# "A COMPARATIVE STUDY BETWEEN PRESSURE OFFLOADING AND CONVENTIONAL DRESSING IN MANAGEMENT OF DIABETIC PLANTAR FOOT ULCERS"

**By**

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In partial fulfilment of the requirements for the degree of

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**IN**

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***Under the guidance of***

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**DR. MRINAL KUMAR V**

## LIST OF ABBREVIATIONS USED

| | |
|---|---|
| DM | : Diabetes Mellitus |
| IDF | : International diabetic federation |
| DFU | : Diabetic foot ulcer |
| NPWT | : Negative pressure wound therapy |
| HBO | : Hyperbaric oxygen therapy |
| NIDDK | : National Institute of diabetes and digestive and kidney diseases |
| QoL | : Quality of life |
| TCC | : Total contact cast |
| PAD | : Peripheral arterial disease |
| MTP | : Metatarso phalangeal joint |
| DCCT | : Diabetes control and complication trial |
| UKPDS | : United Kingdom prospective diabetes study |
| LEA | : Local education authority, UK |
| HBAIC | : Glycohaemoglobin |
| CBC | : Complete blood count |
| ESR | : Erythrocyte sedimentation rate |
| MDP | : Methylene disphosphonate |
| HMPAO | : Hexamethylepropyleneaminoxime |
| PET | : Positron emission topography |
| MRI | : Magnetic resonance imaging |
| TcPO2 | : Transcutaneous oxygen tension |
| ABI | : Ankle Brachial Pressure index |
| CTA | : Computed topography angiography |

| | |
|---|---|
| DSA | : Digital subtraction angiography |
| PTB | : Positive probe to bone |
| UTSA | : University of Texas San Antonio |
| USG | : Ultrasonography |
| TIA | : Transient Ischemic attack |
| MI | : Myocardial infarction |
| MIST | : Minimally invasive surgical trainer |
| FDA | : Food and drug administration |
| VEGF | : Vascular endothelial growth factor |
| FGF | : Fibroblast growth factor |
| KGF | : Keratinocyte growth factor |
| VAC | : Vacuum assisted closure |
| RCW | : Removable cast walker |
| SD | : Standard deviation |

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# INTRODUCTION

Diabetes mellitus (DM) is one of the main problems in health systems and a global public health threat that has increased dramatically over the past 2 decades[1,2]. According to epidemiological studies, the number of patients with DM increased from about 30 million cases in 1985, 177 million in 2000, 285 million in 2010, and estimated if the situation continues, more than 360 million people by 2030. The total number of people in India with diabetes is estimated to be around 50.8 million in 2010, rising to 87 million by 2030 according to the International Diabetes Federation (IDF) [5].

Diabetic foot lesions are actually a complex triad of neuropathy, ischemia and infections with plantar aspect of the foot being the most common site for ulceration.[15]

Recent studies have indicated multiple risk factors associated with the development of Diabetic Foot Ulcer (DFU)[22-25]. These risk factors are as follows: gender (male), duration of diabetes longer than 10 years, advanced age of patients, high Body Mass Index and other co-morbidities such as retinopathy, diabetic peripheral neuropathy, peripheral vascular disease, glycated haemoglobin level (HbA1C), foot deformity, high plantar pressure, infections and inappropriate foot self-care habits.[1,14,24-26]

Patients with DM are prone to multiple complications such as retinopathy, neuropathy, nephropathy, peripheral arterial disease and diabetic foot ulcer being a devastating chronic complication of Diabetes mellitus of them all. DFU is a common complication of DM that has shown an increasing trend over previous decades[7-9]. In total, it is estimated that 15% of patients with diabetes will suffer from DFU during

their lifetime[10]. Although accurate figures are difficult to obtain for the prevalence of DFU, the prevalence of this complication ranges from 4%-27%[10,11,14].

To date, DFU is considered as a major source of morbidity and a leading cause of hospitalization in patients with diabetes[1,7,16,18]. It is estimated that approximately 20% of hospital admissions among patients with DM are the result of DFU[19]. DFU can lead to infection, gangrene, amputation and even death if necessary care is not provided[19].

On the other hand, once DFU has developed, there is an increased risk of ulcer progression that may ultimately lead to amputation. Overall, the rate of lower limb amputation in patients with DM is 15 times higher than patients without diabetes.[10] It is estimated that approximately 50%-70% of all lower limb amputations are due to DFU.[10] Rough estimates are at about 1,00,000 lower limbs are amputated in India every year, of which atleast seventy five percent neuropathic feet with secondary infections and are potentially preventable.[17]

Diabetic foot ulcer commonly affects the toes followed by mid-foot. It usually starts with colonization of neuropathic or ischemic ulcers, traumatic wounds, small fissures between the toes or nail beds, wounds due to burns or chronic pressure. Diabetic foot ulcers can also develop secondary to cellulites, necrotizing fasciitis and abscess.[15]

In addition, it is reported that every 30 second one leg is amputated due to DFU in worldwide.[12] Furthermore, DFU is responsible for substantial emotional and physical distress as well as productivity and financial losses that lower the quality of life.[22]

The previous literature indicates that healing of a single ulcer costs approximately $17500 (1998 United States Dollars). In cases where lower extremity amputation is required, health care is even more expensive at $30000-33500.[23] These costs do not represent the total economic burden, because indirect costs related to losses of productivity, preventive efforts, rehabilitation, and home care should be considered. When all this is considered, 7%-20% of the total expenditure on diabetes in North America and Europe might be attributable to DFU.[24]

Various modalities of wound healing products are in use to treat diabetic foot ulcers like growth factors, skin substitutes, extracellular matrix protein, protease inhibitors, vasoactive compounds, platelet therapies etc. Techniques like Negative pressure wound therapy (NPWT), hyperbaric oxygen therapy (HBO), and Autologous bone marrow cultured cell are also being increasingly used[5]. But around 80% diabetic patients are neuropathic and diabetic patients with a history of foot ulceration have abnormally high pressure under the foot.[21] These have to be reduced to prevent further risk and damage to the foot. One possible solution to this is pressure offloading techniques.

Advantages of using any of the offloading techniques mainly in plantar aspect foot ulcers are such that, the covering prevents injury from heat, objects etc., the padding lessens the effect of muscle wasting and gives a soft surface to any hard bony projections, moulding mainly increases the weight bearing area and takes weight off the affected area and rigidity reduces the effect of shearing stress, stabilises the foot and corrects mobile deformities, ultimately leading to an ulcer free foot.

This study is undertaken to evaluate the safety and clinical efficacy of using pressure offloading techniques with the conventional type of dressing in management of diabetic plantar foot ulcers.

# OBJECTIVES OF THE STUDY

The purpose of the study is to evaluate the safety and clinical efficacy of using pressure offloading techniques with the conventional type of dressing in management of diabetic plantar foot ulcers.

# REVIEW OF LITERATURE

Diabetes is a chronic disease (International Diabetes Federation (IDF) 2012a) and according to IDF (2012b) projections in 2011 the prevalence of DM in the world was of 8.3% and it will increase to 9.90% by 2030. Furthermore, in 2011 the number of deaths related to DM was of 4.593.109 people (IDF 2012b)

DFUs are lesions characterised by a skin break involving loss of epithelium, which can extend through the dermis and deeper tissue, and in some cases involve muscle and even bone (Reiber et al. 1998, Boulton 2004b).

Although, neuropathy and PVD are the primary factors for the presentation of DFUs, other risk factors play an important role in the development, aggravation and healing outcomes of DFUs (Boyko et al. 1999, Reiber et al. 1999, Merza and Tesfaye 2003, Boulton 2004b, Lavery et al. 2008, Wu and Armstrong 2005).

Neuropathy results from continued peripheral nerve damage of motor, sensory and autonomic fibres, that affect sensation, innervations of the muscles of the foot and its circulation (Reiber et al. 1998, Jeffcoate and Harding 2003, Merza and Tesfaye 2003, Boulton 2004b, National Institute of Diabetes and Digestive and Kidney Diseases (NIDDK) 2009).

Motor neuropathy causes muscle wasting, atrophy and weakness which leads to foot deformities, such as claw and hammer toe that in turn predispose the individual to restricted joint mobility, balance problems and gait instability (Reiber et al. 1998, Merza and Tesfaye 2003, Boulton 2004b, Cavanagh et al. 2005, Singh et al. 2005).

Sensory neuropathy leads to decreased or loss of protective sensation to pain, pressure and loss of proprioception (inability to recognize the feet position) (Reiber et al. 1998, Merza and Tesfaye 2003, Boulton 2004b, van Deursen 2004).

The loss of protective sensation places the individual at risk of continuously harming the foot without realising it (Laing 1998, Wu and Armstrong 2005).

Autonomic neuropathy refers to altered micro vascular blood flow that results in warm feet, and decreased sweat production, resulting in dry skin, predisposing callus formation, which is hyperkeratosis that develops around the ulcer, and skin breakdown (Reiber et al. 1998, Merza and Tesfaye 2003, Boulton 2004b, Lavery et al. 2008).

Nevertheless, other factors also play an important role in the development of FUs. DFUs are an important health issue that needs to be addressed. It is fundamental to implement prevention and treatment practices that will improve individuals' Quality of life (QoL) and bring better cost-effectiveness for the health services.

Pressure plays a central role in the development of DFUs and its management becomes essential not only to prevent the development of new ulcers but also to allow the healing process to take place (Caravaggi et al. 2000, Armstrong et al. 2001, Reiber et al. 2002, van Deursen 2004, Piaggesi et al. 2007, Faglia et al. 2010).

Offloading is both a treatment and prevention intervention which relieves, reduces or redistributes plantar pressure to avoid the concentration of high pressures in DFUs, in the diabetic ulcer free foot and also to protect pressure points in the foot (Burden et al. 1983, Cavanagh et al. 2000, Cavanagh et al. 2005, Leung 2007, Edmonds et al. 2008:85). Besides managing plantar ulcers, offloading is also

important when the ulcer is located on the heel and on the lateral aspect of the midfoot and forefoot (Cavanagh et al. 2005).

Offloading is a central intervention in the treatment and management of DFUs (Reiber et al. 2002, Armstrong et al. 2005, Katz et al. 2005, Piaggesi et al. 2007, Faglia et al. 2010).

In terms of offloading devices there is a great variety available that can be used when there is an active ulcer or only when pressure redistribution is necessary. Some of the devices available are included in the following categories casts, therapeutic shoes, orthoses, felt padding and foam (Edmonds et al. 2008, Spencer 2008).

The different offloading devices available are all important for the treatment of DFUs. However, total contact cast (TCC) is considered the most effective device, as it cannot be removed easily by the individual allowing for better compliance (Caravaggi et al. 2000, Armstrong et al. 2001, Reiber et al. 2002, Jeffcoate and Harding 2003, Beuker et al. 2005, Leug 2007, Faglia et al. 2010).

Nonetheless, the consensuses around the best way of offloading DFUs is not well established yet as various authors recommend different devices for offloading DFUs (Caravaggi et al. 2000, Armstrong et al. 2001, Armstrong et al. 2005, Katz et al. 2005, Piaggesi et al. 2007, Faglia et al. 2010).

## ANATOMY OF FOOT

The human foot combines mechanical complexity and structural strength. The ankle serves as foundation, shock absorber and propulsion engine. The foot can sustain enormous pressure (several tons over the course of a one-mile run) and provides flexibility and resilience.

The foot and ankle contain:

- 26 bones
- 33 joints
- More than 100 muscles, tendons and ligaments
- A network of blood vessels, nerves, skin and soft tissue.

These components work together to provide the body with support, balance and mobility. A structural flaw or malfunction in any one part can result in the development of problem elsewhere in the body.

**IMAGE: 1 BONES - MEDIAL VIEW**

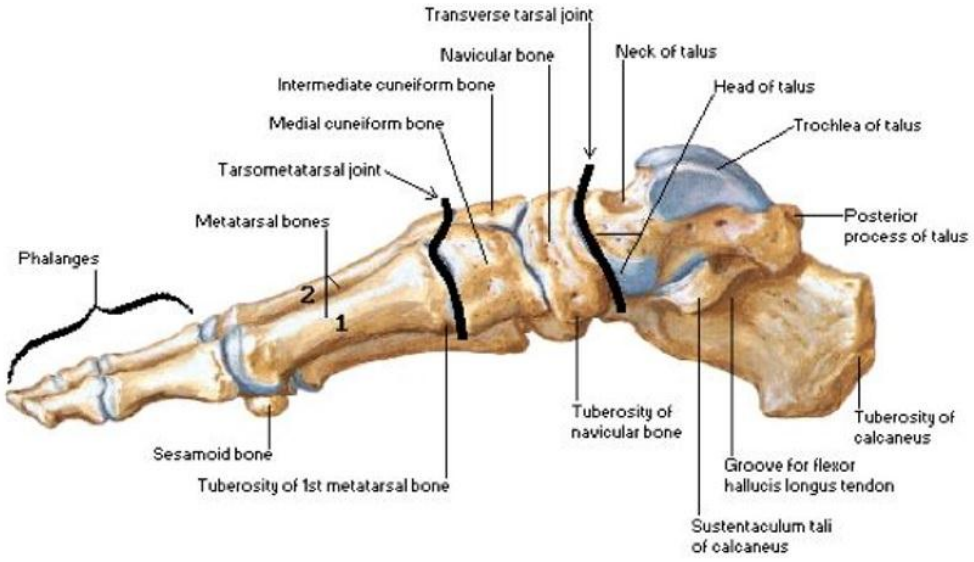
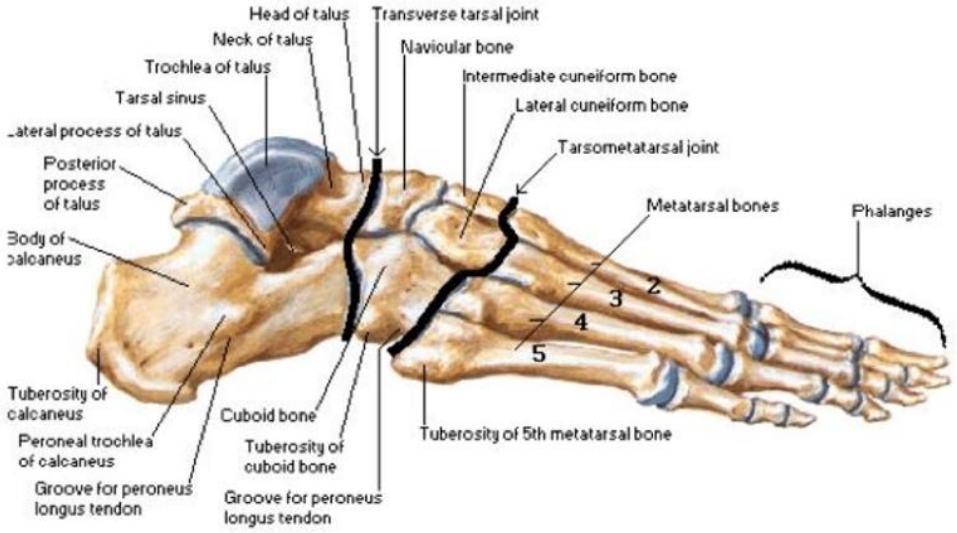


**IMAGE: 2 BONES - LATERAL VIEW**




**Skin**

The skin of dorsum of the foot (hirsute) is thin and highly flexible, containing hair follicles, sweat glands and scanty sebaceous gland. Hairs are sparse and thick. It is less than 2mm thick and few fibrous septa penetrate to deeper fascial structures. The plantar skin (glabrous) is 5mm thick especially over those points which bear weight viz. heel, ball of big toe and lateral margins of the sole. It has no hair follicles of sebaceous glands but sweat glands are numerous. Hypodermis is composed of loose areolar connective tissue, most of this is collagenous and elastic fibers running parallel to the surface of the skin, but some are continuous with the fibers of dermis. Hypodermis is well supplied with blood vessels and nerve endings. Tactile sensation is exceptionally good in the sole.

The subcutaneous tissue in the sole as in the palm differs from that of the rest of body in being more fibrous, tough and stingy. Fibrous septa divide the tissue into small loculi which are filled with fluid fat under tension this makes a shock absorbing pad especially over the heel and over the tips of toes.

**Deep fascia:**

On the dorsum of the foot (fascia dorsalis pedis) is the thin layer continuous above with the inferior extensor retinaculum and at the sides of the foot; it blends with plantar aponeurosis, anteriorly it en-sheathes the dorsal tendons.

**Plantar aponeurosis:**

Cover the whole length of the sole. It arises posteriorly from the medial and lateral tubercles of calcaneous from the back of that bone below the insertion of the tendo-calcaneous. It spreads out over the sole and is inserted by five slips into each of the five toes. A very dense and strong intermediate part is known as plantar aponeurosis.

**Parts of the Foot:**

Structurally, the foot has three main parts:

***The forefoot:***

Forefoot is composed of five toes (called phalanges) and their connecting long bones (metatarsals). Each toe (phalanx) is made up of several small bones. The big toe (hallux) has two phalanges, two joints (interphalangeal joints) and two tiny, round sesamoid bones that enable it to move up and down. The other four toes each have three bones and two joints. The phalanges are connected to the metatarsals by five metatarsal phalangeal joints at the ball of the foot. The forefoot bears half the body's weight and balances pressure on the ball of the foot.

***The Midfoot:***

Forms the foot's arch, and serves as a shock absorber. The bones of the midfoot are cuboid, first, second, third cuneiform and navicular connected to the forefoot and the hind foot by muscles and the plantar fascia.

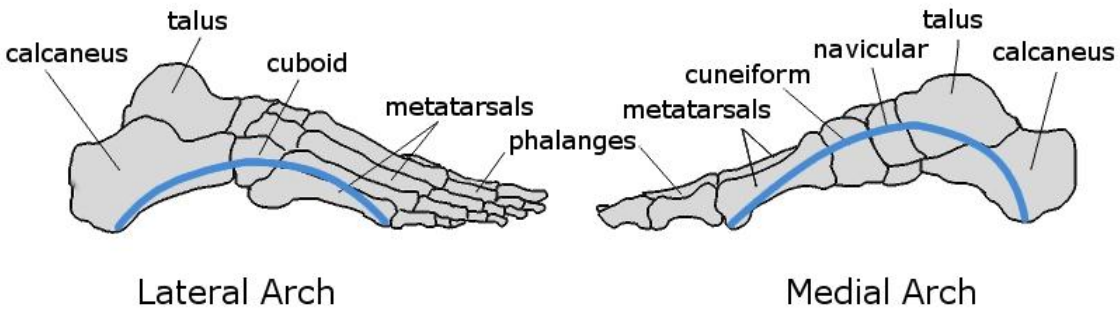
***The Hind foot:***

Hind foot is composed of three joints and links the midfoot to the ankle (talus). The top of the talus is connected to the two long bones of the lower leg (tibia and fibula), forming a hinge that allows the foot to move up and down. The heel bone (calcaneus) is the largest bone in the foot. It joints the talus to form the subtalar joint which enables the foot to rotate at the ankle. The bottom of the calcaneus is cushioned by a layer of fat.

**The Arches:**

The foot has three arches. The medial longitudinal arch is composed of the calcaneus, talus, navicular, cuneiforms, and the first three metatarsals. The lateral longitudinal arch is composed of the calcaneus, cuboid and the fourth and fifth metatarsals. The transverse arch is composed of the cuneiforms, the cuboid and the five metatarsal bones. The arches of the foot are maintained not only by the shapes of the bones as well as by ligaments. In addition, muscles and tendons play an important role in supporting the arches.

**IMAGE 3 : ARCHES OF FOOT**



**Muscles, Tendons and Ligaments:**

There are 20 muscles in the foot that give the foot its shape by holding the bones in position and expand and contract to impart movement. The muscles in the sole of the foot are categorized into four layers: Muscles in the first layer include Flexor digitorium brevis, Abductor hallucis and Abductor digiti minimi. In the second layer are tendon of Flexor hallucis longus, Flexor digitorum accessories and the Lumbricals. In the third layer are Flexor hallucis brevis, Adductor hallucis and Flexor digiti minimi brevis. In the fourth layer are peroneous longus tendon, Tendon of the tibialis posterior, 4 dorsal interossei and 3 plantar interossei.

**Arteries of the sole of the foot:**

***Medial plantar artery:***

This terminal branch of the posterior tibial artery arises beneath the flexor retinaculum. It ends by supplying the medial side of the big toe. During its course it gives off numerous muscular, cutaneous, and articular branches.

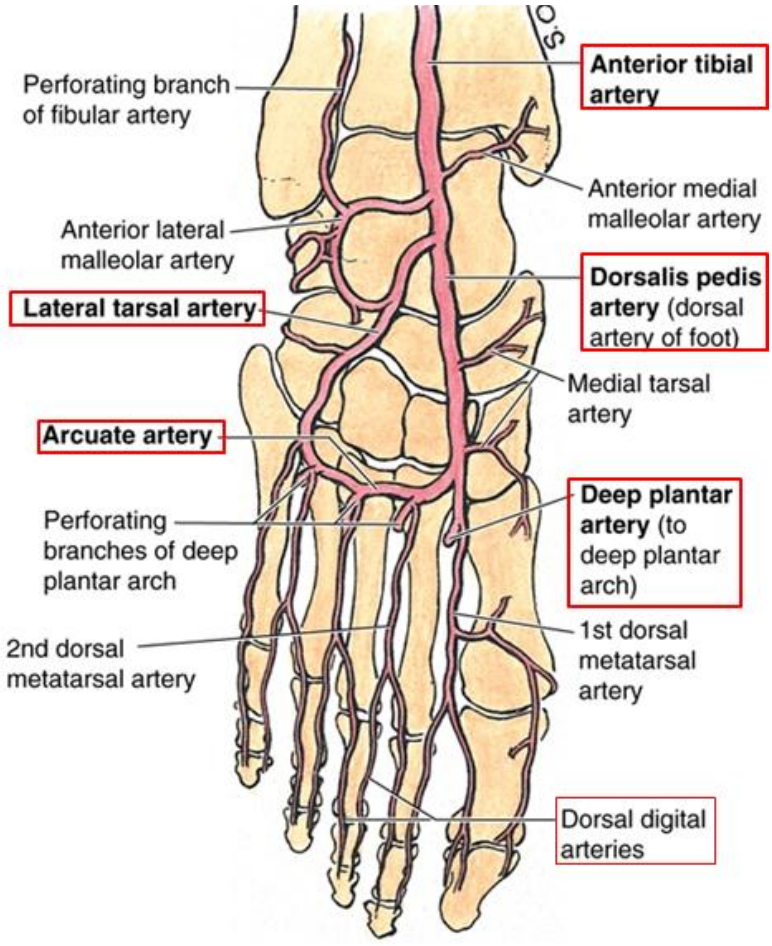
***Lateral Plantar Artery:***

Is the larger of the terminal branches of the posterior tibial artery. During its course, it gives off numerous muscular, cutaneous and articular branches. The plantar arch gives off plantar digital arteries to the adjacent sides of the lateral four toes and the lateral side of the little toe.

***Dorsalis Pedis Artery:***

On entering the sole between the two heads of the first dorsal interosseous muscle, the dorsalis pedis artery immediately joins the lateral plantar artery, gives the first plantar metatarsal artery, which supplies the cleft between the big and second toes.

**IMAGE 4 : ARTERIAL SUPPLY**




**(A) Dorsum of foot**

**Veins of the Sole of the Foot:**

Medial and lateral plantar veins accompany the corresponding arteries, and they unite behind the medial malleolus to form the posterior tibial venae comitantes.

**Nerves of the Sole of the Foot:**

***Medial Plantar Nerve:***

The medial plantar nerve is a terminal branch of the tibial nerve. It gives muscular branches to the abductor hallucis, the flexor digitorium brevis, the flexor hallucis brevis and the first lumbrical muscle. Cutaneous branches: Plantar digital nerves run to the sides of the medial three and one-half toes.

***Lateral Plantar Nerve:***

The lateral plantar nerve is a terminal branch of the tibial nerve.

Branches:

1. From the main trunk to the quadratic plantae and abductor digiti minimi; cutaneous branches to the skin of the lateral part of the sole.
2. From the superficial terminal branch to the flexor digiti minimi and the interosseous muscles of the fourth intermetatarsal space.
3. From the deep terminal branch supplies the abductor hallucis; the second, third and fourth lumbricals; and all the interossei, except those in the fourth intermetatarsal space.

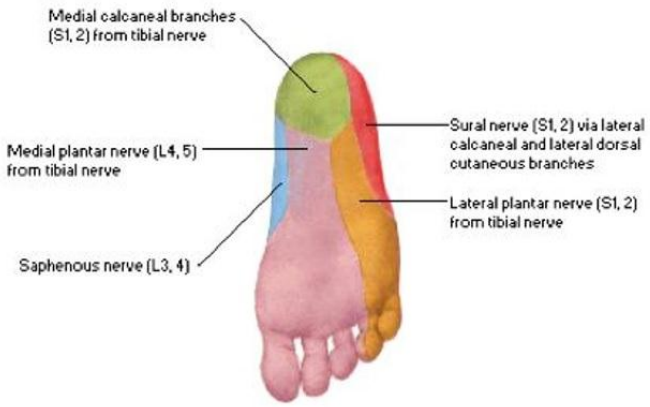
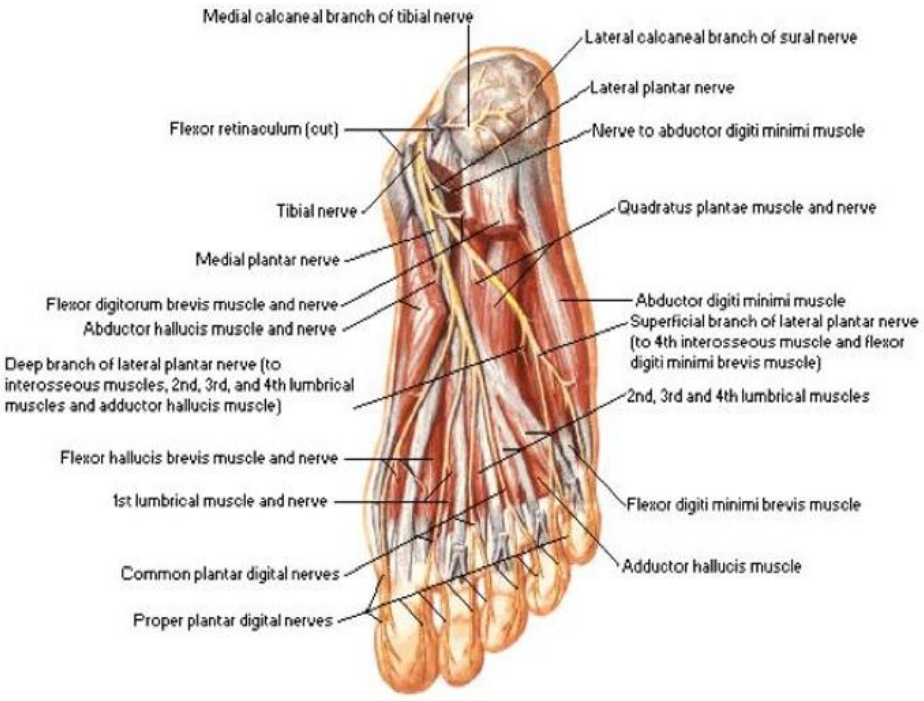


**IMAGE 5 : NERVE SUPPLY**




**Dorsal venous arch:**

The dorsal venous arch lies in the subcutaneous tissue over the heads of the metatarsal bones and drains on the medial side into the great saphenous vein. The great saphenous vein leaves the dorsum of the foot by the ascending into the leg in front of the medial malleolus. The small saphenous vein ascends into the leg behind the lateral malleolus.

**Artery of the dorsum of the foot:**

***Dorsalis Pedis Artery:***

The dorsalis pedis artery begins in front of the ankle joint as a continuation of the anterior tibial artery. It terminates by passing downward into the sole between the two heads of the first dorsal interosseous muscle, where it joins the lateral plantar artery and completes the plantar arch. The Branches are:

1. Lateral tarsal artery.
2. Arcuate artery
3. First dorsal metatarsal artery

***Nerve supply of the dorsum of the foot:***

***Deep Peroneal Nerve:***

It divides into terminal, medial and lateral branches. The medial branch supplies the skin of the adjacent sides of the big and second toes. The lateral branch supplies the extensor digitorium brevis muscle.

**Spaces of the Foot:**

Infections of the foot can be approached and drained effectively. Grodinsky has emphasized the clinical importance of the 4 median fascial spaces on the plantar aspect of the foot and the 2 dorsal spaces.

***Four median Plantar Spaces****:*

1. The first space is located between the plantar aponeurosis and the flexor digitorium brevis.
2. The second space is situated between the flexor digitorium brevis and the conjoined long flexor tendons and quadrates plantae.
3. The third space is found between the flexor digitorium longus (with its associated lumbricals muscles) and the oblique head of the abductor hallucis.
4. The fourth deepest space is situated between the oblique head of the abductor hallucis muscle and the 2nd and the 3rd metatarsal bones and their interosseous muscles.

These spaces are bound both laterally and medially by dense connective tissue septa an infection may travel from one space to another. The sheaths of the entire

flexor tendon extend from the toes and proximal to the distal head of the metatarsal bones; therefore within these sheaths either may remain local or break into one of the four spaces. The 3rd layer of the sole of the foot is enclosed inferiorly by the plantar fascia and superiorly by the metatarsal and small muscles and ligaments of the foot. It is continuous distally into the through the lumbricals and web space along with the long flexor tendons.

**Propulsive action of the foot:**

***Standing immobile:***

The body weight is disturbed via the heel behind and the heads of the metatarsal bones in the front.

***Walking:***

As the body weight is thrown forward, the weight is borne successively on the lateral margin of the foot and the heads of the metatarsal bones. As the heel rises the toes are extended at the metatarso-phalangeal joints and the plantar aponeurosis is pulled on thus heightening the longitudinal arches. The body is then thrown forwards

1. By the actions of the gastroenemius and soleus (and plantaris) on the ankle joint, using foot as a lever.
2. By the toes being strongly flexed by the long and short flexors of the foot, providing the final thrust forward.

The lumbricals and interossei contract and keep the toes extended so that they do not fold under pressure because of the strong action of the flexor digitorium longus. In this action the long flexor tendons also assist in Plantar flexing the ankle joint.

## RISK FOR ULCERATION

Foot ulceration is the most common single precursor to lower extremity amputations among persons with diabetes [2,5]. Treatment of infected foot wound comprises up to one quarter of all diabetic hospital admissions in the US and Britain, making this the most common reason for diabetes-related hospitalization in these countries. The multi-factorial nature of the diabetic foot ulceration has been elucidated by numerous observational studies. Risk factors identified include peripheral neuropathy, vascular disease, limited joint mobility, foot deformities, abnormal foot pressures, minor trauma, a history of ulceration or amputation, and impaired visual acuity.

Peripheral sensory neuropathy in the face of unperceived trauma is the primary factor leading to diabetic foot ulceration. Approximately 45% to 60% of all diabetic ulcerations are purely neuropathic, while up to 45% have neuropathic and ischemic components. According to an important prospective multicenter study, sensory neuropathy was the most frequent component in the causal sequence to ulceration in diabetic patients [2,5].

Other forms of neuropathy may also play a role in foot ulceration. Motor neuropathy resulting in anterior crural muscle atrophy or intrinsic muscle wasting can lead to foot deformities such as foot drop, equinus, hammertoe, and prominent plantar metatarsal heads[57,58]. Ankle equinus, with restricted dorsiflexory range of motion is fairly common in patients with diabetic neuropathy and can be a consequence of anterior crural muscle atrophy. The decreased ankle motion, which confers higher-than-normal plantar pressures at the forefoot, has been implicated as a contributory cause of ulceration as well as recurrence or recalcitrance of existing ulcers[59].

Autonomic neuropathy often results in dry skin with cracking and fissuring, creating a portal of entry for bacteria. Autosympathectomy with attendant sympathetic failure, arteriovenous shunting, and micro vascular thermoregulatory dysfunction impairs normal tissue perfusion and micro vascular responses to injury. These alterations can subsequently be implicated in the pathogenesis of ulceration.

Foot deformities resulting from neuropathy, abnormal biomechanics, congenital disorders, or prior surgical intervention may result in high focal foot pressures and increased risk of ulceration. The effects of motor neuropathy occur relatively early and lead to poor muscle atrophy with consequent development of hammertoes, fat pad displacement, and associated increase in plantar forefoot pressures. Although most deformities cause high plantar pressures and plantar foot ulcerations, medial and dorsal ulcerations may develop as a result of footwear irritation. Common deformities might include prior partial foot amputations, prominent metatarsal heads, hammertoes, Charcot arthropathy, or hallux valgus. A large prospective population based study found that elevated plantar foot pressures are significantly associated with neuropathic ulceration and amputation. The study also revealed a trend for increased foot pressures as the number of pedal deformities increased.

Trauma to the foot in the presence of sensory neuropathy is an important component cause of ulceration. While trauma may include puncture wounds and blunt injury, a common injury leading to ulceration is moderate repetitive stress associated with walking or day-to-day activity. This is often manifested by callus formation under the metatarsal heads. A recent report suggests that even with moderate activity, ulceration may be precipitated by a higher degree of variability in activity or periodic

"bursts" of activity. Shoe-related trauma has also been identified as a frequent precursor to foot ulceration.

Peripheral arterial disease (PAD) rarely leads to foot ulcerations directly. However, once ulceration develops, arterial insufficiently will result in prolonged healing, imparting an elevated risk of amputation[58,59]. Additionally, attempts to resolve any infection will be impaired due to lack of oxygenation and difficulty in delivering antibiotics to the infection site. Therefore, early recognition and aggressive treatment of lower extremity ischemia are vital to lower limb salvage.

Limited joint mobility has also been described as a potential risk factor for ulceration. Glycosylation of collagen as a result of longstanding diabetes may lead to stiffening of capsular structures and ligaments (cheiroarthropathy)[62]. The subsequent reduction in ankle, subtalar, and first metatarso phalangeal (MTP) joint mobility has been shown to result in high focal plantar pressures with increased ulceration risk in patients with neuropathy. Several reports also attribute glycosylation and altered arrangement of Achilles tendon collagen to the propensity for diabetic patients to develop ankle equines.

Other factors frequently associated with heightened ulceration risk include nephropathy, poor diabetes control, duration of diabetes, visual loss and advanced age. Soft tissue changes (other than cheiroarthropathy) in the feet of diabetic patients might also contribute to ulceration through the pathway of altered pressure distributions through the sole of the foot. Such alterations include a reported increased thickness of the plantar fascia with associated limitation of hallux dorsiflexion, decreased thickness of plantar soft tissue, hardness/stiffness of the skin, and a propensity to develop calluses. While these changes are presumably caused by

glycosylation of collagen, their sum effect is to enhance plantar pressures in gait. In the presence of neuropathy, the accentuated plantar pressures can be implicated in the development of ulceration

**Risk Factors for ulceration**

***General or Systemic Contributions***

- Uncontrolled hyperglycemia
- Duration of diabetes
- Peripheral vascular disease
- Blindness or visual loss
- Chronic renal disease
- Older age

***Local tissues***

- Peripheral neuropathy
- Structural foot deformity
- Trauma and improperly fitted shoes
- Callus
- History of prior ulcer amputation
- Prolonged elevated pressures
- Limited joint mobility

**MECHANISMS OF INJURY**

The multifactorial aetiology of diabetic foot ulcers is evidenced by the numerous physiologic pathways that can potentially lead to this disorder. Among

these are two common mechanisms by which foot deformity and neuropathy may induce skin breakdown in persons with diabetes[62].

The first mechanism of injury refers to prolonged low pressure over a bony prominence (i.e., bunion or hammertoe deformity). This generally causes wounds over the medial, lateral, and dorsal aspects of the forefoot and is associated with tight or ill-fitting shoes. Shoe trauma, in concert with loss of protective sensation and concomitant foot deformity, is the leading event precipitating foot ulceration in persons with diabetes[63].

Diabetes mellitus is responsible for a variety of foot pathologies contributing to the complications of ulceration and amputation. Multiple pathologies may be implicated, from vascular disease to neuropathy to mechanism trauma. Regions of high pedal pressure are frequently associated with foot deformity. When an abnormal focus of pressure is coupled with lack of protective sensation, the result can be development of a callus, blister, and ulcer[69,70].

The other common mechanism of ulceration involves prolonged repetitive moderate stress. This normally occurs on the sole of the foot and is related to prominent metatarsal heads, atrophied or anteriorly displaced fat pads, structural deformity of the lower extremity, and prolonged walking. Rigid deformities such as hallux valgus, hallux rigidus, hammertoe, Charcot arthropathy, and limited range of motion of the ankle (equinus), subtalar, and MTP joints have been linked to the development of diabetic foot ulcers. Numerous studies support the significant association between high plantar pressures and foot ulceration. Other biomechanical perturbations, including partial foot amputations, have the same adverse effects.[64,65,66]

## RISK FOR INFECTION

Infections are common in diabetic patients and are often more severe than infections found in non diabetic patients[75]. Persons with diabetes have an increased risk for developing an infection of any kind and a several-fold risk for developing osteomyelitis. With an incidence of 36.5 per 1,000 persons per year, foot infections are among the most common lower extremity complications in the diabetic population (excluding neuropathy), second only to foot ulcers in frequency.

It is well documented that diabetic foot infections are frequently polymicrobial in nature. Hyperglycemia, impaired immunologic responses, neuropathy, and peripheral arterial disease are the major predisposing factors leading to limb-threatening diabetic foot infections. Uncontrolled diabetes results in impaired ability of host leukocytes to fight bacterial pathogens, and ischemia also affects the ability to fight infections because delivery of antibiotics to the site of infection is impaired. Consequently, infection can develop, spread rapidly, and produce significant and irreversible tissue damage. Even in the presence of adequate arterial perfusion, underlying peripheral sensory neuropathy will often allow the progression of infection through continued walking or delay in recognition.

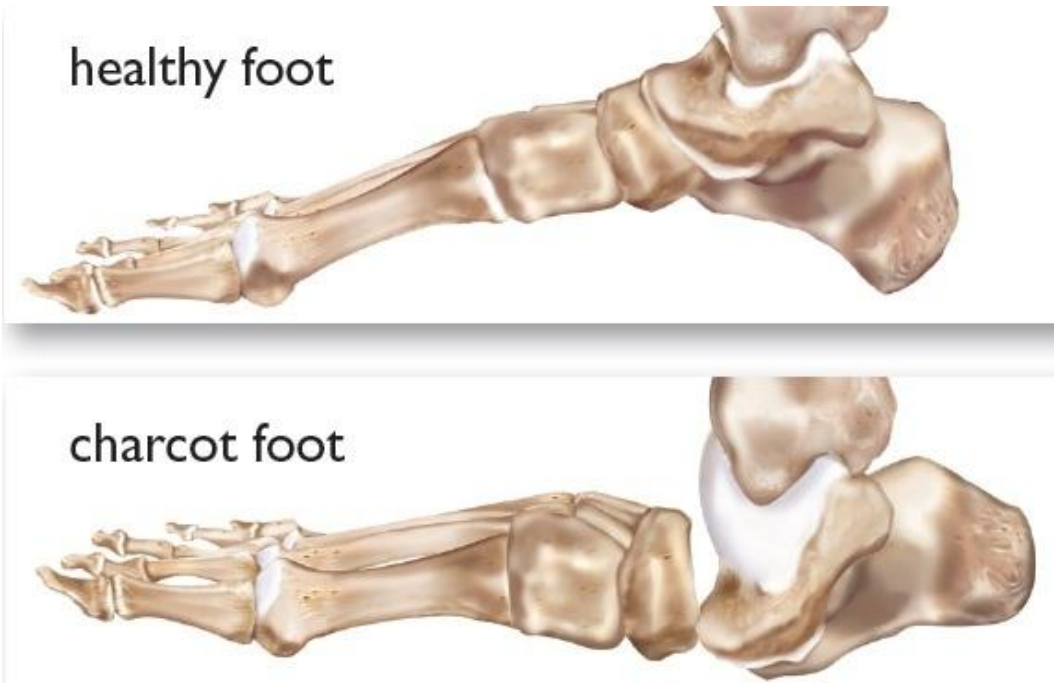
## RISK FOR CHARCOT JOINT DISEASE

It has been estimated that less than 1% of persons with diabetes will develop Charcot joint disease[72,73]. Data on the true incidence of neuroarthropathy in diabetes are limited by the paucity of prospective or population-based studies in the literature. One large population based prospective study found an incidence of about 8.5 per 1,000 persons with diabetes per year; this equates to 0.85% per year and is probably the most reliable figure currently available. Much of the data clinicians rely upon

have been extracted four retrospective studies of small, single centre cohorts. The incidence of reported Charcot cases is likely to be underestimated because many cases go undetected, especially in the early stages.

Primary risk factors for this potentially limb-threatening deformity are the presence of dense peripheral sensory neuropathy normal circulation, and history of preceding trauma often minor on nature. Trauma is not limited to injuries such as sprains or contusions foot deformities prior amputations joint infections, or surgical trauma may result in sufficient stress that can lead Charcot joint disease[74].

**IMAGE 6: CHARCOT DEFORMITY**



## RISK FOR AMPUTATION

The reported risk of lower extremely amputations in diabetic patients ranges from 2% to 16% [79,80]depending on study design and the population studied, Local Education Authority, UK (LEA) rates can be 115 to 40 times higher among the diabetic versus non diabetic populations[64]. Although one author suggests that amputation may be a marker not only for disease severity but also for diseases management, it is clear that amputation remains a global problem for all persons with diabetes. The same risk factors that predispose to ulceration can also generally be considered contributing causes of amputation, albeit with several modifications.

While peripheral arterial disease may not always be an independent risk factor for ulceration when controlling for neuropathy, it can be significant risk factor for amputation. PAD affecting the feet and legs is present in 8% of adult diabetic patients at diagnosis and in 45% after 20 years. The incidence of amputation is 4 to 7 times greater for diabetic men and women than for their non diabetic counterparts[81]. Impairment of arterial perfusion may be an isolated cause for amputation and a predisposing factor for gangrene. Early diagnosis, control of risk factors, and medical management as well as timely revascularization may aid in avoiding limb loss.

While infection is not often implicated in the pathway leading to ulceration, it is a significant risk factor in the causal pathway to amputation. Lack of wound healing, systematic sepsis, or unresolved infection can lead to extensive tissue necrosis and gangrene, requiring amputation to prevent more proximal limb loss. This includes soft tissue infection with severe tissue destruction, deep space abscess, or osteomyelitis. Adequate debridement may require amputation at some level as a means of removing all infected material[82].

Another frequently described risk factor for amputation is chronic hyperglycemia. Results of the Diabetes control and complications Trial (DCCT) and the United Kingdom prospective Diabetes Study (UKPDS) support the long held theory that chronic poor control of diabetes is associated with a host of systemic complications[82,83].

The link between degree of glucose control and incidence or progression of numerous diabetic complications has been well established by these and other studies. Such complications include peripheral neuropathy, microangiopathy, microcirculatory disturbances, impaired leukocyte phagocytosis, and glocosylation of tissue proteins. Each has adverse effects on the diabetic foot. They can contribute to the etiology of foot ulceration, delay normal wound healing and subsequently lead to amputation.

Several studies have reported a significant correlation between elevated glucose and IFA. Amputation has also been associated with other diabetes related co-morbidities such as nephropathy, retinopathy and cardiovascular disease.[84]Aggressive glucose control, management of associated co-morbidities, and appropriate lower extremity care coordinated in a team environment may indeed lower overall risk for amputation.

The best predictor of amputation is a history of previous amputation. A past history of a lower extremity ulceration or amputation increases the risk for further ulceration, infection and subsequent amputation. It may also be inferred that patients with previous ulceration possess all the risk factors for developing ulceration, having demonstrated that they already have the component elements in the causal pathway. Up to 34% of patients develop another ulcer within 1 year after healing an index wound, and the 5 year rate of developing a new ulcer is 70%[84].

The recurrence rate is higher for patients with a previous amputation because of abnormal distribution of plantar pressures and altered osseous architecture. The cumulative risk of neuropathy, deformity, high plantar pressure, poor glucose control and male gender are all additive factors for pedal ulceration in these diabetic patients.

***Re-amputation***[84,85]: it can be attributed to disease progression, non healing wounds and additional risk factors for limb loss that develop as a result of the first amputation. Tragically the 5 year survival rate after a diabetes related LEA has been reported to be as low as 28% to 31%. Persons with renal failure or more proximal levels of amputation have a poor prognosis and higher mortality rate. Those who undergo a diabetes related amputation have a 40% to 50% chance of undergoing a contralateral amputation within 2 years[86].

**ASSESSMENT OF THE DIABETIC FOOT**

The pedal manifestations of diabetes are well documented and potentially limb threatening when left untreated. Recognition of risk factors and treatment of diabetic foot disorders require the skills of a specialized practitioner to diagnose, manage, treat and counsel the patient. Integration of knowledge and experience through a multidisciplinary team approach promotes more effective treatment, thereby improving outcomes and limiting the risk of lower extremity amputation[81].

The evaluation of the diabetic foot involves careful assimilation of the patient's history and physical findings with the results of necessary diagnostic procedures. Screening tools may be valuable in evaluating the patient and determining risk level. Early detection of foot pathology, especially in high- risk patients, can lead to earlier intervention and thereby reduce the potential for hospitalization and amputation. This is also facilitated by an understanding of the

underlying pathophysiology of diabetic foot disorders and associated risk factors. Identification of abnormal historical and or physical findings can therefore improve the prognosis for a favourable outcome through appropriate and early referral.

**History**

A through medical and foot history must be obtained from the patients. The history should address several specific diabetic foot issues

**Medical History**

***Global History***

- Diabetes- duration
- Glycaemia management
- Cardiovascular, renal and ophthalmic evaluations
- Other co morbidities
- Treating physician
- Nutritional status
- Social habits alcohol, tobacco, drugs
- Current medications
- Allergies
- Previous hospitalization/ surgery

**Foot Specific history**

***General***

- Daily activities including work
- Footwear
- Chemical exposures

- Callus formation
- Foot determines
- Previous foot infections, surgery
- Neuropathic Symptoms
- Claudication/ rest pain

***Wound ulcer history***

- Location
- Duration
- Inducing trauma
- Infection
- Hospitalization
- Wound care
- Off loading techniques
- Wound response
- Patient compliance
- Interference with wound care (family or social problems for patient)
- Previous foot tissue or surgery
- Charcot foot previous or active
- Charcot treatment

**Physical Examination**

All patients with diabetes require a pedal inspection whenever they present to any health care practitioner, and they should receive a through lower extremity examination at least once annually[90]. Patients with complaints relating to the diabetic

foot require more frequent detailed evaluations. The examination should be performed systematically so that important aspects are not overlooked.

It begins with a gross evaluation of the patient and extremities. Any obvious problem can then receive closer scrutiny. Key components of the foot examination are presented below. Although not specifically mentioned in this section, it is assumed that a general medical assessment (including vital sign measurement) should be obtained.

**Vascular Examination**

- Palpation of pulses: common femoral, popliteal, Dorsalis pedis, posterior tibial
- Handheld Doppler examination
- Skin/limb colour changes: cyanosis, erythema, elevation pallor, dependendent rubor
- Presence of edema
- Temperature gradient (ipsilateral and contralateral extremity)
- Dermal thermometry
- Ischemic changes: Skin atrophy, thin, smooth, parchment like skin, abnormal wrinkling, absence of hair growth, onychodystrophy

**Neurological examination**

- Vibration perception: Tuning fork 125 Hz/cps, measurement of vibration perception threshold (biothesiometer)
- Light pressure Semmes-Wenstein 10gm monofilament
- Light touch: cotton wool
- Two point discrimination
- Pain: pin prick (sterile needle)

- Temperature perception: hot and cold
- Deep tendon reflexes: patella, Achilles
- Clonus testing
- Babinski's test
- Romberg's test

**Dermatological examination**

- Skin appearance
  - o Color, texture, turgor, quality
  - o Dry skin
- Calluses
  - o Discoloration/ subcallus hemorrhage
- Fissures (especially posterior heel)  Nail appearance
  - o Onychomycosis, dystrophic gryphotic
  - o Atrophy or hypertrophy
  - o Paronychia
- Hair growth
- Ulceration, gangrene, infection
  - o Note location, size, depth, infection status, etc
- Interdigital lesions
- Tinea pedis
- Markers of diabetes
  - o Shin spots: diabetic dermopathy
  - o Necrobiosis lipodica diabeticorum
  - o Bullosum diabeticorum

- o Granuloma annulare
- o Acanthosis nigricans

**Muskuloskeletal examination**

- Biomechanical abnormalities
- Structural deformities
  - Hammertoe, bunion, tailor's bunion
  - Hallux limitus/ rigidus
  - Flat or high arched feet
  - Charcot deformities
  - Posts surgical deformities (amputations)
- Prior amputation
- Limited joint mobility
- Tendo-Achliles contractures/ equinus
- Gait evaluation
- Muscle group strength testing
  - Passive and active non weight bearing
  - Foot drop
  - Atrophy: intrinsic muscle atrophy
- Plantar pressure assessment
  - Computerized devices
  - Harris ink mat, pressure sensitive foot mat

**Foot wear examination**

- Type of shoe (athletic, oxford, comfort etc)
- Fit
- Depth of toe box

- Shoe wear, pattern of wear
- Lining wear
- Foreign bodies
- Insoles, orthoses.

**Diagnostic procedure**

Diagnostic procedure may be indicated in the assessment and care of the diabetic foot. Consideration should be given to the following test in concert with those suggested by members of the consulting team. It should be noted that many of the following tests lack the ability to impart a definite diagnosis, necessitating clinical correlation.

**Laboratory tests**

Clinical laboratory tests that may be needed in appropriate clinical situations include fasting or random blood glucose, glycohemoglobin (HbA1c), complete blood count (CBC) with or without differential erythrocyte sedimentation rate (ESR), serum chemists, C-reactive protein, alkaline phosphatase, wound and blood cultures and urinalysis. Caution must be exercised in the interpretation of laboratory tests in the patients, because several reports have documented the absence of leukocytosis in the presence of severe foot in infections. A common sign of persistent infection is recalcitrant hyperglycemia despite usual anti hyperglycaemic regimens.

**Imaging Studies**

The diabetic foot may be predisposed to both common and unusual infections or non-infectious processes, partially because of the complex nature of diabetes and its associated vascular and neuropathic complications. As a result, imaging

presentations will vary due to lack of specificity in complex clinical circumstances. Such variability creates a challenge in the interpretation of imaging studies, therefore, imaging studies should only be ordered to establish or confirm a suspected diagnosis and or direct patient management. Distinguishing osteomyelitis from aseptic neuropathic arthropathy is not easy, and all imaging studies must be interpreted in conjunction with the clinical findings.

Plain radiographs should be the initial imaging study in diabetic patients with signs and symptoms of a diabetic foot disorder. Radiographs can detect osteomyelitis osteolysis, fractures, dislocations seen in neuropathic arthropathy, medial arterial calcification, soft tissue gas and foreign bodies as well as structural foot deformities, presence of arthritis, and biomechanical alterations. Acute osteomyelitis might not demonstrate osseous changes for up to 14 days. Serial radiographs should be obtained in the face of an initial negative radiographic image and a high clinical suspicion of osseous disease.

Technetium 99 methylene disphosphonate (Tc-99 MDP) bone scans are often used in diabetic foot infection to determine the presence of osteomyelitis. Although highly sensitive, this modality lacks specificity in the neuropathic foot. Osteomyelitis, fractures, arthritis and neuropathic arthropathy will all demonstrate increased radiotracer uptake. However, a negative bone scan is strong evidence against the presence of infection. To improve the specificity of nuclear imaging, white blood cells can be labelled with Tc-99 hexamethylpropyleneaminoxime (tc-99 HMPAO), Indium-111 oxime or gallium-67 citrate.

Indium-111 selectively labels polymorphonuclear leucocytes and is more specific for acute infections than Tc-99 MDP scanning, chronic infections and

inflammations are not well imaged with indium-111 because chronic inflammatory cells (lymphocytes) predominate and are not well labelled with indium58. Combining Tc-99 MDP and Indium-111 increases the specificity of diagnosing osteomyelitis. This combined technique is useful because the Tc-99 MDP scan localizes the anatomic site of inflammation and the Indium-111 labels the infected bone. The Indium-111 scan is not typically positive in aseptic neuropathic arthropathy although false positive Indium scans can occur. 100% sensitivity and 89% specificity have been reported with a combined technique in evaluating diabetic infections.

In Tc-99 HMPAO scanning, white blood cells are labelled in a similar manner as in Indium scanning. However, with Tc-99 HMPAO scans, imaging occurs 4 hours following administration vs 24 hours post administration with Indium scanning. Tc-99 HMPAO uses a smaller radiation dose, is less expensive and offers improved resolution compared with Indium scanning. The sensitivity and specificity of both techniques are comparable. Tc-99 HMPAO scans cannot be combined with Tc-99 MDP scans because of similar labelling characteristics. Tc-99 sulphur colloid is useful in distinguishing osteomyelitis from neuropathic arthropathy. This tracer is picked up by the bone marrow and any haemopoietically active marrow will be positive. Infected bone replaces normal bone marrow, so it shows up as a relative “cold spot”. This technique is best combined with Indium scanning and osteomyelitis would appear as a “hot” indium scan and a “cold” sulphur colloid scan.

CT scans may be indicated in the assessment of suspected bone and joint pathology not evident on plain radiograph. CT offers high anatomic detail and resolution of bone with osseous fragmentation and joint subluxation. Subluxation of the transverse tarsal or tarso metatarsal joints can be seen prior to being visualized on readiographs[76].

MRI is usually preferred over CT for the investigation of osteomyelitis because of its enhanced resolution and its ability to visualize the extent of any infectious process. Despite its high cost, MRI has gained wide acceptance in the management of diabetic foot infections. When neuropathic arthropathy is present, the T1 and T2 bone images are hypo intense (decreased signal) and the soft tissue show edema. Increased signal on T2 bone images is seen is osteomyelitis, however, tumours and avascular necrosis can also be hyper intense on T2. Post contrast fat suppression images should be obtained if available.

PET scan is a promising new technique for distinguishing osteomyelitis from neuropathic arthopathy. A recent meta-analysis comparing the diagnostic accuracy of PET scanning with bone leucocyte scanning found that PET scans were the most accurate modality for diagnosing osteomyelitis, providing a sensitivity of 96% and specificity of 91%.

When PET scanning was unavailable, an Indium labelled leucocyte scan was found to be an acceptable alternative, offering a sensitivity of 84% and specificity of 80% in the peripheral skeleton. The use of USG for detecting chronic osteomyelitis has been shown to be superior to plain radiographs, providing sensitivity comparable to Tc-99 MDP bone scanning. Although ultrasound is a widely available cost effective imaging modality, MRI is more accurate and is the imaging study of choice if radiographs are normal and clinical suspicion is high for bone or soft tissue infection[76,77].

**Vascular evaluation**

The lower extremity must be assessed for vascular and neuropathic risk factors. Although positive findings in the neurologic examination rarely require

further evaluation, positive findings of vascular insufficiency may require further consultation. The indications for vascular consultation include an ABI of less than 0.7, toe blood pressures less than 40mmHg, or transcutaneous oxygen tension (TcPO2) levels less than 30mmHg [77,78].

Since these measures of arterial perfusion are associated with impaired wound healing. If history and physical examination suggest ischemia (i.e. absent pedal pulses) or if a non healing ulcer is present, further evaluation in the form of non invasive testing is warranted.

Non invasive arterial studies should be performed to determine lower extremity perfusion. Such studies may include Doppler segmental arterial pressures and waveform analysis, ABI, toe blood pressures and TcPO2[77]. ABI may be misleading because ankle pressures can be falsely elevated due to medial arterial calcinosis and non compressibility of affected arteries. A growing body evidence suggest that toe blood pressures in diabetic patients may have a role in predicting foot ulceration risk as well as predicting successful wound healing. TcPO2 measurements have received similar support in the literature. Both tests can be performed distally and the foot regardless of arterial calcification in the major pedal arteries and they are both favourable at pressures in the range of 40mmHg.

Laser Doppler velocimetry and measurement of skin perfusion pressure (SPP) have primarily been used in research settings, but can accurately assess blood flow and oxygen tension in the superficial arterioles and capillaries of the skin. Several recent reports indicate that laser Doppler measurement of SPP can be highly predictive of CLI and wound healing failure at levels less than 30mmHg. Arteriography with clearly visualized distal run off allows appropriate assessment for

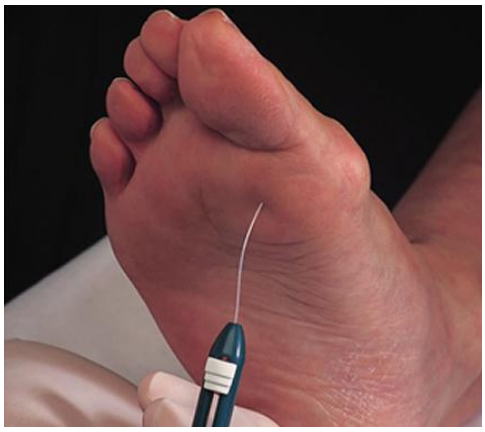
potential re vascularisation. MRI or CT angiogram are alternatives for evaluation of distal arterial perfusion[78,79.]

**IMAGE 7 : HAND HELD DOPPLER**

**Neurological evaluation**

Peripheral sensory neuropathy is the major risk factor for diabetic foot ulceration. The patient history and physical examination utilizing the 5.07 Semmes-Weinstein monofilament (10gm) wire are sufficient to identify individuals at risk for ulceration. Vibration perception threshold assessment with bio thesiometer is also useful in identifying patients at high risk for ulceration. More sophisticated studies such as nerve conduction studies are rarely necessary to diagnose peripheral sensory neuropathy. Patients with neuropathic ulcerations usually have such profound sensory neuropathy that these studies add little to their clinical management.

**IMAGE 8 : SEMMES WEINSTEIN MONOFILAMENT**



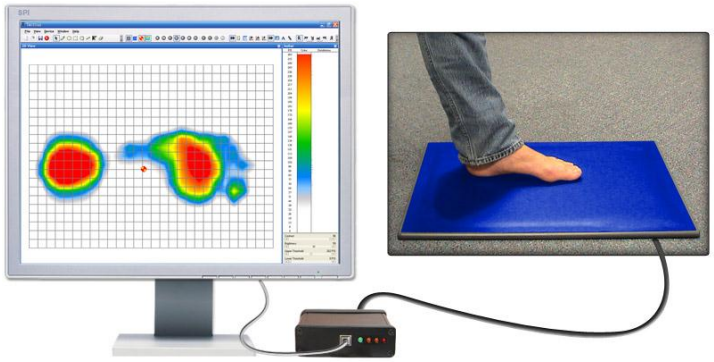
**Plantar foot pressure assessment**

High plantar foot pressure is a significant risk factor for ulceration. Normal plantar foot pressure is 5-35 kPa. Measurement of high plantar foot pressure is possible using a variety of modalities. While these measurements may be important to identifying areas of the foot at risk for ulceration and possibly in evaluation orthotic adjustment, they are primarily used in diabetic foot research. The Harris mat, while not as sophisticated, can provide qualitative measurement of plantar foot pressures and can identify potentially vulnerable areas for ulceration.

A newer non computerized device (PressureStat, foot logic), which is similar to Harris mat and uses pressure sensitive contact sheets that provide a semi quantitative estimation of pressure distribution under the foot, has been suggested as an inexpensive screening tool for identifying areas at high risk for ulceration.

Computerized plantar force technology is available in the form of in-shoe testing systems. These systems help to evaluate plantar foot pressure digitally.

**IMAGE 9 : HARRIS MAT**



## THE HEALTHY DIABETIC FOOT : PREVENTION

A healthy, intact diabetic foot is best maintained by a consistent and recurrent preventive treatment strategy. This is best accomplished through a multi disciplinary approach involving a team of specialists and personnel who provide a co-ordinated process of care. Team members may include a podiatrist, internist, ophthalmologist, endocrinologist, infectious disease specialist, cardiologist, nephrologists, vascular surgeon, orthopaedic surgeon, nurse (educator, wound care and home care) and pedorthist/orthotist.

Patient and family education assumes a primary role in prevention. Such education encompasses instruction in glucose assessment, insulin administration, diet,

daily foot inspection and care, proper footwear and the necessity for prompt treatment of new lesions. Regularly scheduled podiatric visits including debridement of calluses and toe nails are opportunities for frequent foot examination and patient education. Diabetes is a lifelong problem and the incidence of diabetic foot complications increases with age and duration of disease. A recent Markov analysis of the cost effectiveness of foot care according to published guidelines found that such preventive care can improve survival, reduce ulceration and amputation rates, is cost effective and can even save on long term costs when compared with standard care.

Risk stratification based on the presence of predisposing casual risk factors, including prior history of ulceration also serves as a guide to the frequency of foot care visits. By identifying high risk patients and tailoring a total foot care prevention program accordingly, the incidences of ulceration and lower extremity amputations can be reduced.

Therapeutic shoes with pressure relieving insoles and high toe boxes are important adjunctive treatments that can reduce the occurrence of ulceration and resultant amputation in high risk patients. While most studies support the efficacies of protective footwear in this regard, two reports suggest that shoes in the absence of a comprehensive prevention program might not be sufficient to prevent a new lesion. Nevertheless, patients with foot deformities that cannot be accommodated by standard therapeutic foot wear should have custom shoes that provide that appropriate fit, depth and a rocker insole. If structural deformities cannot be accommodated by therapeutic footwear, prophylactic surgical correction should be considered but patients must be carefully selected. Diabetic patients at risk for foot lesions must be educated about risk factors and the importance of foot care. Including the need for self inspection and surveillance, monitoring foot temperature, appropriate daily foot

hygiene, use of proper footwear, good diabetic control and prompt recognition and professional treatment of newly discovered lesions.

Home temperature assessment of the foot has been shown to reduce the incidence of foot ulcers tenfold compared with standard preventive care. Patients with visual or physical impairments preclude their own care should encase the assistance of family or friends to aid in the regard when compared with a comprehensive approach when compared with a comprehensive approach to preventive foot care, patient education and reduce the frequency and morbidity of limb threatening diabetic foot lesions.

Provider education is equally important in prevention, since not all clinicians are cognizant of important signs and risk factors for pedal complication. Furthermore, provider education is effective in reinforcing proper diabetes management and foot care practices, resulting in reduction in ulceration and adverse lower extremity outcomes.

## DIABETIC FOOT ULCER

### *Evaluation of ulcers*

The initial evaluation of a DFU should be comprehensive and systematic to ascertain the parameters that may have led to its onset as well as to determine the factors that may impair its healing. Critical in this regard are assessments for vascular perfusion (ischemia) infection/osteomyelitis and neuropathy. Thorough vascular evaluation must be performed. This includes palpation of pulses, clinical evaluation of capillary filling time, venous filling time, pallor on elevation and dependent rubor[59].

If pulses are not palpable or if clinical findings suggest ischemia, non invasive arterial evaluation (segmental Doppler pressures with wave forms, ABI, toe pressures,

TcPO2 measurements) and vascular surgical consultations are warranted. When required, these physiologic and anatomic data can be supplemented with the use of magnetic resonance angiography or CT angiography (CTA) and subsequent use of arteriography with digital subtraction angiography as necessary (DSA)[66,67].

Description of the ulcer characteristics on presentation is essential for the mapping of the ulcer's progress during treatment. While some characteristics are more important than others, they all have prognostic value during management. The presumed etiology of the ulcer (i.e. chemical vs mechanical) and character of the lesion (neuropathic, ischemic or neuro ischemic) should be determined.

The evaluation should also describe the size and depth of the ulcer as well as the margins, base and geographic location on the extremity or foot. All but the most superficial ulcers should b examined with a blunt, sterile probe. The description should note whether the sterile probe detects sinus tract formation, undermining of the ulcer margins or dissection of the ulcer into tendon sheaths, bone or joints. A positive probe to bone (PTB) finding is highly predictive of osteomyelitis, although the frequency of false negative tests reduces its sensitivity. Perhaps most importantly the positive predictive value for PTB falls off significantly when the prevalence of osteomyelitis decreases. The existence and character of odour and exudate should be noted.[59,67,68]

Cultures may be necessary when signs of inflammation re present. Generally, clinically uninfected ulcers without infection should not be cultured. Current recommendations for culture and sensitivity include thorough surgical preparation of wound site with curettage of the wound base for specimen or aspiration of abscess material.

***Classification of ulcers***

Appropriate classification of the foot wound is based on a thorough assessment. Classification should facilitate treatment and be generally predictive of expected outcomes. Several systems of ulcer classification are currently in use in the U.S.A and abroad to describe these lesions and communicate severity. Perhaps the easiest system is to classify lesions as neuropathic, ischemic or neuro-ischemic, with descriptors of wound size, depth and infection[37]. Regardless of which system is used, the clinician must be able to easily categorize the wound and once classified, the ensuring treatment should be directed by the underlying severity of pathology.

Although no single system has been universally adopted, the classification system most often used was described and popularized by Wagner. In the Wagner's system, foot lesions are divided into six grades based on the depth of the wound and extent of tissue necrosis. Since these grades failed to consider the important roles of infection, ischemia and other co morbid factors, subsequent authors have modified the classification system by including descriptors for these considerations. For example, the University of Texas San Antonio (UTSA) system, associates lesion depth with both ischemia and infection. This system has been validated and is generally predictive of outcome, since increasing grade and stage of wounds are less likely to heal without re vascularisation or amputation[61]. The UTSA system is now widely used in many clinical trials and diabetic foot centres.

Another hybrid system, the PEDIS system, evaluates five basic characteristics: perfusion, extent/size, depth/tissue loss, infection and sensation. While this system has yet to be validated, it provides the benefit of having been developed by a consensus body. Imaging studies play an important role in the assessment and

evaluation of the diabetic foot ulcer. Plain xrays are indicated based on the extent and nature of the ulcer. Clinical change in the appearance of the ulcer or failure to heal with appropriate treatment may dictate repeating the radiograph periodically to monitor for osseous involvement. Additional imaging modalities such as nuclear medicine scans, USG, MRI and CT may be indicated depending on the clinical picture. These modalities have been previously discussed in this document.

**WAGNER CLASSIFICATION SYSTEM**

| | |
|---|---|
| **Grade 0** | Foot symptoms like pain only |
| **Grade 1** | Superficial ulcers |
| **Grade 2** | Deep ulcers |
| **Grade 3** | Ulcer with bone involvement |
| **Grade 4** | Forefoot gangrene |
| **Grade 5** | Full foot gangrene |

## TREATMENT OF DIABETIC ULCERS: GUIDING PRINCIPLES

The primary treatment goal for diabetic foot ulcers is to obtain wound closure as expeditiously as possible. Resolving foot ulcers and decreasing the recurrence rate can lower the possibility lower extremity amputation in the diabetic patient. The Wound Healing Society defines a chronic wound as one that has failed to proceed through an orderly and timely repair process to produce anatomic and functional integrity.

A chronic wound is further defined as one in which the healing cascade has been disrupted at some point, leading to prolonged inflammation and failure to re-epithelialize and allowing for further breakdown and infection. Early, advanced or appropriate wound care practices may be more cost effective than standard care practices for decreasing the incidence of lower extremity amputations.

The essential therapeutic areas of diabetic ulcer management are as follows: management of co-morbidities, evaluation of vascular status and appropriate treatment, assessment of lifestyle factors, ulcer assessment and evaluation, tissue management/wound bed preparation and pressure relief.

### Management of co-morbidities

Because diabetes is a multi organ systemic disease, all co morbidities that affect wound healing must be assessed and managed by a multi disciplinary team for optimal outcomes in the diabetic foot ulcer. Among the most common co morbidities are hyperglycemia and vascular diseases such as cerebral vascular accident, TIA, MI, angina, valvular heart disease, atrial fibrillation, aneurysms, renal dysfunction, hypertension, hyper cholesterolemia and hyperlipidemia.

**Evaluation of vascular status**

Arterial perfusion is a vital component of healing and must be assessed in the ulcerated patient, since impaired circulation contributes significantly to non healing of ulcers and subsequent risk for amputation. Early evaluation and reference are important. Symptoms of vascular insufficiency may include edema, altered skin characteristics (lack of hair, diseased nails, altered moisture), slow healing, cool or cold extremities and impaired arterial pulsations. Vascular reconstructive surgery of the occluded limb improves prognosis and may be required prior to debridement, foot sparing surgery and partial amputation.

**Assessment of lifestyle/psycho social factors**

Lifestyle and psycho-social factors may influence wound healing. For example, smoking has a profound effect on wound healing due to its associated vasoconstriction and low oxygen carrying capacity of blood. Other factors (eg: alcohol and drug abuse, eating habits, obesity, malnutrition and mobility) should also be noted. In addition, depression and mental illness may impact the outcome of treatment since these conditions can directly affect the patient's adherence to recommendations and attitude towards healing.

**Ulcer assessment and evaluation**

The importance of a thorough and systemic evaluation of any ulceration cannot be over emphasized. Indeed, the findings of an ulcer, specific examination will directly guide subsequent treatment. Initial evaluation and detailed description of any ulcer should encompass location, size, depth, shape, inflammation, edema, exudate

(quality and quantity), past treatment and duration. The margins of the ulcer should be assessed for callus formation, maceration and erythema.

The presence of erythema among other size such as tenderness and warmth might suggest infection. The quality of the tissue (i.e. moist, granular, desiccated, necrotic, undermining, slough, eschar or liquefied) should be noted. Thorough evaluation is used to determine the presence of sinus tract or deep abscess [66,71,72]. Frequent re evaluation with response directed treatment is essential. Once the ulcer is healed, management consists of decrease in the probability of recurrence.

**Tissue management/ wound bed preparation**

***Debridement:*** debridement of a necrotic tissue is an integral component in the treatment of chronic wounds since they will not heal in the presence of unviable tissue, debris or critical colonization. Undermined tissue or closed wound spaces will otherwise harbour bacterial growth. Debridement serves various functions- removal of necrotic tissue and callus, reduction of pressure, evaluation of the wound bed, evaluation of tracking and tunnelling and reduction of bacterial burden. Debridement facilitates drainage and stimulates healing.

However, debridement may be contra indicated in arterial ulcers. Additionally, except in avascular cases, adequate debridement must always precede the application of topical wound healing agents, dressings or wound closure procedures. Of the five types of debridement (surgical, enzymatic, autolytic, mechanical, biological) only surgical debridement has been proven to be efficacious in clinical trials.

***Surgical debridement:*** surgical debridement is the cornerstone of management of diabetic foot ulcers. Thorough, sharp debridement of all non viable soft tissue and

bone from the open wound is accomplished primarily with a scalpel, tissue nippers, curettes and curved scissors[87]. Excision of necrotic tissue extends as deeply and proximally as necessary until healthy, bleeding soft tissue and bone are encountered. Any callus tissue surrounding the ulcer must also be removed. The main purpose of surgical debridement is to turn a chronic ulcer into an acute, healing wound[87]. A diabetic ulcer associated with a deep abscess requires hospital admission and immediate incision and drainage. Joint resection or partial amputation of the foot is necessary if osteomyelitis, joint infection or gangrene is present.

The principles guiding the surgical management of diabetic foot ulcers are discussed under "surgical management of the diabetic foot". Necrotic tissue on a regular basis can expedite the rate at which a wound heals and has been shown to increase the probability of attaining full secondary closure[88,89]. Less frequent surgical debridement can reduce the rate of wound healing and secondarily increase the risk of infection. Surgical debridement is repeated as often as needed if new necrotic tissue continues to form. Frequent debridement, referred to as "maintenance debridement" is commonly required.

***Hydrosurgery:*** Is a novel system indicated for the surgical debridement of the damaged and necrotic tissue in traumatic, ulcerated, chronic wound, surgical incisions and burns.

Assessment of a diabetic foot ulcer includes not only a description of the skin lesion but also the accurate assessment of the contributing factors and aetiology[91-93]. Amongst its properties are precision, selective cutting and minimal thermal damage to the tissues[93]. When surgical or sharp debridement is not indicated, other types of debridement can be used. For example, vascular wounds may benefit from enzymatic

debridement while an extremely painful wound may benefit from autolytic debridement. Mechanical debridement is often used to cleanse wounds prior to surgical or sharp debridement.

***Enzymatic debridement:*** a highly selective method, it consists of the application of exogenous proteolytic enzymes manufactured specifically for wound debridement. Various enzymes have been developed including bacterial collagenase. Collagenases are enzymes that are isolated from Clostridium histolyticum. These display high specificity for the major collagen types (I and II) but they are not active against keratin, fat or fibrin[91,94].

Papain obtained from the papaya plant is effective in the breakdown of fibrinous material and necrotic tissue. When combined with urea, it denatures non viable protein matter. The enzymatic compounds are inactivated by hydrogen peroxide, alcohol and heavy metals including silver, lead and mercury. One study found that papain-urea developed granulation tissue faster than those treated with collagenase but no contrast between rates of complete wound healing were made.

***Autolytic debridement***: autolytic debridement occurs naturally in a healthy, moist wound environment when arterial perfusion and venous drainage are maintained

***Mechanical debridement:*** a non selective, physical method of removing necrotic tissue, mechanical debridement may include wet to dry dressings and high pressure irrigation or pulsed lavage and hydrotherapy. Wet to dry is one of the most commonly prescribed and over used methods of debridement in acute care settings. Hydrotherapy is a form of whirlpool and may remove surface skin, bacteria, wound exudates and debris.

***Biological (larval) therapy:*** It utilizes the sterile form of the Lucilia sericata blow fly for the debridement of necrotic and infected wounds[98]. Maggots secrete a powerful proteolytic enzyme that liquefies necrotic tissue. It has been noted that wound odour and bacterial count including MRSA, diminish significantly with larval therapy larval therapy seems to be beneficial, but there is paucity of controlled studies to support its routine use in the diabetic foot wound.

***Moisture balance****:* One of the major breakthroughs in wound management over the past 50 years was the demonstration that moisture accelerates re-epithelialisation in a wound. Tissue moisture balance is a term used to convey the importance of keeping wounds moist and free of excess fluids. A moist wound environment promotes granulation and autolytic processes[95]. Effective management of chronic wound fluids is an essential part of wound bed preparation. It also helps in addressing the issues of cellular dysfunction and bio chemical imbalance.

***Wound dressing:*** Can be categorized as passive, active or interactive. Passive dressings primarily provide a protective function. Active and interactive dressings and therapies are capable of modifying a wounds physiology by stimulating cellular activity and growth factor release.

***Advanced wound care modalities***: wound bed preparation offers clinicians a comprehensive approach to removing barriers to healing and stimulating the healing process so that the benefits of advanced wound care can be maximized[98]. Advanced care may sometimes be the only means of rapidly and effectively attaining wound closure. The advent of therapeutic growth factors, gene therapy, tissue engineered constructs, stem cell therapy and other drugs and devices that act through molecular

and cellular based mechanisms is enabling the modern surgeon and wound care provider to actively promote wound angiogenesis to accelerate healing.

***Growth factor therapy*:** chronic ulcers have demonstrated benefits from autologous platelets releasates or genetically engineered products such as recombinant DNA platelet derived growth factor becaplermin gel[96]. This agent has been shown to stimulate chemotaxis and mitogenesis of neutrophils, fibroblasts, monocytes and other components that form the cellular basis of wound healing. In one pivotal randomized placebo controlled blinded trial involving patients with full thickness diabetic foot ulcers, recombinant human platelet derived growth factor (becaplermin) demonstrated a 43% increase in vascular endothelial growth factor (VEGF), fibroblasts growth factor (FGF) and keratinocyte growth factor (KGF) have been under study[96,97].

***Bioengineered tissues**:* These have been shown to significantly increase complete wound closure in venous and diabetic foot ulcers. Currently, two bioengineered tissues have been approved to treat diabetic foot ulcers in the US- ApligrafTM and DermagraftTM. Both have demonstrated efficacy in randomized controlled trials. Tissue engineered skin substitutes can provide the cellular substrate and molecular components necessary to accelerate wound healing and angiogenesis[99]. They function both as biologic dressings and has delivery systems for growth factors and extra cellular matrix components through the activity of live human fibroblast contained in their dermal element.

***Bilayered skin substitutes:*** These include bilayered skin equivalents (ApligrafTM) and cultured composite skin (OrcelTM). ApligrafTM has been shown to significantly reduce the time to complete the wound closure in venous and diabetic ulcers[99,100]. DermagraftTM is no longer available in the US. Extracellular matrices are generally

derived from devitalized tissues to produce an immunologically inert acellular dermal matrix. These include dermal regeneration template, allogenic dermal matrix, matrix of human dermal fibroblast and porcine small intestine submucosa (OasisTM). OasisTM composed of structural cellular components and growth factors utilized to promote natural tissue remodelling recently completed a randomised trial that showed non inferiority to becaplermin gel in the healing of diabetic foot ulcers[101].

***Adjunctive modalities***: regenerative tissue matrix (GraftJacketTM) is a new therapy used in diabetic foot ulcers, although it has not undergone any randomized clinical trials to date. This allograft skin is minimally processed to remove epidermal and dermal cells while preserving the bio active components and structure of dermis. This results in a framework that supports cellular repopulation and vascularisation.

***Hyperbaric Oxygen (HBO) Therapy:*** Has shown promise in the treatment of diabetic foot wounds with hypoxia severe enough to interfere with healing. However, most of the HBO studies were hampered by methodological errors that preclude any definite role for this modality in the routine treatment of diabetic foot ulcers[102]. Nevertheless in 2003, Medicare and Medicaid coverage for HBO extended to ulcers classified as Wagner's grade III or higher that failed standard wound care therapy.

***Ultrasound devices:*** new ultrasound devices are being used to debride the wound and provide ultrasonic therapy. The MIST TherapyTM system is an ultrasonic device approved by the FDA for wound debridement and cleansing. MIST TherapyTM uses a fine saline spray that allows ultrasound to be administered directly to the wound bed without contact to the affected tissue, thus minimizing potential trauma to delicate capillary buds and emerging islands of epithelium[103].

***Negative Pressure Wound Therapy (NPWT):*** Has become a common adjunctive treatment modality for diabetic foot ulcerations. Use of a vacuum assisted closure device (V.A.C) promotes wound healing through the application of topical, sub atmospheric or negative pressure to the wound base. This therapy removes edema and chronic exudate, reduces bacterial colonization, enhances formation of new blood vessels, increases cellular proliferation and improves wound oxygenation as a result of applied mechanical force[104]. These actions are synergistic. Numerous applications of these modalities have proven successful including use over exposed bone, tendon, and hardware to generate granulation tissue. It is also frequently used to facilitate adherence of split thickness skin grafts, rotational flaps or tissue substitutes to a wound bed.

A recent clinical trial of the V.A.C device for the treatment of open amputation wound in the diabetic foot showed significantly faster healing and development of granulation tissue with NPWT compared with standard moist wound care. The rationale for using electrical stimulation in wound healing stems from the fact that the human body has an endogenous bio electric system that enhances healing of bone fractures and soft tissue wounds. Laboratory and clinical studies provide an abundance of support for the use of electrical stimulation in wound care. In a randomized control study evaluating wound healing using electrical stimulation in neuropathic ulcers, significant differences in healed ulcer areas and number of healed ulcers at 12 weeks were in the group receiving electrical stimulation compared with the control group.

**IMAGE 10 : VAC DRESSING**

## OFF LOADING

The reduction of pressure to the diabetic foot is essential to treatment[23,60]. Proper off loading and pressure reduction prevents further trauma and promotes healing. This is particularly important in the diabetic patient with decreased or absent sensation in the lower extremities[16,18] The choice of offloading modality should be determined by the patient's physical characteristics, ability to comply with the treatment and location and severity of the ulcer.

Clinicians must alternate treatment based on the clinical progress of the wound. Even a simple method like using a felted aperture foam pad has been found to be effective in removing pressure and promote wound healing. A study conducted in 2001 noted that the use of Total Contact Cast (TCC) healed a higher portion of wounds in shorter time than a half shoe or a removable cast walker (RCW)[64]

More recently investigators compared the use of TCC with an RCW that was rendered irremovable (iTCC) by circumferential wrapping of an RCW with a single strip of fiber glass casting material. They concluded that the latter may be equally efficacious, faster to place, easier to use and less expensive than TCC in the treatment of diabetic neuropathic plantar foot ulcers[16,18]. The findings of this study and another study also suggest that the modification of the RCW into an irremovable device may improve the patient compliance, thereby increasing the proportion of healed ulcers and the rate of healing of neuropathic wounds.

Regardless of the modality selected, the patient should not return to an unmodified shoe until complete healing of the ulcer has occurred. Furthermore, any shoe that has resulted in the formation of an ulcer should never again be worn by the patient[20].

***Types of offloading devises:***

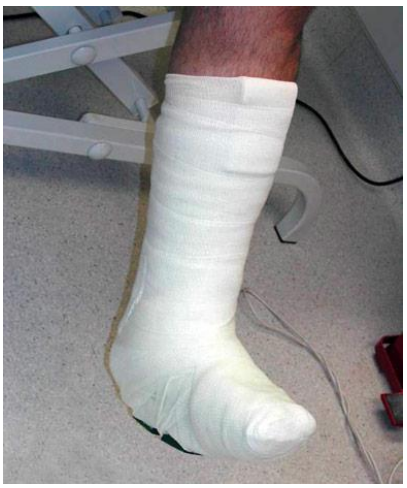
- **Total Contact Cast (TCC)**

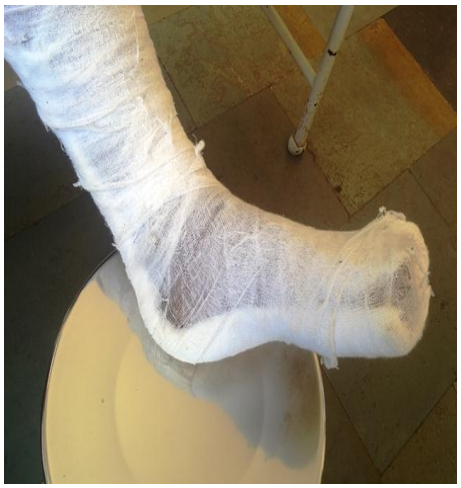
This method is reportedly the "gold standard" form of offloading for neuropathic plantar ulcers reportedly healing upto 90% of ulcers within 6-8 weeks. It is a below knee cast that incorporates the whole lower limb including the foot. It was traditionally made of plaster of Paris bandage immediately moulded to the lower leg and foot with padding only over the toes, malleoli and tibial crest. Now-a-days, this inner layer of plaster of Paris is covered with a quickly setting outer and very rigid fibreglass casting bandages so that the patient can mobilize within half an hour.

A piece of plywood is placed under the whole foot and is incorporated into the cast with all hollows and cavities filled with plaster of paris bandages and a small rubber rocket placed centrally under the cast to weight bear upon. Plaster of paris takes 48 hrs to fully set and thus the outer layer of the fibreglass tape bandage is in use. In developing countries where fibreglass is not available crutches should be used to prevent weight bearing in the first two days. Although evidence clearly supports TCC efficacy it is still not widely used, even though these are relatively cheap to make from a resource perspective.

Contraindications for this and other type of cast includes: infection, ischemia, loss of sight or balance. It should be noted that in patients with heavily exuding wounds cast may need frequent changes, but this is not contraindication by itself.

**IMAGE 11 : TCC AND TCS**





- **Total Contact Slab (TCS)**

These are similar to total contact casts in principle but differs from them in a way that it can be removed and reapplied regularly in patients with severe infection and producing heavy exudates.

- **Removable Cast Walkers / Slings**

These are commercially available below knee cast/ splints/ walkers, which usually have a rocker sole and a rigid plastic outer shell with either soft or pneumatic lining.

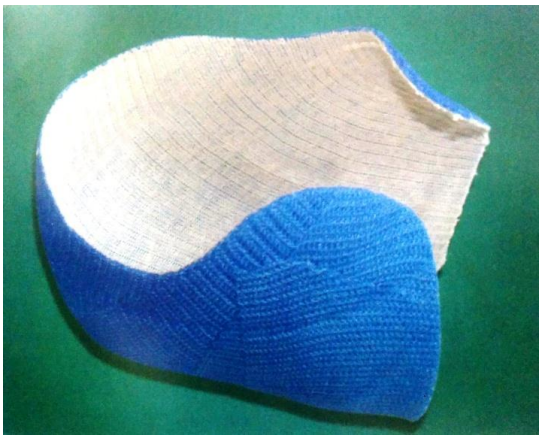
Velcro straps attached to the plastic outer rigid shell are used to secure the cast walker in place. These can be effective for forefoot ulcers but equally as they can be taken off and worn only at clinic visits, can make them equally ineffective. These are also expensive, although require very little training and are instant put-ons and therefore time saving.

**IMAGE 12 : REMOVABLE CAST WALKER**

- **Removable Heel Casts**

These are new technique using a semi-flexible cast bandage with a focus rigidity area over the heel ulcer site. They have no incorporated padding and need to be replaced when exudate soiling occur.

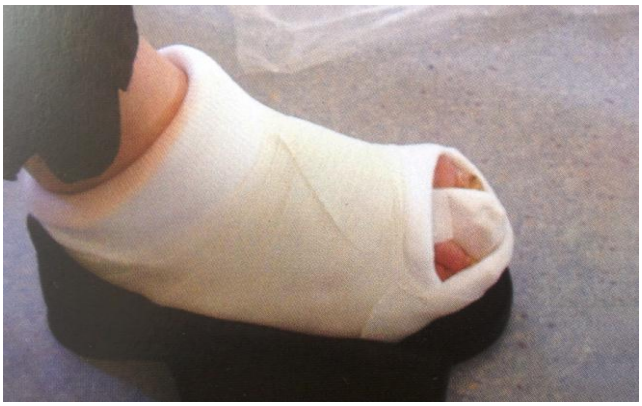
**IMAGE 13 : REMOVABLE HEEL CAST**



- **Fiberglass Boots**

These are made from fibreglass bandage and are either removable or non-removable devises. They have padding incorporated into the cast to save the ulcer site from ground contact. Removable casts allow wound inspection and easy dressing changes. They can slip away or be left off by the patient. Non-removable casts do not allow slippage and can be aperture to the ulcer site to allow dressing changes. They require a over cast sandal.

**IMAGE 14 : FIBERGLASS BOOTS**



- **Below Knee Walking Plaster**

These are the same type of below casts used for setting fractures and may or may not be aperture at the ulcer site.

- **Blueprint For Cast Care**

All non-removable casts should be checked daily by the patient or their care, looking for cast rubs/sores, exudate strike through, swelling at the open end of the cast, malodour from the cast, pain or loosening. If any of these occur, the cast must be removed immediately and the limb inspected.

All non-removable non-aperture casts are removed after one week. Then, if all is well, reapply for 2 weeks and then every 4 weeks until healed.

Removable casts with incorporated felt padding should be checked at each visit. When the padding is compressed, it needs to be replaced by either a complete new cast or stripping and relining the original cast.

The danger signs and symptoms are the same for the non-removable casts.

- **Half Shoes**

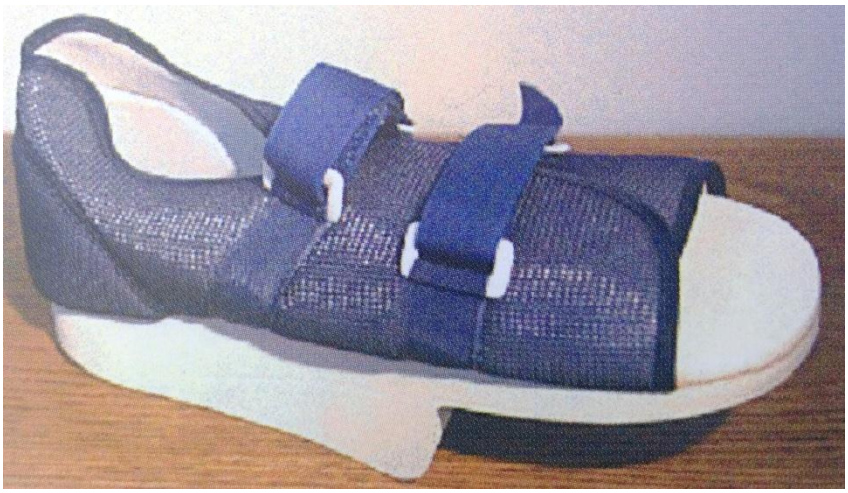
These are commercially available sandal type shoes with a sole unit that is thick at the hell and angles posteriorly and a thin forefoot platform that is raised from the ground. This type of device offloads the forefoot. There is also a similar devise in reverse that can be used for the heel. They create a large limb discrepancy, so caution needs to be taken in patients with poor proprioception, sight or balance.

**IMAGE 15: HALF SHOES**


- **Healing Sandals**

This type of footwear is made by application of a rigid rocker to the bottom of a shoe or a sandal. It limits dorsiflexion, therefore distributing pressure evenly, especially over the metatarsal heads. Although this footwear is light and stable, it is not as efficient as other methods of offloading and it also requires significant amount of time and experience to produce and is therefore not easily accessible.

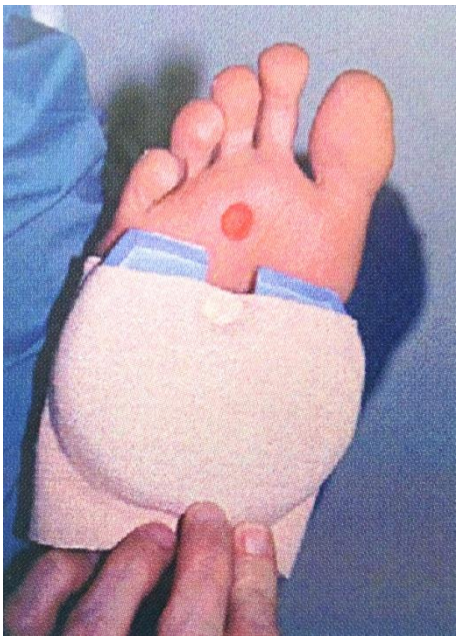
**IMAGE 16 : HEALING SANDALS**



- **Felted Foam** [109]

It consists of bilayered foam, placed over the plantar surface of the foot with an opening to accommodate the wound. It is relatively inexpensive and is easily accessible. However, it has the disadvantage that it can cause or produce pressure and shear at the wound edges and there are no authentic case studies reported to suggest its efficacy in offloading.

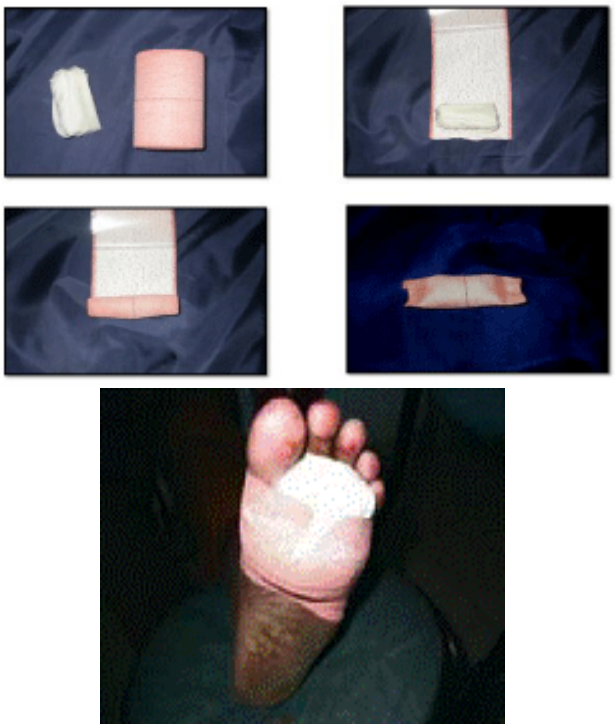
**IMAGE 17 : FELTED FOAM**



- **Mandakini Offloading Device**[110]

This one of the indigenous technique of offloading designed using used pair of gloves and dynaplast adhesive plaster. Here a used gloves are rolled as we do for autoclaving. It is then placed on adhesive surface of dynaplast and covered circumferentially with dynaplast. Number of gloves to be used will be decided on weight of patient. Edges of dynaplast are approximated by sharp pressure. Thus the Mandakini off-loading device is ready to place. It acts like a soft air-cushion, off-loads body weight. Fore foot lesions are attended by applying the device proximal to lesion and hind foot lesions are attended by applying device distal to lesion. Frequency of application is every week and results in complete healing of ulcers in 4–6 weeks.

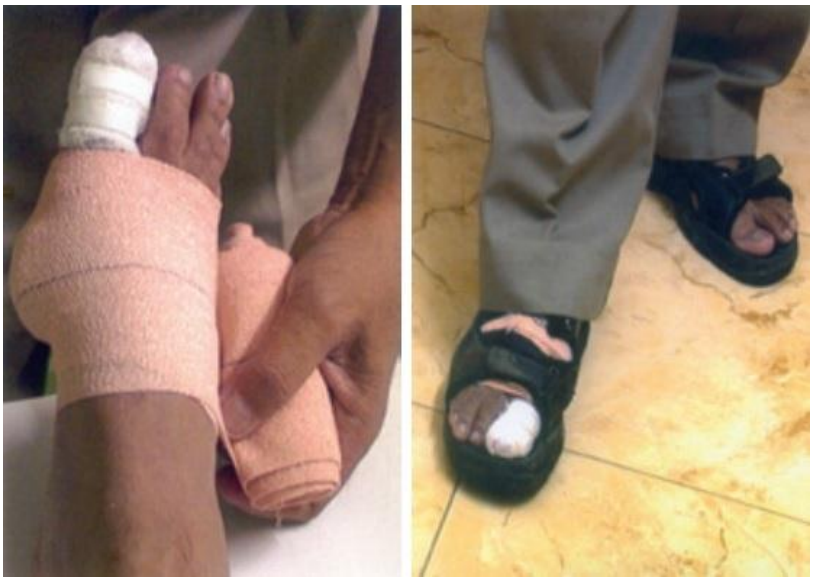
**IMAGE 18 : MANDAKINI OFFLOADING DEVICE**



- **Samadhan System** [111]

This is based on the principles of simplicity, ease of application, affordability and effectiveness, and which requires no training. The Samadhan System of offloading was developed in 2000. The Hindi word ‘samadhan’ means ‘solution’. The system incorporates both a removable (Samadhan-R) and a non-removable offloading device (Samadhan-IR). It has 3 components- A foam cylinder, called “Samadhan Unit,” a piece of elastocreppe bandage, called “Retainer“ and metallic clips, provided with the elastocreppe bandage, called “Fastner,” which keep edge of the elastocreppe bandage in position. These Samadhan Units are kept ready and are cut to size of the sole of the patients when he arrives. Next Samadhan Unit is placed at a point which offloads the wound and then it is retained with retainer and fastners. It is very easy to manufacture Samadhan Units. We take a piece of rubberized foam, density 40, size 4 inchesX6 inches, apply liquid adhesive on one side and role up carefully into a cylinder, keeping adhesive side inwards. Then we leave it pressed by some weight for a few hours and the Unit is ready. More than 70% of people with diabetes in the Samadhan-R group achieved complete healing, compared to only 10% in the common footwear group. In another prospective clinical trial, we compared the Samadhan IR versus common footwear. More than 85% of people in the Samadhan-IR group achieved complete healing, compared with 10% in the common footwear group.

**IMAGE 19: SAMADHAN SYSTEM OF OFFLOADING**



- **Crutches**

These are simple, tried and tested and can arguably be quite effective if used correctly and fully. These are cheap and require little expertise to produce. There are no issues of cast applications and dressing changes can go unhindered.

- **Wheel Chair**

A wheel chair is also another simple but effective method of offloading but is expensive, limiting and not very practical for home or work. Of course, here the foot is off ground as well as off the wheelchair foot plate.

# MATERIALS AND METHODS

**Source of data**

All patients attending the surgery OPD and/ or admitted patients in BLDEU's Shri B. M. Patil Medical College, Hospital and Research centre, Vijayapur with diabetic plantar foot ulcers during the period of October 2014 to May 2016.

**Methods of collection of data.**

- Period of study was from October 2014 to may 2016.
- The patients were allocated to each group in such a way that all odd numbers were included in the study group and all even numbers were included in the control group.
- And while allocating cases, age of patient and size of the ulcer was matched.
- A proforma was used to collect all the relevant data from the patients.
- Detailed history was taken; thorough clinical examination and investigations were performed on all the patients included in the study.
- All the cases were followed up to discharge and subsequently for a follow up till wound healing.
- "Primary efficacy end point" was complete ulcer closure and "Secondary efficacy end point" was time taken to achieve ulcer closure by either secondary suturing or skin grafting.

**Inclusion criteria**:

- Diabetic foot ulcers on the plantar aspect of the foot that come under Wagner grade I and II

**Exclusion criteria:**

- Diabetic foot ulcers other than on the plantar aspect of the foot.
- Diabetic patients with foot ulceration resulting from electrical, radiation burns.
- Patients on medications such as corticosteroids, immunosuppressant or chemotherapy.
- Pregnant or nursing mothers.
- Diabetic foot ulcer patients with gross ischemia who need amputation,
- Case of diabetic foot ulcers with skin cancer.

**RESEARCH HYPOTHESIS.**

Reducing pressure at the site of ulcer can promote healing and prevent further ulceration in diabetic plantar foot ulcers.

**SAMPLING**:

- Prospective, interventional study.
- Incidence of diabetic foot ulcer = 5% [105]
- Formula for estimating sample size:

$$\mathbf{n = \frac{(Z\alpha + Z\beta)^2 2SD^2}{MD^2}}$$

$\alpha$ = level of significance = 1%

$1-\beta$ = power of test = 90%

Anticipated mean difference of treatment = 30 days[106, 107]

Anticipated standard deviation = 30[106, 107]

- Calculated sample size is 30

- Hence in this study 60 cases were studied overall, of which 30 were allocated to each group in such a way that all odd numbers were included in the study group and all even numbers were included in the control group.
- And while allocating cases, age of patient and size of the ulcer were matched.

**INVESTIGATIONS OR INTERVENTIONS REQUIRED IN THE STUDY.**

No animal experiments involved in the study.

All patients will undergo the following investigations:

- Complete hemogram and blood group.
- Pus for culture and sensitivity.
- Random blood glucose at admission to the hospital / HbA1c levels at the time of admission.
- Regular blood glucose monitoring with FBS / PPBS / RBS.
- Urine routine, renal function test, ECG.

**IMAGING STUDIES**

- X-RAY of the affected limb / part bearing the diabetic foot ulcer.
- Other relevant investigations when required like Doppler USG of the affected limb, chest x-ray, USG abdomen.

**SURGICAL INTERVENTION IN THE FORM OF**

- Thorough debridement of the foot ulcers to remove all necrotic debris.
- Skin grafting or secondary suturing if required.

# RESULTS

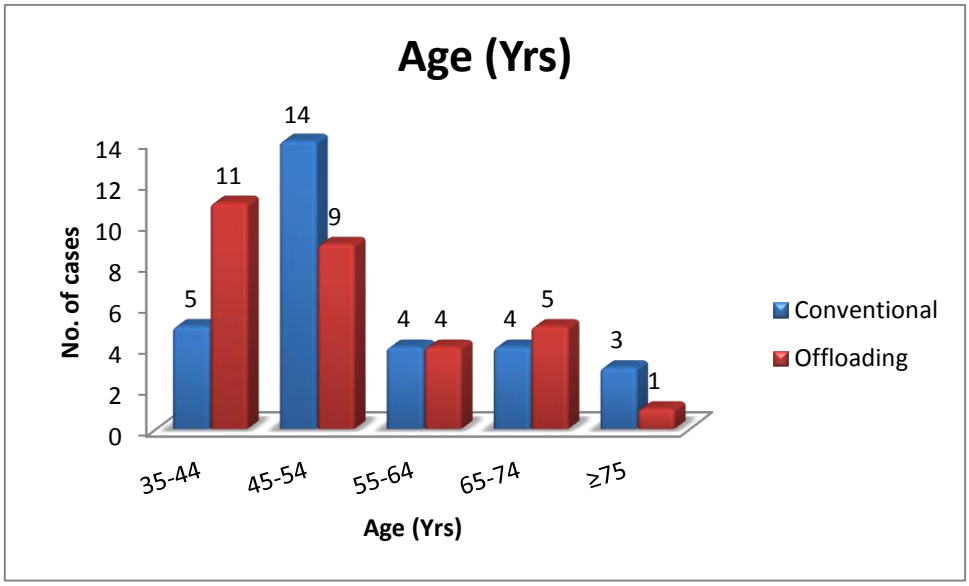
## STATISTICAL ANALYSIS

All characteristics were summarized descriptively. For continuous variables, the summary statistics of N, mean, standard deviation (SD) were used. For categorical data, the number and percentage were used in the data summaries. Chi-square ($\chi^2$)/ Freeman-Halton Fisher exact test was employed to determine the significance of differences between groups for categorical data. The difference of the means of analysis variables was tested with the unpaired t-test. If the p-value was $< 0.05$, then the results will be considered to be significant. Data were analyzed using SPSS software v.23.0.

**Table 1: Distribution of cases by Age among study groups**

| Age (Yrs) | Conventional | | Offloading | | p value |
|---|---|---|---|---|---|
| | N | % | N | % | |
| 35-44 | 5 | 16.7% | 11 | 36.7% | 0.349 |
| 45-54 | 14 | 46.7% | 9 | 30.0% | |
| 55-64 | 4 | 13.3% | 4 | 13.3% | |
| 65-74 | 4 | 13.3% | 5 | 16.7% | |
| ≥75 | 3 | 10.0% | 1 | 3.3% | |
| Total | 30 | 100.0% | 30 | 100.0% | |

**Figure 1: Distribution of cases by Age among study groups**

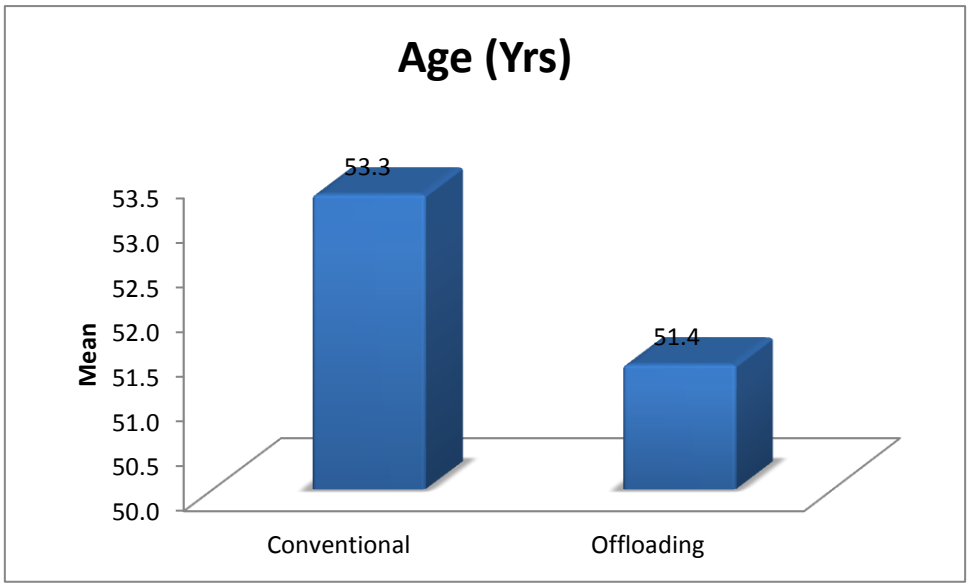



In this study, it is noted that about 60% of the patients in conventional dressing group and 43.3% of patients included in the offloading group belong to the age group of 45 to 65 yrs of age.

**Table 2: Mean Age among study groups**

| Age (Mean±SD) | Conventional | Offloading | p value |
|---|---|---|---|
| | 53.3±11.7 | 51.4±11.3 | 0.533 |

**Figure 2: Mean Age among study groups**



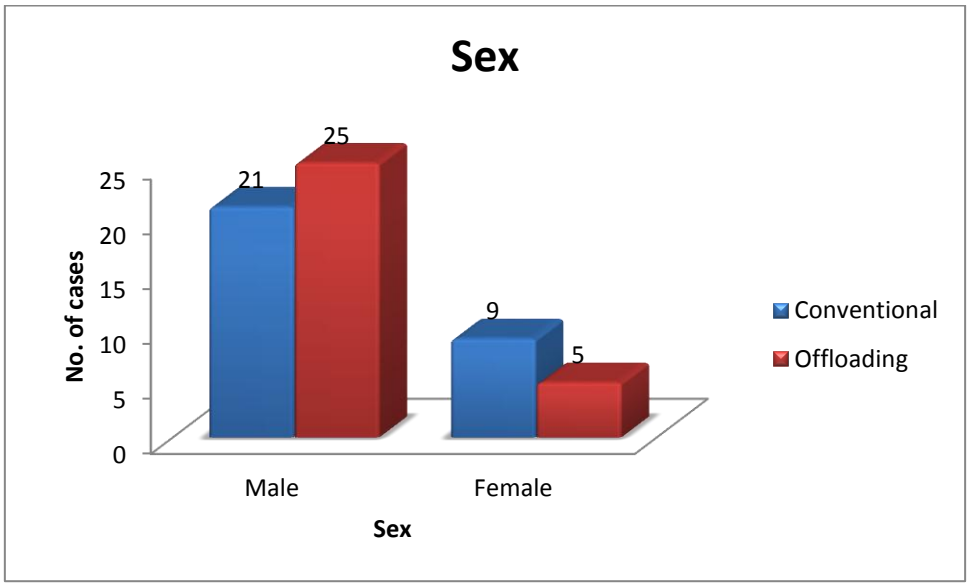

The mean age of the patients in years included in the study were 53.3±11.7 and 51.4±11.3 for the conventional dressing group and offloading group respectively.

**Table 3: Distribution of cases by Sex among study groups**

| Sex | Conventional | | Offloading | | p value |
|---|---|---|---|---|---|
| | N | % | N | % | |
| Male | 21 | 70.0% | 25 | 83.3% | 0.222 |
| Female | 9 | 30.0% | 5 | 16.7% | |
| Total | 30 | 100.0% | 30 | 100.0% | |

| Male female ratio | Conventional | Offloading |
|---|---|---|
| | 2.3:1 | 5.0:1 |

**Figure 3: Distribution of cases by Sex among study groups**




This study population showed a male preponderance with 76.66 % of patients included in the study being males with male female ratio being 3:1.

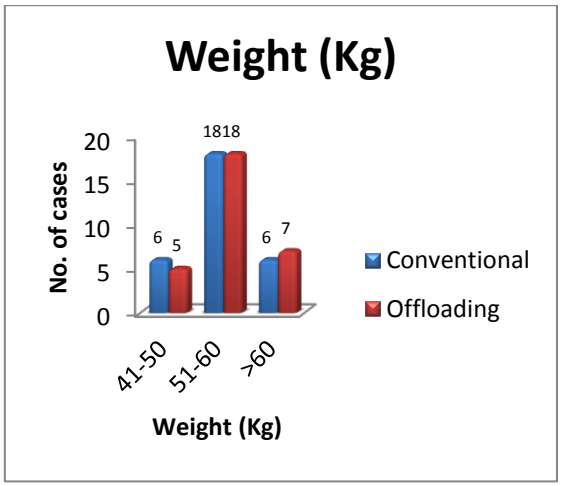
**Table 4: Distribution of cases by Weight among study groups**

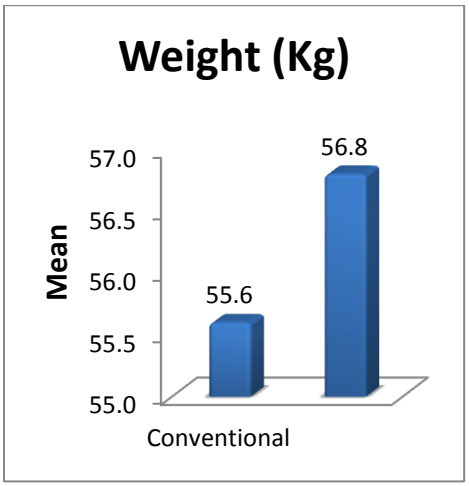
| Weight (Kg) | Conventional | | Offloading | | p value |
|---|---|---|---|---|---|
| | N | % | N | % | |
| 41-50 | 6 | 20.0% | 5 | 16.7% | 0.92 |
| 51-60 | 18 | 60.0% | 18 | 60.0% | |
| >60 | 6 | 20.0% | 7 | 23.3% | |
| Total | 30 | 100.0% | 30 | 100.0% | |

**Table 5: Mean Weight among study groups**

| Weight(Mean±SD) | Conventional | Offloading | p value |
|---|---|---|---|
| | 55.6±6.3 | 56.8±5.5 | 0.463 |

**Figure 4 and 5: Distribution of cases by Weight among study groups, Mean Weight among study groups**



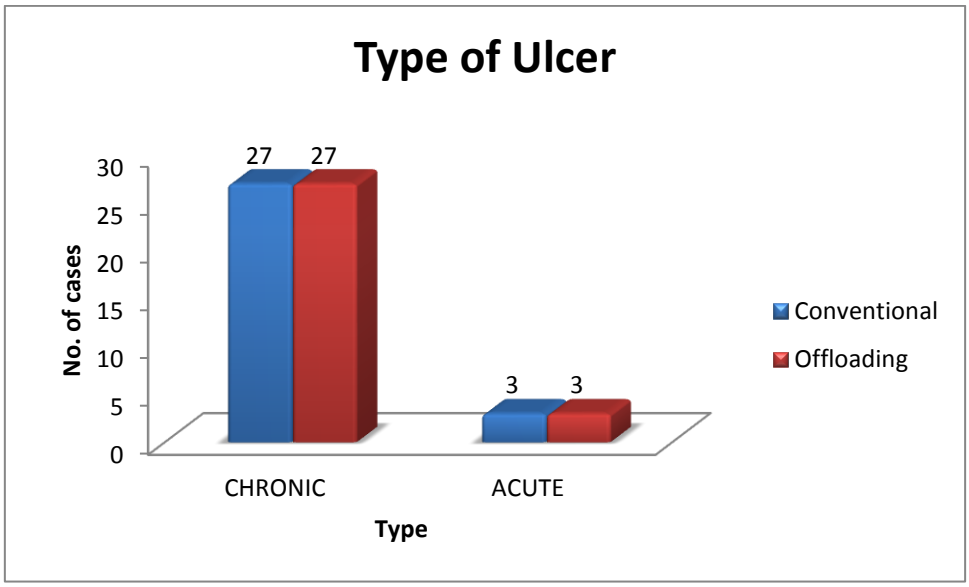




In this study the mean weight in kg of the patients included in the study was 55.6±6.3 and 56.8±5.5 for conventional dressing group and offloading group respectively and was comparably same. Therefore the plantar pressure are assumed to be uniform in both the groups taking in to consideration that no foot deformity was noted in any of the patients include in the study.

**Table 6: Distribution of cases by Type of Ulcer among study groups**

| Type of Ulcer | Conventional | | Offloading | | p value |
|---|---|---|---|---|---|
| | N | % | N | % | |
| Chronic | 27 | 90.0% | 27 | 90.0% | No Difference |
| Acute | 3 | 10.0% | 3 | 10.0% | |
| Total | 30 | 100.0% | 30 | 100.0% | |

**Figure 6: Distribution of cases by Type of Ulcer among study groups**

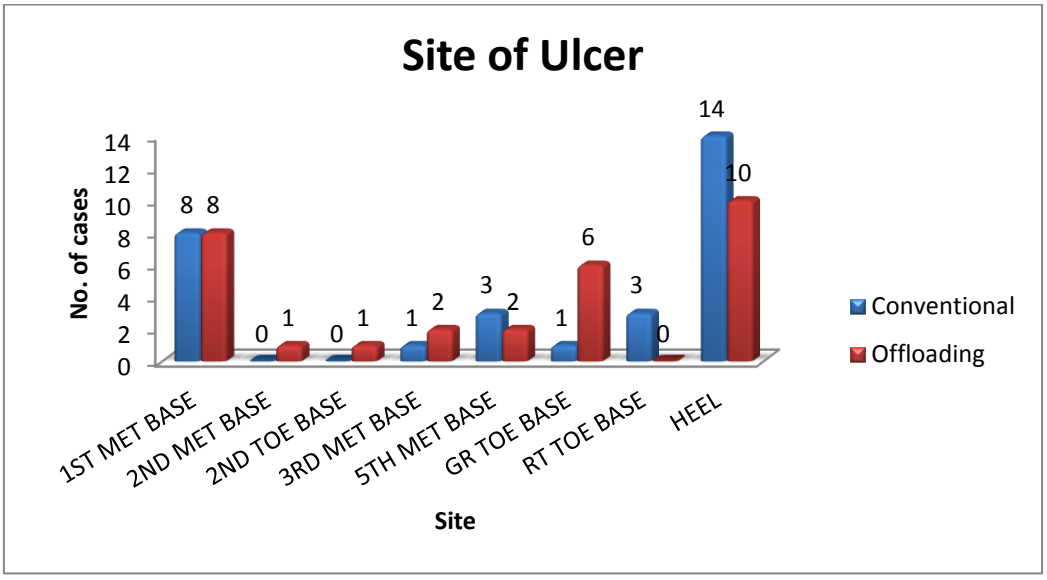



90% of the ulcers included in this study were chronic plantar foot ulcers i.e. trophic ulcers. Demarcation between acute and chronic ulcers was that all the ulcers of less than 1week old were considered to be acute, 3 patients in conventional dressing group and 3 patients in offloading group were acute which were secondary to trauma.

**Table 7: Distribution of cases by Site of Ulcer among study groups**

| Site of Ulcer | Conventional | | Offloading | | p value |
|---|---|---|---|---|---|
| | N | % | N | % | |
| 1ST MET BASE | 8 | 26.7% | 8 | 26.7% | 0.202 |
| 2ND MET BASE | 0 | 0.0% | 1 | 3.3% | |
| 2ND TOE BASE | 0 | 0.0% | 1 | 3.3% | |
| 3RD MET BASE | 1 | 3.3% | 2 | 6.7% | |
| 5TH MET BASE | 3 | 10.0% | 2 | 6.7% | |
| GR TOE BASE | 1 | 3.3% | 6 | 20.0% | |
| FIFTH TOE BASE | 3 | 10.0% | 0 | 0.0% | |
| HEEL | 14 | 46.7% | 10 | 33.3% | |
| Total | 30 | 100.0% | 30 | 100.0% | |

**Figure 7: Distribution of cases by Site of Ulcer among study groups**

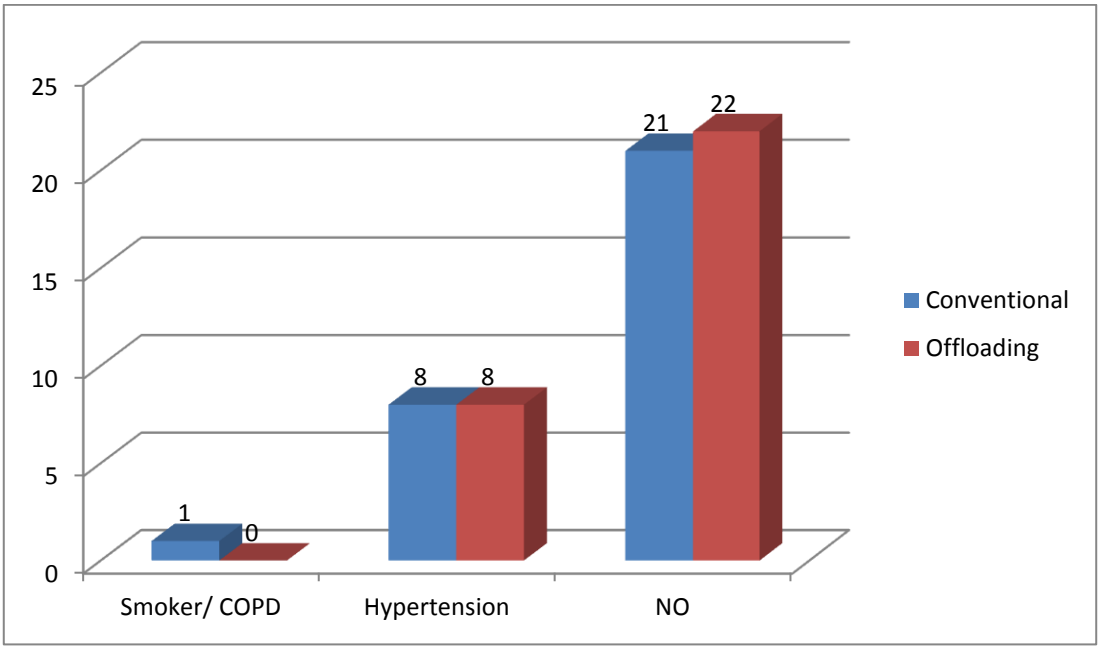



It was noted in this study that the most common site for ulceration was the heel with incidence of 40% followed by base of the 1st metatarsal head with 26.66%.

**Table 8: Distribution of cases by Co-morbidities among study groups**

| Co-morbidities | Conventional | | Offloading | | p value |
|---|---|---|---|---|---|
| | N | % | N | % | |
| Smoker/ COPD | 1 | 3.3% | 0 | 0.0% | 0.478 |
| Hypertension | 8 | 26.7% | 8 | 26.7% | |
| No co-morbidities | 21 | 46.7% | 22 | 76.7% | |
| Total | 30 | 100.0% | 30 | 100.0% | |

**Figure 8: Distribution of cases by Co-morbidities among study groups**

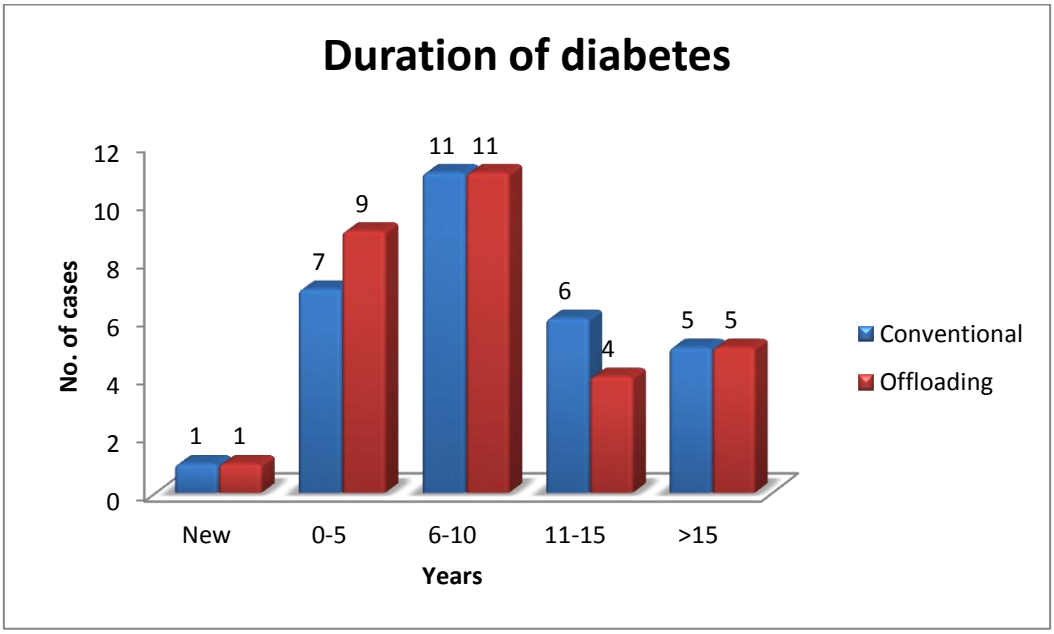



Of the patients included in the study, one patient in the conventional group was a known case of COPD, and 8 patients in each group were hypertensive. Rest of the patients did not have any co-morbidities.

**Table 9: Distribution of cases by Duration of diabetes among study groups**

| Duration of diabetes (Yrs) | Conventional | | Offloading | | p value |
|---|---|---|---|---|---|
| | N | % | N | % | |
| New | 1 | 3.3% | 1 | 3.3% | 0.957 |
| 0-5 | 7 | 23.3% | 9 | 30.0% | |
| 6-10 | 11 | 36.7% | 11 | 36.7% | |
| 11-15 | 6 | 20.0% | 4 | 13.3% | |
| >15 | 5 | 16.7% | 5 | 16.7% | |
| Total | 30 | 100.0% | 30 | 100.0% | |

**Figure 9: Distribution of cases by Duration of diabetes among study groups**

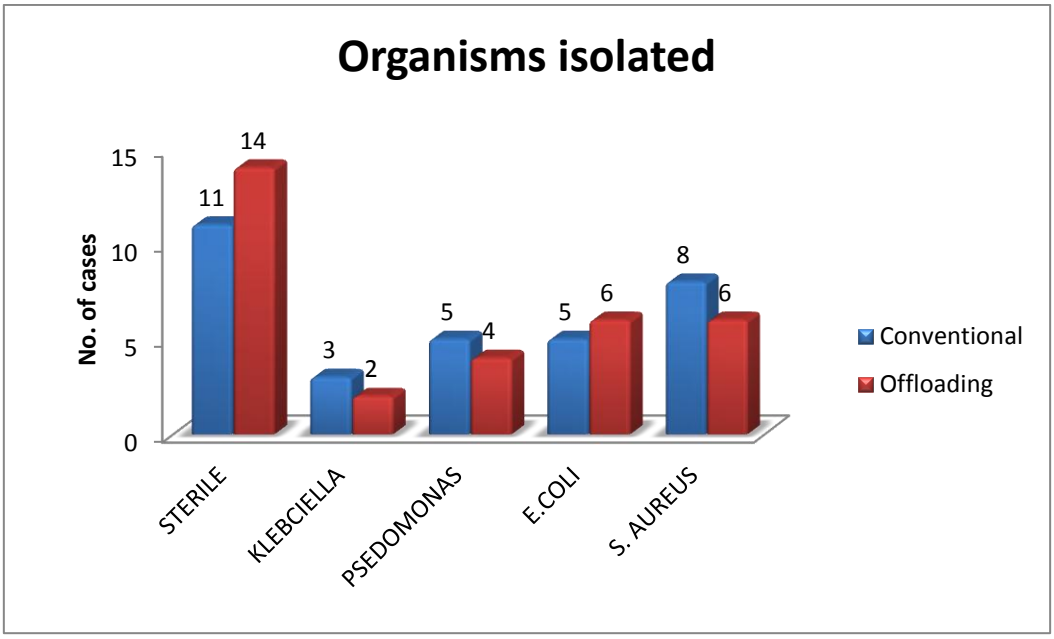



It was noted that patient included in the study where chronic type two diabetes mellitus patients with 70% having the disease for more than 6 to 10 years.

**Table 10: Distribution of cases by Organisms isolated among study groups**

| Organisms isolated | Conventional | | Offloading | | p value |
|---|---|---|---|---|---|
| | N | % | N | % | |
| STERILE | 11 | 36.7% | 14 | 46.7% | 0.432 |
| KLEBCIELLA | 3 | 10.0% | 2 | 6.7% | 0.640 |
| PSEDOMONAS | 5 | 16.7% | 4 | 13.3% | 0.718 |
| E.COLI | 5 | 16.7% | 6 | 20.0% | 0.739 |
| S. AUREUS | 8 | 26.7% | 6 | 20.0% | 0.542 |

**Figure 10: Distribution of cases by Organisms isolated among study groups**

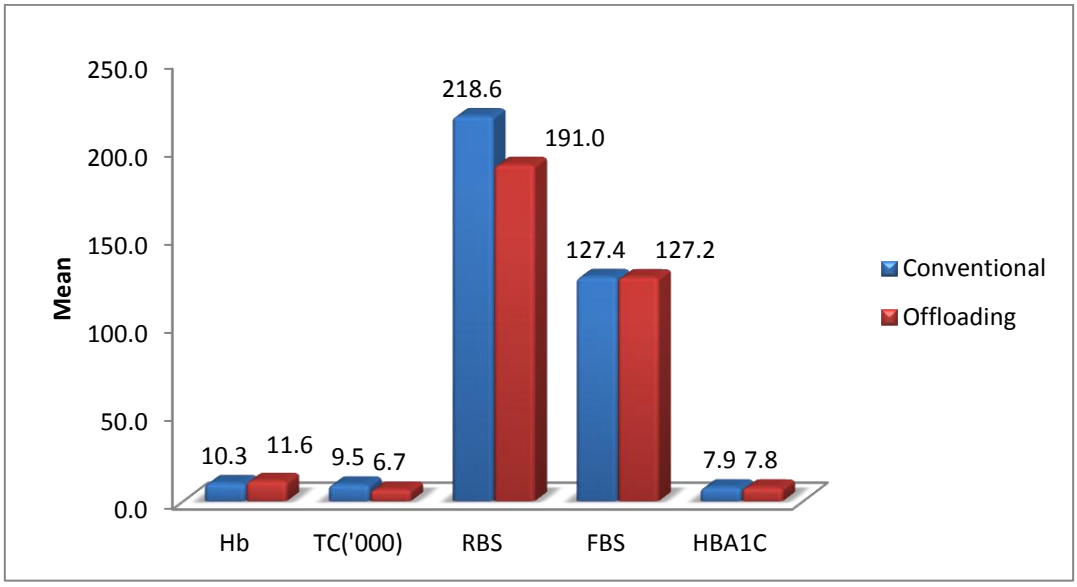



When it comes to organisms isolated from the ulcers included in the study, 41.66% cultures were sterile for any organisms. Staphylococcus Aureus was isolated from 23.33% of patients making it the most common organism isolated followed by E. Coli and Pseudomonas Aeruginosa with 18.33% and 15% respectively being the next most common organisms isolated. It was noted that two of the ulcers had more than one organisms isolated.

**Table 11: Mean Parameters among study groups**

| Parameters (Mean±SD) | Conventional | Offloading | p value |
|---|---|---|---|
| Hb gm% | 10.3±2 | 11.6±1.8 | 0.01 (Sig) |
| TC('000) | 9.5±5.7 | 6.7±2.9 | 0.02 (Sig) |
| RBS | 218.6±64.5 | 191±58.8 | 0.089 |
| FBS | 127.4±17.6 | 127.2±18.4 | 0.96 |
| HBA1c | 7.9±1.1 | 7.8±1.1 | 0.719 |

**Figure 11: Mean Parameters among study groups**

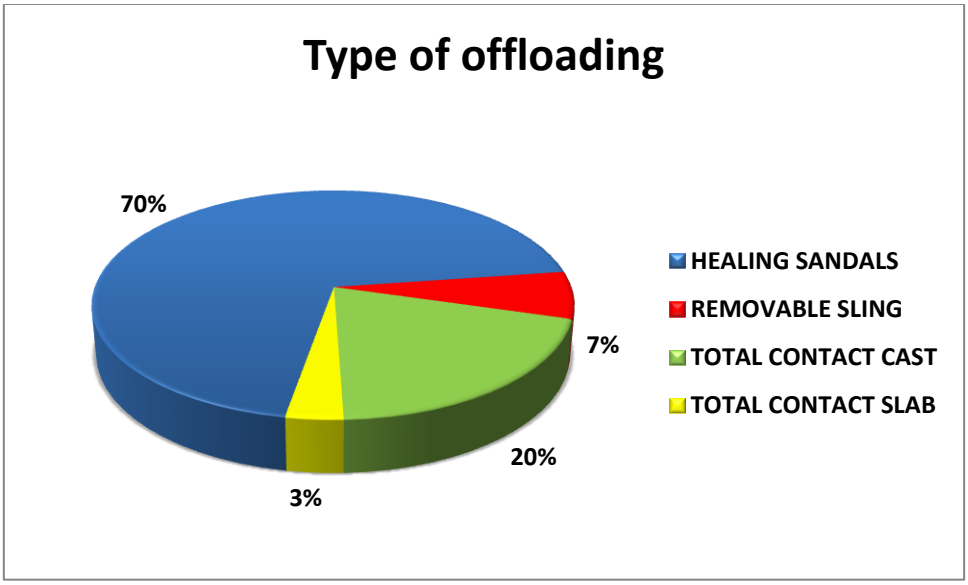



Study noted that the patients were significantly anaemic with mean Hb of 10.3±2 and 11.6±1.8 in conventional dressing group and offloading group respectively. Patients were also chronic uncontrolled diabetic with mean FBS of 127.4±18 and mean HBa1c of 7.85±1.1

**Table 12: Distribution of cases by Type of offloading**

| Type of offloading | Offloading | |
|---|---|---|
| | N | % |
| HEALING SANDALS | 21 | 70.0% |
| REMOVABLE SLING | 2 | 6.7% |
| TOTAL CONTACT CAST | 6 | 20.0% |
| TOTAL CONTACT SLAB | 1 | 3.3% |
| Total | 30 | 100.0% |

**Figure 12: Distribution of cases by Type of offloading**

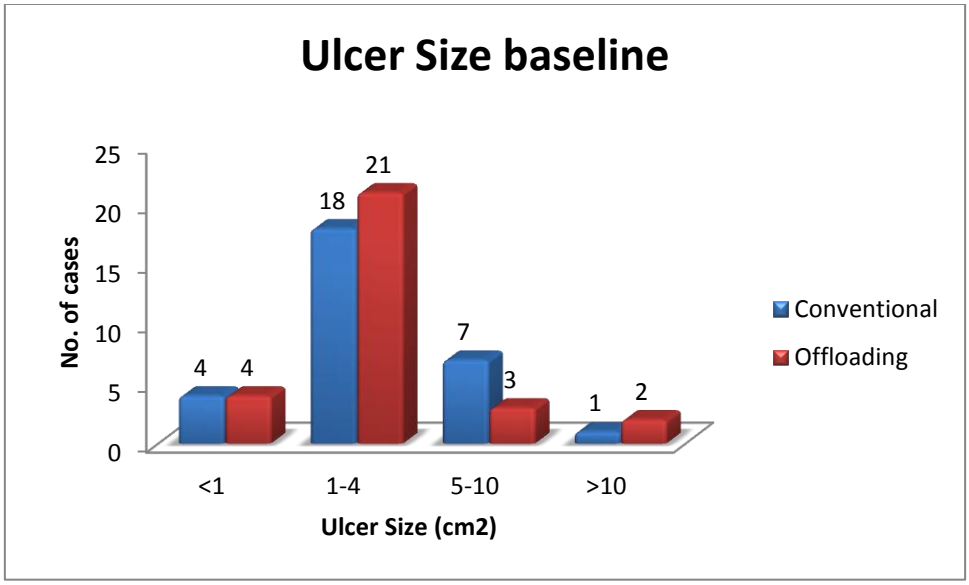



Four different types of offloading techniques were used in patients in this study with healing sandals being the most common type with 70% followed by total contact cast used in 20% of patients and then removable slings and slabs in 6.7% and 3.3% patients respectively.

**Table 13: Distribution of cases by Ulcer Size at baseline among study groups**

| Ulcer Size (cm2) baseline | Conventional | | Offloading | | p value |
|---|---|---|---|---|---|
| | N | % | N | % | |
| <1 | 4 | 13.3% | 4 | 13.3% | 0.539 |
| 1-4 | 18 | 60.0% | 21 | 70.0% | |
| 5-10 | 7 | 23.3% | 3 | 10.0% | |
| >10 | 1 | 3.3% | 2 | 6.7% | |
| Total | 30 | 100.0% | 30 | 100.0% | |

**Figure 13: Distribution of cases by Ulcer Size at baseline among study groups**

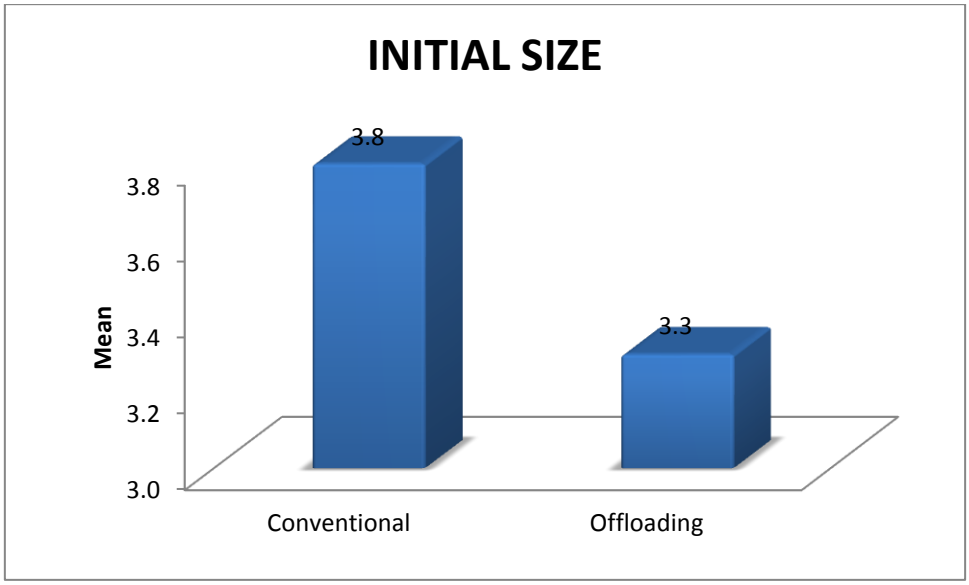



Of the ulcers included in the study, it was noted that 78.33% of ulcers were smaller than 4 $cm^2$.

**Table 14: Mean Ulcer Size at baseline among study groups**

| Mean Ulcer Size (cm2) baseline (Mean±SD) | Conventional | Offloading | p value |
|---|---|---|---|
| | 3.8±3.5 | 3.3±3.6 | 0.632 |

**Figure 14: Mean Ulcer Size at baseline among study groups**

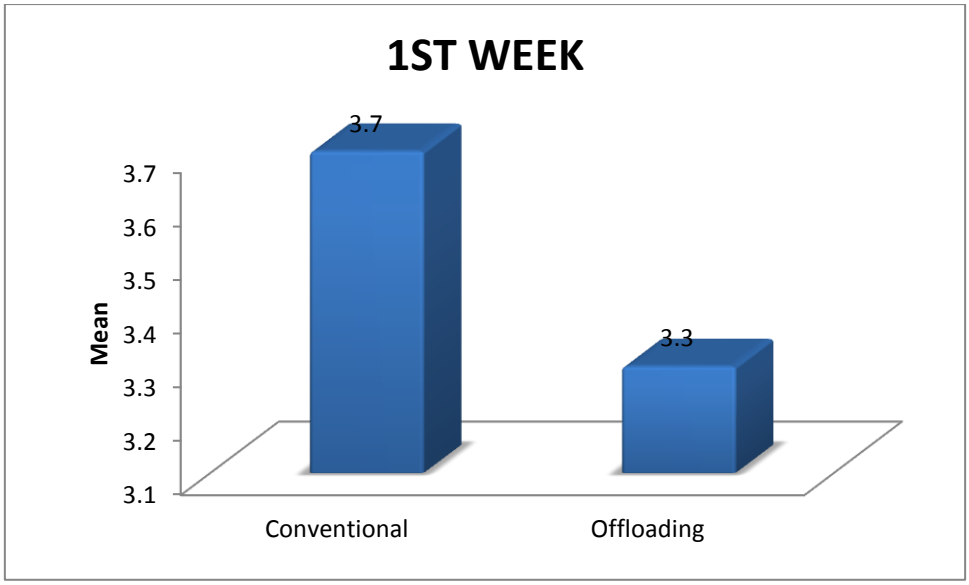



The mean size of the ulcers included in the both the study groups were 3.8 and 3.3 respectively and were comparable.

**Table 15: Mean Ulcer Size at 1st week among study groups**

| Mean Ulcer Size (cm2) Ist week (Mean±SD) | Conventional | Offloading | p value |
|---|---|---|---|
| | 3.7±3.5 | 3.3±3.3 | 0.651 |

**Figure 15: Mean Ulcer Size at 1st week among study groups**

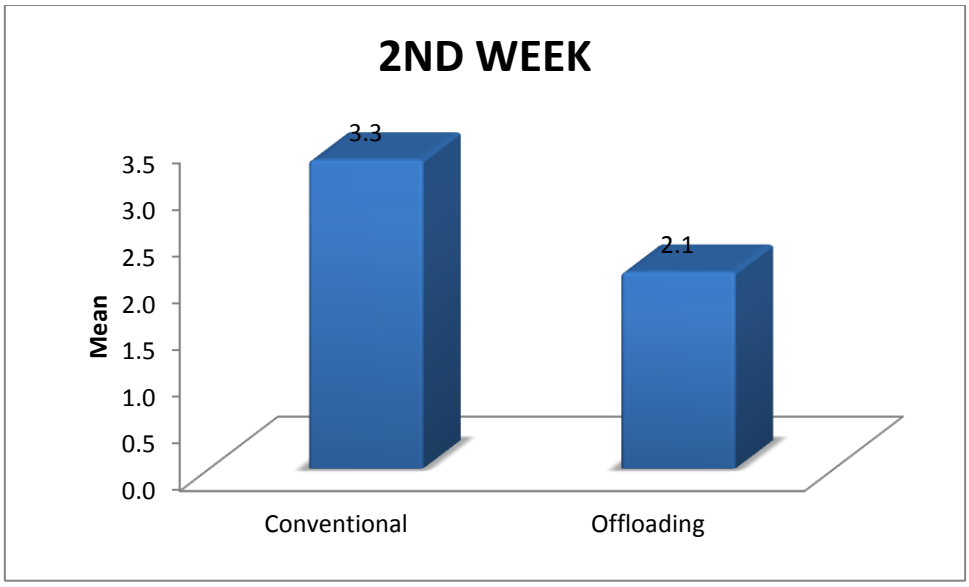



Mean ulcer size by the end of 1st week did not have any significant change in the ulcer of both the study groups.

**Table 16: Mean Ulcer Size at 2nd week among study groups**

| Mean Ulcer Size (cm2) 2nd week (Mean±SD) | Conventional | Offloading | p value |
|---|---|---|---|
| | 3.3±3.3 | 2.1±2.7 | 0.13 |

**Figure 16: Mean Ulcer Size at 2nd week among study groups**

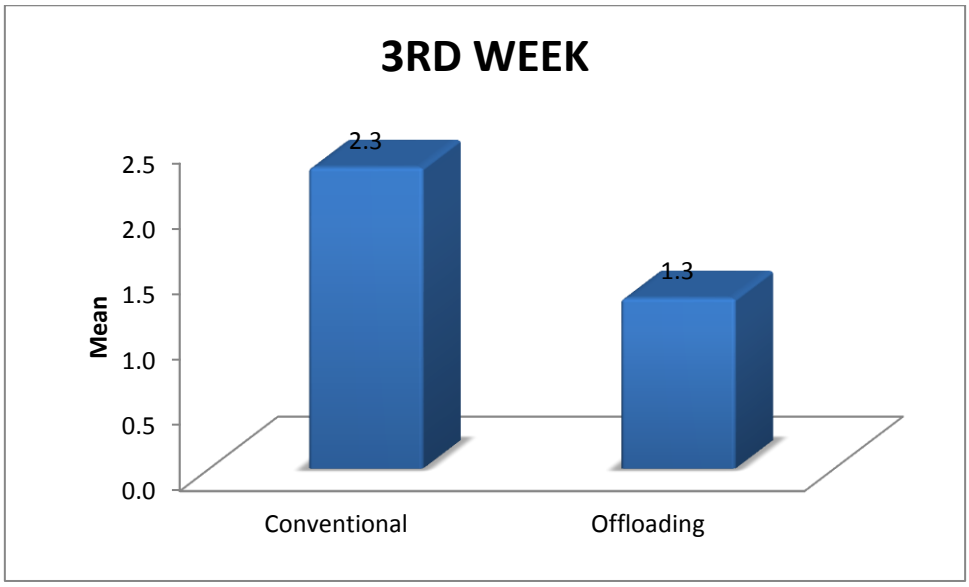



Mean reduction in size by the end of 2nd week is comparatively better in the offloading group though not significant.

**Table 17: Mean Ulcer Size at 3rd week among study groups**

| Mean Ulcer Size (cm2) 3rd week (Mean±SD) | Conventional | Offloading | p value |
|---|---|---|---|
| | 2.3±2.4 | 1.3±1.9 | 0.071 |

**Figure 17: Mean Ulcer Size at 3rd week among study groups**

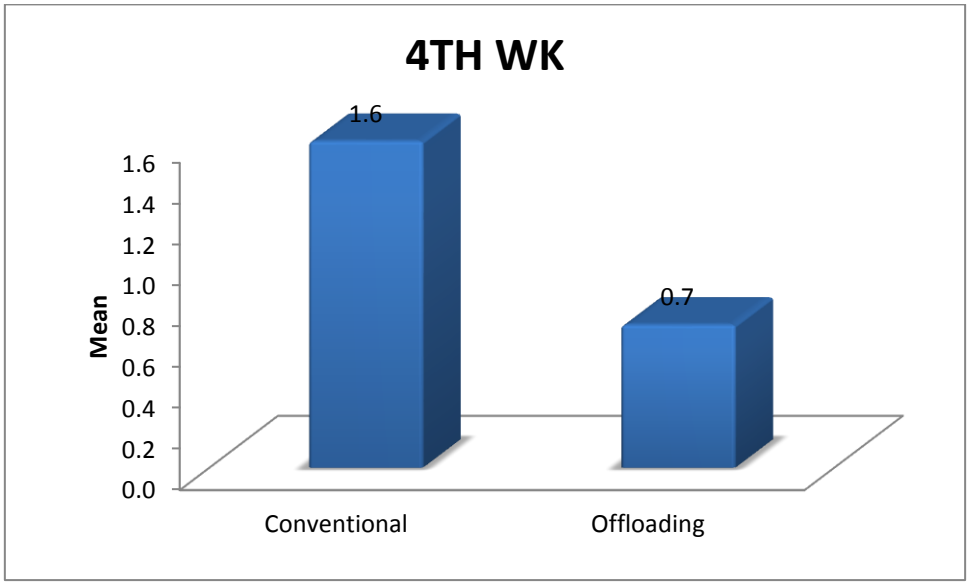



Mean reduction in size by the end of 3rd week is again comparatively better in the offloading group but not significant.

**Table 18: Mean Ulcer Size at 4th week among study groups**

| Mean Ulcer Size (cm2) 4th week (Mean±SD) | Conventional | Offloading | p value |
|---|---|---|---|
| | 1.6±1.6 | 0.7±1.1 | 0.025 (Sig) |

**Figure 18: Mean Ulcer Size at 4th week among study groups**

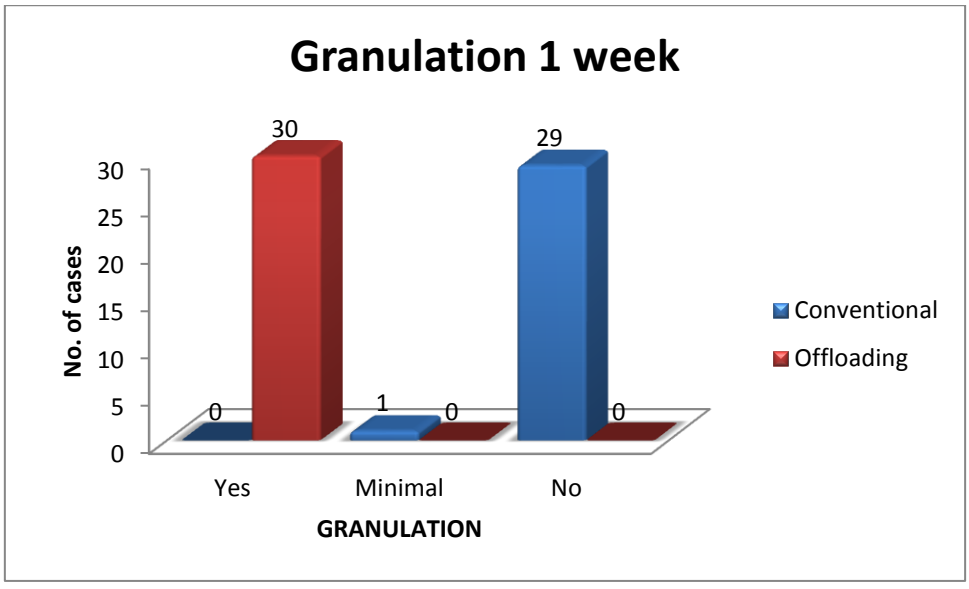



Mean reduction in size by the end of 4th week is statistically significant in the offloading group when compared to conventional dressing group with P value of 0.025

**Table 19: Distribution of cases by amount of Granulation at 1st week among study groups**

| Granulation 1 week | Conventional | | Offloading | | p value |
|---|---|---|---|---|---|
| | N | % | N | % | |
| Complete | 0 | 0.0% | 30 | 100.0% | <0.001 (Sig) |
| Minimal/Partial | 1 | 3.3% | 0 | 0.0% | |
| No | 29 | 96.7% | 0 | 0.0% | |
| Total | 30 | 100.0% | 30 | 100.0% | |

**Figure 19: Distribution of cases by amount of Granulation at 1st week among study groups**

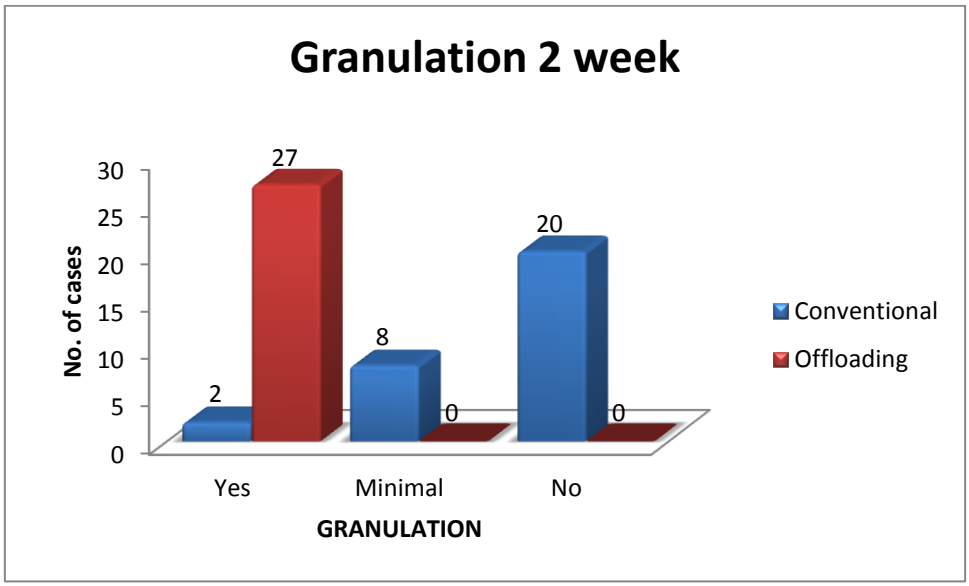



It is noted that there is significant improvement in formation of granulation tissue in the offloading group when compared to the conventional dressing group by the end of 1st week with P value of <0.001

**Table 20: Distribution of cases by amount of Granulation at 2nd week among study groups**

| Granulation 2 week | Conventional | | Offloading | | p value |
|---|---|---|---|---|---|
| | N | % | N | % | |
| Complete | 2 | 6.7% | 27 | 100.0% | <0.001 (Sig) |
| Minimal/Partial | 8 | 26.7% | 0 | 0.0% | |
| No | 20 | 66.7% | 0 | 0.0% | |
| Total | 30 | 100.0% | 27 | 100.0% | |

**Figure 20: Distribution of cases by amount of Granulation at 2nd week among study groups**

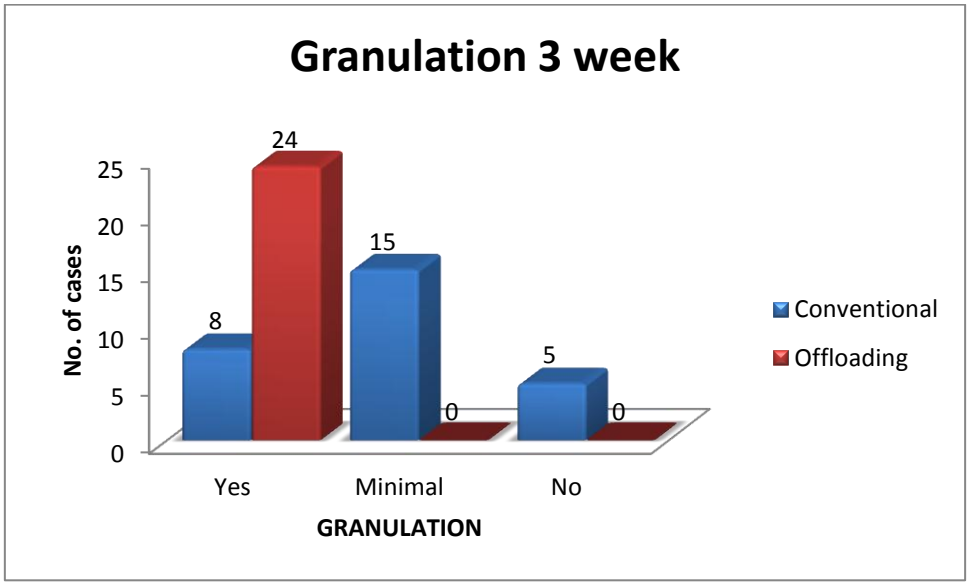



It is again noted that there is significant improvement in formation of granulation tissue in the offloading group when compared to the conventional dressing group by the end of 2nd week with P value of <0.001 . However minimal pale granulation tissue is seen in 26.7% of ulcers in the conventional dressing group.

**Table 21: Distribution of cases by amount of Granulation at 3rd week among study groups**

| Granulation 3 week | Conventional | | Offloading | | p value |
|---|---|---|---|---|---|
| | N | % | N | % | |
| Complete | 8 | 26.7% | 24 | 80.0% | <0.001 (Sig) |
| Minimal/Partial | 15 | 46.7% | 0 | 0.0% | |
| No | 5 | 16.7% | 0 | 0.0% | |
| Total | 30 | 100.0% | 30 | 100.0% | |

**Figure 21: Distribution of cases by amount of Granulation at 3rd week among study groups**

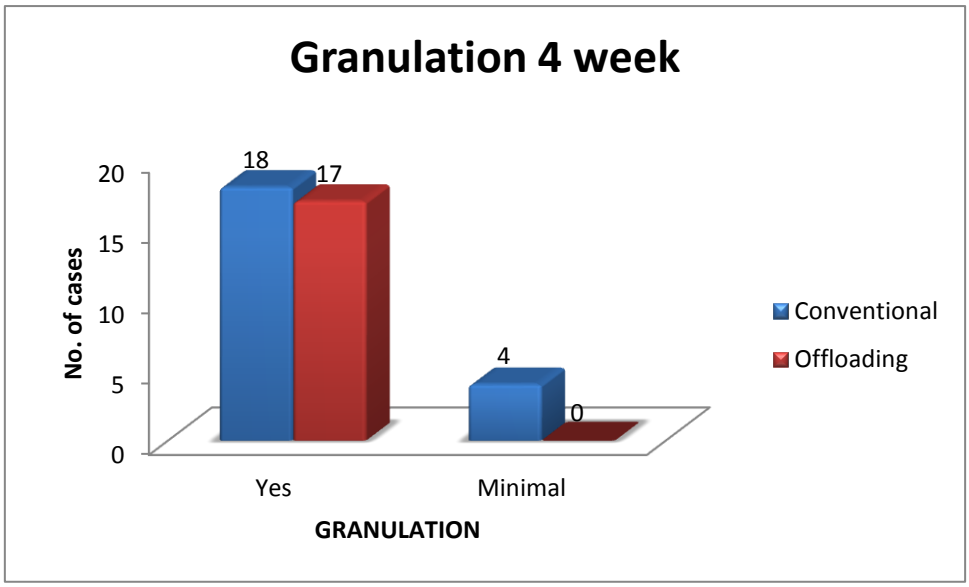



By the end of 3rd week 20% of the ulcers in the offloading group have healed and rest of 80% are completely covered with healthy granulation tissue compared to 46.7% pale and 26.7% ulcers with healthy granulation tissue in the conventional dressing group which is again statistically significant with P value of <0.001

**Table 22: Distribution of cases by amount of Granulation at 4th week among study groups**

| Granulation 4 week | Conventional | | Offloading | | p value |
|---|---|---|---|---|---|
| | N | % | N | % | |
| Complete | 18 | 60.0% | 17 | 56.7% | 0.074 |
| Minimal/Partial | 4 | 13.3% | 0 | 0.0% | |
| Total | 30 | 100.0% | 30 | 100.0% | |

**Figure 22: Distribution of cases by amount of Granulation at 4th week among study groups**

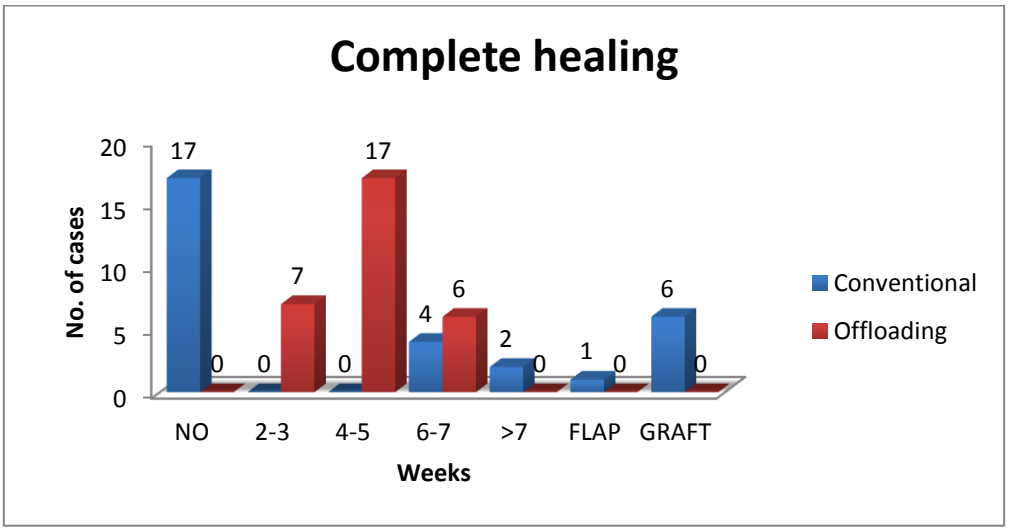



By the end of 4th week 73.3% of ulcers in the conventional group are still healing with 13.3% still having unhealthy granulation tissue. On the other hand the offloading group of patients noted significantly good healing with 43.3% ulcers healed and rest 56.7% ulcers nearing complete healing.

**Table 23: Distribution of cases by complete healing among study groups**

| Complete healing (Weeks) | Conventional | | Offloading | | p value |
|---|---|---|---|---|---|
| | N | % | N | % | |
| No | 17 | 56.70% | 0 | 0.00% | <0.001 (Sig) |
| 2-3 | 0 | 0.00% | 7 | 23.30% | |
| 4-5 | 0 | 0.00% | 17 | 56.70% | |
| 6-7 | 4 | 13.30% | 6 | 20.00% | |
| >7 | 2 | 6.70% | 0 | 0.00% | |
| Flap | 1 | 3.30% | 0 | 0.00% | |
| Graft | 6 | 20.00% | 0 | 0.00% | |
| Total | 30 | 100.00% | 30 | 100.00% | |

**Figure 23: Distribution of cases by complete healing among study groups**

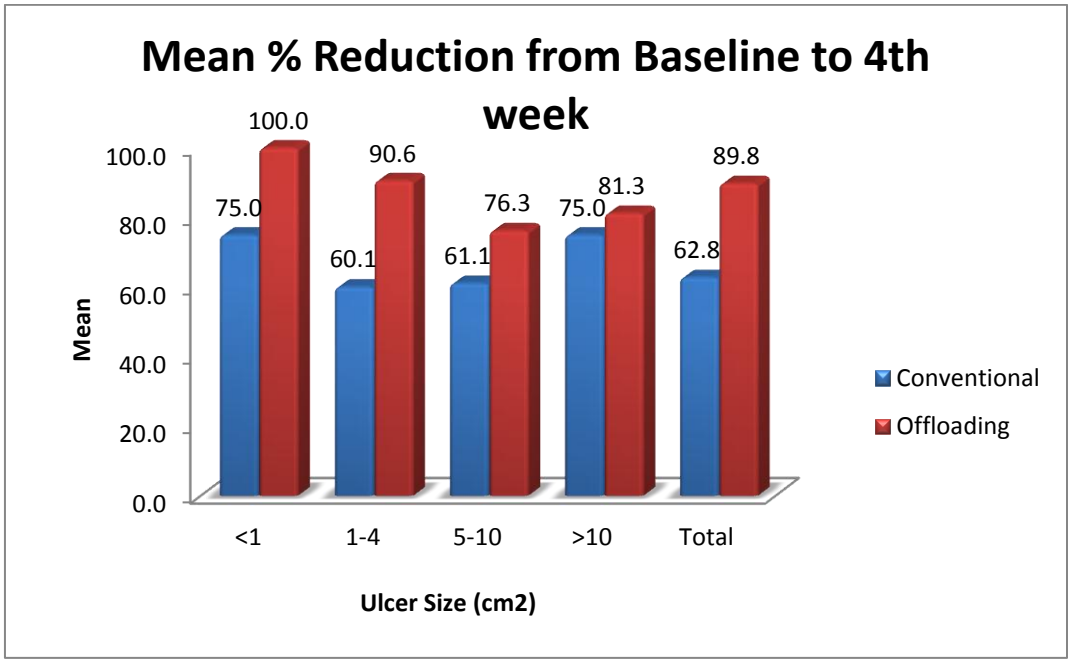



On follow up of the patients included in the study, it was noted that ulcers included in the offloading group completely healed by the end of 6 to 7 weeks where as the ulcers in the conventional group, only 20% healed, 23.3% patients underwent flap or split thickness skin grafting to attain complete healing, with 56.7% ulcers remained unhealed. This was again statistically significant with P value of <0.001

**Table 24: Percent Reduction in Ulcer size from Baseline to 4th week**

| Ulcer Size (cm2) | Conventional | | | Offloading | | | p value |
|---|---|---|---|---|---|---|---|
| | N | Mean Reduction (%) | SD | N | Mean Reduction (%) | SD | |
| <1 | 4 | 75.0 | 50.0 | 4 | 100.0 | 0.0 | 0.356 |
| 1-4 | 18 | 60.1 | 30.2 | 21 | 90.6 | 12.5 | <0.001 (Sig) |
| 5-10 | 7 | 61.1 | 22.4 | 3 | 76.3 | 21.0 | 0.349 |
| >10 | 1 | 75.0 | NA | 2 | 81.3 | 8.8 | 0.667 |
| Total | 30 | 62.8 | 30.5 | 30 | 89.8 | 13.4 | <0.001 (Sig) |

**Figure 24: Percent Reduction in Ulcer size from Baseline to 4th week**




In this study it was noted that when the ulcer size is less than 1cm2 the mean percentage in reduction of the ulcer size was 75% in the conventional group when compared to 100% in the offloading group.

In ulcers size between 1 to 4 cm2, mean percentage in reduction of the ulcer size was 60.1% in the conventional group when compared to 90.6% in the offloading group which was statistically significant with P value of <0.001.

In ulcers of size between 5 to 10 cm2, mean percentage in reduction of the ulcer size was 61.1% in the conventional group when compared to 81.3% in the offloading group.

In ulcers size above 10 cm2, mean percentage in reduction of the ulcer size was 75% in the conventional group when compared to 81.3% in the offloading group.

With this the mean reduction in size in the conventional dressing group was found to be 62.8% when compared to 89.8% in the offloading group which was again statistically significant (P value <0.001).

# DISCUSSION

In this study, it is noted that about 60% of the patients in conventional dressing group and 43.3% of patients included in the offloading group belong to the age group of 45 to 65 yrs of age suggesting that diabetic foot ulcers are more common in older age groups. 90% of the ulcers included in this study were chronic plantar foot ulcers i.e. trophic ulcers.

The mean age of the patients in years included in this study were 53.3±11.7 and 51.4±11.3 for the conventional dressing group and offloading group respectively. This is found to be in accordance with the mean age of patients as in different similar studies conducted like 57.2±12.9 years noted in Lindy Begg et al 2016 and slightly lower when compared to 65.6±9.9 years noted in David Armstrong et al 2005.

| | |
|---|---|
| Our Study | 53.3±11.7 and 51.4±11.3 years |
| Lindy Begg et al 2016 | 57.2±12.9 years |
| David Armstrong et al 2005. | 65.6±9.9 years |

The sex ratio of the patients included in the study showed a male preponderance with 76.66 % of patients included in the study being males with male female ratio being 3:1. Other similar studies also noted high number of male patients included in the study with 88% in David Armstrong et al 2005 and 82.4 % in Lindy Begg et al 2016.

| | |
|---|---|
| Our Study | 76.66 % |
| David Armstrong et al 2005 | 88% |
| Lindy Begg et al 2016 | 82.4 % |

The mean weight of the patients included in this study in kilograms were 55.6±6.3 and 56.8±5.5 respectively for conventional dressing group and offloading group respectively, which was comparably same in both groups. Therefore the plantar pressure are assumed to be uniform in both the groups taking in to consideration that no foot deformity was noted in any of the patients include in the study. Weights of the patients were found to be on the lower side when compared to study conducted by Lindy Begg et al 2016.

The most common site for ulceration in the patients included in both the groups collectively was the heel with incidence of 40% followed by base of the 1st metatarsal head with 26.66%. These results were again consistent with the findings of study conducted by Lindy Begg et al 2016 with heel ulcerations at 35% and base of 1st metatarsal head at 23%.

| | |
|---|---|
| Our study | heel 40%<br>base of the 1st metatarsal head 26.66%. |
| Lindy Begg et al 2016 | heel 35%<br>base of 1st metatarsal head 23%. |

It was observed that the patients who were included in the study and patients in general who are found to have a tendency to develop foot ulcerations were chronically diabetic. In our study more than 70% of the patients were known to be on diabetic medication for more than 6 to 10yrs and possibly undiagnosed diabetic for a much longer time. This was again noted in almost all the studies that conducted studies on diabetic foot.

Our study noted that the patients were significantly anaemic with mean haemoglobin levels at 10.3±2 and 11.6±1.8 in conventional dressing group and offloading group respectively. Almost all patients in the study groups were uncontrolled diabetic and on irregular treatment with mean FBS values of 127.4±18 and mean HBA1c of 7.85±1.1 which was in consistent with mean HBA1c of 8.2±1.4 in the study conducted by David Armstrong et al in 2005.

| | |
|---|---|
| Our study | HBA1c of 7.85±1.1 |
| David Armstrong et al in 2005. | HBA1c of 8.2±1.4 |

When it comes to organisms isolated from the ulcers included in the study, 41.66% cultures were found to be sterile for any organisms. However, Staphylococcus Aureus was isolated from 23.33% of patients making it the most common organism isolated followed by E.Coli and Pseudomonas Aeruginosa with 18.33% and 15% respectively being the next most common organisms isolated. This was again in accordance with the study conducted by Onkar Singh et al in 2011 which showed Staphylococcus Aureus as most common isolated organism with 45% followed by E.Coli and Pseudomonas Aeruginosa with 20% each.

| | |
|---|---|
| Our study | Staphylococcus Aureus 23.33%<br>E.Coli 18.33%<br>Pseudomonas Aeruginosa 15% |
| Onkar Singh et al in 2011 | Staphylococcus Aureus 45%<br>E.Coli 20%<br>Pseudomonas Aeruginosa 20% |

Four different types of offloading techniques were used on patients in this study with healing sandals being the most common type with 70% followed by total contact cast used in 20% of patients and then removable slings and slabs in 6.7% and 3.3% patients respectively.

Of the ulcers included in the study, it was noted that 78.33% of ulcers were smaller than 4 cm2. The mean size of the ulcers included in the both the study groups were 3.8±3.5 and 3.3±3.6 cm2 respectively. These sizes were larger than the size of the ulcers included in different similar studies like 2.3±1.2 cm2 as in David Armstrong et al 2005, 1.8 to 2.8 cm2 as in Mueller et al 1989, 1.4 cm2 to 2.2 cm2 included in Faglia et al 2010 and comparable with 4.2±3 cm2 in Van de Weg et al 2008.

| | |
|---|---|
| Our study | 3.8±3.5 and 3.3±3.6 $cm^2$ |
| David Armstrong et al 2005 | 2.3±1.2 $cm^2$ |
| Mueller et al 1989 | 1.8 to 2.8 $cm^2$ |
| Faglia et al 2010 | 1.4 cm2 to 2.2 $cm^2$ |
| Van de Weg et al 2008 | 4.2±3 $cm^2$ |

It was noted that there was significant improvement in formation of granulation tissue in the offloading group when compared to the conventional dressing group by the end of 1$^{st}$ week and kept improving over the next 3 weeks with P value of <0.001 . In the conventional dressing group by the end of 2$^{nd}$ week minimal pale granulation tissue was seen in 26.7% of ulcers which marginally improved over 3$^{rd}$ week with 46.7% ulcers had pale unhealthy granulation and 26.7% ulcers with healthy granulation tissue.

By the end of 4th week 73.3% of ulcers in the conventional group are still healing with 13.3% still having unhealthy granulation tissue. On the other hand the offloading group of patients noted significantly good healing with 43.3% ulcers completely healed and rest 56.7% ulcers nearing complete healing.

Mean ulcer size by the end of 1st week did not have any significant change in the ulcer of both the study groups. Mean reduction in size by the end of 2nd and 3rd week are comparatively better in the offloading group though not significant. But mean reduction in size by the end of 4th week is statistically significant in the offloading group when compared to conventional dressing group with P value of 0.025

Healing rate of the ulcers in this study when compared between the conventional dressing with offloading for trophic ulcers were found to be statistically significant with healing of 89.8% in the offloading group and P value of <0.001 . It was noted that ulcers had significantly faster healing in the offloading group when the size of the ulcers were between 1-4 cm2 with P value of <0.001. The average healing time in study was 35±14 days. This is consistent with the most the study mentioned below.

| | |
|---|---|
| Our study | Complete healing in 5-6 weeks |
| Mandakini offloading device | Complete healing in 4–6 weeks. |
| Samadhan system | 85% achieved complete healing in 6weeks |

| STUDY | PERCENTAGE HEALING | HEALING TIME |
|---|---|---|
| Our Study | 89.8% | 35 ± 14 days |
| Meuller et al 1989 | 90% | 42±29 days |
| Armstrong et al 2001 | 89.5%. | 33.5±5.9 days |
| Birke et al 2002 | 81% | 45.5±43.4 days |
| Zimny et al 2003 | - | 75.2 days |
| Armstrong et al 2005 | - | 41.6±18.7 days |
| Katz et al 2005 | - | 28-35 days |
| Piaggesi et al 2007 | 95% | 6.5±4.4wks |
| Van de Weg et al 2008 | - | 59-90 days |
| Dumont et al 2009 | 70.1% | 43-99 days |
| Faglia et al 2010 | 73.9% | 39±4.2 days |
| Miyan et al 2013 | 95% | 34-45 days |

On follow up of the patients included in the study, it was noted that ulcers included in the offloading group completely healed by the end of 6 to 7 weeks where as the ulcers in the conventional group, only 20% healed, 23.3% patients underwent flap or split thickness skin grafting to attain complete healing, and 56.7% ulcers remained unhealed. This was again statistically significant with P value of <0.001

# SUMMARY

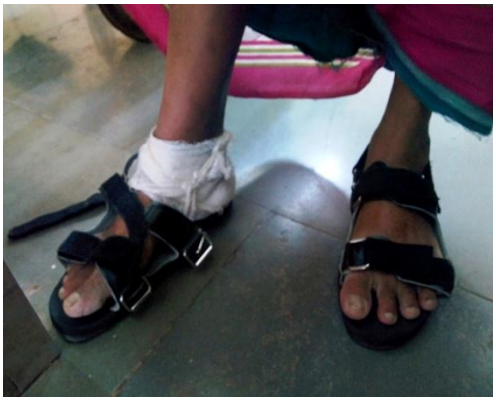
- Pressure offloading of diabetic plantar foot ulcers facilitates faster granulation tissue fill up.
- Pressure offloading of diabetic plantar foot ulcers causes significant reduction in surface area of ulcer in a shorter duration
- Diabetic plantar foot ulcer patients treated with Pressure offloading showed faster recovery time compared to conventional dressing.

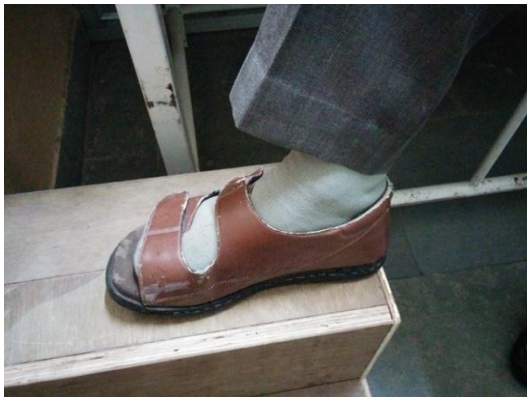
# CONCLUSION

Pressure offloading of plantar foot ulcers of patients with diabetes mellitus can be considered as a superior option and can used regularly even on outpatient basis for faster wound healing and further prevention of reulcerations in these patients.

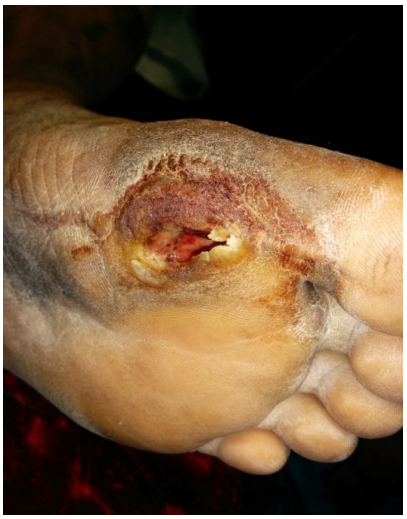
# 20: IMAGES TAKEN DURING THE STUDY

**METHOD OF OFFLOADING : HEALING SANDAL**

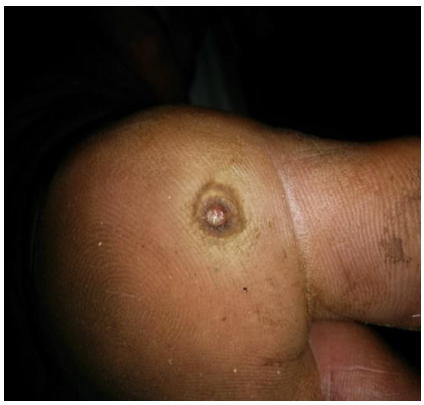




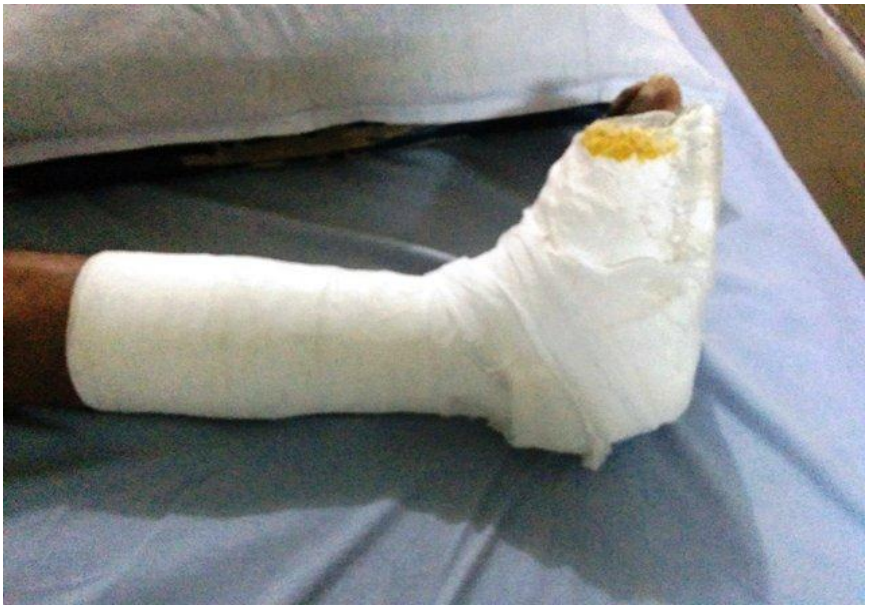
INITIAL SIZE OF THE ULCER



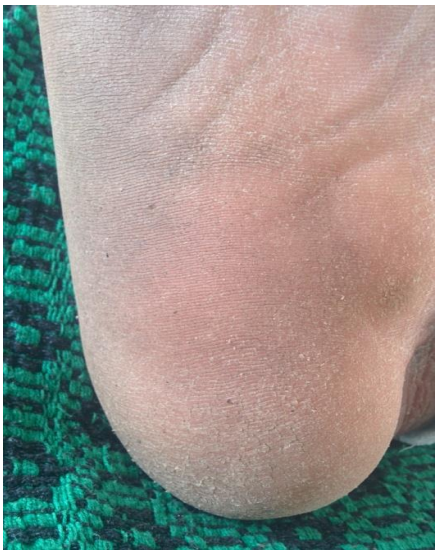
POST OFFLOADING



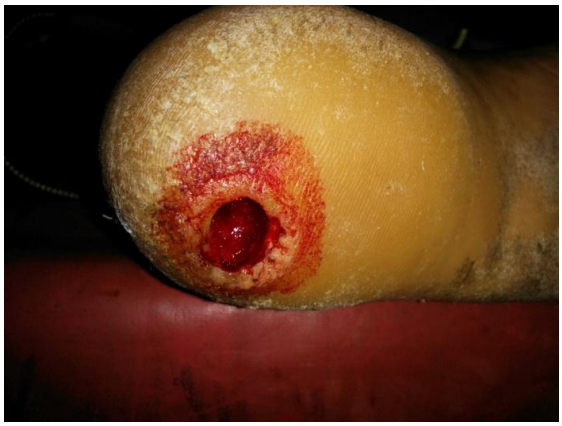
**METHOD OF OFFLOADING : TOTAL CONTACT CAST**



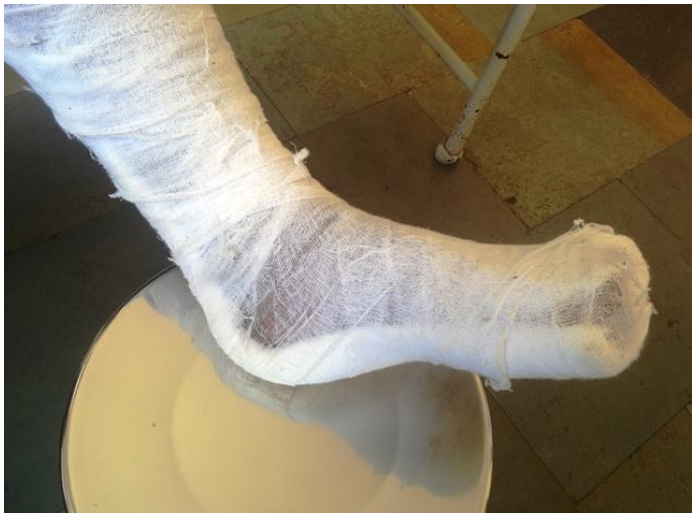
POST OFFLOADING



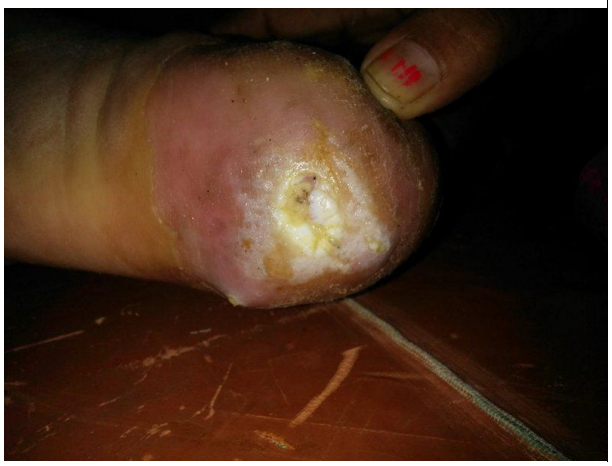
INITIAL SIZE OF THE ULCER:



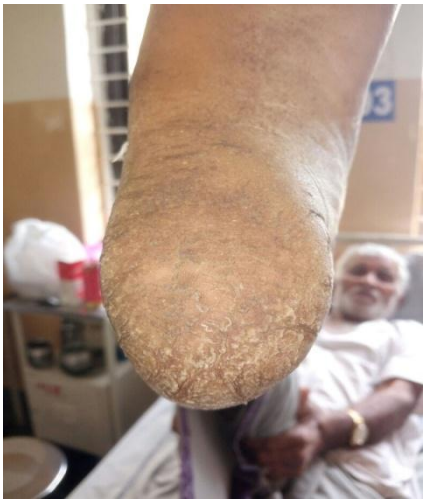
**METHOD OF OFFLOADING : TOTAL CONTACT SLAB**



INITIAL SIZE OF THE ULCER

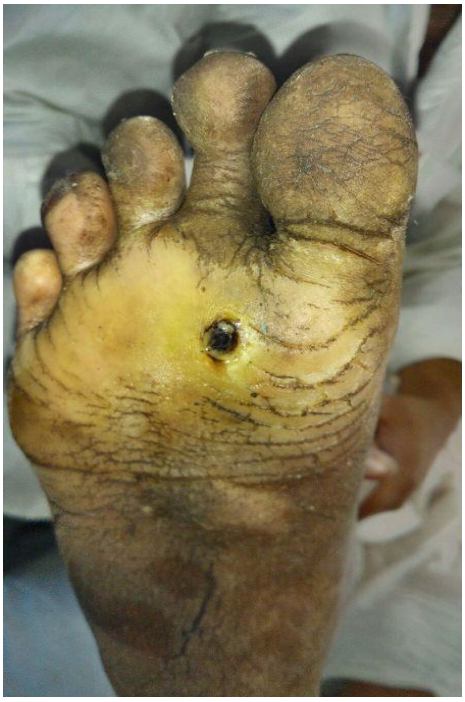


POST OFFLOADING

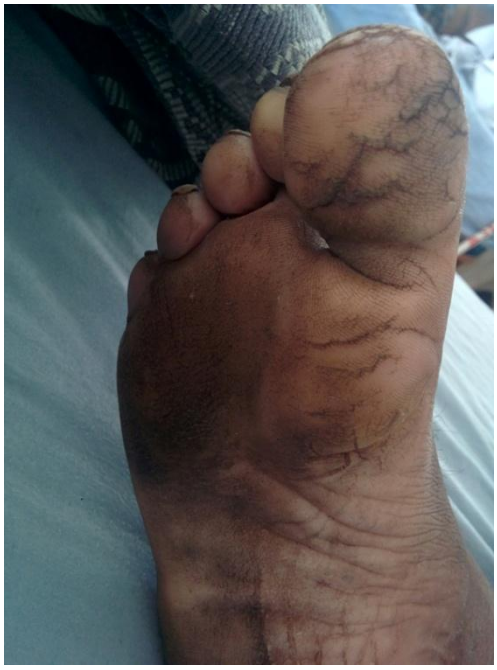


**METHOD OF OFFLOADING : REMOVABLE SLING**

INITIAL SIZE OF THE ULCER:



POST OFFLOADING



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# ANNEXURES

# ETHICAL CLEARANCE CERTIFICATE

Institutional Ethical Committee
BIJAPUR-586 103.
OUTWARD
No. 5414
Date 24/11/14
B.L.D.E.U's Shri B.M. Patil Medical College

B.L.D.E. UNIVERSITY'S
SHRI.B.M.PATIL MEDICAL COLLEGE, BIJAPUR-586 103
INSTITUTIONAL ETHICAL COMMITTEE

## INSTITUTIONAL ETHICAL CLEARANCE CERTIFICATE

*The Ethical Committee of this college met on* 22-11-2014 *at* 3-30pm *to scrutinize the Synopsis of Postgraduate Students of this college from Ethical Clearance point of view. After scrutiny the following original/corrected & revised version synopsis of the Thesis has been accorded Ethical Clearance.*

*Title* "A Comparative Study Between Pressure offloading and Conventional Dressing in Management of Diabetic Plantar Foot Ulcers

*Name of P.G. student* Dr. Mrinal Kumar V.
Dept of General Scogery.

*Name of Guide/Co-investigator* Dr M.S. Kotennavar Professor
Dept of General Surgery.

for
DR.TEJASWINI. VALLABHA
CHAIRMAN
INSTITUTIONAL ETHICAL COMMITTEE
BLDEU'S, SHRI.B.M.PATIL
MEDICAL COLLEGE, BIJAPUR.

Following documents were placed before E.C. for Scrutinization
1) Copy of Synopsis/Research project.
2) Copy of informed consent form
3) Any other relevant documents.

## RESEARCH INFORMED CONSENT FORM

**TITLE OF THE PROJECT** **:** A COMPARATIVE STUDY BETWEEN PRESSURE OFFLOADING AND CONVENTIONALDRESSING IN MANAGEMENT OF DIABETIC PLANTAR FOOT ULCERS

**PRINCIPAL INVESTIGATOR** **:** Dr. MRINAL KUMAR V

P.G. DEPARTMENT OF GENERAL SURGERY

**P.G.GUIDE** **:** DR.M.S.KOTENNAVAR

PROFESSOR

DEPARTMENT OF GENERAL SURGERY

**PURPOSE OF RESEARCH:**

I have been informed that this study is comparative study between pressure offloading and conventional dressing. I have also been given a free choice of participation in this study. This will help in proper understanding regarding the treatment outcome of plantar diabetic foot ulcers.

**PROCEDURE:**

I have been explained that after thorough debridement of the ulcer, one of the pressure offloading techniques will be used to offload pressure from my foot ulcer. Then after 7, 14, 21, 28 days, reduction in size of wound, granulation tissue is observed and compared with control who receive conventional method of dressing.

Total contact casting will be used for non infected wounds and either slings or offloading foot wares will be used in case of an infected wound.

**RISK AND DISCOMFORTS:**

I understand that I may experience some pain and discomfort during the examination or during my treatment. This is mainly the result of my condition and the procedure of this study is not expected to exaggerate theses feelings that are associated with the usual course of the disease.

**BENEFITS:**

I understand that my participation in the study will have no direct benefits to me other than the potential benefits of the diagnosis and treatment which is planned to reduce my pain. The major potential benefit is to find out which treatment is more effective.

**ALTERNATIVES:**

I understand that the two modes of treatment being studied are standard ways of treating my problem that is plantar diabetic foot ulcers.

**CONFIDENTIALITY:**

I understand that medical information produced by this study will become a part of this hospital records and will be subjected to the confidentiality and privacy regulation of this hospital. Information of a sensitive, personal nature will not be a part of the medical records, but will be stored in the investigator's research file and identified only by a code number. The code key connecting name to numbers will be kept in a separate secure location.

If the data are used for publication in the medical literature or for teaching purpose, no names will be used and other identifiers such as photographs and audio or video tapes will be used only with my special written permission. I understand that I may see the photograph and videotapes and hear audiotapes before giving this permission.

**REQUEST FOR MORE INFORMATION:**

I understand that I may ask more questions about the study at any time. Dr. Mrinal Kumar V is available to answer my questions or concerns. I understand that I will be informed of any significant new findings discovered during the course of this study, which might influence my continued participation.

If during this study, or later, I wish to discuss my participation in or concerns regarding this study with a person not directly involved, I am aware that the social worker of the hospital is available to talk with me.

And that a copy of this consent form will be given to me to keep it and for careful reading.

**REFUSAL OR WITHDRAWL OF PARTICIPATION:**

I understand that my participation is voluntary and I may refuse to participate or may withdraw consent and discontinue participation in the study at any time without prejudice to my present or future care at this hospital.

I also understand that Dr. Mrinal Kumar V will terminate my participation in this study at any time after he has explained the reasons for doing so and has helped arrange for my continued care by my own physician or therapist, if this is appropriate.

**INJURY STATEMENT:**

I understand that in the unlikely event of injury to me/my ward, resulting directly to my participation in this study, if such injury were reported promptly, then medical treatment would be available to me, but no further compensation will be provided.

I understand that by my agreement to participate in this study, I am not waiving any of my legal rights.

I have explained to _____ the purpose of this research, the procedures required and the possible risks and benefits, to the best of my ability in patient's own language.

Date: Dr. M.S.Kotennavar (Guide) Dr. Mrinal Kumar V (Investigator)

**STUDY SUBJECT CONSENT STATEMENT:**

I confirm that Dr. Mrinal Kumar V has explained to me the purpose of this research, the study procedure that I will undergo and the possible discomforts and benefits that I may experience, in my own language.

I have been explained all the above in detail in my own language and I understand the same. Therefore I agree to give my consent to participate as a subject in this research project.

_____ _____

(Participant) Date

_____ _____

(Witness to above signature) Date

# PROFORMA

NAME: CASE NO.:

AGE: IP NO./ OP NO. :

SEX: DOA:

RELIGION: DOD:

OCCUPATION: WEIGHT:

ADDRESS:

CHIEF COMPLAINTS WITH HISTORY OF PRESENTING ILLNESS:

PAST HISTORY:

- DIABETES MELLITUS:
- HYPERTENSION:
- HISTORY OF ANY DRUG INTAKE:

PERSONAL HISTORY:

- DIET:
- APPETITE:
- SLEEP:
- HABITS:

FAMILY HISTORY:

GENERAL PHYSICAL EXAMINATON:

VITALS

- PULSE RATE:
- BLOOD PRESSURE:
- RESPIRATORY RATE:
- TEMPERATURE:

LOCAL EXAMINATION:

- INSPECTION OF FOOT WITH ULCER

  SITE:

  SIZE:

  SHAPE:

  SURROUNDING SKIN:

  FOOT DEFORMINTY:

  SURFACE AREA:
- PALPATION

  SENSATION:

  FILAMENT:

  BIOTHESIOMETER:

  PULSATIONS:

  HAND HELD DOPPLER:
- PRESSURE POINTS ( HARRIS MAT):

CLINICAL DIABETIC FOOT GRADING (WAGNER'S):

OTHER SYSTEMIC EXAMINATION:

- RESPIRATORY SYSTEM:
- CARDIOVASCULAR SYSTEM:
- CENTRAL NERVOUS SYSTEM:
- PER ABDOMINAL EXAMINATION:

INVESTIGATIONS:

- BLOOD

  HB%:

  TC:

  DC:

  ESR:

  BT:

  CT:

- URINE

  ALBUMIN:

  SUGAR:

  MICROSCOPY:

  KETONE BODIES:

- HIV : HBSAg:

- GYCEMIC LEVELS

  RBS:

  FBS:

  PPBS:

  HBA1C:

- PUS CULTURE AND SENSITIVITY:

- COLOUR DOPPLER:

- XRAY FOOT AP AND OBLIQUE VIEW:

- ECG:

- ECHOCARDIOGRAPHY (WHENEVER REQUIRED)

FINAL DIAGNOSIS:

FOLLOW UP:

1ST WEEK

2ND WEEK:

3RD WEEK:

4TH WEEK:

COMMENTS:

# KEY TO MASTER CHART

| | |
|---|---|
| Y | : YES / PRESENT |
| GR | : GREAT |
| MET | : METATARSAL |
| HTN | : HYPERTENSION |
| TOB | : TOBACCO CHEWER |
| ALC | : ALCOHOLIC |
| SMO | : SMOKER |
| TC | : TOTAL LEUCOCYTE COUNTS |
| Hb | : HEAMOGLOBIN |
| RBS | : RANDOM BLOOD SUGAR |
| FBS | : FASTING BLOOD SUGAR |
| GRA | : COMPLETE GRANULATION TISSUE |
| MIN | : MINIMAL/PARTIAL GRANULATION TISSUE |
| NO | : NO GRANULATION TISSUE |
| HS | : HEALING SANDAL |
| RS | : REMOVBLE SLING |
| TCC | : TOTAL CONTACT CAST |
| TCS | : TOTAL CONTACT SLAB |