“EVALUATION OF USEFULNESS OF LASER THERAPY IN MANAGEMENT OF DIABETIC FOOT ULCER”

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IN

GENERAL SURGERY

Under the Guidance of

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LIST OF ABBREVATION

- DF-Diabetic foot
- WHO-World health organization
- DFS - Diabetic foot syndrome
- DM - Diabetes mellitus
- LLLT - Low level laser therapy
- NM - Nanometer
- CAD - Coronary artery disease
- CVD - Cerebrovascular disease
- RFT - Renal function test
- LFT - Liver function test
- WHO - World health organization
- FBS - Fasting blood sugar
- PPBS - Postprandial blood sugar
- RBS - Random blood sugar
- HBA1C - Glycated hemoglobin
- HB - Hemoglobin
- HE:AG - Helium argon
- ND YAG - Neodymium yettrium argon
- DM - Diabetes mellitus
- DFS - Diabetic foot syndrome
- DFU - Diabetic foot ulcer
- PVD - Peripheral vascular disease
- C&S - Culture and sensitivity

- ATP - Adenosine triphosphate
- CO2 - Carbon dioxide
- MM - millimeter
- MW - Milliwatts

ABSTRACT

BACKGROUND:

Low-level laser treatment (LLLT) is phototherapy that uses low-power monochromatic and coherent light to treat injuries and lesions. Low laser therapy given to soft tissues in vitro and in vivo has been reported to stimulate certain healing pathways in wound healing. Studies published in the LLLT literature have examined the effects of Low light laser therapy on fibroblast growth, locomotion and production of collagen as early as 24 hours after laser treatment and accelerates wound healing. LLLT proved promising in many studies, and its cost effectiveness in a developing nation like India, which could soon changed the way of treating diabetic foot ulcers .

OBJECTIVES:

To determine the efficacy of LLLT in terms of ulcer healing rate and ulcer size reduction.

METHODS: A "Comparative Prospective Observational study" was conducted in "Department of General Surgery" at a tertiary care center among patients with diabetic foot ulcers for long time with Grade I-III (Megitt-Wagner Grade). 60 patients were divided in to 2 groups. Study group was treated with LLLT and Control group was treated with Conventional Dressing. Clearance was obtained from Ethical committee of the Institutuion before starting the study. Patients were explained the procedure and consent was taken from all the patients involved prior to the start of the study.

RESULTS:

Mean Age in Study group was 50.5 ± 15.19 and in Control group was 54.9 ± 10.33. In Both groups majority were males. In Study Group, 50.00% had Spontaneous and 50.00%

and Traumatic onset. In Control Group, 70.00% had Spontaneous and 30.00% and Traumatic onset. Mean Area of wound % Decreased in Study group (22.19 ± 7.21) and in Control group (15.28 ± 6.54). There was significant difference in mean Area of wound % Decreased comparison between two groups.

CONCLUSION:

Based on the results from the above study , we conclude that highest percentage of decrease in wound area was observed in Laser Group compared to Conventional Group.

KEYWORDS: Low Laser Therapy, Conventional Therapy, Diabetic Foot Ulcer

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INTRODUCTION

"The foot of a diabetic patient is potentially at risk of pathologic consequences, including infection, ulceration, &/or damage of deep tissues associated with neurologic abnormalities, various degrees of peripheral vascular disease, &/or complications caused by diabetes metabolically in the lower limb," according to the World Health Organization[1].

The chance of diabetes and its side effects are raising & the chance of lower limb amputations is 15-fold more in patients with diabetics as compared with non – diabetics. It is essential to notify that DF ulcers with prolonged duration are the number one cause of amputations in diabetes patients, & also that 15% of patients with diabetics develop ulcers & are most commonly seen over foot.[2] The DF ulcers are very resistant to regular conventional treatment and may develop lot number of complications with more severity if not treated appropriately .

In India, diabetic foot care is one of the most neglected and underappreciated components of diabetes management. Many people walk barefoot due to social, religious, and economic pressures[3]. Poverty and a lack of education lead to the wearing of unsuitable footwear and the development of foot lesions later in life. Patients can also try home treatments before going to the doctor. It estimated that 90% of diabetic patients in India do not see a specialist in their lifetime .Problem is further worsened by a delay in accessing healthcare due to patient approaching informal care providers and alternative medicine prescribers.

Reports of LLLT applied to soft tissues in laboratory studies suggest stimulations of

specific healing processes in healing wounds. Major changes seen in ulcers which are exposed with LLLT include increased healing by more granulation in the tissues , increased fibroblast proliferation, collagen synthesis, epithelialization and enhanced neovascularization.

NEED FOR THE STUDY:

Diabetes is seen in around 387 million individuals in the world and is one requires highest concern in health care system. The chance of diabetes & its side effects are on a rise and the risk of lower extremity amputations is 15-fold higher as compared to non – diabetics. It is essential to mention, that DF ulcer with chronic time is the most leading cause of amputations, & 15% of all diabetics develop diabetic ulcer, and are mostly seen in the foot[3]. Hence DF ulcers needs strict management which includes serial monitoring of sugars and dressings.

These DF ulcers are most non responding to regular conventional therapy & may lead to severe complications if not wisely managed. LLLT is a mode of therapy in which we apply light of low intensity to wounds caused by trauma & breach of continuity. Previous researches of low laser therapy provided to cells & tissues in laboratory set up concludes stimulations of processes of healing in healing wounds. Mostly the studies on the LLLT & its history have explained the outcome of LLLT on growth of fibroblast , locomotion & production of collagen as fast as 24 hours period after treated with light and accelerates wound healing. Low light laser therapy has proved promising in many studies, and its cost effectiveness is a boon in a developing nation like India, which could soon change the way DF ulcers are being treated [4,5].

AIM AND OBJECTIVES

AIM:

To determine the efficacy of low light laser therapy in treatment of Diabetic Foot ulcer

OBJECTIVES:

To assess the efficacy of LLLT in terms of

1. Rate of ulcer healing
2. Decreasing of size of the ulcer and
3. Duration of healing in diabetic foot

REVIEW OF LITERATURE

HISTORICAL ASPECTS OF WOUND

Diabetes is the most commonly noticed disease and is famous from its antiquity. It is described as a disease with polyuria in the ebers papyrus which also explains medical compilations described from very long back i.e second millennium BC. In India it is described as sweet urine about 500 AD[6]. Initially Egyptians are the one who differentiated infected wounds from diabetic wounds compared to noninfected wounds. In 1650 B.C. Edwin Smith who is a Surgical Papyrus, enlightened 48 different varieties of wounds. A later (Ebers Papyrus, 1550 B.C.) elaborates use of concoctions honey which got which are antibacterial , lint which got absorbent properties, and grease which acted as a barrier for treating wounds. In contemporary daily wound management these properties are still followed and are considered very essential.

Galen of Pergamum (120-201 A.D), emphasized the importance of maintaining a moistness for proper sufficient healing of wounds. They almost needed 19 centuries for proving this important concept scientifically, & was shown that the epithelialization incidence also raised by 50% in wounds with environment with wet content when they are compared with wounds without wet content environment. The relation btw Diabetes and side effects of it in the limbs was first recognized John Rollo (1978). The link between ulceration in the foot , neurological complications and vascular diseases was described by Pryce (1887). Oakley is the first person to classify foot lesions as infection, compromised blood supply, and neurological complications and both vascular and neurological in 1956. Charcot joints is deformity which is seen in Diabetes were first noticed and elaborated by Weinberg in 1980. Celsus described the technique of removing the effected limb.

The history of wound healing is very ancient. 48 cases and their reports are included in the

Edwin smith Papyrus (1700 B.C) with diabetes out of which 7 cases describe wounds and their management. Ancient physicians of Egypt, Greece, India and Europe evolved very subtle methods for healing these wounds , they include foreign bodies removal , approximating the wounds , protecting wounds with uninfected materials and damaged tissues are also protected from corrosive agents[7].

The Three Gesture Theory was described 4000 years ago on clay tablets since 2200 B.C. The theory states:

- Washing the wound
- Making plasters
- Bandaging the wound[8].

Healing without moisture is advised by Greeks from Hippocrates theories , and he also elaborated as the only function of regular dressings is to safe guard the injury from injuries caused by external force and dust and prevention of more damage[9] . The wound healing methods and the care which we give at present for the wounds has been followed from mid – sixteenth century by Ambroise Pare, who is a great French surgeon. His methods are application of egg yolk, rose oil, honey and turpentine which has digestive properties locally to amputation stumps which has showed dramatic results.

John Hunter, William Stewart Halsted, who are biologists proved that by decreasing injury to the tissues helps in fast and effective healing which lead in development of minimal interference concept in care of wounds .

Joseph Lister (advocating cleanliness in the hospital) and Louis Pasteur in their research on diabetes and wounds have established a solid basis in the infection management by knowing the exact cause & method how it developed for preventing it. Thus taking care of bacteria and reducing the infection by asepsis, antiseptic measures and antimicrobials hold a new era in

management of diabetic wounds. A Belgian surgeon Antione Depage mentioned–The debridement decompresses the contused center by widening the tissues which are strangulated by the constrictions of the fascia. The surgeon aims to avoid septic and dangerous complications, by placing the wound in best possible settings for healing and suturing[10].

In the last two decades, wound healing and care have advanced dramatically.

This is due to:

- Anatomic, biochemical, and molecular levels are all used to define the biologic mechanisms of repair.
- Extensive wound healing research has resulted in conclusion of commercially viable therapeutic techniques.
- New pharmacological drugs are being developed to improve the developing process in short and long duration wounds.
- Improved reconstructive surgical procedures, thanks to the introduction of muscular and musculocutaneous flaps, as well as microvascular free-tissue transfers [11].

The concept of a wound with wet content for healing was not enlightened until the 1960s. A wet wound environment would aid to facilitate debridement, minimise inflammation, reduce pain, and reduce scarring, in addition to protecting the area from infection [12].

ANATOMY

Foot: The part of the lower limb distal to the ankle joint is known as the foot. It is separated into three sections: the ankle, metatarsus, and digits. The great toe (digit I) is positioned medially, followed by four additional digits that are positioned laterally, finishing with the little toe (digit V). The foot has a superior (dorsum of foot) and inferior (sole of foot) surface (sole)

Bones: In the foot, there are three groups of bones.

• Metatarsals (I to V), which are the bones of the metatarsus;

• Seven tarsal bones, which create the skeletal framework for the ankle;

• Phalanges, or toe bones—except for the great toe, which has two phalanges—each toe has three phalanges.

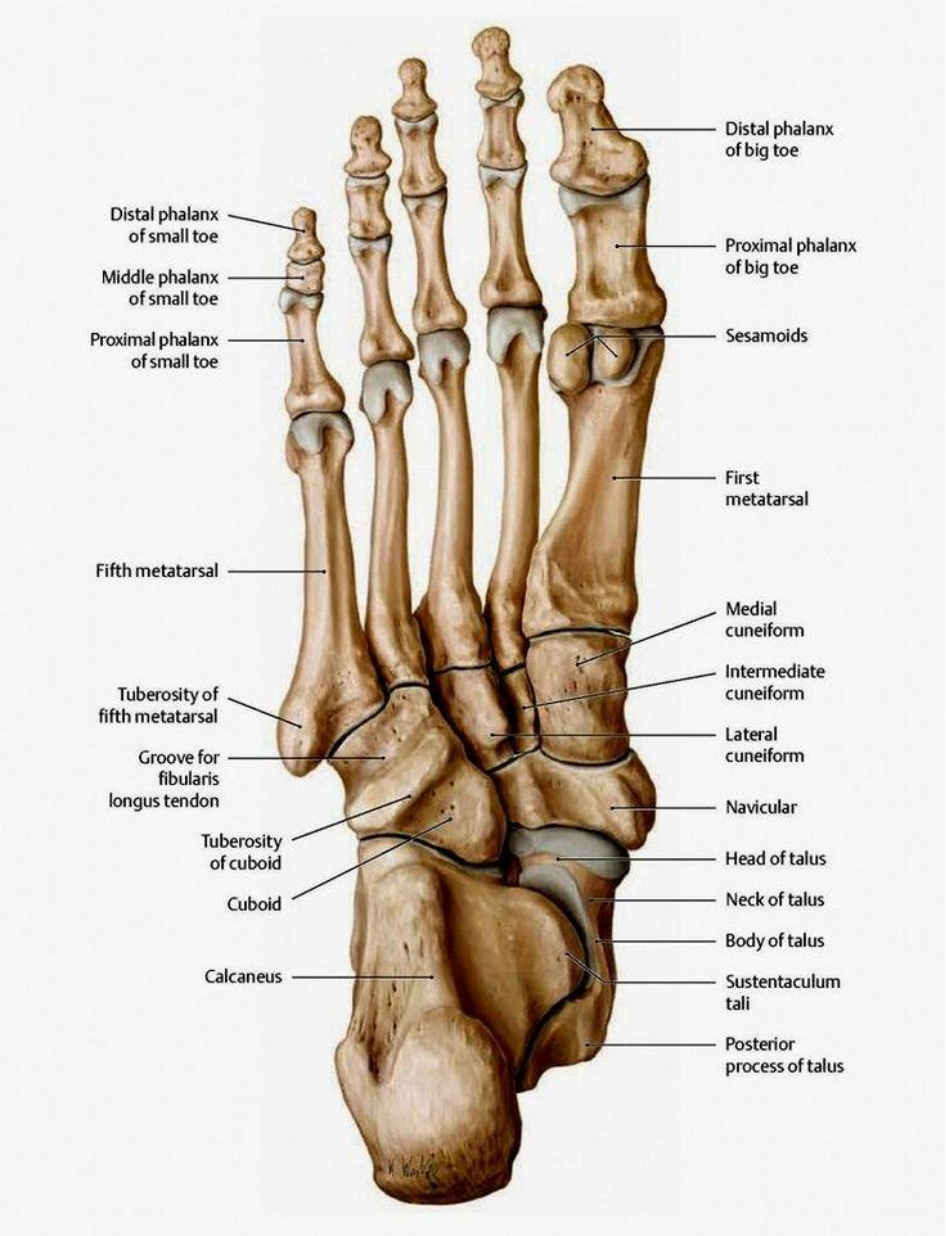


Fig. 1 Bones of foot

Joints

- Ankle joint
- Intertarsal joints
- Subtalar joint (Talo calcaneo navicular joint)
- Calcaneocuboid joint
- Tarsometatarsal joints
- Metatarsophalangeal joints
- Interphalangeal joints

Arches of The Foot

The bones of the foot do not lie in a horizontal plane. Instead, they form longitudinal and transverse arches relative to the ground, which absorb and distribute downward forces from the body during standing and moving on different surfaces.

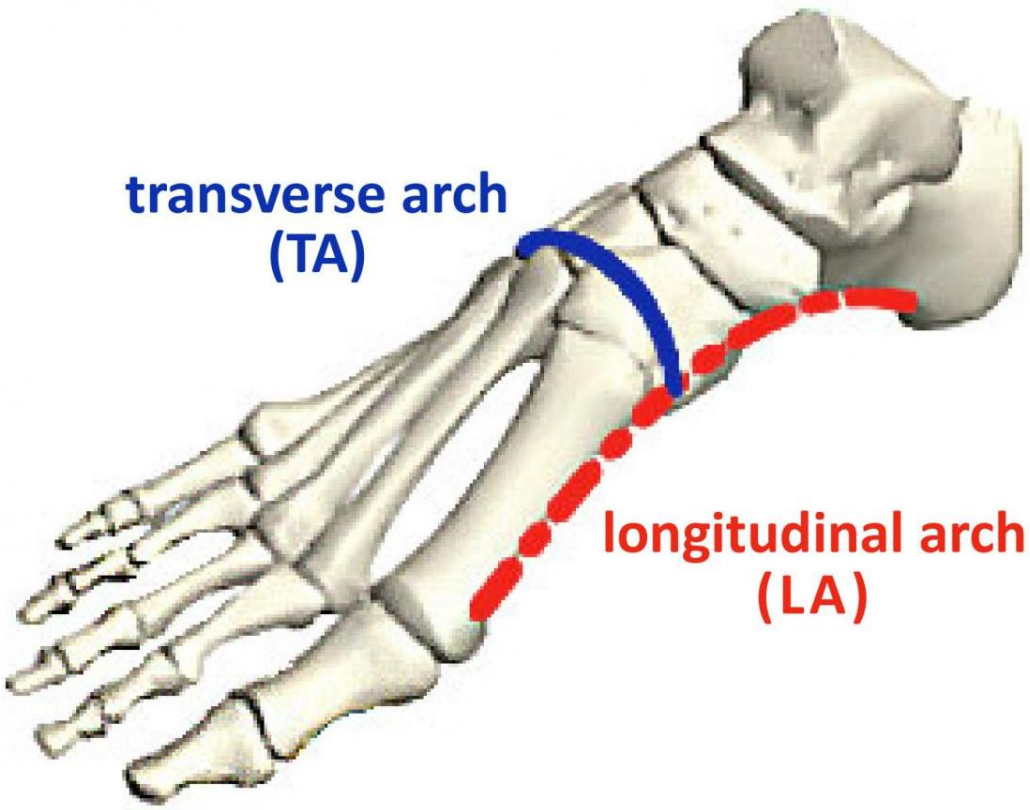


Fig. 2 Arches of foot

Plantar Aponeurosis

A thickening of deep fascia in plantar aspect of foot is known as the plantar aponeurosis. It is firmly attached to the calcaneal tuberosity's medial process and extends forward like a thick band of longitudinally organised connective tissue fibres. As the fibres pass anteriorly, they diverge and form digital bands, which penetrate the toes and connect with bones, ligaments, and the skin's dermis.

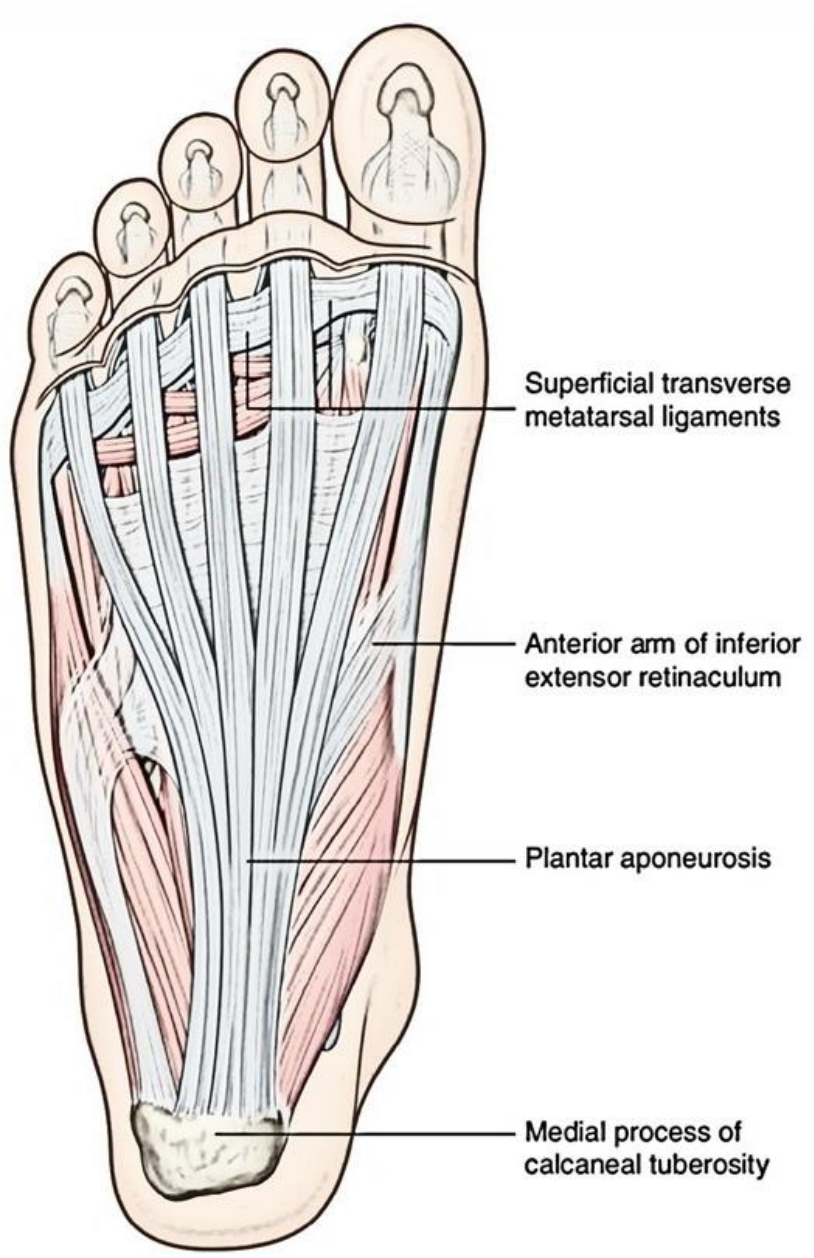


Fig. 3 Plantar aponeurosis

Arteries: The foot is supplied by posterior tibial and dorsalis pedis arteries branches .

Veins: In the foot, there are interwoven networks of deep and superficial veins. The arteries are followed by the deep veins. Over the metatarsals, superficial veins drain into a dorsal venous arch on the dorsal surface of the foot.

Nerves: The tibial, deep fibular, superficial fibular, sural, and saphenous nerves supply the foot[14].

Table 1: Muscles of footMuscle on dorsal aspect of foot

Muscle	Origin	Insertion	Innervations	Function
Extensor digitorum brevis	Superolateral surface of the calcaneus first muscle layer in the sole of the foot	The tendons of the extensor digitorum longus of toes II to IV are located at the base of the proximal phalanx of the great toe and on the lateral to it	Deep fibular nerve [S1, S2]	Extension of metatarsophalangeal joint of great toe and flextion of toes II to IV
LAYERS OF THE FOOT				
FIRST LAYER				
Abductor hallucis	Medial process of calcaneal tuberosity	Medial side of base of proximal phalanx of great toe	Medial plantar nerve from the tibial nerve [S2, S3]	Abducts and flexes great toe at metatarsophalangeal Joint
Flexer digitorum brevis	Medial process of calcaneal tuberosity and plantar Aponeurosis	Sides of plantar surface of middle phalanges of lateral four toes	Medial plantar nerve from the tibial nerve [S2, S3]	Flexes lateral four toes at proximal interphalangeal Joint
Abductor digitiminimi	Lateral and medial processes of calcaneal tuberosity, and band of connective tissue connecting calcaneus with base of metatarsal V Second layer of muscles in the sole of the foot	Lateral side of base of proximal phalanx of little toe	Lateral plantar nerve from tibial nerve [S2, S3]	Abducts little toe at the metatarsophalangeal joint
SECOND LAYER				

Quadrates plantae	Medial surface of calcaneus and lateral process of calcaneal tuberosity	Lateral side of tendon of flexor digitorum longus in proximal sole of the foot	Lateral plantar nerve from tibial nerve [S1 toS3]	Assists flexor digitorum longus tendon in flexingtoes II to V
Lumbricals	First lumbrical-medial side of tendon of flexor digitorum longus associated with toe II; second, third, and fourth lumbricals-adjacnetsurfaces of adjacenttendons of flexor digitorum longus third layer of muscles in the sole of the foot			Flexion of metatarsophalangeal joint and extension of interphalangeal joints.
THIRD LAYER				
Flexor hallucis brevis	Plantar surface of cuboid and lateral cuneiform; tendon of tibialis posterior	Lateral and medial sides of base of proximal phalanx of the great toe	Lateral plantar nerve from tibial nerve [S1, S2]	Flexes metatarsophalangeal joint of the great toe
Adductor hallucis	Transverse head-ligaments associated with metatarsophalangeal joints of lateral three toes; oblique head-bases of metatarsals II to IVand from sheath covering fibularis longus	Lateral side of base of proximal phalanx of great toe	Lateral plantar nerve from tibial nerve [S2, S3].	Adducts great toe at metatarsopha-langeal joint
Flexor digiti minimi brevis	Base of metatarsal V and related sheath of fibularis longus tendon Fourth layer of muscles in the sole of the foot	Lateral side of base of proximal phalanx of little toe	Lateral plantar nerve from tibial nerve [S2, S3]	Flexes little toe at metatarsophalangeal joints

FOURTH LAYER				
Dorsal interossei	Sides of adjacent metatarsals	Dorsal expansions and bases of proximal phalanges of toes II to IV	Lateral plantar nerve from tibial nerve; first and second dorsal interossei also innervated by deep fibular nerve [S2, S3]	Abduction of toes II to IV at metatarsophalangeal joints; resist extension of metatarsophalangeal joints and flexion of interphalangeal joints
Plantar interossei	Medial sides of metatarsals of toes III to V	Dorsal expansion and bases of proximal phalanges of toes III to V	Lateral plantar nerve from tibial nerve [S2, S3]	Adduction of toes III to V at metatarsophalangeal joints; resist extension of the metatarsophalangeal joints and flexion of the interphalangeal joints

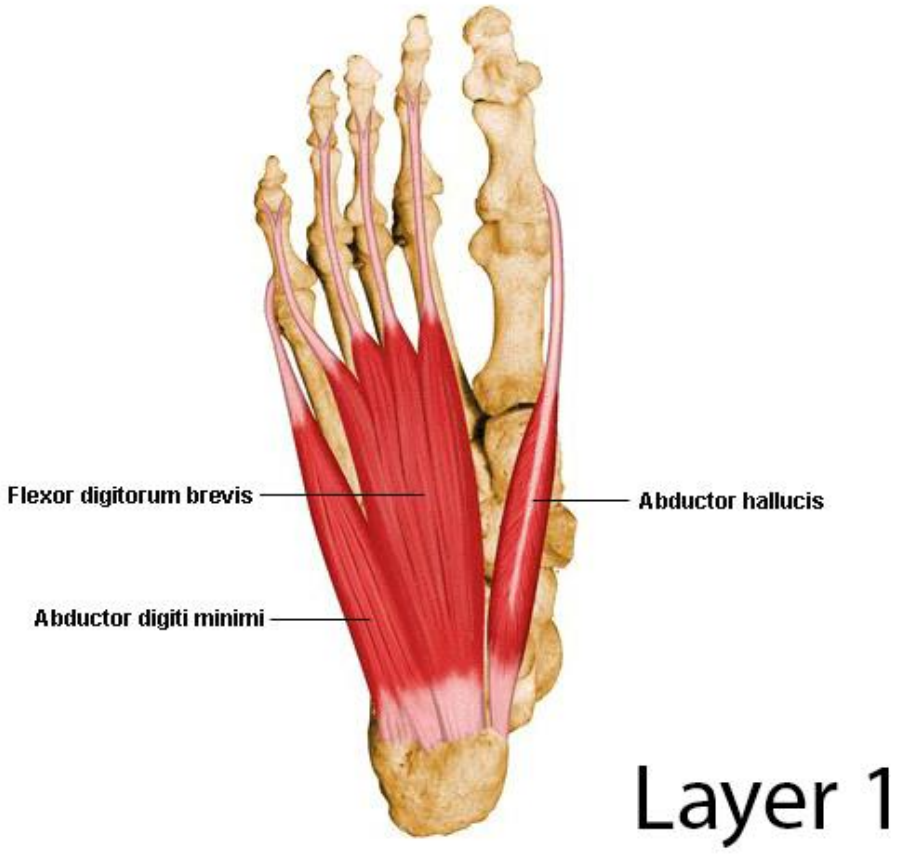


Fig. 4: Layer-1 of Foot

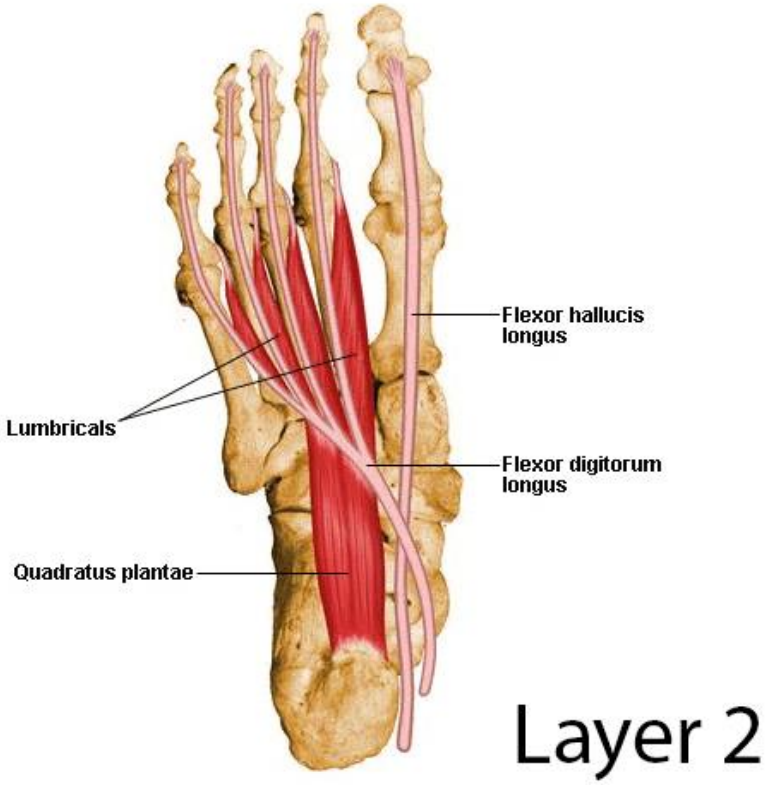


Fig. 5: Layer-2 of Foot

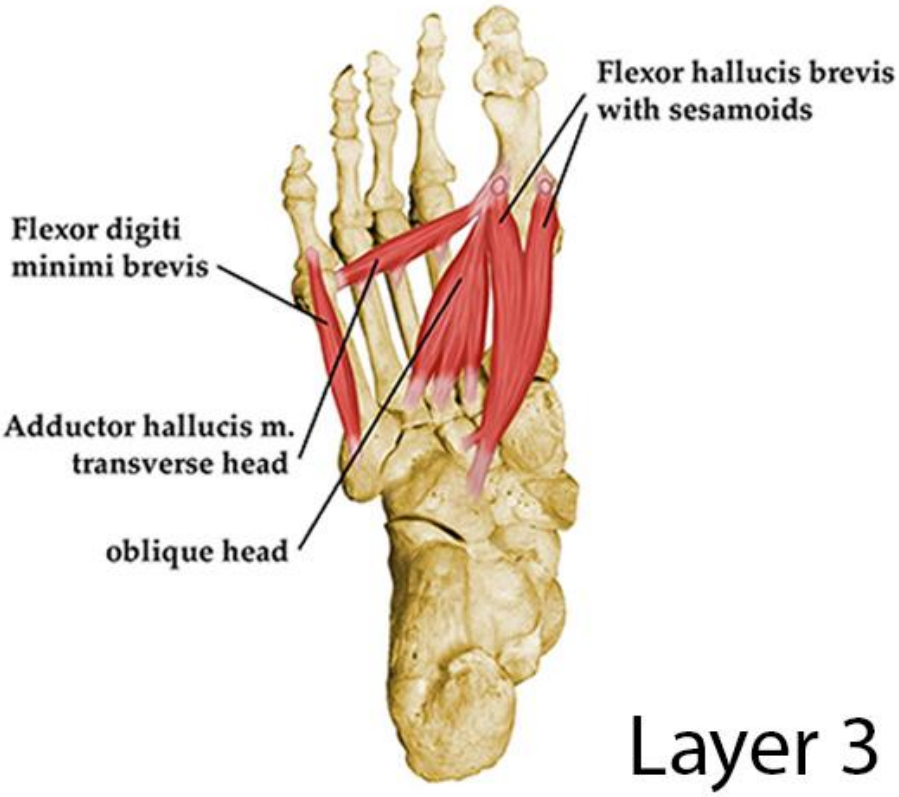


Fig. 6: Layer-3 of Foot

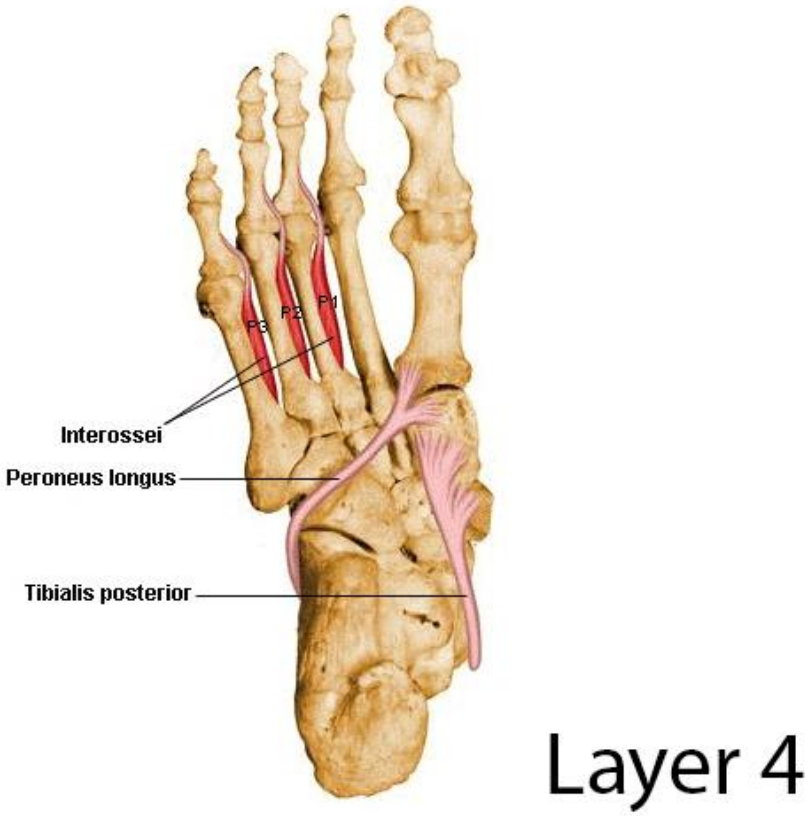


Fig. 7: Layer-4 of Foot.

PATHOPHYSIOLOGY :

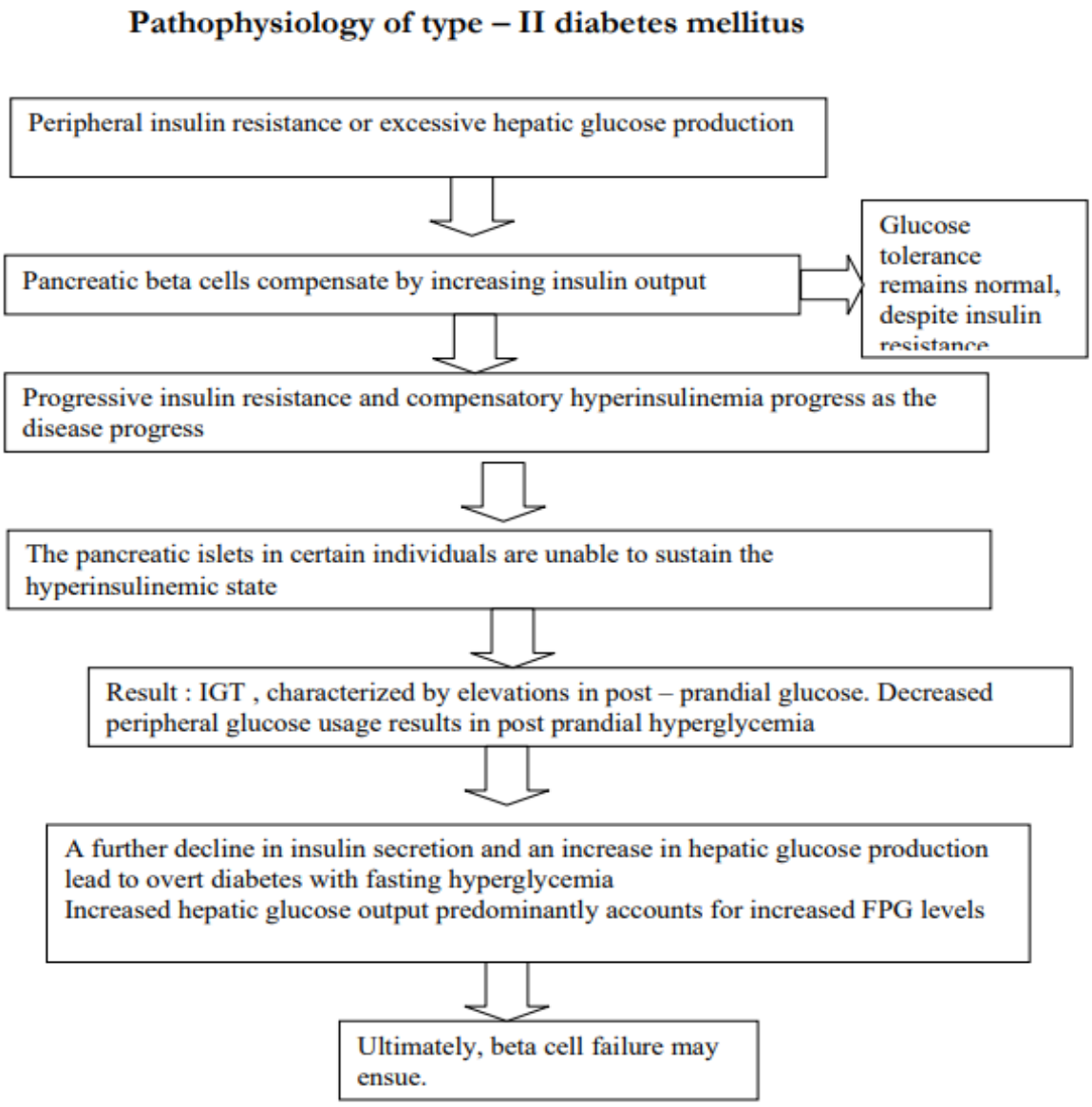


Fig. 8 :Pathophysiology of type II diabetes mellitus

Pathophysiology

Pathogenesis is a complicated process that involves several interconnected stages.

A. Angiopathy

B. Neuropathy

C. Immunopathy

A. Angiopathy:

Diabetic angiopathy is the most common morbidity and mortality among diabetic patients. There are two types of angiopathy:

Macroangiopathy and Microangiopathy.

Macroangiopathy: Diabetic macroangiopathy is a more diffuse illness with more multisegmental involvement and compromised collateral circulation than nondiabetic macroangiopathy. It is more commonly seen bilaterally in the lower limbs, with the infrapopliteal arteries being implicated more frequently than in nondiabetic patients. The development of DF ulcers was found to be linked to vascular impairment as measured by resting Doppler ankle pressure. Diabetes patients with large vessel disease have more chance to develop foot lesions which finally leads to ischemic skin changes, which can lead to ulceration and infection.

Microangiopathy: The hemodynamic hypothesis of diabetic microangiopathy causation was explored by Tooke and Brash. According to this theory, diabetes patients' capillaries have increased microvascular pressure and flow in the early stages of the disease. An injury reaction occurs inside the microvascular endothelium due to increased capillary pressure. Microvascular sclerosis develops as the release of extravascular matrix proteins caused by injury. The ultrastructural hallmark of diabetic microangiopathy is sclerosis, which manifests itself in the arteriole as hyalinosis and the capillary as basement membrane thickening. The sclerotic process

causes decreased maximum hyperemia and loss of autoregulatory capacity as the duration of diabetes increases. A crucial finding is that in the early stages of type 1 diabetes, nailfold capillary pressure is higher. This has been linked to glycemic management, as measured by glycosylated haemoglobin at the time of blood pressure measurement. Furthermore, the pressure in such areas looks to be particularly high in those individuals at high risk for microangiopathy, yet relatively normal in patients who have avoided the clinical complications of diabetes over many years.

B. Neuropathy

Neuropathy is the most common risk factor for diabetic foot ulcers and starts early in the pathophysiology of diabetic foot issues. Sensory, motor, and autonomic neuropathy are the three types of diabetic neuropathy. The result of this trifecta is a foot that is unable to respond to pain and is biomechanically impaired, with higher foot pressure, reduced joint mobility, and poorly hydrated skin that is unable to adapt effectively to damage.

Sensory neuropathy: The damage from sensory neuropathy affects the large myelinated alpha fibres. Its distribution is usually symmetric in stocking and glove pattern as a result, patients are unable to perceive injury to their feet because primary protective or warning systems are defective. This fundamental pathophysiologic impairment is referred to as the loss of protective sensation. Affected patients sustain repetitive, unrecognized injuries to their feet that culminate in full thickness ulceration. Ulcer in an insensate foot is usually painless. Neuropathy can have a wide range of severities and symptoms. Loss of protective sensation does not necessarily mean complete absence of sensation or pain. So called painful or painless ulcers may develop because of ischemia or deep sepsis; these require prompt attention and intervention. This scenario can also represent damage to both large myelinated nerves and small unmyelinated

nerves, so the patient may have burning sensation because of small fibre damage and deep, gnawing pain and numbness because of large-fibre neuropathy.

Motor neuropathy: Often occurs in late in form of neuropathy caused peripherally in DF and contribute to intrinsic muscle wasting of the feet and hands. Short, weak flexors and extensors that are over powered by long, strong flexors and extensors in the foot contributes to structural foot deformities such as claw toes, dislocated metatarsophalangeal joints and ankle equines. Motor neuropathy changes the biomechanics of the foot and directly contributes to increased shear and pressure under the balls of toes, the most common site of neuropathic foot ulcers. Severe motor neuropathy contributes to the form of 'intrinsic minus' foot, or the appearance of high arch structures because of muscle wasting and weakness.

Autonomic neuropathy: Early on in the course of neuropathy, autonomic dysfunction arises. Autonomic dysfunction in the foot causes blood to shunt through direct arteriole venule connections, reducing perfusion effectiveness. Hair, sweat, and oil gland function are lost, resulting in dry, scaly skin that cracks and fissures easily. Touch and proprioception are less affected than vibration, pain, and temperature sensations.

C. Immunopathy

It's debatable whether immunopathy in the development of infection in a diabetic patient. The majority of researchers feel that poor glucose control makes patients more susceptible to infection. Humoral immunity appears to be normal in patients with diabetes. The levels of circulating immunoglobulins are normal to slightly raised, and B lymphocytes number is normal. Elevated glucose concentrations can have a major impact on cell-mediated immune responses. The diabetic patient's host defence mechanism appears to be compromised at the cellular level, as evidenced by decreased leukocyte function and intracellular killing. In

association with hyperglycemia, phagocytosis & the leukocyte's intracellular killing activity appear to be considerably changed. Improved diabetes control has partially or totally reversed some abnormalities.

In poorly controlled diabetes patients, MacCuishet found a reduction in phytohemagglutinin-induced lymphocyte transformation, but not in well-controlled diabetic patients or healthy people. T lymphocyte immunodeficiency in type 1 diabetes proved to have a poor response to staphylococcal antigen in diabetic individuals, regardless of glycemic management[14] .

Precipitating factors:

Even if the predisposing variables are present, an injured foot may not develop major issues. Physical trauma, on the other edge, is a powerful source of trouble.

a. A puncture wound, such as one caused by an ingrowing toe nail, allows germs to enter and cause illness. Furthermore, if there is extensive ischemia of the tissues, necrosis may develop.
b. Ischemic necrosis is result of localized pressure, such as from tight shoes.
c. Repetitive mechanical trauma, such as walking, can result in inflammation and necrosis.
d. In case of diminished feeling of warmth and pain sensation, heat, such as from a hot water bottle, may hurt the skin.

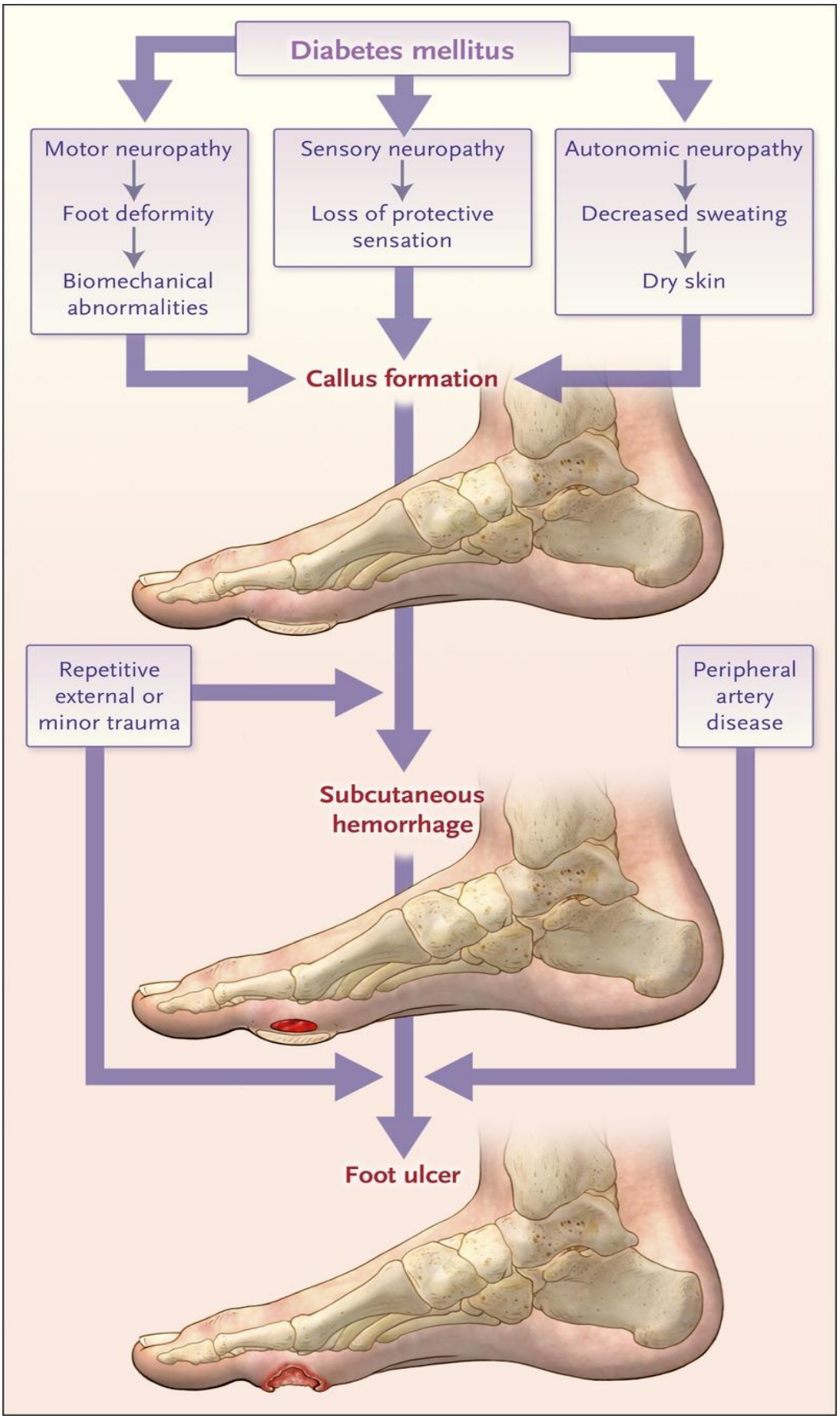


Fig. 9: Pathogenesis of diabetic ulcer

The essential premise is that predisposing variables impair one's ability to cope with minor injuries that develop into big lesions. The exception to this rule is not having of mechanical damage , significant vascular disease can cause gangrene.

DIABETES AND WOUND HEALING:

Infection plays a significant role in the morbidity of diabetes individuals with foot problems.

It's unclear whether they're more susceptible to infection due to weakened resistance or whether the lower blood flow allows infections to take hold and the neuropathy allows the infection to go unnoticed.

Diabetes may disrupt the inflammatory and wound-healing processes by decreasing-

a. The blood flow to the afflicted area.

b. The inflammatory response's efficacy.

c. Fibrous tissue development as the repair process.

a. Impairment of blood supply:

Small wounds may not be able to heal properly due to a lack of blood flow, resulting in necrosis and infection.

The growth of anaerobic organisms is promoted in ischemic tissue, especially if there is concurrent growth of aerobes. There are various routes by which diabetes-related microvascular alterations can impede damage response. Small vessel obstructions may restrict blood flow from growing sufficiently to facilitate healing. Furthermore, thickening of the capillary basement membrane may affect permeability, obstructing leukocyte movement and fluid exudation. Frank wound ischemia is harmful to all wound healing & a role in progress of chronic wounds in the first place. Relative hypoxia, on the other hand, is a more

common clinical condition that in the creation and failure to cure wounds. Hypoxia is a powerful stimulator of fibroblast growth and angiogenesis at first**.**

Hypoxia slows wound healing. Fibroblasts cannot reproduce and collagen production is significantly reduced in an environment with 30 to 40 mmHG of oxygen.

Hypoxia in the wound also makes it more susceptible to bacterial invasion.

a. Formation of Fluid-Cellular Exudates:

There is final proof that diabetes impairs the various stages of inflammation.

a. Polymorph adhesion to vessel walls is reduced, and the rate of escape from vessels is reduced. This is joined to the level of fasting blood glucose and can be treated to return to normal.
b. White cell movement in response to a chemical stimulus (chemo taxis) is hindered.
c. The polymorphs' ability to eat and kill germs is diminished. Although the findings of the numerous studies are not unequivocal, it is likely that hyperglycemic individuals' leucocytes are less effective at both engulfing and killing bacteria. Diabetes control may help to improve this deficiency.

In wounds with both short and long duration, wound infection has been found to hinder wound contraction. The release of bacterial enzymes and metalloproteinases, which may dissolve fibrin and promote wound development, is thought to be the process.

b. Formation of Fibrous Tissue:

In insulin deficiency - Diabetes, Goodson and Hunt (1979) found that the development of strength in an incised wound, which was closely related collagen produced in the tissues closest to the wound edge, was reduced.

Goodson and Hunt's tests showed that if insulin was administered immediately after wounding, granulation tissue production could be restored to normal. There was no increase in collagen quantity generated when insulin replacement was delayed until the time of maximal collagen synthesis (approximately 10 days after injury). If this finding holds true in humans, it suggests that diabetes control should be prioritised in the early postoperative period. In practise, this is the time when maintaining good control is most challenging[14]

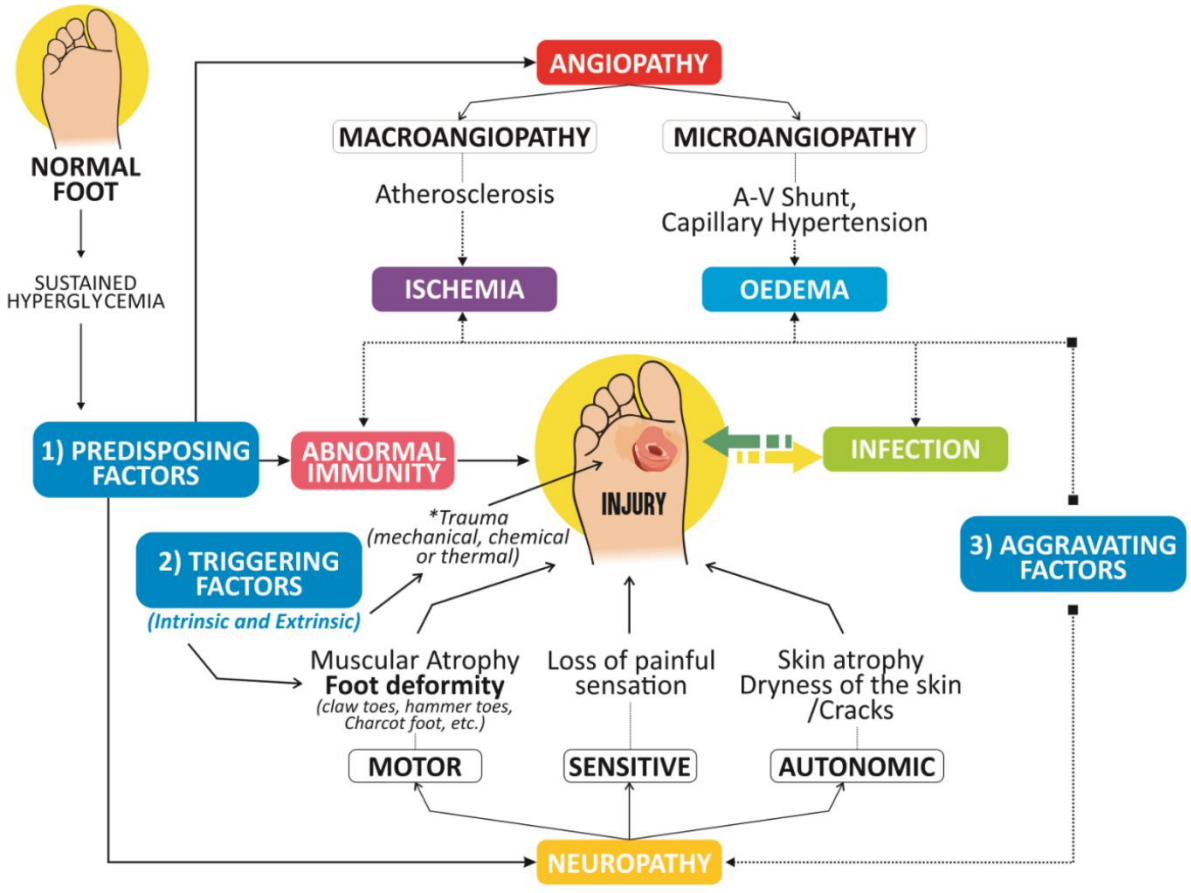


Fig 10. Pathogenesis of diabetic neuropathy

Diabetic Gangrene

This is seen in neuropathic foot where the arterial tree appears perfectly normal and so are the pulsations throughout the limb. The gangrene is due to primary infection followed by secondary thrombosis ofthe digital vessels. It is slowly progressive and generally remains limited to the foot in which it began.

Necrotizing Fascitis

It's a rather uncommon diabetic foot infection consequence.

It's an infection that causes necrosis of subcutaneous tissue and fascia, as well as noncrepitus gangrene of the overlying skin. It's mainly caused by Streptococcus pyogenes, however staphylococcus aureus can also cause it. The inflammatory area is the pathogonomonic indicator, and the affected area is initially red, hot, swollen, and painful. This can lead to full-blown gangrene. This condition, if left untreated, might result in death within days.

The neuropathic foot

• Due to arteriovenous shunting and swollen dorsal veins, it is a warm, well-perfused foot with bounding pulses.

• Sweating is reduced, and the skin may be dry and prone to fissuring.

• The toes can be clawed, and the arch of the foot can be lifted.

• Despite strong circulation, necrosis can develop as severe infection, and it is also prone to bone and joint problems (the charcot foot).

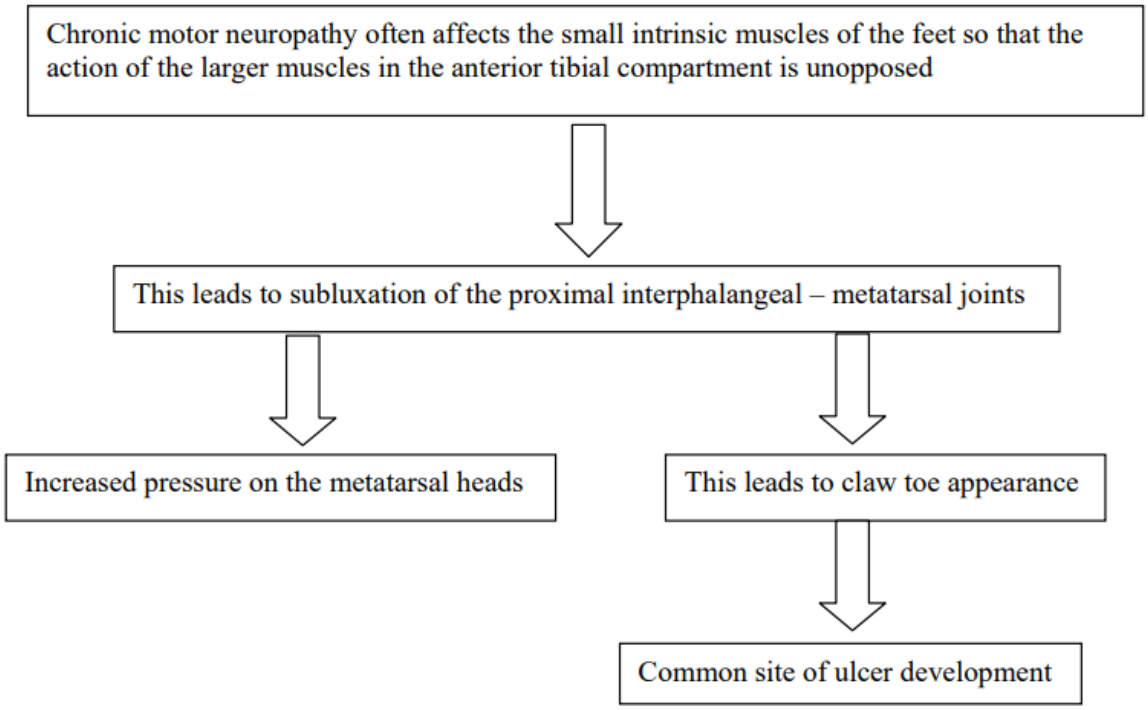


Fig. 11Charcot foot

The neuroischaemic foot:

- The colour of the severely ischemic foot might be a deceptively healthy pink or red, generated by dilation of capillaries in an attempt to enhance perfusion. It is a chilly, pulseless foot with diminished perfusion and invariably has neuropathy.
- Swelling, which is typically caused by cardiac or renal failure, can make the ischemic foot appear deceptively warm if it is severely infected.
- Ulceration is the most common presenting symptom.
- Ischaemic ulcers are most commonly found on the foot's margin, which comprises the tips of the toes and the area around the heel backside , and are typically caused by trauma or wearing inappropriate footwear.
- Because of neuropathy and the distal distribution of vascular disease in the leg, intermittent claudication and rest discomfort may be absent.

- Plantar ulceration develops necrosis if associated with infection or if tissue perfusion is critically diminished, even if neuropathy is present and plantar pressures are high[16].

Biomechanics of Diabetic Foot Gait cycle:

1. Stance phase
2. Swing phase

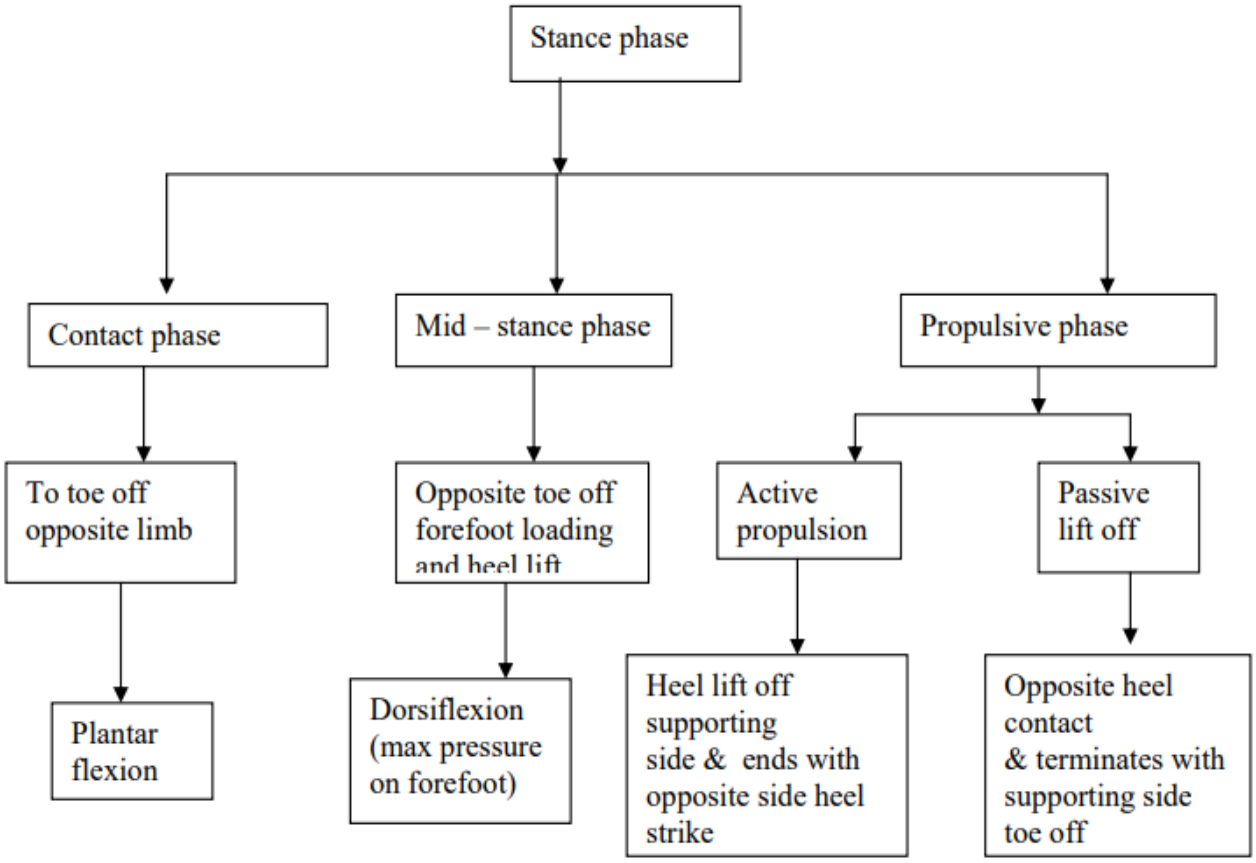


Fig. 12: Biomechanics of diabetic foot gait cycle

WOUND HEALING

Wound healing is a complicated physiological process that is influenced by several interconnected elements. The examination and management of wounds should be based on a knowledge of natural tissue repair and the factors that influence it.

The process of wound healing:

The body's tissues can mend through one of two mechanisms:

Regeneration or Repair.

Regeneration is more limited than repair in that it involves the restoration of damaged tissues with identical cells. Complete regeneration occurs in a few number of human cells, such as epithelial, liver, and nerve cells.

The main healing method is repair, which involves the replacement of injured tissue with connective tissue, which eventually produces a scar. The process by which the body replaces and restores function to damaged tissues is known as wound healing[17].

The major goal of wound management should be to maintain a controlled set of local conditions that can support the complicated cellular activity that occurs during wound healing. When striving to enhance a wound's healing capacity, providing a supportive microenvironment at the wound surface is critical[18]

Phases of Wound Healing

John Hunter (1728-1793), a keen observer of biologicphenomena, “the injury alone has in all cases a tendency to produce the disposition & the means of a cure”. Normal wound healing follows a predictable pattern that can be divided into overlapping phases defined by characteristic cellular populations and biochemical activities:

(a) Hemostasis and inflammation

(b) Proliferation

(c) Maturation and remodeling.

All wounds need to progress through this series of biochemical events at cellular level that characterize phases of healing to successfully re-establish tissue integrity.

A. Hemostasis and Inflammation

Hemostasis occurs before and during inflammation, which is followed by the release of chemotactic substances from the wound site. Wounding, by definition, compromises tissue integrity, resulting in blood vessel division and direct extracellular matrix exposure to platelets. Platelet aggregation, degranulation, and activation of the coagulation cascade occur when sub endothelial collagen is exposed to platelets. Platelet-derived growth factor (PDGF), transforming growth factor beta (TGF), platelet-activating factor, fibronectin, and serotonin are among the wound-active chemicals released by platelet granules. In addition to attaining hemostasis, the fibrin clot acts as a scaffold for inflammatory cells such as polymorphonuclear leukocytes (PMNs or neutrophils) and monocytes to migrate into the wound.

Following an injury, cellular infiltration follows a predictable pattern. PMNs are the first cells to infiltrate the wound site, peaking between 24 and 48 hours. Neutrophil migration is local prostaglandin production, and the presence of chemotactic chemicals such as complement factors, interleukin-1 (IL-1), tumour necrosis factor alpha (TNF-), TGF, platelet factor 4, or bacterial products. The phagocytosis of microorganisms and tissue debris is thought to be neutrophils' principal function. Early in the course of inflammation, PMNs are a key source of cytokines, particularly TNF, have a considerable impact on later angiogenesis and collagen formation.

In the early stages of wound healing, PMNs also release proteases such as collagenases, which aid in matrix and ground material disintegration. These cells do not appear to have a function in collagen deposition or the acquisition of mechanical wound strength, other from restricting infections. Neutrophil factors, have been linked to a delay in wound epithelial closure. Macrophages are the second type of inflammatory cell to penetrate the wound, and they are known to be critical for effective healing. Macrophages, which are derived from circulating

monocytes, reach large numbers in the wound 48 to 96 hours after the damage and remain there until the wound heals completely.

Macrophages, like neutrophils, help to debride wounds by phagocytosis and contribute to microbial stasis by generating oxygen radicals and nitric oxide. The most important role of the macrophage is to activate and recruit other cells through mediators, as well as directly through cell-cell interaction and intercellular adhesion molecules. Macrophages influence cell proliferation, matrix synthesis, and angiogenesis by producing mediators such as TGF, vascular endothelial growth factor (VEGF), insulin-like growth factor, epithelial growth factor, and lactate. Macrophages are also important regulators of angiogenesis, matrix deposition, and remodeling . T lymphocytes are a type of inflammatory/immune cell that invades the site on a regular basis. T-lymphocyte counts, which are less abundant than macrophages, peak at around 1 week after injury and truly bridge the inflammatory to proliferative phases of healing. Although lymphocytes are recognized to be important in wound healing, their involvement in wound healing is not entirely understood.

T lymphocytes have an active part in the regulation of the wound environment, according to a large body of evidence. Most wound T lymphocytes are depleted, which reduces wound strength and collagen content, whereas selective depletion of the CD8+ suppressor T lymphocyte subset improves wound healing. Depletion of the CD4+ helper subset, on the other hand, had no effect. Lymphocytes also inhibit fibroblast collagen synthesis by secreting cell-associated interferon, TNF-, and IL-1. This impact is abolished, indicating that extracellular matrix formation is controlled not just by soluble substances but also by direct cell-cell interaction between lymphocytes and fibroblasts.

B. Proliferation

The proliferative phase is the second stage of wound healing, and it lasts around four to twelve days. The re-establishment of tissue continuity occurs at this period. The last cell groups to invade the healing wound are fibroblasts and endothelial cells, and the strongest chemotactic agent for fibroblasts is PDGF. To carry out their primary job of matrix synthesis remodelling, recruited fibroblasts must first proliferate and then become activated once they enter the wound area. These are produced by wound macrophages are primarily responsible for this activation.

Wound fibroblasts produce more collagen, proliferate less, and actively carry out matrix contraction than non-wounded fibroblasts. Although it is obvious that the cytokine-rich wound environment plays a key role in phenotypic modification and activation, the particular mediators are yet unknown. Lactate is also a powerful regulator of collagen synthesis through a process requiring adenosine 5'-diphosphate-ribosylation, which accumulates in considerable amounts over time (10mmol).

During this stage of repair, endothelial cells also grow in large numbers. These cells assist in the formation of new capillaries (angiogenesis), which is critical for wound healing. Endothelial cells migrate from the wound's intact venules . TNF, TGF, and VEGF and their migration, replication, and creation of new capillary tubules. VEGF is produced by numerous cells, although macrophages are a primary source in the healing wound, and VEGF receptors are only found on endothelial cells.

MATRIX SYNTHESIS

Biochemistry of Collagen

Collagen, the most abundant protein in the body, is essential for adult wound healing to be completed successfully. The wound's functional integrity depends on its deposition, maturation, and subsequent remodelling. Although there are at least 18 different forms of collagen, types I

and III are the most important for wound healing. The main component of the extracellular matrix in skin is type I collagen. During the mending process, Type III, which is also found in skin, becomes more prominent and important.

Biochemically, each collagen chain has a glycine residue every third position. Proline or lysine takes the second position in the triplet during translation. Protocollagen is a polypeptide chain made up of residues of one thousand amino acids that is produced via messenger RNA transcription (mRNA). Specific hydroxylases hydroxylate proline to hydroxyproline and lysine to hydroxylysine once protocollagen is released into the endoplasmic reticulum. Prolyl hydroxylase requires oxygen and iron as cofactors, a co substrate in the form of ketoglutarate , and an electron donor in the form of ascorbic acid (vitamin C). The protocollagen chain is similarly glycosylated in the endoplasmic reticulum by connecting galactose and glucose at certain hydroxylysine residues. The hydrogen bonding forces within the chain are altered by the hydroxylation and glycosylation processes, resulting in steric alterations that push the protocollagen chain to acquire a helical form. Three helical chains entwine to form "procollagen," a right-handed super helical structure. Registration peptides are nonhelical peptide domains found on both ends of this structure. The covalent cross-linking of lysine residues strengthens the procollagen molecule, which is initially connected by weak ionic bonds. Extracellularly, a procollagen peptidase cleaves the nonhelical registration peptides, and the procollagen strands are further polymerized and cross-linked. By forming intra molecular and intermolecular covalent connections, the resultant collagen monomer is further polymerized and cross-linked.

Systemic variables such as appropriate oxygen supply, the presence of sufficient nutrition (amino acids and carbohydrates), cofactors (vitamins and trace metals), and the local wound environment all have a role in collagen synthesis and posttranslational alterations (vascular supply and lack of infection). Collagen production and deposition can be improved by addressing these factors and correcting dietary deficits.

Proteoglycan Synthesis

Glycosaminoglycans make up a significant component of "ground substance" in granulation tissue. Rarely found free, they couple with proteins to form proteoglycans. The polysaccharide chain is made up of repeating disaccharide units composed of glucuronic or iduronic acid and a hexosamine, which is usually sulfated. The disaccharide composition of proteoglycans varies from about 10 units in the c/o heparan sulfate to as much as 2000 units in the case of hyaluronic acid.

Dermatan and chondroitin sulphate are the most common glycosaminoglycans found in wounds. These chemicals are synthesized by fibroblasts, and their concentration rises dramatically during the first three weeks of recovery. The interaction between collagen and proteoglycans is currently being investigated. The lattice supplied by sulfated proteoglycans is to be necessary for formation of fibrils and fibres. Furthermore, it appears that the degree of sulfation is important in determining the collagen fibril structure. Proteoglycans are integrated into the collagen framework as collagen is deposited in scar. However, when scars mature and collagen remodels, the amount of proteoglycans in the scar decreases.

C. Maturation and Remodelling

The scar grows and remodels during the fibroplastic phase, which is distinguished by a reorganisation of previously generated collagen. Collagen is broken down by matrix metalloproteinases, and the net wound collagen content is the result of a balance between collagenolysis and collagen synthesis. As a result, there is a net shift toward collagen synthesis, resulting in the re-establishment of an extracellular matrix made up of a relatively acellular collagen-rich scar.

A fresh wound's wound strength and mechanical integrity are determined by the quantity and quality of newly deposited collagen. The pattern of matrix deposition at the wound site is predictable: Fibronectin and collagen type III make up the early matrix scaffolding;

glycosaminoglycans and proteoglycans are the next major matrix components; and collagen type I is the ultimate matrix. Several weeks after the damage, the amount of collagen in the wound reaches a peak, although the tensile strength continues to rise for several months. The benefits of fibril formation and cross-linking include decreased collagen solubility, greater strength, and higher resistance to enzymatic breakdown of the collagen matrix. Scar remodelling continues after a few months (6 to 12 months), finally resulting in a mature, avascular, and acellular scar. The mechanical strength of a scar is never equal to that of healthy tissue.

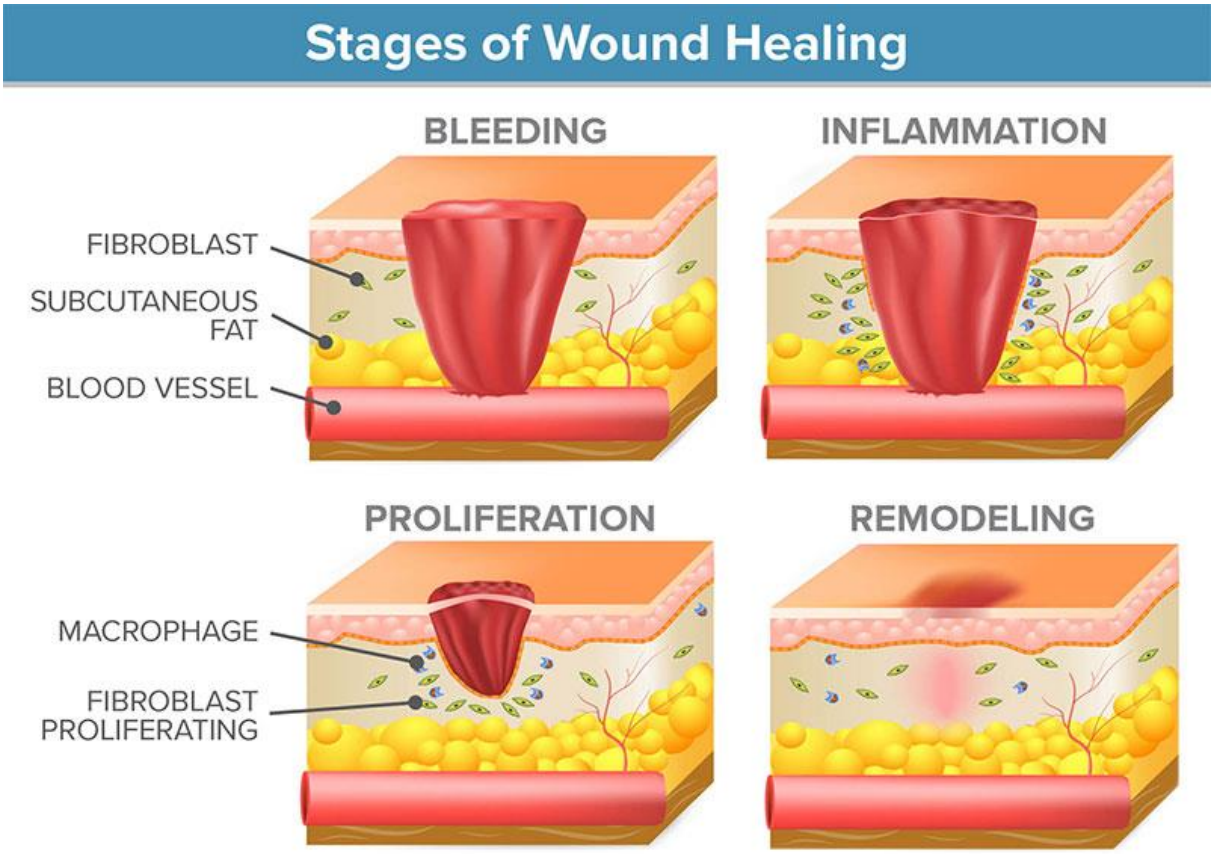


Fig. 13 Stages of wound healing

Extracellular matrix is a form of collagen constantly changing, in wound which is healing & tissue homeostasis. Collagenolysis is caused by collagenase activity, which is a type of matrix metalloproteinase that needs to be activated. These are in charge of both collagen synthesis and lysis. Collagen remodelling is influenced by a number of factors. TGF, for example, stimulates the creation of inhibitors of tissue metalloproteinase, which enhances new collagen transcription

& lowers collagen degradation. The ultimate determinant of wound strength and integrity is the balance of collagen deposition and breakdown.

Epithelialization

Tissue integrity and strength are being re-established, the external barrier must also be restored. Proliferation and migration of epithelial cells near to the wound are the hallmarks of this process. The process begins within one day after injury and manifests itself as epidermal thickening at the wound edge. Marginal basal cells at the wound's edge lose their strong attachment to the underlying dermis, expand, and migrate over the temporary matrix's surface. Fixed basal cells in a zone near the cut edge undergo a series of rapid mitotic divisions and these cells appear to migrate by moving over one another in a leapfrog fashion until the defect is covered. Once the defect is bridged, the migrating epithelial cells lose their flattened appearance, become more columnar in shape and increase their mitotic activity. Layering of the epithelium is re-established and the surface layer eventually keratinizes.

In the case of approximated incised wounds, re-epithelialization takes less than 48 hours, but it might take much longer in the event of bigger wounds with a severe epidermal/dermal defect. Repair consists predominantly on re-epithelialization with limited or no fibroplasia and granulation tissue formation if only the epithelium and superficial dermis are injured, as in split-thickness skin transplant donor sites or superficial second-degree burns. The stimuli for re-epithelialization remain incompletely defined; however, it appears that the process is mediated by a combination of a loss of contact inhibition; exposure to constituents of the extracellular matrix, particularly fibronectin and cytokines produced by immune mononuclear cells. In particular, epithelial growth factor, TGF, basic fibroblast growth factor, PDGF and insulin-like growth factor I have beenshown to promote epithelialization.

Wound Contraction

All wounds undergo some degree of contraction. For wounds that do not have surgically approximated edges, the wound area will be decreased by this action (healing by secondary intention); the decrease in length of scar itself results in contracture. The myofibroblast has been postulated as being the major cell responsible for contraction and it in that it possesses a cytoskeletal structure. Typically, this cell contains smooth muscle actin in thick bundles called stress fibers, giving myofibroblasts contractile capability.

Smooth muscle actin is undetectable until day 6 of wound healing, after which it becomes significantly expressed over the next 15 days. This expression declines after 4 weeks, and the cells are thought to be undergoing apoptosis. One perplexing aspect is that the detection of myofibroblasts in the wound does not always match to the onset of wound contraction, which occurs virtually immediately after injury. In vitro, fibroblasts placed in a collagen lattice actively move and compress the lattice without expressing stress fibres. Contraction is thought to be caused by the movement of cells and the remodelling of the cytoskeleton that occurs as a result[19].

COMPLETION OF HEALING

In wound healing, growth factors normally follow fundamental rules, and healing comes to a halt at the appropriate time. Local hypoxia and lactic acidosis appear to be the ultimate triggers for the production of growth factors and cytokines in most cases. Healing should come to a halt as these impulses fade away as the new microcirculation grows[20] .

CLINICAL FEATURES:

The DF syndrome or disease (DFD) includes several pathologies, mainly neuropathy which is seen peripherally in diabetic foot and peripheral arterial disease which result in foot ulceration. Diabetic foot ulceration which results to amputation, especially when wound infection or

osteomyelitis are involved. DF ulcer is defined as a full thickness wound which is present at a level distal to the ankle in patients with diabetes. Patients with DF are also more likely to present with other diabetes related complications such as nephropathy, retinopathy, ischemic heart disease and cerebrovascular disease[21].

Evidence of neuropathy

- Foot posture, claw toes, and callus over pressure sites are all signs of neuropathic alterations.
- Sensory neuropathy glove and stocking peripheral type of distribution
- Patients describe a feeling senseless walking when they have cold or lifeless feet.
- With adequate supply of blood, the foot may feel dry & heated on rare instances.
- On the toes and foot, there is a loss of mild touch and a discomfort (pin prick) sensation.
- This loss sensation may extend to the calf in more severe situations. Pain perception loss could lead to harm that goes unnoticed.
- Reflexes of tendons of ankle and plantar are absent ; loss of vibration perception in the foot; at the ankle and knee

Evidence of ischemia:

- Patient complains of rest discomfort or intermittent claudication
- Coldness of the foot
- Absence of ankle pulses
- Dependent rubor

VARIOUS PRESENTATIONS OF DIABETIC FOOT

Nail Problems

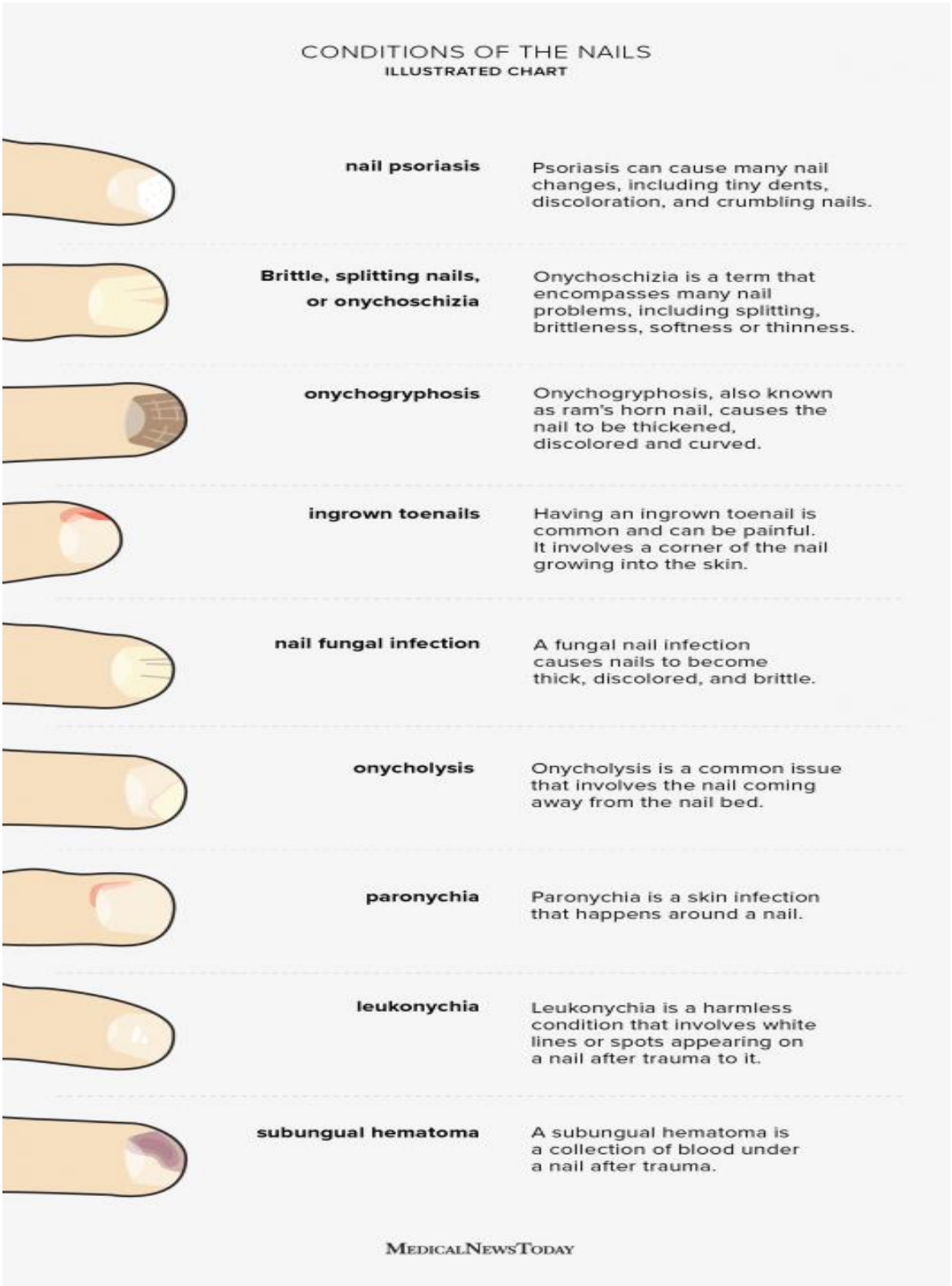


Fig 14: Nail problems in diabetes

Lesions under the nail

These can be due to: haematoma , necrosis, melanoma, exostosis.

Fissures: Points where the skin is strained tend to develop fissures which are moist or dry cracks of epidermis. I n deep cracks dermis will be involved . Dry skin can develop fissures. Emollients, such as E45 cream, olive oil, or coco butter, are used to treat dry skin, whereas an astringent or antiperspirant, such as aluminum chloride, is used to treat wet skin.

Verrucae: Warts can emerge as circular flattened papules or plaques anywhere on the foot, and they can be single or many. Without therapy, the majority of cases will resolve within two years. Cryotherapy with liquid nitrogen is the suggested treatment for persons with diabetes who have painful or spreading verrucae. The wart may need to be surgically removed in some cases.

Bullae (blisters): Following skin trauma, these are collection of clear fluid superficially within or beneath epidermis. Unsuitable footwear, inability to wear socks, and walking in wet footwear are also common reasons. Cleaning and covering little flaccid bullae with a sterile non-adherent dressing is possible. Large bullae (over 1 cm in diameter) and all tense bullae should be lanced and drained with a scalpel before dressing; aspiration using a syringe is less effective because the hole frequently shuts. Fluid builds up again, and the blister expands due to unrelieved hydrostatic pressure. Blister causes should always be determined and addressed.

Bullosis diabeticorum: Following skin trauma, these are superficial accumulations of clear fluid within or beneath the epidermis. Unsuitable footwear, inability to wear socks, and walking in wet footwear are also common reasons. Cleaning and covering little flaccid bullae with a sterile non-adherent dressing is possible. Large bullae (over 1 cm in diameter) and all tense bullae should be lanced and drained with a scalpel before dressing; aspiration using a syringe is less effective because the hole frequently shuts. Fluid builds up again, and the blister expands due to unrelieved hydrostatic pressure. Blister causes should always be determined and addressed.

Chilblains (perniosis): Cold and injudicious reheating cause these localized inflammatory sores. Chilblains are a common ailment that affects the toes.

Malignancy: Malignancies of foot are very rare compared to other malignancies with an order of squamous cell carcinoma,malignant melanoma and rarely basal cell carcinoma if present .

Hyperhydrosis: It is condition in which profuse sweating of the feet is seen and is most common in tropical climates population where humidity is high . In this condition, skin will be white, macerated and rubbery in texture and prone to blistering and fungal infections. It may be due to hyperthyroidism or anxiety.

Hammer toe: Hammer toe is a complex deformity consisting of contraction (hyperflexion) of the proximal interphalangeal joint, while the metatarsophalangeal joint is either dorsiflexed or in the neutral position. The distal interphalangeal joint may be in the neutral position, hyperextended or in plantar flexion. Hammer toe may be flexible or rigid. It is due to loss of balancing lumbrical functions.

Claw toes: Claw toes resemble hammer toes, however they have more buckling and deformity. The interphalangeal joint has a fixed flexion deformity, which is linked with callus and ulceration on the apex and dorsal surface of the joint. Although claw toes and neuropathy may be linked, they are frequently unrelated, especially when the clawing is unilateral and accompanied with forefoot trauma or surgery. Claw toes are a rare complication of a plantar fascia rupture.

Hallux valgus: Hallux valgus is a malformation of the first metatarsophalangeal joint characterized by lateral hallux displacement and a medial protrusion on the foot's border. In the neuroischaemic foot, this location is particularly fragile, and it commonly breaks down under the strain of a tight shoe.

Limited joint mobility (including hallux rigidus): Both feet & hands are affected by limited joint mobility. The subtalar and first metatarsophalangeal joints have a reduced range of motion. Loss of dorsiflexion & high pressures on the plantar surface of the first toe cause callus

development & ulceration due to limited joint mobility of the first metatarsophalangeal joint. It's most common in barefoot & sandal-wearing people.

Charcot foot: This is a type of deformity noted in DF persons. The deformity is " rocker bottom deformity" , which presents with displaced tarsus bone with downwards sublaxation , and the medial convexity, which is caused by talonavicular joint displacement or tarsometatarsal dislocation, are two classic deformities caused by damaged bones and joints in the foot . Both are frequently linked with a bony protrusion which is prone to ulceration and difficult to repair. Weakness and Instability of the hind foot will be seen in this condition as the joints of ankle and subtalar are involved.

Major Infections

1. **Cellulitis:** It is a subcutaneous infection associated with fluid collection which increases the compartment pressures and results in ischemic changes. Patients presents with swelling of limbs with stretched and shiny skin which usually associated with pain. If this is left untreated patients lands up into MODS. Most of the untreated cellulitis limbs require amputations either at level below or above the knee.
2. **Abscess:** Abscesses can affect one or multiple toes, as well as areas which are superficial and deep of the sole. Persons with this condition usually presents with discomfort , pain , discharge from the area with fluctuation positive . Swelling and redness usually noted over dorsal aspect of web spaces of the foot are the most common symptoms.

3. **Ulcer:** The dorsal or plantar aspect of the foot may be affected by an ulcer. Plantar ulcers are sometimes known as penetrating or trophic ulcers. They are usually painless and develop where weight is normally carried. A patch of hyperkeratosis, usually above a metatarsal head, is the first alteration to appear. The proximal interphalangeal joint of a clawed toe is the most

prevalent site for a toe ulcer. Hyperkeratosis, or inflammation, can occur before skin breakdown and the formation of a tiny ulcer.

4. **Gangrene:** The term gangrene refers to the death of tissue. Gangrene can be divided into two types. Infection is the cause of wet gangrene. A lack of blood supply causes dry gangrene. When there isn't enough blood reaching a portion of the foot, the skin and flesh might die and turn dark or black. On regions of the foot that are subjected to pressure, gangrene can develop. The heel, malleoli, and portions of the first metatarsal head medially and the base of the fifth metatarsal are all common places. Because of embolism of athermanous debris, small patches of gangrene may develop on regions of the foot that are not subjected to pressure. In the inter digital clefts, gangrenous patches can occur. If an infection develops, it may spread via tissue that is unable to contain the process due to a lack of blood flow, resulting in wet gangrene[22].

Wagner's Classification of Diabetic Foot Wounds[23].

"Grade 0 – No open lesions; may have deformity or cellulitis.

Grade 1 - Superficial, full thickness ulcer limited to the dermis, not extending to the subcutis.

Grade 2 - Ulcer of the skin extending through the subcutis with exposed tendon orbone and without osteomyelitis or abscess formation

Grade 3 - Deep ulcers with osteomyelitis or abscess formation

Grade 4 - Localized gangrene of the toes or the forefoot

Grade 5 - Foot with extensive gangrene"[23].

INVESTIGATIONS

I. BLOOD EXAMINATION:

a. Hemoglobin: This test is used to assess a patient's overall health as well as his or her suitability for certain surgical procedures.

b. Total WBC Count: This figure represents the body's defence system.

c. Differential WBC count: Provides a clue to diagnosis, such as an increase in lymphocyte count in tuberculosis and a decrease in neutrophil count in malnutrition.

d. ESR: In tuberculosis and other disorders, the ESR is elevated.

e. Bleeding and coagulation times: These timings may need to be adjusted if the patient is having surgery.

f. Fasting blood sugar: Used to evaluate the presence (or absence) of diabetes as well as the level of diabetic management.

g. HbAlc to assess prior diabetes status.

h. Serum creatinine: An indicator which is most sensitive of renal function that may be impacted by diabetic nephropathy.

i. Blood urea: This is another indicator of renal function, albeit it changes depending on the patient's hydration status.

j. Rheumatoid factor test: This test is performed to determine whether or not a person has rheumatoid arthritis.

k. VDRL test: This test is performed when a syphilitic ulcer is suspected.

l. HbsAg and HIV .

II. EXAMINATION OF URINE;

In v/o sugars and ketone bodies which are medical complications associated with diabetes.

III. BACTERIOLOGICAL:

Examination of the discharge: Inflammation & ulcer spreading necessitate this investigation. It's helpful to have a baseline bacterial culture with sensitivity results. It serves as a guide for choosing the right chemotherapy.

IV. BIOPSY:

When in doubt, a biopsy of the ulcer is helpful. It should be done to rule out any odd ulceration cases.

V. X-RAY OF FOOT:

Gross disintegration, fragmentation, periosteal new bone growth, and other deformity associated with diabetes are all common bone abnormalities seen on foot X rays in diabetic individuals. If infection is suspected, abnormalities in soft tissue shape and loss of tissue planes might be visible on plain images. Edema, or swelling, is a common occurrence. Other abnormalities include osteosclerosis, fragmentation, periositis, & regions which are radiolucent within the soft tissues that could be air as a result of debridement / open wound (or) microbes producing gas. Calcification of the arteries (also known as Monckeberg's diabetes or medial arterial calcification). The feet are usually the first and most common location of involvement.

VI. DOPPLER ULTRASOUND:

Is a good supplement to a physical examination. Because calcific stenosis might create artificially increased pressures, measuring the ankle-brachial ratio is less useful than in non-diabetic patients. The usual Doppler pulse is triphasic, but it becomes monophasic below a significant obstruction, and this can be heard. If all of the patients' foot pulses are triphasic on Doppler, the patients are unlikely to have severe ischemia. Formal noninvasive assessment is required if the pulses are

monophasic.

VII. DUPLEX IMAGING: (Duplex Ultrasound scanning)

This is a critical investigative approach in vascular disease. B-mode ultrasound is used in a duplex scanner to create a picture of the vasculature. The varying ability of the tissues to reflect the ultra sound radiation creates this image. The vessels are subsequently insolated with a second type of ultrasound, Doppler ultrasound, and the Doppler shift is analyzed by a dedicated computer in the Duplex scanner. These shifts can provide precise information on vascular blood flow and turbulence. Color coding is a feature on some scanners that enables for observation of blood flow on the image. The different colors represent changes in flow direction and velocity: high flow points usually imply stenosis. It enables for the measurement of the cross-sectional area of the artery lumen. The flow towards or away from the transducer may be easily discriminated using color, allowing for the detection of peripherally flowing artery flow.

TREATMENT:

The following are the treatment options for DF ulcers that are currently available.

- Off – loading
- Debridement
- Use of appropriate dressings
- Medical and surgical treatment of infection
- Vascular reconstruction and / or amputation or reconstructive foot surgery when necessary.

Offloading therapy:

Neuropathic DF wounds on the foot's plantar aspect occurbecause of a combination of focal pressure and repetitive stress at a given site[24]. The mitigation of either of these variables

(pressure or repetitive stress) may reduce risk for ulceration. Mechanical stress that occurs at right angles to the integument is termed "vertical stress." This tends to damage healthy tissue through repetitive compressive forces. Stress that is imparted parallel to the foot's plantar aspect is termed "shear." This shearing of soft tissue is equally damaging and is evidenced by the characteristic undermined nature of the periphery of poorly off-loaded diabetic foot wounds. Shear and vertical stress work in tandem in the pathogenesis of a diabetic foot wound. So relieving areas of elevated plantar pressure (off-loading) can prevent and heal plantar ulceration.

Methods to offload the foot include bed rest, the use of a wheelchair, crutch assisted walking, total contact casts, felted-foam, half-shoes, therapeutic shoes, custom splints and removable cast walkers. Total contact casts (TCCs) are considered the gold standard of the offloading and treatment of neuropathic ulcers[25].

The method is called "total contact casting" as it uses a cast which is padded and well molded , maintains contact with the whole foot's plantar aspect & the lower leg. Total contact casting have efficient results in treating lot of wounds which are not infected , non-ischemic plantar DF wounds, with healing rates ranging from 72% to 100% over a course of 5-7 week. High amount of peak plantar pressures are noted in the forefoot and are less significant in the hindfoot and medial arch TCC distributes pressures uniformly and allows walking and are more effective over the foot's plantar aspect . TCCs are effective for a number of other reasonsbesides their ability to off-load. They may help reduce or control edema that can impede healing and, thus, potentially protect the foot from infection. However, the most important attribute of this technique may be its ability to ensure appropriate patient compliance.

OFF LOADING TECHNIQUES.

- Accommodative dressings - patellar tendon – bearing braces
- Assistive devices - removable walking braces
- Callus removal - scotch cast boot
- Foot casts - shoe cutouts
- Half, wedge or surgical shoes - surgical correction of deformity
- Orthoses - therapeutic shoes
- Padded hosiery - total contact casting

Debridement:

Debridement is widely accepted as the most definitive treatment for the diabetic foot ulcer. Inadequate debridement may lead to prolonged infection, increasing risk for limb amputation. Sharp debridement of the DF ulcer stimulates the non migratory edge epithelium, releases growth factors and reduces the local inflammatory and proteolytic environment. The goal of operative debridement is to remove all hyperkeratotic tissue (ie : callus), necrotic tissue, functionally abnormal senescent cells and infected tissue, allof which inhibit wound healing. In this manner, the remaining tissue, although physiologically impaired, can respond to exogenous topical treatment, (ie, growth factors or cell therapy).

Clinical judgment has traditionally defined the margin of debridement, which is recognized as tissue with punctuate bleeding the margin of debridement of the skin edge should extend to the soft tissue beyond the callus. The depth of wound bed debridement should extend to tissue that is free of fibrosis and infection, eg, osteomyelitis, as confirmed by pathology and microbiology[23].

Infection Control:

Diabetic foot infections (DFIs) are usually a consequence of skin ulceration from ischemia or trauma to a neuropathic foot. The compartmentalized anatomy of the foot, with its various spaces, tendon sheaths and neurovascular bundles, allows ischemic necrosis to affect tissues within a compartment or spread along anatomic tissue planes. Recurrent infections are common, and 10% to 30% of affected patients eventually require amputation.

Diabetic patients are predisposed to foot infections, not only because of the portal of entry and poor blood supply, but also because of defects in humoral immunity (e.g., impaired neutrophil chemotaxis, phagocytosis, intracellular killing) and impaired monocyte macrophage function, which correlate with the adequacy of glycemic control. Cell-mediated immunity and complement function may also be impaired.

Acute infections are usually caused by gram-positive cocci- Staphylococcus aureus is the prioritized pathogen in DFIs. It is often present as a monomicrobial infection, but usually it is also an important pathogen in polymicrobial infections. Chronic wounds, recurrent infections and infections in hospitalized persons are more prone to harbor complex flora, including aerobic and anaerobic flora. Among gram-negative bacilli, bacteria of the family Enterobacteriaceae are common and Pseudomonas aeruginosa may be isolated from wounds that have been treated with hydrotherapy or wet dressings. Antibiotic-resistant bacteria, especially MRSA, may be isolated from patients who have received antibiotics previously or who have been hospitalized or reside in long-term care facilities.

Agents are proven to be effective for therapy of DFIs in clinical trials include cephalosporins, {3-lactamase inhibitor combination antibiotics, fluoroquinolones, clindamycin, carbapenems, vancomycin and linezolid. The time required for the therapy for DFIs has not been determined; common practice is to treat mild infections for 1 week, whereas serious infections

may require up to a 2-week course of therapy. Adequate debridement, resection or amputation can shorten the necessary durations of therapy.

Wound bed preparation:

The aim of preparation of bed of the wound is having granulation tissue which is well vascularized with no adjacent cellulites, drainage, or odor with removal of scar tissue. Proper debridement concurrently prepares the wound bed and stimulates the healing process. The four approaches of wound bed preparation, which address the different pathophysiological abnormalities underlying chronic wounds, are as follows:

1. Tissue management
2. Inflammation and infection control
3. Moisture balance
4. Epithelial (edge) advancement.

After cleaning of an infected wound and wound bed preparation, application of antibiotics topically may be efficacious[26].

DRESSINGS:

To avoid the creation of wound worsening as a result of devitalized tissue, tissues should be kept wet after debridement. A wet wound allows epidermal cells to migrate more quickly over the wound bed, promoting angiogenesis and connective tissue creation. Individualized dressings are advised in the treatment of DF ulcers. A lesion that is granulating adequately requires a dressing which is different than wound which is epithelializing; A wound that produces excessive amounts of exudates should be handled differently than a deep sinus wound.

The location of the wound, the depth of the wound, the amount of Escher or slough present, the amount of exudate, the state of the wound edges, the presence of infection, the need for adhesiveness, and the conformability of the dressing are all elements to consider while choosing the correct dressing.

Because the wound environment changes frequently during therapy, selection of dressing should be reevaluated on a regular basis to keep up with these changes.

The granulation tissue stimulation (new dressing technology has substantially improved, & other different products which are new have been created for the managing of various forms of ulcers with chronic duration) is an important part of optimal wound bed preparation.

Different variety of dressings which can kill bacteria while also assisting in wound healing.

In addition, some of these dressings have been discovered to provide protection against pollutants, bacteria, and viruses.

CLASSIFICATION OF DRESSINGS.

- Wound dressings have changed over time to provide protection to the wound's bare surface, absorb exudates, limit infection, promote granulation tissue production, and provide an optimum healing environment. Dressings can be categorised into two categories based on their intended use:
    1. Dressings for short-term use: These dressings must be replaced at regular intervals.
    2. Long-term applications, often known as skin substitutes, are classified into two categories: temporary and permanent. Temporary - used on fresh 'partial thickness wounds' until complete healing is achieved.
- Semi-permanent - used on a "full thickness wound" until autografting can be done.
- Dressings can be classified as conventional, synthetic, or biological depending on the type of material utilised in their manufacture. The dressings can be further classified within

each category as follows: primary dressing - a dressing that comes into direct touch with the wound bed.

- A secondary dressing is one that is applied after the primary dressing has been removed.

TYPES OF DRESSINGS [28]

Type of dressing	Advantages	Disadvantages
Traditional dressings (gauze and absorbent cellulose)	Cheap and widely available appropriate for gangrenous lesions	Adhere to the wound bed and may cause bleeding o removal. Provide little protection against bacteria contamination
Films	Semi – permeable,. Form bacteria barrier. Durable, require changing every 4-5 days. Cheap	Useful on flat or superficial wounds only. Some patients are allergic to the adhesive in the dressing
Foams	Appropriate for ulcers with high production of exudates. Provide thermal insulation. Easily conformable, may be used to fill cavities without sinus tracts	Effect difficult to quantify. Not as effective and rapid as surgical debridement. Not appropriate for neuro – ischemic ulcers, which produce minimal exudates. Wound must be monitored closely for signs of infection
Hydrocolloids	Safe and selective, using the body's own defense mechanisms. Good for necrotic lesions, with light to moderate exudates. May be used to fill cavities without sinus tracts. Can be easily used with a shoe. Adhesive surface prevents slippages. Do not require daily dressing changes. Cost effective	Their occlusive and opaque nature prevents daily observation of the wound. Wound must be monitored closely for signs of infection. May promote anaerobic growth and mask a secondary infection.
Alginates	Useful as absorbents of exudates. Good for infected ulcers. Some products have hemostaic properties.	Not appropriate for neuro – ischemic ulcers, which produce minimal exudates. May dry out and form plug with in the wound bed. Requires painstaking removal with the use of large amounts of saline.
Enzymatic dressings.	Good for any wound with a large amount of necrotic debris, and for eschar formation. Promote autolysis and fast healing decrease maceration of the skin and risk of infection	Costly, must be applied carefully only to the necrotic tissue. May require a specific secondary dressing. Irritation and discomfort may occur.
Medicated dressings		Data based on animal models and cell cultures only.

Fig 15: Types of dressings

Hyperbaric oxygen therapy:

Hyperbaric oxygen therapy (HBOT) is based on the premise that the delivery of supra physiological concentrations of oxygen to diseased tissues will result in beneficial physiological changes. The therapy is based on achieving an atmospheric pressure of 2-3 atmospherespressure which is administered using a sealed polyethylene bag over the affected area and administering 100 percent oxygen to a pressure between 20 and 30 mmHg. Treatments last 2 to 2 1/2 hours.

HBOT can be offered to patients who have DF ulcers for whom at least 30 days of standard wound care has failed and who have a Wagner grade III lesion or higher (meaning the ulcer must penetrate to tendon, bone, or joint and may be associated with deep abscess, osteomyelitis, gangrene, or septic arthritis) In thecase of diabetic foot ulceration, it is believed both that the function of phagocytic cellsis improved, assisting in the fight against any infection and that wound healing is independently aided through effects on cellular processes. Thus, it has been suggestedthat HBOT is useful for treating the infection and for the chronic diabetic wounds healing [29].

TOPICAL NEGATIVE PRESSURE WOUND THERAPY

Vacuum assisted closure (VAC), may also be known as “negative pressure wound therapy” (NPWT) or Microde-formational wound therapy or vacuum therapy (VT) or vacuum sealing (VS), which has brought a revolution in wound care since past 15 years. This modality was enlightened initially and elobrated by Fleischmann et al. in 1993[30].

INDICATIONS FOR NEGATIVE PRESSURE WOUND THERAPY

- Acute and Traumatic wounds.
- Pressure ulcers.
- Chronic wounds
- Flaps

- Grafts
- Wound Dehiscence

Contraindications to NPWT:

- Untreated osteomyelitis
- Wounds with malignancy
- Necrotic tissue along with eschar
- Exposed blood vessels and nerves
- Fragile skin: Before using the VAC system, the skin's integrity should be checked. When the adhesive dressing is peeled from the skin during dressing changes, it can induce shearing or skin avulsion in patients with thin skin due to ageing, chronic corticosteroid usage, or attributable to a collagen vascular problem. As a result, people who are allergic to skin adhesives should avoid using the VAC device.

SURGICAL MANAGEMENT OF DIABETIC FOOT:

AMPUTATION:

The following are the reasons for amputations:

I. Life-threatening infection: Although antibiotics have made this an uncommon occurrence, it is nevertheless essential on occasion, especially if there is surgical crepitus or air within the tissues.

II. Only severely ill and elderly individuals, who can't endure the shock physically and physiologically caused by amputation, will have dead tissue removed (including osteomyelitic bone).

III. Intractable pain is a rare complication of infections, but it can happen as a result of dry gangrene or acute ischemia.

IV. Lifestyle needs: These include everything from a wage earner's inability to take the long

periods of rest sometimes required for conservative management to undue boredom and personality deterioration in an elderly person who is excessively confined, whether in a hospital or at home, due to the immobility required to heal.

LOCAL AMPUTAION:

Pre-operative management:

i. Control diabetes: Good diabetes control will not be achieved until the infection has been drained, also underlying medical condition like ketosis must be treated & hyperglycemia lowered, which is normally accomplished with insulin infections.

ii. Infection control: Antibiotic medication is required for roughly 24-48 hrs before initiation of the surgery, unless anaerobic infection is present.

A. Ray Amputation: Many people consider it as very helpful of all local amputations because it eliminates a toe & metatarsal shaft distal half.

B. Toe Amputations: Amputation of only one toe

C. All (remaining) toes: Claw toes of foot with neuropathic changes have a minimal part in walking, but they make the foot vulnerable to mild damage, which can lead to morbidity. Disarticulation of toes which are healthy is occasionally justified if there is no uncertainty regarding the foot's blood supply.

D. Transmetatarsal amputation: For diabetics, this is a traditional local amputation through the metatarsals.

E. Symes amputation:It is performed on a foot with gangrenous changes that is too widespread for a transmetatarsal amputation to be performed safely, but the supply of blood is deemed to be adequate to allow recovery.

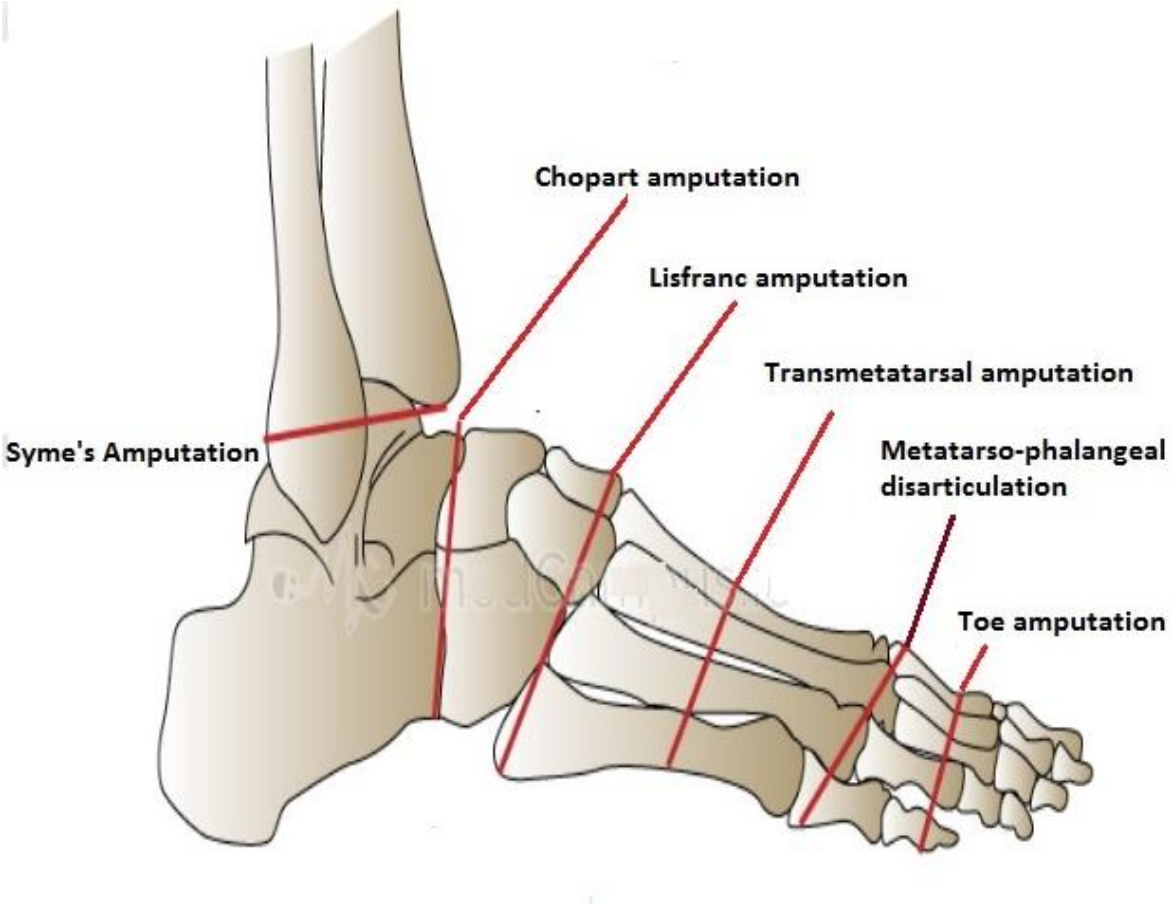


Fig. 16:Types of foot amputations

1. MAJOR AMPUTATIONS:

Below knee amputation (BKA).

2. HIGHER AMPUTATIONS:

A. Through knee disarticulation.

B. A supracondylar (Gritti-Strokes) amputation.

C. Mid-thigh amputation.[15]

3. VASCULAR SURGERY:

A diabetic's arterial disease is characterized by a relative sparing of the aorta and iliac arteries, as well as an increase in atherosclerosis occlusions in the calf arteries.

INDICATION FOR ARTERIAL SURGERY:

There are the same in the diabetic as in the non-diabetic limb :

- Intermittent claudication
- Threatened loss of limb.

Types of operations:

The type of surgery performed will be determined by the degree of the arterial occlusion as seen on an arteriogram; the criteria are that there must be appropriate pressure above the area to be rebuilt and that there must be patent vessels distal to the reconstructions. In order for the enhanced blood flow to reach the peripheral tissues.

I. For aorta-iliac stenosis or occlusion:
   - Aorta-femoral bypass (or)
   - Thrombo-endarterectomy.

II. For femoral artery occlusion:
   - Femoropopliteal bypass.
   - Profundoplasty[15].

THERAPY IN DF ULCER USING LASER :

"Light Amplification by Stimulated Emission of Radiation" Wound healing effect of laser is explained by its bio stimulation effect. It acts on cellular level and diminishes inflammation. It increases cell proliferation and formation of granulation tissue .

CLASSIFICATION OF LASER:

Class I- very low power laser

Class II- Low power laser <1mw

Class III 3a-Laser <5mw

3b-5mw-500mw

Class IV- High power laser >500mw

RED LASER:

Because of its high penetrance power of 10mm, a red laser with a wavelength of 635nm is employed for wound healing. It works by encouraging mitochondrial ATP release.

- Angiogenesis
- Increased protein synthesis
- Increased blood flow
- Improved fibroblast proliferation
- Increased collagen formation
- Reduced oxidative stress

AT CELLULAR LEVEL:

- Production of ATP is increased
- Macrophages releases growth factor
- Proliferation of keratinocytes

- Vasodilatation by NO synthesis
- Reduction of edema and inflammation

LASER DELIVERIES:

CONTACT METHOD: The probe is positioned directly over the target. This technique improves laser penetration. The disadvantages of this procedure are cross infection and discomfort.

NON-CONTACT METHOD: The probe is put at a safe distance from the target area. Multiple iodide cluster units are employed, & the non-contact approach is beneficial in open wounds.

TYPES OF LASERS:

GAS LASERS:

- Commonly used gas lasers are Helium Neon laser, carbon dioxide laser, argon ion laser
- Helium neon laser 633nm used in optical engineering
- Co2 lasers emits hundreds of watts, used for cutting and welding
- Argon ion lasers operates between 351 and 528nm

CHEMICAL LASERS:

- Formed by chemical reactions which emits high amplitude of energy
- Examples are hydrogen fluoride laser, deuterium fluoride laser

EXCIMER LASERS:

- Special sort of gas laser formed by electric discharges
- These are all noble gas compounds, active only in excited state, otherwise chemically inert
- Examples are ArF 193NM, KrCl 232nm, KrF 248NM, XeF 351nm

SOLID STATE LAESRS:

- Crystalline or rod chemicals combined with ions releases energy states
- Examples are ruby, chromium, neodymium, yttrium, aluminium Garnet (Nd:YAG)

FIBER LASER:

- Fiber lasers are total internal reflection in single mode optical fibre

DYE LASERS:

- Organic dyes are used as lasers, single or multiple mixtures can be used.

OTHER USES:

1. Tennis elbow -82 % pain relief
2. Fibromyalgia-66 % pain relief
3. Headache -70 % pain relief
4. Low back ache-71 % relief
5. Rheumatoid arthritis-stiffness was reduced
6. Hair loss-improves hair growth

PHYSICAL CHARACTERISTICS OF LASER:

There are three characteristics of laser light that clearly differentiate it from ordinary light.

1. **MONOCHROMATICITY**: Ordinary light is comprised of a conglomeration of many wave lengths, commonly known as VIBGYOR, i.e. Red, Orange, Yellow, Green, Blue, Indigo, and Violet, all merging to produce "white" light. Wave length of laser light is 6,328 A units. This wave length falls within the visible spectrum (3,900 to 7,700 A units) in R section, the laser light of 6,328 A units is a brilliant red color.
2. **COHERENCE:** Wave lengths of ordinary light are so variable and do not "match" in wave forms, frequencies, or shapes, there is much scrambling of wave forms, cancellation and reinforcements of individual waves, and interference in the energy production in general; this

factor minimizes the power of ordinary light as an energy source. The identical wave lengths and forms, that comprise laser light, lead to great amplification since the “waves and troughs” of the radiation are reinforced. Because they are parallel and in line with each other, they are termed coherent.

3. **NON-DIVERGENCE:** The laser beams unique in the absolute “straightness” of the directed radiation. Ordinary light shines in all directions (e.g. consider a light bulb radiating in all directions). The sun is another example of omni directional radiation. The laser , shines in only one direction, not unlike a flashlight, although its beam is far more concentrated and narrowed. The divergence of a laser beamed to the Moon surface and Earth showed a deflection of just a few meters after a journey of more than 260,000 miles.

OUTPUT POWER = STRENGTH (W/Mw)

Power is measured in watts (W). the strength or power output of a laser is thus measured in watts or milli watts (mW=A thousand of watt). Higher output power means higher power density, which is often desirable. In addition, a higher output power means that a certain desired dose (input energy. Measured in joules per cm2 of skin) is more quickly reached because energy is the same as power multiplied by time.

ENERGY:

Joules = Power (mW) X Time (Sec) Eg Joules = 2.5mW X4sec = 10J/cm2

THE FOLLOWING AREAS SHOULD NEVER BE IRRADIATED:

- Eyes
- Pregnant uterus
- Thyroid glands
- Male external genitalia
- Skin if there is cutaneous or subcutaneous bacterial infection.

- Growing cartilage in children.

WOUND HEALING & REPAIR

PRINCIPLE OF ARNDT – SCHULTZ

- If the quantity of energy is too small to stimulate the absorbing tissues, NO significant reaction will take place.
- If the quantity of energy absorbed per unit of time is adequate to stimulate, the absorbing tissue will perform its normal function.
- It the quantity for energy is too great, per unit time, the absorbing tissue will be disrupted and cannot perform its normal function.
- Local tissue temperatures SHOULD NOT be elevated above 45oc that tissue destruction is likely to occur.
- Therefore proper selection of laser dose is necessary to prevent photo – inhibitory effects (as it works on the principle of ARNDT – SCHULTZ).
- Effects of LLLT are not thermal and are completely photochemical & the responses of cells occurs due to changes in photo acceptor molecules (also known as chromophores molecules which are able to absorb photonic energy) such as porphyrin .
- Mechanism of action in detail of LLLT is not completely understood.
- However, it is known that during laser irradiation cells absorb photonic energy that is incorporated into chromophores , which in turn, stimulates cellular metabolism
- The chromophores are able to transfer the absorbed energy to other molecules and thus cause chemical reactions in surrounding tissue.

DOSAGE: Actual dosages with the cold laser depend on the power factor, duration of radiation, and tissue resonance.

WOUND HEALING

In most wound healing applications, the prescribed dosage is 10,600 nm wavelength, 2-10 sec/cm^2 of open lesion. This may require several minutes of hand – held direction of the beam over wound surface so that each square centimeter is exposed for the same 2-10 seconds. The probe tip is held approximately 2 to 3 mm from the surface to obtain a “Disc” of laser light about 1 cm in diameter on the wound surface . The non – divergent nature of the laser is modified by a lens – like spreading of the beam for therapeutic purposes.

MATERIALS AND METHODS

Source of Data:

Patients with diabetic ulcer were selected for this study from Dept. of General Surgery, R. L. Jalappa Hospital and Research Centre, Tamaka, Kolar attached to Sri Devraj Urs Medical College.

Study Population: Patient with DF ulcers of chronic duration with Grade I-III (Megitt-Wagner Grade)

Inclusion Criteria:

1. Type 2 diabetes mellitus
2. Patients with ulcer duration > 3weeks
3. Grade I-III Meggitt-wagner classification
4. Negative wound cultures

Exclusion Criteria:

1. Cellulitis, septicemia, shock
2. Immunocompromised patients
3. Diabetic gangrene
4. Pulseless limb
5. Associated osteomyelitis
6. Pregnant women

Duration of study: December 2019 and June 2021

Study Design: Observation study

Sampling technique: Purposive sampling

Sample size:

Formula

$$n = \frac{2s_p^2\left[Z_{1-\alpha/2} + Z_{1-\beta}\right]^2}{\mu_d^2}$$

$$s_p^2 = \frac{s_1^2 + s_2^2}{2}$$

Where,

s_1^2 : Standard deviation in the first group

s_2^2 : Standard deviation in the second group

μ_d^2 : Mean difference between the samples

α : Significance level

1- β : Power

Two Means – Hypothesis testing for two means (equal variances)

Standard deviation in group I = 366.4

Standard deviation in group II=366.4

Mean difference = 267 (to detect at least 30% increase in mean decrease in the area of ulcer in study group)

Effect size = 1.413

Alpha error (%) = 5

Power (%) =80

Sided = 2

Required sample size per group = 30

Method of Data Collection:

Data was collected using structured questionnaire. A complete detailed history, examination findings were recorded. All patients underwent clinical examination with relevant investigations after obtaining an informed consent about the need for study & the methodology & which group they are placed under. Persons with diabetes were categorised into 2 groups using even odd method to include similar type of cases in both groups, where, in group A-low light laser and in group B-conventional dressing is used.

DATA COLLECTION:

1. Measurement of size of ulcers in each group before the start of the treatment
2. Grading of the ulcers was done by Meggitt-Wagner

WAGNER' S GRADING OF ULCER

- Grade 1 -Partial skin thickness
- Grade 2 - Full skin thickness
- Grade 3 -Underlying tissues involved (fascia , ligaments, tendons)
- Grade 4 - Grade 3 +abscess or osteomyelitis
- Grade 5 - Grade 4 + necrotic tissues
- Grade 6 - Gangrenous tissues found

Laboratory Investigations Needed in The Study:

1. **HEMATOLOGICAL**:

Complete blood count with grouping and typing-
Blood Urea - Serum Creatinine -
Serum Sodium- Serum Potassium-
FBS - PPBS- HbA1c-

2. URINE EXAMINATION:

Albumin - Sugar - Ketones -

3. DISCHARGE/ BIOPSY FROM THE ULCER FOR CULTURE AND SENSITIVITY

4. X- RAY OF FOOT (IF REQUIRED)

5. DOPPLER STUDIES (IF REQUIRED)

STATISTICAL ANALYSIS: [31, 32, 33]

Data was entered into Microsoft excel data sheet and was analyzed using SPSS 22 version software. Categorical data was represented in Frequencies and proportions. **Chi-square test** was used as test of significance for qualitative data. Continuous data was represented as mean and standard deviation. **Independent t test** was used as test of priority to similarity the mean difference between two quantitative variables.

Graphical representation of data: MS Excel and MS word was used to obtain various types of graphs such as bar diagram and Pie diagram.

p value(Probability that the result is true) of <0.05 was considered as statistically significant after assuming all the rules of statistical tests.

Statistical software: MS Excel**,** SPSS version 22 **(**IBM SPSS Statistics, Somers NY, USA) was used to analyze data.

RESULTS

Table 2: Mean Age Comparison between two groups

	Group				p value
	Study Group		Control Group		
	Mean	SD	Mean	SD	
Age	50.5	15.19	54.9	10.33	0.195

Mean Age in group of study was 50.5 ± 15.19 and in Control group was 54.9 ± 10.33. There was no difference with significant importance in mean Age comparison between two groups.

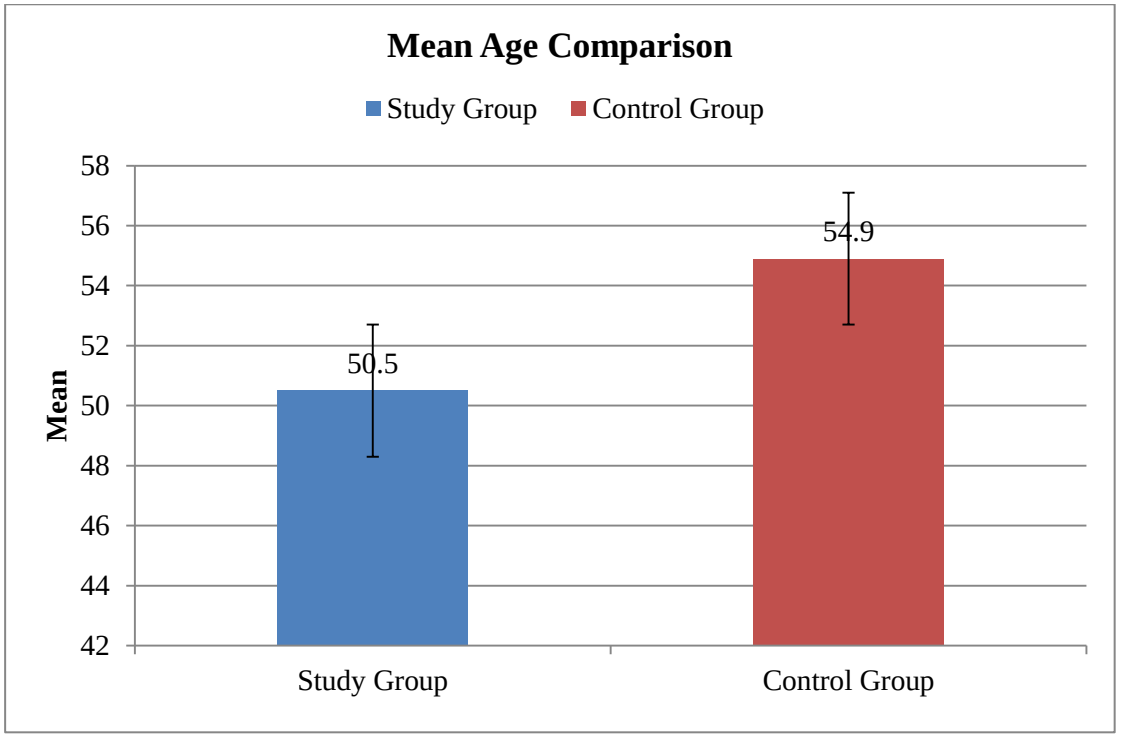


Fig. 17: Bar Diagram Showing Mean Age Comparison between two groups

Table 3: Sex Distribution between two groups

		Group			
		Study Group		Control Group	
		Count	%	Count	%
Sex	Female	9	30.00%	4	13.33%
	Male	21	70.00%	26	86.67%

$\chi 2 = 2.455$, df = 1, p = 0.117

In Study Group, 30.00% were Female and 70.00% and Male. In Control Group, 13.33% were Female and 86.67% and Male. There was no difference with significant importance in Sex Distribution between two groups.

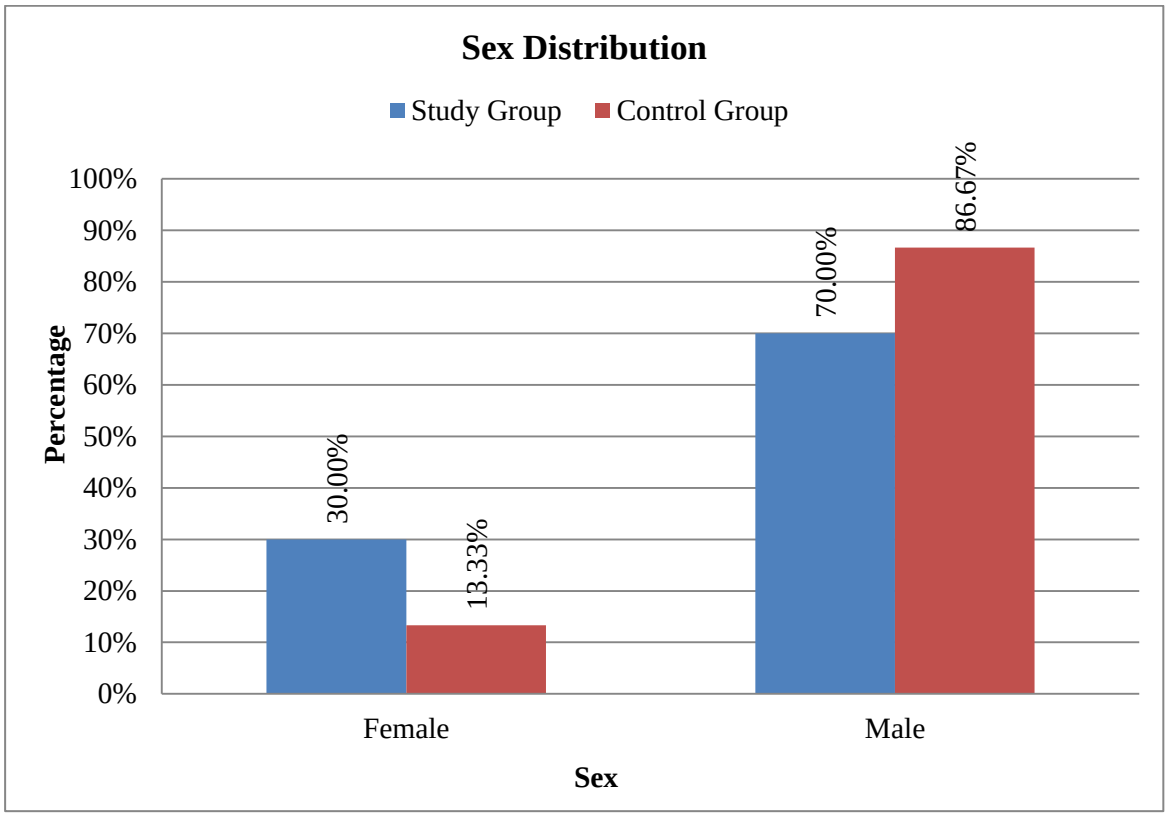


Fig. 18: Bar Diagram Showing Sex Distribution between two groups

Table 4: Mode of Onset Distribution between two groups

		Group			
		Study Group		Control Group	
		Count	%	Count	%
Mode of Onset	Spontaneous	15	50.00%	21	70.00%
	Traumatic	15	50.00%	9	30.00%

$\chi 2$ = 1, df = 2, p = 0.114

In Study Group, 50.00% had Spontaneous and 50.00% and Traumatic. In Control Group, 70.00% had Spontaneous and 30.00% and Traumatic. There was no difference with significant importance in Mode of Onset Distribution between two groups.

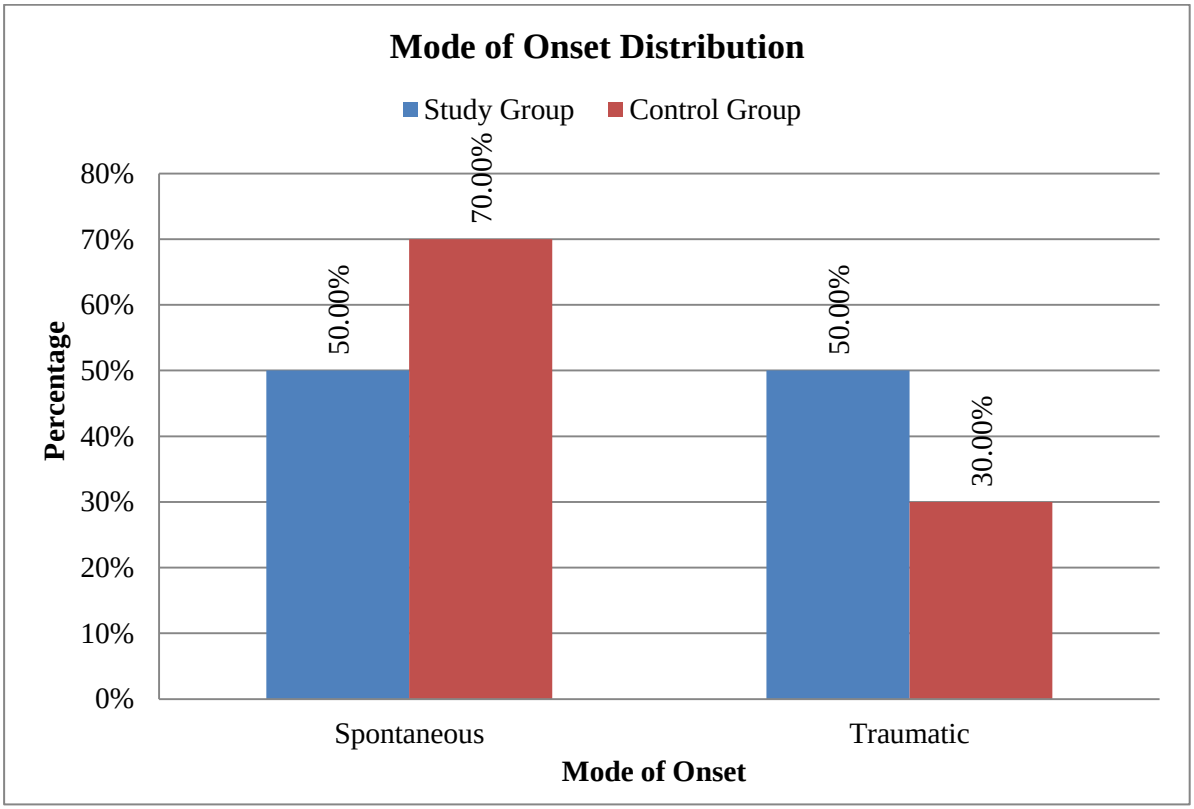


Fig.19: Bar Diagram Showing Mode of Onset Distribution between two groups

Table 5: Mean HbA1c Comparison between two groups

	Group				p value
	Study Group		Control Group		
	Mean	SD	Mean	SD	
HBA1C	6.93	2.52	6.7	2.29	0.709

Mean HBA1C in Group LF was 6.93 ± 2.52 and in Group LB was 6.7 ± 2.29. There was no difference of significant importance in mean HBA1C comparison between two groups.

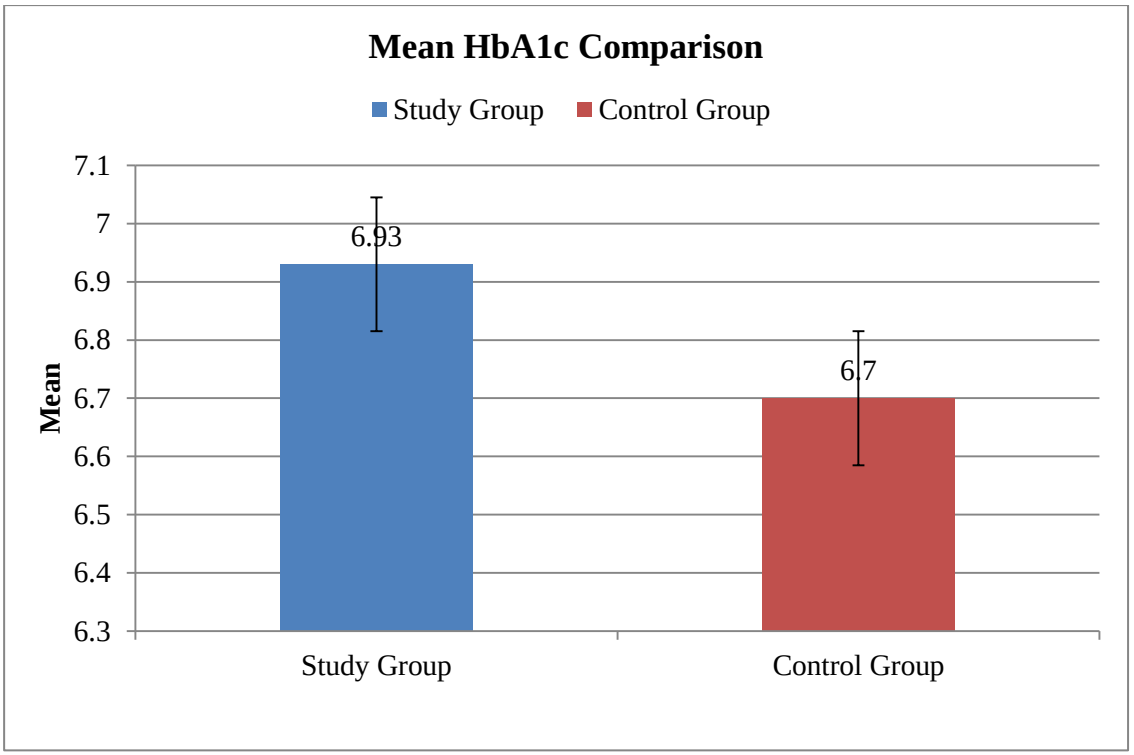


Fig.20: Bar Diagram Showing Mean HBA1C Comparison between two groups

Table 6: Type of Anti Diabetic Agent Distribution between two groups

		Group			
		Study Group		Control Group	
		Count	%	Count	%
Type of Anti-Diabetic Agent	Insulin	14	46.67%	20	66.67%
	OHA	10	33.33%	10	33.33%
	No	6	20.00%	0	0.00%

$\chi 2 = 7.059$, df = 2, p = 0.029*

In Study Group, 46.67% had Insulin, 33.33% had OHA and 20.00% had none. In Control Group, 66.67% had Insulin and 33.33% had OHA. There was a difference which make significance importance in Type of Anti Diabetic Agent Distribution between two groups.

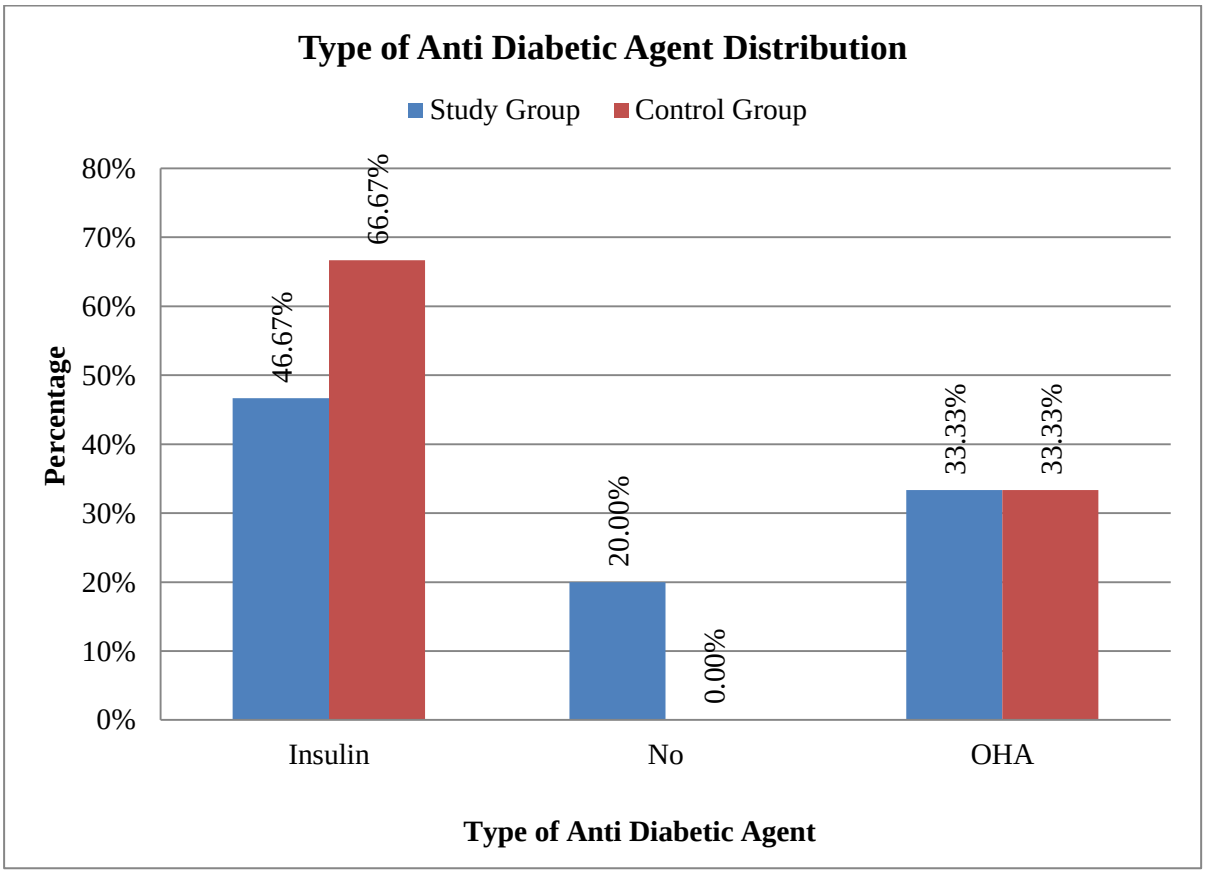


Fig.21: Bar Diagram Showing Type of Anti Diabetic Agent Distribution between two groups

Table 7: Mean Area of wound Comparison between two groups

Area (sqcms)	Group						p value b/w 2 groups
	Study Group			Control Group			
	Mean	SD	P value with in the group	Mean	SD	P value with in the group	
Initial Area	51.07	34.44		46.57	24.6		0.563
Day1	49.6	34.39	<0.001*	45.5	24.38	<0.001*	0.596
Day 4	47.33	32.06	0.002*	43.87	24.15	0.001*	0.638
Day 7	45.43	29.88	0.008*	42.53	23.81	<0.001*	0.679
Day 10	42.43	29.9	0.157	41.43	23.26	<0.001*	0.886
Day 13	32.86	20.65	0.096	40.33	22.9	<0.001*	0.194
Day 16	41.93	28.99	0.299	39.7	22.35	<0.001*	0.739
Day 20	40.17	29.1	0.001*	39.03	21.98	<0.001*	0.865

Mean Initial area (sq cms) of wound in group of study was 51.07 ± 34.44 & in Control group was 46.57 ± 24.6. There was no difference in mean Initial area (sq cms) of wound btw two groups. Similarly, there was no difference in area of wound between two groups from 1st day to Day 20. In group of study , there was significant difference in mean Area of wound compared to the previous value at Day 1, Day 4, Day 7 and Day 20. In Control group, there was significant difference in mean area of wound compared to previous values from 1st day to Day 20.

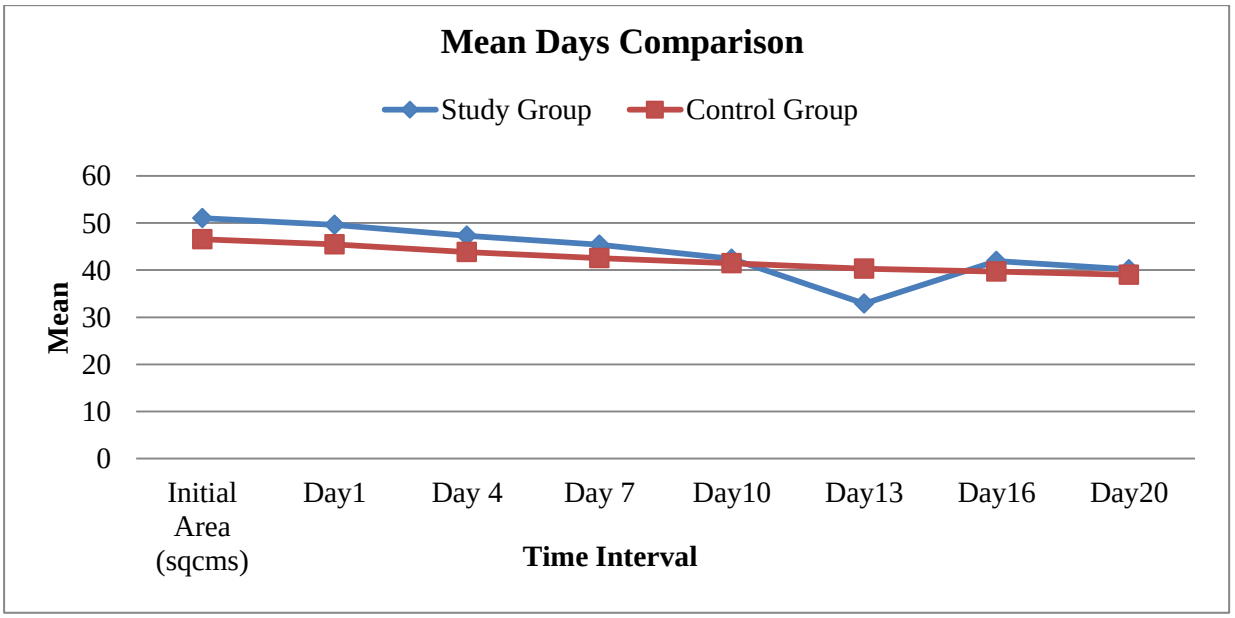


Fig.22: Line Diagram Showing Initial area (sqcms) of wound Comparison between two groups

Table 8: % Decrease in area of wound Comparison between two groups

	Group				p value
	Study Group		Control Group		
	Mean	SD	Mean	SD	
% Decrease in Area of wound	22.19	7.21	15.28	6.54	< 0.001*

Mean Area of wound % Decreased in group of study was 22.19 ± 7.21 and in Control group was 15.28 ± 6.54. There was significant difference in mean Area of wound % Decreased comparison between two groups.

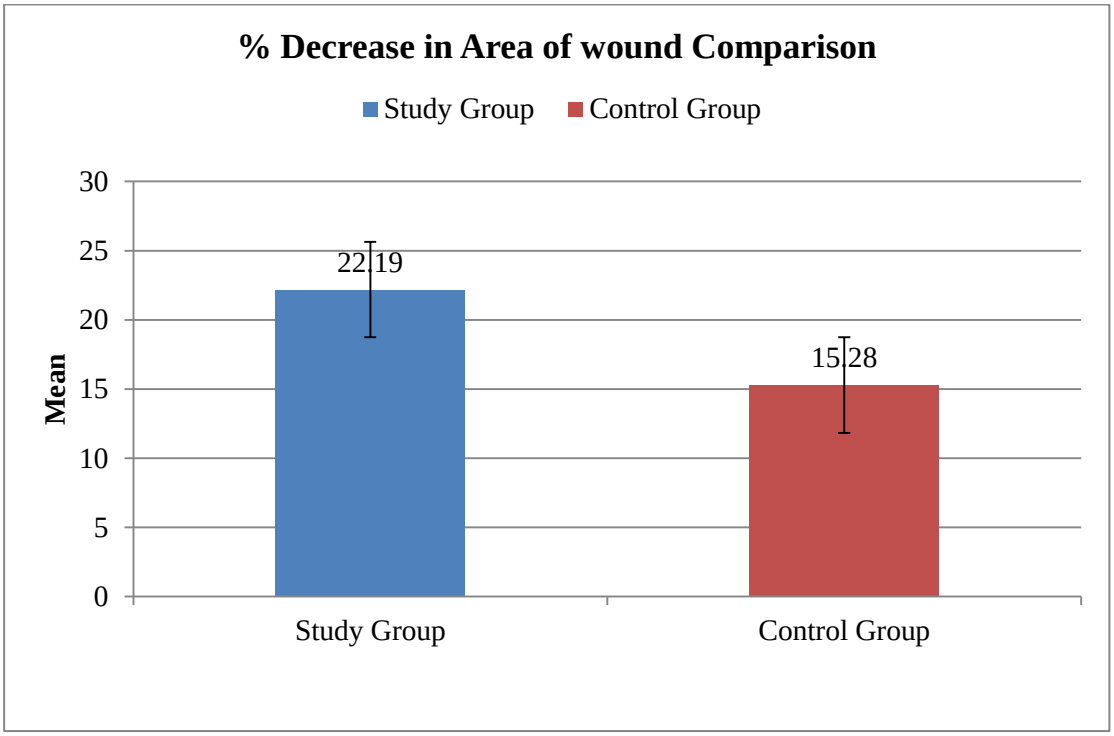
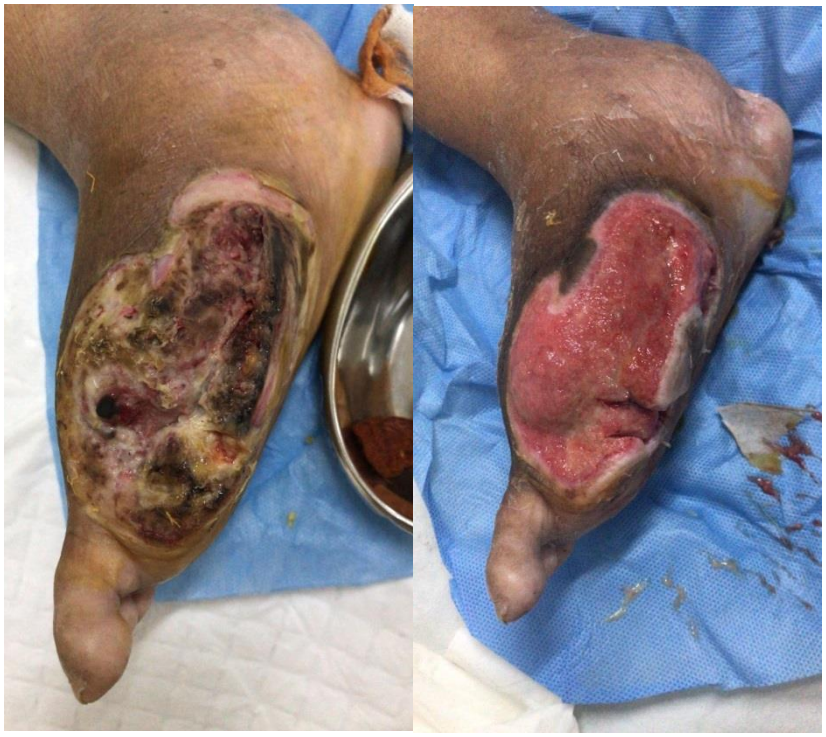


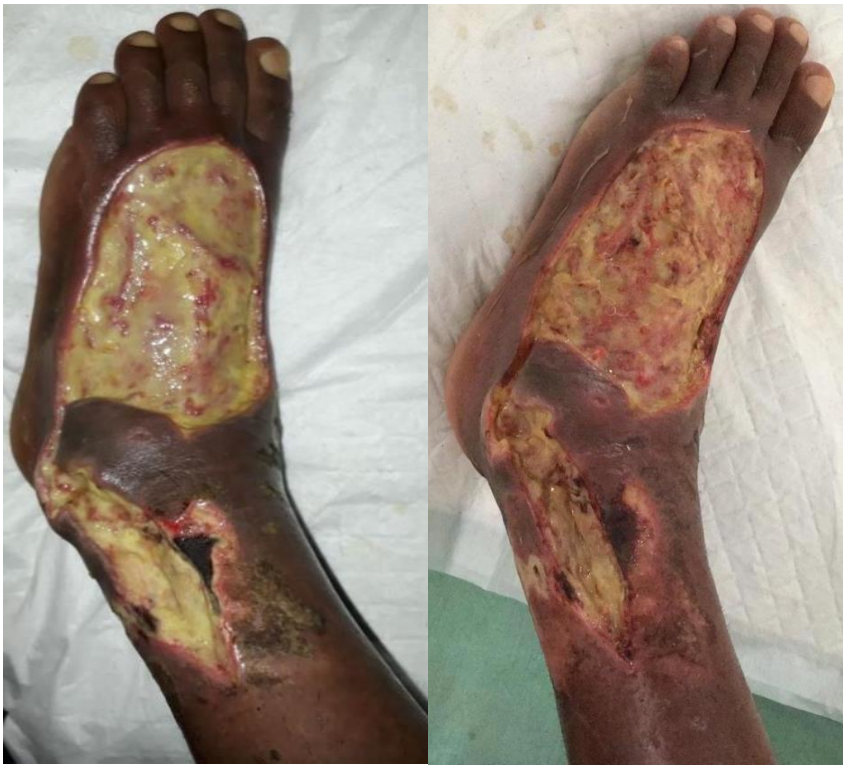
Fig.23: Bar Diagram Showing % Decrease in Area of wound Comparison between two groups

PHOTO GALLERY

STUDY GROUP



CONTROL GROUP



DISCUSSION

A Comparative Prospective Observational study which was performed in Department of surgery at a tertiary care centre among persons admitted with DF ulcers of chronic duration with Grade I-III (Megitt-Wagner Grade). 60 persons with DF included in this study were segregated in to 2 groups. Study group received Low Light Laser Therapy and Control group received Conventional Dressing. Clearance from institutional ethical committee was taken prior to the start of the study. Consent was informed and taken from all the persons with DF ulcers recruited prior to the start of the study.

Neuropathy, ischemia, and sepsis are the most common causes of diabetic foot problems. In diabetes, infection spreads quickly due to a sluggish healing process and poor vascularity. Controlling hyperglycemia is a crucial element in wound healing. In addition to wound debridement, antibiotics, and regular dressings are required. LLLT, hyperbaric oxygen therapy, vacuum aided closure, and growth factor administration are just a few of the various supplementary therapies that have been tested.

Low-level laser treatment (LLLT) is a type of phototherapy that uses monochromatic and coherent low-power light to treat injuries and lesions. Low laser therapy given to soft tissues in vitro and in vivo has been reported to stimulate certain healing pathways in wound healing. Most of the studies published in the Low light laser therapy researches have explained the effects of Low light laser therapy on fibroblast growth, locomotion & production of collagen as early as 24 hrs post treatment with laser and accelerates wound healing. LLLT has proved promising in many studies, and its cost effectiveness is a boon in a developing nation like India, which could soon change the way DF ulcers are being treated[4,5].

Profile of Subjects:

Mean Age in group used for study was 50.5 ± 15.19 and in Control group was 54.9 ± 10.33. In Study Group, 30.00% were Female and 70.00% and Male. In Control Group, 13.33% were Female and 86.67% and Male. There was no difference noted in Age & Gender btw two groups hence matching was achieved.

Kajagar BM et al.,[4] in their study observed that Mean age of subjects in group used for study was 54.35±6.84 and in Control group was 50.94±8.11 years. Male: Female ratio in group used for study was 2:1 & in Control group was 2:1. There was no significant difference in Age and gender distribution. The findings were similar to the present study.

Priyadarshini LMJ et al.,[34] in the study observed that the mean Age was 52.1±8.940 in cases and 52.48±11.57 years in controls. The proportion of male in cases was 26 (52%) and female was 24 (48%) whereas the proportion of male in controls was 31 (62%) and female was 19 (38%). There was no significant difference in age and gender distribution between two groups. The findings were similar to the present study.

Mode of Onset:

In Study Group, 50.00% had Spontaneous and 50.00% and Traumatic onset. In Control Group, 70.00% had Spontaneous and 30.00% and Traumatic onset.

Kajagar BM et al.,[4] observed that in both Study group mode of onset was Insidious in 32% and Traumatic in 68%.

Diabetic Profile of subjects:

Mean HBA1C in Group LF was 6.93 ± 2.52 and in Group LB was 6.7 ± 2.29. In Study Group, 46.67% had Insulin, 33.33% had OHA and 20.00% had none. In Control Group, 66.67% had Insulin and 33.33% had OHA.

Wound area:

Mean Initial area (sq cms) of wound in Study group was 51.07 ± 34.44 and in Control group was 46.57 ± 24.6. There was no significant difference in mean Initial area (sq cms) of wound between two groups. Similarly, there was no significant difference in area of wound between two groups from Day 1 to Day 20. In Study group, there was significant difference in mean Area of wound compared to the previous value at Day 1, Day 4, Day 7 and Day 20. In Control group, there was significant difference in mean area of wound compared to previous values from 1st day to Day 20.

Mean Area of wound % Decreased in Study group was 22.19 ± 7.21 and in Control group was 15.28 ± 6.54. There was no difference noted in mean Area of wound % Decreased comparison between two groups.

Kajagar BM et al.,[4] in-study group observed that Initial ulcer area (mm2) was 2608.03±683.14 and in Control group was 2747.17±603.79 mm^2. There was no difference noted in Initial ulcer size b/w 2 groups. Final ulcer size in Study group was 1564.79±437.30 and in control group was 2424.75±551.26. There was no difference noted in final ulcer size between two groups. Mean decrease in area of ulcer (mm^2) in group of study was 1043.20±266.62 and in Control group was 322.44±85.84. There was significant difference in Mean decrease in area of Ulcer b/w two groups. The findings were similar to the present study.

Priyadarshini LMJ et al., [34] mean ulcer area on day-1 was 13.74±11.88 in cases and 19.09±15.03 in controls. There was no difference noted in initial ulcer area between two groups. The mean day-15 area was 3.97±5.41 in cases and 18.80±17.70 in controls. The mean difference across the group is (-14.83). It is statistically significant (p value <0.001). The mean change in area was 9.77±7.83 in cases and 0.28±11.37 in controls. The mean difference across the group is

(9.48). It is statistically significant (p value <0.001). The findings were similar to the present study.

A study by **Hopkins et al.,**[35] has reported results in subjects numbered 22 and shown 55% greater wound size reduction in group used for study as compared to group used as control . **Gupta et al.,** [36] have demonstrated a significantly greater reduction (p<0.002) in the surface area of leg ulcers treated with red light and infrared light than in sham-irradiated controls. The leg ulcers treated with LLLT showed an average decrease in surface area of 193.0 mm2, whereas in controls it was only 14.7 mm^2.

Kamalakannan M et al., [37] in their study 40 participants, received LLIT for 4weeks, 6 days/week. Outcome measures were taken at the baseline and after 4weeks of the treatment protocol. This research work states that LLLT (40.65 SD 13.5) showed significant improvement in wound contraction after 4 weeks. Final result that LLLT is more effective in reducing the time of healing and improving the QOL.

CONCLUSION

From the study it can be concluded that highest percentage of decrease in wound area was observed Laser Group compared to Conventional Group. Also, with in the Laser group percentage decrease was high compared to Control group. Hence from the study it is clearly evident that Low Laser Therapy for Diabetic wounds was better compared to Conventional Therapy.

SUMMARY

A Comparative Prospective Observational study was performed in Department of surgery at a tertiary care center among persons who got admitted with long standing DF ulcers with Grade I-III (Megitt-Wagner Grade). 60 persons with diabetes were segregated in to 2 groups. Study group received Low Light Laser Therapy and Control group received Conventional Dressing.

The following observations were made in the study

1. Mean Age in Study group was 50.5 ± 15.19 and in Control group was 54.9 ± 10.33.
2. In Both groups' majority were males.
3. In Study Group, 50.00% had Spontaneous and 50.00% and Traumatic onset. In Control Group, 70.00% had Spontaneous and 30.00% and Traumatic onset.
4. Mean HBA1C in Group LF was 6.93 ± 2.52 and in Group LB was 6.7 ± 2.29.
5. In Study Group, 46.67% had Insulin, 33.33% had OHA and 20.00% had none. In Control Group, 66.67% had Insulin and 33.33% had OHA.
6. Mean Initial area (sq cms) of wound in Study group was 51.07 ± 34.44 and in Control group was 46.57 ± 24.6. There was no significant difference in mean Initial area (sq cms) of wound btw two groups. Similarly, there was no significant difference in area of wound between two groups from 1st day to Day 20.
    a. In Study group, significant difference was noted in mean Area of wound compared to the previous value at Day 1 , Day 4, Day 7 and Day 20.
    b. In Control group, there was significant difference in mean area of wound compared to previous values from Day 1 to Day 20.
7. Mean Area of wound % Decreased in Study group was 22.19 ± 7.21 and in Control group was 15.28 ± 6.54. There was significant difference in mean Area of wound % Decreased comparison between two groups.

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SRI DEVARAJ URS MEDICAL COLLEGE, TAMAKA,

KOLAR-563101

PROFORMA

Particulars of the patients:

Name:

Age:

Gender:

Occupation:

UHID NO:

Date of admission:

Date of discharge:

Religion:

Socio Economic status:

Socio – Economic Class	**No of patients**	**Percentage**
Poor socio economic status		
Lower middle class		
Upper middle class		

HISTORY:

Chief complaints:

History of presenting illness: Cause of ulcer: Traumatic

Spontaneous

Duration of ulcer:

Past History: Duration of diabetes

Type of diabetic medication

Family History:

Personal History

LOCAL EXAMINATION

FOOT EXAMINATION:

Skin dryness:

Heel fissures/Cracks:

Scratch marks:

Signs of infection:

Edema:

Erythema:

Induration:

Exudate:

WEEKLY ANALYSIS OF ULCER:

	1^{ST} WEEK	2^{ND} WEEK	3^{RD} WEEK	4^{TH} WEEK	5^{TH} WEEK	6^{TH} WEEK
POSITION						
SIZE						
EDGE						
FLOOR						
DEPTH						
GRANULATION TISSUE						
DISCHARGE FROM ULCER						
SLOUGH						
MARGINS						
SURROUNDING SKIN						
FBS AND HBA1C						

INVESTIGATION:

1.BLOOD:

HEAMOGLOBIN	
RBC	
PCV	
TOTAL COUNT	
PLATELET COUNT	
ESR	
SERUM CREATINE	
BLOOD UREA	
BLOOD GROUPING	
BT CT	
FBS	
PPBS, HBA1C	

2.URINE:

ALBUMIN	
KETONE	
SUGARS	
RBC	
EPITHELIAL CELLS	
PUS CELLS	

3.CULTURE AND SENSITIVITY OF DISCHARGE/BIOPSY FROM ULCER:

4. X RAY OF FOOT(IF REQUIRED):

5. DOPPLER STUDIES (IF REQUIRED)

SRI DEVARAJ URS MEDICAL COLLEGE, TAMAKA,

KOLAR-563101

PATIENT INFORMATION SHEET

TITTLE:"EVALUATION OF USEFULLNESS OF LASER THERAPY IN MANAGEMENT OF DIABETIC FOOT ULCER"

Name:

Address:

Age: Ward and UHID no:

Sex: Study number:

The purpose of the study is explained in detail to the patient and all information collected is for study purpose only. The data collected is submitted to the department of surgery, SDUMC, Kolar and confidentiality ensured. The merits and demerits explained briefly to the patient.

The low light laser therapy has positive impact on healing by reducing the bacterial count, activating fibroblasts. It is safe and effective and no need for dressing to be changed often.

The patient/patient attenders have been counselled about the aim and methods of research, expected duration of the subject participation and the benefits to be expected from the study. They have also been explained about the risks involved to the patient under study, provision of free treatment for research related injury, freedom of individual to participate and to withdraw from the study at any point of time, the necessity of investigations with cost and source of investigations.

This study is done for evaluation of usefulness of laser therapy in management of diabetic foot ulcers. Patients will be divided into two groups as per even odd method. Patient will be placed either in one of the groups and will be managed with respective material application. The size of the ulcer is measured on weekly basis till the ulcer is ready for grafting.

Person for contact for queries:

Dr. Murakonda Sowmya chowdary

Ph: 9493447634

Department of general surgery

SDUMC, KOLAR

SRI DEVARAJ URS MEDICAL COLLEGE, TAMAKA,

KOLAR-563101

PATIENT INFORMED CONSENT FORM

PATIENT UHID NO : .

TITLE:

"EVALUATION OF USEFULLNESS OF LASER THERAPY IN MANAGEMENT OF DIABETIC FOOT ULCER"

PRINCIPAL INVESTIGATOR: DR.MURAKONDA SOWMYA CHOWDARY

The contents of the information sheet that was provided have been read carefully by me/explained in detail to me, in a language that I comprehend, and I have fully understood the contents. I confirm that I have had the opportunity to ask questions.

The nature and purpose of the study and its potential risks/benefits and expected duration of the study, and other relevant details of the study have been explained to me in detail. I understand that my participation is voluntary and that I am free to withdraw at any time, without giving any reason, without my medical care or legal right being affected.

I understand that the information collected about me from my participation in this research and sections of any of my medical notes will be confidential & may be looked at by responsible individuals. I give permission for these individuals to have access to my records.

I agree to take part in the above study.

Subject name and signature/ thumb impression: Date:

Name and signature/ thumb impression of witness: Date:

Place:

Name and signature of person obtaining consent: Date:

Place:

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