± 93.71 with calcium alginate group. However this difference increase in mean bacterial load was statistically not significant (p=0.787).

An experimental study[5] has demonstrated the bacterial absorption and retaining ability of the calcium alginate over artificially created infected wound.

Another experimental study[6] has documented the bacterial retaining ability of calcium alginate dressing and supported its antibacterial property.

Another experimental study[7] has also supported the bacterial retaining property of calcium alginate dressings, and its passive mechanism for reducing the bacterial load over the wound and advocated for the in vivo study to explain the antibacterial effect of calcium alginate over wound in clinical surgical settings.

The difference in the results of previous experimental studies[5,6,7] and present study, may be due to moisture retaining property of calcium alginate itself, which promote the growth of bacteria in moist environment. To our knowledge this was the first randomized control trial which compared the effect of calcium alginate dressing on bacterial load in infected diabetic ulcer with conventional gauze dressing.

It must be emphasized that other previous studies[5,6,7] were over artificially created infected wound. However this present study was conducted over infected ulcer in patients with diabetes who have tissue hypoxia along with diminished phagocytic response of the neutrophils and macrophages, along with diminished neovascualrization, all of which greatly contribute to poor control of infection.

Also a recent randomized controlled trial[60] has concluded that the occlusive moist environment dressing (Calcium alginate) principle in the clinical surgical setting does not lead to quicker wound healing and it is not cost effective.

**Limitation of this study**

The limitations of the present study were short period of intervention to derive the conclusion and smaller sample size.

**Recommendation**

Further studies on larger population with longer duration of intervention may emphasize better interpretation of antibacterial property of calcium alginate dressing as compared to conventional gauze dressing.

# CONCLUSION

The present study has shown that dressing with calcium alginate is ineffective in reducing the bacterial load of the infected diabetic foot ulcers. This study has also shown that conventional gauze dressing can be used in patients with diabetic foot ulcers thereby reducing the cost to the patients.

# SUMMARY

Diabetic foot ulcer is frequently seen complication in patients with diabetes. Infection is one of the important factor for delay healing of diabetic foot ulcer. Dressing of foot ulcer is an important part of its management for local control of infection which will accelerate the healing. Various dressings like, occlusive and non occlusive group are available for the dressing of diabetic foot ulcers.

The objectives of the present study was to measure the effectiveness of calcium alginate dressing on bacterial load in infected diabetic foot ulcer in comparison to conventional gauze.

A total of 60 patients with infected diabetic foot ulcers (bacterial load more than 1 x $10^5$ CFU per gram of tissue) were studied. The patients were divided into two groups of 30 each. Group 1 patients were dressed with calcium alginate while other group patients with conventional gauze dressing. Bacterial load before the first dressing and after the third dressing was determined in one gram of ulcer tissue in both the groups. A comparative study was done between both groups regarding change in bacterial load before and after the dressing.

In this study male preponderance was seen in both the groups. Patient between age group of 56 to 65 years were most commonly affected. The duration of DM was 6 to 10 years in majority of the patients. Trauma was the main cause of onset of diabetic foot ulcer in group 1 while in group 2 the diabetic foot ulcers were more commonly spontaneous in onset. Dorsum aspect of the foot was most

often affected. Peripheral neuropathy was the most common diabetes associated foot complication.

In this present study it was observed that in both the groups, diabetic foot ulcers were heavily infected. Mean bacterial load (X $10^5$ CFU per gram of tissue) before the first dressing over ulcer in calcium alginate group was 513.3 ± 122.4 while in conventional gauze group it was 516.7 ± 117.7. There was increase in mean bacterial load (X $10^5$ CFU per gram of tissue) over the ulcer after three consecutive dressings in both the groups. The increase in mean bacterial load (X $10^5$ CFU per gram of tissue) was 13.33 ± 93.71 in calcium alginate group and 20 ± 96.13 in conventional gauze group. However this increase was statistically not significant ($p > 0.05$).

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# ANNEXURE I

## CONSENT FORM

**A randomized control trial to assess the efficacy of calcium alginate dressing versus conventional gauze dressing on bacterial load in infected diabetic foot ulcer**

Principal Investigator - **Dr. Rakesh Kumar Pandey**

You are being asked to be a subject in a research study of "**A randomized control trial to assess the efficacy of calcium alginate dressing versus conventional gauze dressing on bacterial load in infected diabetic foot ulcer**" at KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belgaum between Jan-2008 and Dec-2008 conducted by Dr. Rakesh Kumar Pandey, Post Graduate student in General Surgery at Jawaharlal Nehru Medical College, Belgaum.

Your participation in this research is your voluntary decision whether or not to participate will not affect your current or future relationship with KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belgaum. If you decide to participate, you are free to withdraw at any time.

The purpose of research is to compare the efficacy of two different types of dressing over diabetic foot in reduction of bacterial count.

**Procedures involved**

Two different type of dressings will be used in this study. Either of these two dressing will be applied over your ulcer depending upon which study group you belong. Later change in bacterial count in the ulcer will be assessed by taking small biopsy from the floor of the ulcer before and after the three dressings.

**Risk and benefits**

There is no risk involved in the procedure and likely benefits being faster healing of the ulcer.

**Alternatives**

Even if you decline the participation you will get the routine line of management.

**Privacy and confidentiality**

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

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

**Authorization to publish results**

When the results of the research are published or discussed in conferences no information will be disclosed that would disclose your identity. Any information that is obtained in connection with this study and that can be identified with you will remain confidential and will be disclosed only with your permission or if required by law.

**Financial incentives for participation**

You will not be paid / offered any free gifts for participating in the research. You will not be reimbursed for expenses.

**Consent statement**

I undersigned _____ have been explained in my vernacular language about the study and my participation in the study is voluntary. If I want, I can withdraw at any time. Also I have been given enough time to clear my doubts and rights as study participant.

In case you have any questions related to the study, you can contact Dr. Rakesh Kumar Pandey (Mobile No. 9916260291).

In case if you have any questions about your research subject, you may call Dr. V. D. Patil, Principal and Chairman, J. N. Medical College, Institutional Ethical Committee for Human Subjects Research, Phone No. 0831 2471350 at Jawaharlal Nehru Medical Co11ege, Belgaum.

Participants name _____

Signature _____

Witness Name _____

Signature _____

Experimenters Name _____

Signature _____

Date _____

Place _____

# ANNEXURE II - PROFOMA

## I. Patient identification data

Name : IP No. :

Age : DOA :

Sex : DOD :

Occupation :

Address :

## II. Chief complaints

## III. Medical history

Peripheral neuropathy : ( )

Nephropathy : ( )

Retinopathy : ( )

PVD : ( )

CVD : ( )

## IV. Diabetic status

Type : Duration :

Medication:

Oral Hypoglycemics : ( )

Insulin : ( )

Complication:

Neuropathy : ( )

Vasculopathy : ( )

**V. Ulcer detail**

1. Mode of onset

Traumatic : ( )

Spontaneous : ( )

Pressure : ( )

Others : ( )

2. Duration

3. Progress

**VI. Wound observation**

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

**VII. Neurological examination**

**VIII. Vascular examination** Left Right

Popliteal artery

Anterior Tibial artery

Posterior Tibial artery

Dorsalis Pedis artery

**IX. Any foot deformity present**

Toe deformity

Bunion

Charcots foot

Foot drop

**X. If amputation has been done**

Specify, Date :

Side :

Level :

Cause for amputation :

**XI. Investigation**

CBC

FBS Date Time

Blood Urea Sr. Creatinine

UKB

HbA1c

Urine:

Routine

Microscopy

X-ray Foot (If indicated)

Antero Posterior view

Lateral View

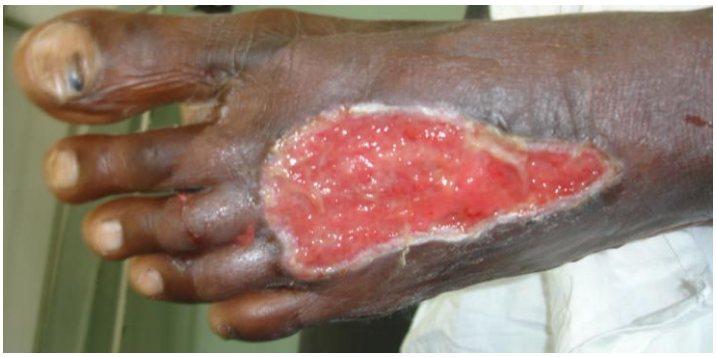
Tissue culture/sensitivity and bacterial load

Before first dressing

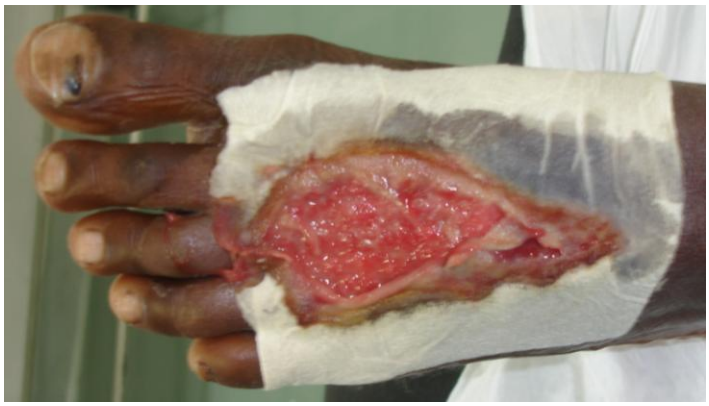
After third dressing

# ANNEXURE III - PHOTOGRAPHS


**Photograph 1: Calcium alginate**

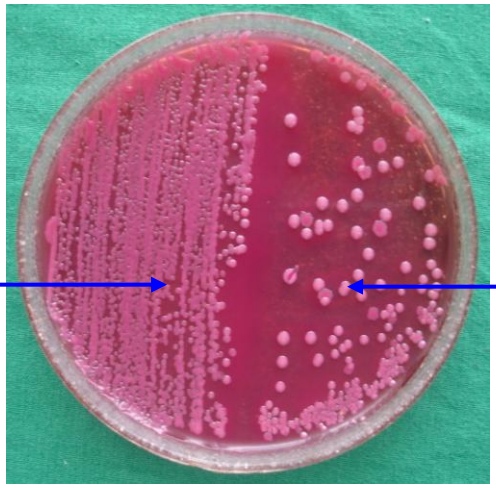


**Photograph 2: Diabetic foot ulcer**

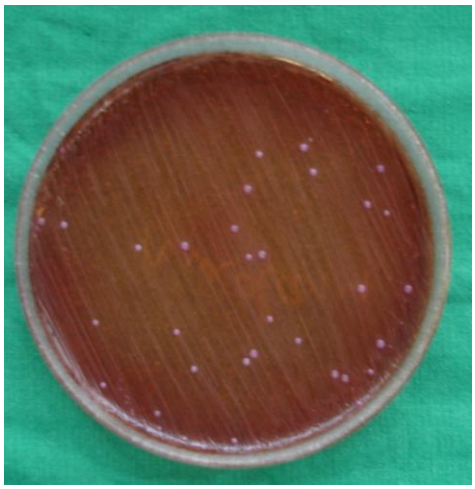


**Photograph 3: Calcium alginate dressing over diabetic foot ulcer**


**Photograph 4: Glucose broth (5 ml)**




**Photograph 5: Bacterial colonies over MacConkey agar**



**Photograph 6: Two different bacterial colonies over blood agar**

# ANNEXURE IV MASTER CHART – GROUP 1

# GROUP 2

| Sl. No. | IP No. | Gender | Age (Years) | Complications | | | | DM | | Ulcer details | | | | | | Investigations | | | | | | | | | | | Outcome | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | | Peripheral neuropathy | Vasculopathy | Foot deformity | Amputation | Duration (Years) | Medication | Onset | Duration (Days) | Side | Site | Size ($cm^2$) | Discharge | Haemoglobin (gm%) | TLC (/cmm) | FBS (mg/dL) | HbA1c (%) | Sr. Creat (mg/dL) | Urine albumin | X-ray | Bacterial load | | Type of bacteria | Bacterial load change ($10^5$/gm tissue) | Bacterial load | Percentage |
| | | | | | | | | | | | | | | | | | | | | | | | Before dressing ($10^5$ /gm tissue) | After 3rd dress. ($10^5$ /gm tissue) | | | | |
| 1 | 340047 | M | 62 | - | - | - | - | 10 | O | S | 10 | R | D | 4x4 | + | 11.0 | 8400 | 134 | 6.3 | 1 | - | N | 200 | 100 | EC,KP,KO | -100 | IN | -50.0 |
| 2 | 257189 | M | 65 | + | - | TD | GTD | 16 | IO | PR | 22 | R | P | 3x2 | + | 10.0 | 4900 | 122 | 7.0 | 0.9 | ++ | N | 600 | 600 | STP, KP | 0 | U | 0.0 |
| 3 | 262184 | F | 52 | - | - | - | - | 8 | O | T | 19 | L | P | 5x4 | + | 11.0 | 7000 | 149 | 7.2 | 0.7 | - | N | 600 | 600 | PA | 0 | U | 0.0 |
| 4 | 339949 | M | 36 | - | - | - | - | 5 | O | T | 18 | R | D | 5x5 | + | 12.0 | 5800 | 135 | 6.8 | 1.1 | - | N | 600 | 600 | PM,KP | 0 | U | 0.0 |
| 5 | 264050 | M | 50 | + | - | - | - | 6 | O | PR | 20 | R | P | 4x2 | + | 14.0 | 10000 | 160 | 7.2 | 1.5 | - | N | 600 | 600 | KP, CF | 0 | U | 0.0 |
| 6 | 271455 | M | 60 | - | - | - | - | 16 | I | T | 45 | R | D | 6x5 | + | 12.0 | 6800 | 128 | 6.4 | 1.8 | - | N | 600 | 600 | PM | 0 | U | 0.0 |
| 7 | 262663 | M | 59 | - | - | - | - | 12 | O | T | 25 | L | D | 7x4 | + | 13.0 | 7800 | 120 | 7.0 | 0.8 | - | N | 600 | 600 | EC,PA | 0 | U | 0.0 |
| 8 | 270587 | M | 60 | - | - | - | - | 14 | O | T | 5 | R | D | 8x6 | + | 12.0 | 8600 | 164 | 7.9 | 1.2 | + | N | 500 | 600 | PM | 100 | IN | 20.0 |
| 9 | 338935 | M | 65 | - | - | - | - | 20 | I | T | 20 | R | D | 4x4 | + | 14.0 | 9000 | 136 | 6.9 | 1.3 | - | N | 600 | 600 | EC,KP,PV | 0 | U | 0.0 |
| 10 | 278839 | M | 52 | - | - | - | GTD | 7 | O | S | 6 | L | D | 5x3 | + | 14.0 | 10500 | 138 | 7.5 | 1 | - | N | 400 | 600 | EC | 200 | IN | 50.0 |
| 11 | 282131 | M | 52 | + | - | - | - | 10 | O | T | 7 | L | P | 6x4 | + | 13.0 | 11000 | 110 | 7.8 | 1.1 | - | N | 600 | 600 | EC, PA | 0 | U | 0.0 |
| 12 | 282898 | M | 65 | + | - | TD | - | 16 | I | PR | 40 | L | P | 6x5 | + | 10.0 | 9400 | 146 | 7.2 | 0.8 | + | N | 600 | 600 | EC | 0 | U | 0.0 |
| 13 | 272139 | F | 55 | - | - | - | - | 14 | O | S | 4 | L | D | 5x5 | + | 10.0 | 8700 | 154 | 7.5 | 0.9 | - | N | 300 | 400 | EC | 100 | IN | 33.3 |
| 14 | 272192 | M | 55 | - | - | TD | - | 8 | O | T | 50 | R | D | 7x4 | + | 9.0 | 4800 | 130 | 6.8 | 1.4 | - | N | 600 | 600 | EC | 0 | U | 0.0 |
| 15 | 328811 | M | 60 | - | - | - | - | 18 | I | T | 10 | R | D | 6x5 | + | 12.0 | 5600 | 134 | 6.6 | 1.1 | - | N | 300 | 500 | STP,PA,KP | 200 | IN | 66.7 |
| 16 | 328976 | M | 59 | - | - | - | - | 10 | I | S | 9 | L | D | 5x5 | + | 11.0 | 9000 | 120 | 6.8 | 1.4 | - | N | 500 | 400 | CD,KP | -100 | DE | -20.0 |
| 17 | 333884 | M | 63 | - | - | - | STD | 8 | O | T | 14 | L | D | 7x4 | + | 12.0 | 10500 | 100 | 7.8 | 1.3 | - | N | 400 | 600 | EC,KO | 200 | IN | 50.0 |
| 18 | 340537 | M | 50 | - | - | - | - | 8 | O | S | 14 | R | D | 8x5 | + | 14.0 | 8400 | 160 | 7.7 | 0.6 | - | N | 400 | 600 | CF,MRSA | 200 | IN | 50.0 |
| 19 | 337578 | M | 38 | + | - | TD | - | 3 | O | PR | 22 | R | P | 6x3 | + | 15.0 | 4800 | 148 | 6.9 | 1.6 | + | N | 600 | 600 | PA,PM | 0 | U | 0.0 |

| Sl. No. | IP No. | Gender | Age (Years) | Complications | | | | DM | | Ulcer details | | | | | | Investigations | | | | | | | | | | | Outcome | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | | Peripheral neuropathy | Vasculopathy | Foot deformity | Amputation | Duration (Years) | Medication | Onset | Duration (Days) | Side | Site | Size ($cm^2$) | Discharge | Haemoglobin (gm%) | TLC (/cmm) | FBS (mg/dL) | HbA1c (%) | Sr. Creat (mg/dL) | Urine albumin | X-ray | Bacterial load | | Type of bacteria | Bacterial load change ($10^5$/gm tissue) | Bacterial load | Percentage |
| | | | | | | | | | | | | | | | | | | | | | | | Before dressing ($10^5$ /gm tissue) | After 3rd dress. ($10^5$ /gm tissue) | | | | |
| 20 | 327870 | M | 40 | - | - | - | - | 3 | I | T | 20 | L | D | 4x4 | + | 11.0 | 11500 | 128 | 7.0 | 0.7 | + | N | 600 | 600 | STP | 0 | U | 0.0 |
| 21 | 328334 | F | 65 | - | - | TD | - | 11 | O | T | 13 | L | P | 5x5 | + | 13.0 | 9600 | 148 | 7.0 | 0.8 | - | N | 400 | 300 | EC,PA | -100 | DE | -25.0 |
| 22 | 338800 | M | 39 | - | - | - | - | 1 | I | T | 13 | L | D | 4x4 | + | 12.0 | 6000 | 104 | 6.7 | 1.3 | - | N | 500 | 400 | MRSA | -100 | DE | -20.0 |
| 23 | 280173 | M | 47 | - | - | - | - | 5 | I | T | 12 | R | D | 10x8 | + | 14.0 | 5800 | 96 | 6.8 | 1.2 | - | N | 400 | 200 | PM | -200 | DE | -50.0 |
| 24 | 338969 | M | 55 | - | - | - | - | 6 | O | S | 10 | R | D | 7x6 | + | 10.0 | 4600 | 134 | 7.6 | 1.3 | - | N | 600 | 600 | MRSA | 0 | U | 0.0 |
| 25 | 338440 | F | 60 | - | - | - | - | 20 | I | T | 8 | L | D | 6x4 | + | 11.0 | 11500 | 156 | 7.7 | 1.1 | - | N | 600 | 600 | CD,KP | 0 | U | 0.0 |
| 26 | 334720 | M | 65 | + | - | TD | LTD | 3 | O | PR | 24 | L | P | 5x4 | + | 10.0 | 9800 | 168 | 6.4 | 1.6 | ++ | N | 300 | 300 | EC,PM | 0 | U | 0.0 |
| 27 | 340486 | M | 45 | - | - | - | - | 3 | I | T | 16 | L | D | 8x4 | + | 13.0 | 9300 | 126 | 7.3 | 0.7 | - | N | 600 | 600 | PA | 0 | U | 0.0 |
| 28 | 335529 | F | 35 | - | - | - | - | 10 | O | T | 6 | R | P | 4x2 | + | 9.0 | 7600 | 145 | 7.3 | 1 | + | N | 600 | 600 | STP | 0 | U | 0.0 |
| 29 | 332271 | M | 40 | - | - | - | - | 2 | I | S | 4 | R | D | 8x6 | + | 14.0 | 7000 | 122 | 7.8 | 1.2 | - | N | 600 | 600 | EC,PA | 0 | U | 0.0 |
| 30 | 341618 | M | 62 | - | - | - | - | 10 | O | S | 5 | L | D | 6x4 | + | 12.0 | 6800 | 156 | 7.6 | 1.1 | - | N | 600 | 600 | EC,KP,PA | 0 | U | 0.0 |

| Sl. No. | IP No. | Gender | Age (Years) | Complications | | | | DM | | Ulcer details | | | | | | Investigations | | | | | | | | | | | Outcom | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | | Peripheral neuropathy | Vasculopathy | Foot deformity | Amputation | Duration (Years) | Medication | Onset | Duration (Days) | Side | Site | Size ($cm^2$) | Discharge | Haemoglobin (gm%) | TLC (/cmm) | FBS (mg/dL) | HbA1c (%) | Sr. Creat (mg/dL) | Urine albumin | X-ray | Bacterial load | | Type of bacteria | Bacterial load change ($10^5$/gm tissue) | Bacterial load |
| | | | | | | | | | | | | | | | | | | | | | | | Before dressing ($10^5$/gm tissue) | After 3rd dress. ($10^5$/gm tissue) | | | |
| 1 | 286347 | F | 65 | - | - | - | - | 15 | I | T | 4 | L | D | 8x5 | + | 12.0 | 5900 | 140 | 7.4 | 0.9 | - | N | 600 | 600 | EC, KP | 0 | U |
| 2 | 337766 | M | 42 | - | - | - | - | 4 | I | T | 7 | L | D | 5x3 | + | 11.5 | 9800 | 124 | 7.8 | 1 | - | N | 600 | 600 | EC,KO | 0 | U |
| 3 | 288478 | F | 58 | - | - | - | - | 11 | I | S | 12 | L | D | 4x4 | + | 12.5 | 10400 | 149 | 7.1 | 1.2 | - | N | 600 | 600 | STP | 0 | U |
| 4 | 266293 | M | 56 | - | - | TD | - | 10 | O | S | 20 | R | D | 7x4 | + | 10.0 | 11000 | 168 | 7.1 | 1.1 | - | N | 600 | 600 | PM | 0 | U |
| 5 | 293366 | M | 50 | - | - | - | - | 8 | O | S | 15 | R | D | 8x5 | + | 11.0 | 9600 | 122 | 7.0 | 1.4 | - | N | 600 | 600 | EC, STP | 0 | U |
| 6 | 259122 | M | 60 | + | - | TD | - | 12 | O | PR | 30 | L | P | 2x2 | + | 10.0 | 8500 | 156 | 7.2 | 0.8 | + | N | 600 | 600 | EC, PM | 0 | U |
| 7 | 274046 | M | 50 | - | - | - | - | 7 | IO | T | 12 | L | D | 9x5 | + | 12.0 | 7800 | 138 | 7.5 | 1.2 | - | N | 600 | 600 | EC | 0 | U |
| 8 | 340729 | M | 41 | - | - | - | - | 5 | O | S | 13 | R | D | 10x8 | + | 13.5 | 7600 | 128 | 6.4 | 0.8 | - | N | 500 | 400 | STP | -100 | DE |
| 9 | 341148 | M | 60 | - | - | - | - | 10 | O | S | 10 | L | D | 4x2 | + | 11.0 | 11500 | 100 | 7.6 | 1.1 | - | N | 600 | 600 | MRSA,PA | 0 | U |
| 10 | 337984 | M | 50 | - | - | - | GTD | 12 | I | T | 8 | R | P | 5x5 | + | 10.0 | 10000 | 160 | 7.6 | 1.4 | + | N | 400 | 600 | EC,KP | 200 | IN |
| 11 | 275222 | M | 60 | + | - | - | - | 12 | O | PR | 38 | L | P | 4x2 | + | 12.0 | 5600 | 110 | 7.8 | 1.5 | - | N | 400 | 400 | EC | 0 | U |
| 12 | 275988 | F | 40 | - | - | - | - | 2 | O | T | 5 | R | D | 4x3 | + | 11.0 | 6000 | 148 | 7.4 | 1.6 | - | N | 300 | 600 | EC | 300 | IN |
| 13 | 267928 | M | 55 | - | - | - | - | 7 | O | T | 6 | L | P | 4x4 | + | 12.0 | 7400 | 150 | 7.3 | 1.1 | + | N | 600 | 600 | EC | 0 | U |
| 14 | 341600 | F | 55 | - | - | - | - | 7 | I | S | 7 | R | D | 8x4 | + | 10.0 | 8200 | 98 | 7.6 | 1 | - | N | 400 | 500 | KO,PA,EC | 100 | IN |
| 15 | 265717 | M | 61 | - | - | TD | - | 13 | IO | T | 12 | R | P | 4x3 | + | 14.0 | 7600 | 160 | 7.1 | 1.8 | ++ | N | 500 | 500 | CF | 0 | U |
| 16 | 334470 | M | 40 | - | - | - | - | 3 | O | S | 15 | L | D | 9x4 | + | 13.0 | 8400 | 124 | 6.4 | 1.6 | - | N | 600 | 600 | EC | 0 | U |
| 17 | 340370 | F | 42 | - | - | - | - | 3 | I | S | 5 | R | D | 7x5 | + | 11.0 | 9600 | 128 | 7.2 | 1 | - | N | 600 | 600 | MRSA | 0 | U |
| 18 | 342562 | M | 65 | + | - | TD | STD, TTD | 18 | O | PR | 45 | R | P | 5x4 | + | 12.0 | 10500 | 148 | 6.5 | 0.9 | ++ | N | 300 | 300 | KO, KP, MRSA | 0 | U |
| 19 | 339934 | M | 55 | - | - | - | - | 10 | I | S | 10 | R | D | 6x3 | + | 12.0 | 6400 | 144 | 6.4 | 0.8 | - | N | 600 | 600 | PA | 0 | U |

| Sl. No. | IP No. | Gender | Age (Years) | Complications | | | | DM | | Ulcer details | | | | | | Investigations | | | | | | | | | | Outcom | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | | Peripheral neuropathy | Vasculopathy | Foot deformity | Amputation | Duration (Years) | Medication | Onset | Duration (Days) | Side | Site | Size ($cm^2$) | Discharge | Haemoglobin (gm%) | TLC (/cmm) | FBS (mg/dL) | HbA1c (%) | Sr. Creat (mg/dL) | Urine albumin | X-ray | Bacterial load | | Type of bacteria | Bacterial load change ($10^5$/gm tissue) | Bacterial load |
| | | | | | | | | | | | | | | | | | | | | | | | Before dressing ($10^5$/gm tissue) | After 3rd dress. ($10^5$/gm tissue) | | | |
| 20 | 295203 | M | 48 | - | - | - | - | 8 | O | S | 12 | L | D | 5x2 | + | 11.0 | 9800 | 136 | 6.7 | 0.7 | - | N | 200 | 200 | EC, CF | 0 | U |
| 21 | 337464 | F | 65 | - | - | - | - | 20 | O | T | 8 | R | D | 4x3 | + | 13.0 | 10800 | 128 | 6.8 | 1.1 | - | N | 400 | 200 | KO,STP,PA | -200 | DE |
| 22 | 341048 | F | 62 | - | - | - | - | 12 | IO | T | 11 | L | P | 5x5 | + | 12.0 | 10000 | 104 | 7.4 | 1.1 | - | N | 600 | 600 | PA | 0 | U |
| 23 | 341137 | M | 40 | - | - | - | - | 6 | O | S | 14 | R | D | 8x5 | + | 11.0 | 8200 | 100 | 7.4 | 1.2 | - | N | 600 | 600 | EC,PA | 0 | U |
| 24 | 340594 | M | 65 | + | - | TD | GTD | 18 | O | T | 20 | R | P | 4x2 | + | 14.0 | 8000 | 140 | 7.3 | 1.6 | - | N | 600 | 600 | MRSA,KP | 0 | U |
| 25 | 340195 | M | 62 | - | - | - | - | 14 | I | S | 8 | L | D | 9x4 | + | 12.0 | 6600 | 178 | 6.8 | 1.2 | - | N | 600 | 600 | MRSA,KP,PA | 0 | U |
| 26 | 263488 | M | 45 | - | - | - | - | 6 | O | S | 11 | L | D | 8x4 | + | 13.0 | 7600 | 176 | 7.0 | 1.3 | - | N | 400 | 600 | STP | 200 | IN |
| 27 | 337094 | M | 37 | - | - | - | - | 2 | O | T | 20 | R | P | 4x4 | + | 12.0 | 9200 | 156 | 7.2 | 1.4 | - | N | 600 | 600 | KP | 0 | U |
| 28 | 337477 | M | 65 | - | - | TD | - | 16 | O | S | 12 | R | D | 7x6 | + | 15.0 | 9800 | 100 | 6.2 | 1.5 | - | N | 500 | 400 | MRSA,PA | -100 | DE |
| 29 | 221650 | F | 35 | - | - | - | - | 1 | I | S | 7 | R | D | 6x3 | + | 14.0 | 10200 | 98 | 7.3 | 1.1 | - | N | 600 | 600 | PA, EC | 0 | U |
| 30 | 280155 | M | 53 | - | - | - | FTD | 10 | O | T | 6 | R | P | 3x2 | + | 13.0 | 8800 | 180 | 7.6 | 1 | - | N | 400 | 600 | EC, PA | 200 | IN |

| ıe |
| --- |
| Percentage |
| 0.0 |
| 0.0 |
| 0.0 |
| 0.0 |
| 0.0 |
| 0.0 |
| 0.0 |
| -20.0 |
| 0.0 |
| 50.0 |
| 0.0 |
| 100.0 |
| 0.0 |
| 25.0 |
| 0.0 |
| 0.0 |
| 0.0 |
| 0.0 |
| 0.0 |

| ıe |
| --- |
| Percentage |
| 0.0 |
| -50.0 |
| 0.0 |
| 0.0 |
| 0.0 |
| 0.0 |
| 50.0 |
| 0.0 |
| -20.0 |
| 0.0 |
| 50.0 |

# ANNEXURE IV

## KEY TO MASTER CHART

| | | |
|---|---|---|
| CD | : | Citrobacter Diversus |
| CF | : | Citrobacter Freundii |
| D | : | Dorsum |
| DE | : | Decreased |
| DM | : | Diabetes Mellitus |
| EC | : | Escherichiae Coli |
| F | : | Female |
| FBS | : | Fasting Blood Sugar |
| FTD | : | Fourth Toe Disarticulation |
| GTD | : | Great Toe Disarticulation |
| $HbA_{1C}$ | : | Glycosylated haemoglobin |
| I | : | Insulin |
| IN | : | Increased |
| IO | : | Oral hypoglycaemic drug + Insulin |
| IP No | : | In-Patient Number |
| KO | : | Klebsiella Oxytoca |
| KP | : | Klebsiella Pneumoniae |
| L | : | Left |
| M | : | Male |
| N | : | Normal |
| O | : | Oral hypoglycaemic Drug |

| | | |
|---|---|---|
| P | : | Plantar |
| PA | : | Pseudomonas Aeruginosa |
| PM | : | Proteus Mirabilis |
| PR | : | Pressure |
| PV | : | Proteus vulgaris |
| R | : | Right |
| S | : | Spontaneous |
| Sl. No. | : | Serial Number |
| Sr. Creat | : | Serum Creatinine |
| STD | : | Second Toe Disarticulation |
| STP | : | Staphylococcus Aureus |
| T | : | Traumatic |
| TD | : | Toe Deformity |
| TLC | : | Total Leucocyte count |
| TTD | : | Third Toe Disarticulation |
| U | : | Unchanged |