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The Role of Surgical Drainage, Decompression, Debridement and Dressings in the Reduction of Disability and Death as a Result of Diabetic Foot

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ABSTRACT

Introduction: Obesity and diabetes are very common in the world. Diabetic foot is becoming increasingly common. The lifetime risk of developing a diabetic foot ulcer is between 19% and 34%. Recurrence is common after initial healing; approximately 40% of patients have a recurrence within 1 year after ulcer healing, almost 60% within 3 years, and 65% within 5 years.[1] It is a major burden on health resources.

The objective of this study was to analyze the causative factors contributing to the development of diabetic foot and to find the role of early surgical intervention in the form of drainage, decompression, debridement and aseptic dressings in the reduction of morbidity and mortality of this disease.

Method: 100 patients of diabetic foot were included in this study. Superficial wound infections and ulcers which were healed within 2 weeks were excluded from this study. All patients underwent some sort of surgical procedures like debridement, drainage of pus and decompression of closed compartments, toe amputation, BKA or AKA, TCC and RCW in minor OR. Repeated debridement of dead and gangrenous tissue was accomplished. Povidone iodine as an antiseptic and saline soaked or dry gauze dressing was opted according to the condition of the wound and close monitoring continued untill complete wound healing.

Result: Repeated debridement and dressings of the wound with povidone soaked gauze in infected wounds and saline soaked gauze dressing when healthy granulation tissue develops are very effective in rapid healing. Tight

dressings aggravate already existing foot ischemia and should be avoided. Antibiotics have no place until and unless local infected gangrenous tissue is dealt with surgically. Systemic complications of DM like CKD, hypertension, IHD are associated with increased morbidity and mortality. Mortality was 3 percent in this study.

INTRODUCTION

International working group on the diabetic foot (IWGDF) defined DF as a set of symptoms secondary to current or previous diabetes including skin chapping, ulceration, infection or destruction of foot tissue. Diabetic foot ulcer (DFU) is a complicated and multi factorial clinical problem that affects many patients with diabetes who experience neuropathy, peripheral arterial disease(PAD) that disrupt the foot epidermis and dermis causing breach in the skin envelope or protective barrier exposing the underlying tissue to infection. If surgical intervention is delayed, destruction of soft tissue extends proximally to put the limb or life in danger, as infection and edema will affect further the already compromised blood supply by thrombosis of local vessels.

Surgical debridement include drainage of pus, excision of dead and necrotic tissue, decompression by opening the tight closed compartments surgically and wound irrigation with saline to wash away the local inflammatory mediators. It blocks the local bacterial biofilm formation and initiates the process of wound healing by promoting granulation tissue. Simple loose dressings following repeated debridement are very effective for wound healing by protecting the wound from surrounding infection and controlling soilage and smell. Dressing should be changed when soaked.

The WIFi, Wagner, and University of Texas classification systems of diabetic foot are good predictors of major amputation with WIFi as the most predictive. These are powerful tools to grade diabetic foot. Treatment may be planned accordingly that could reduce the length of hospital stay and economic burden. Cruz PN et al [1] The WIFI classification is based on three clinical factors: Wound, Ischemia, and Foot Infection.

Wound

Grade1. Small shallow ulcer on the distal foot with no exposed bone, limited to the distal phalanx and no gangrene.

Grade2. Deeper ulcer with exposed bone, joint or tendon or gangrenous changes limited to the digits.

Grade3. Extensive, deep ulcer or with extensive gangrene involving forefoot, mid foot or full thickness heel ulcer ± calcaneal involvement or all together.

Ischemia

Graded using ABI, a grade of 0 was given if ≥.80, 1 if 0.6-0.79, 2 if 0.4-0.59, and 3 if ≤0.39.

Foot infection

Grade 1 if at least 2 of the following signs of infection are present: local swelling, induration or erythema >0.5 to 2cm around the ulcer, local tenderness or pain, local warmth, or purulent discharge.

Grade2 if there are involvement of deeper structures deeper than skin and subcutaneous tissues but no systemic inflammatory response signs available.

Grade 3 if there were signs of Systemic Inflammatory Response Syndrome manifested by two or more of the following: Temperature >38° or <36°C, Heart rate >90 beats/min, Respiratory rate >20 breaths/min or PaCO2 <32mm Hg, or White blood cell count >12,000 or <4000 cu/mm or Immature (band) forms 10%

Injuries and infections go unnoticed initially because of neuropathy. Repeated foot injuries, pressure sores, friction or scald burns and local ischemia trigger ulceration leading to diabetic foot. Hyperglycemia, poor water intake, hot weather, sweating, poor hygienic conditions and infections provoke the existing situation. All these problems are avoidable to some extent if proper guidelines and preventive measures are followed.

Approximately 20% of moderate or severe diabetic foot infections result in lower extremity amputations. After every 20 s a lower limb is amputated due to complications of diabetes. Of all the lower extremity amputations in persons with diabetes, 85% are preceded by a foot ulcer. The mortality at 5 years for an individual with a diabetic foot ulcer is 2.5 times as high as the risk for an individual with diabetes who does not have a foot ulcer [2]. The economic burden exacted on health care systems is considerable and includes direct and indirect costs, with loss of personal earnings and burden to care givers. The diabetic foot is a significant contributor to the global burden of disability and reduces the quality of life. It remains a considerable public health problem [3].More than half patients develop recurrent ulcers within 5 years. Katherine MC Dermott et al [4]. Glycemic control is an established method of primary prevention of microvascular complications and has shown to reduce amputation rates when combined with other cardiovascular disease prevention strategies [5].

Anatomy of the Foot

The foot supports the body weight and provides leverage for walking and running. It is uniquely constructed in the form of arches, which enable it to adapt its shape to uneven surfaces. It also serves as a resilient spring to absorb shocks, such as in jumping.

Skin of the sole of the foot is thick and hairless, which is firmly bound down to the underlying deep fascia by a number of fibrous bands. Numerous sweat glands are present.

The sensory nerve supply to the sole of the foot is derived from the posterior tibial nerve branches mainly. Medial calcaneal branch of the posterior tibial nerve, which innervates the medial side of heel; branches from the medial plantar nerve innervate the medial two-thirds of the sole and branches from the lateral plantar nerve innervate the lateral third of the sole.

Plantar aponeurosis is a triangular sheath formed as a thickening of the deep fascia and occupies the central area of the sole. The deep fascia covering the abductors of the big and little toe is thinner and weak.

The apex of the plantar aponeurosis is attached to the medial and lateral tubercles of the calcaneum. The base of the aponeurosis divides at the bases of the toes into five slips. Each slip divides into two bands, one passing superficially to the skin and the other passing deeply to the root of the toe and attaches to the fibrous flexor sheath and deep transverse ligaments. From the medial and lateral border of the aponeurosis fibrous septa pass superiorly in to the sole and take part in the formation of fascial spaces of the sole. The function of the planter aponeurosis is to give firm attachment to the overlying skin, to protect the underlying vessels, nerves, tendons and the synovial sheaths and to assist in maintaining arches of the foot. Muscles of the sole of the foot are described in four layers from inferior layer superiorly.

First layer

Abductor hallucis

Flexor digitorum brevis

Abductor digiti minimi

Second layer

Quadratus plantae

Lumbricals

Flexor digitorum longus tendon.

Flexor hallucis longus tendon

Third Layer

Flexor hallucis brevis

Adductor halluccis

Flexor digiti minimi brevis

Fourth Layer

Interossei

Peroneus longus tendon

Tibial is posterior tendon.

Posterior tibial artery gives its calcaneal branch and then divides in to medial and lateral planter arteries which pass deep to the plantar aponeurosis over the flexor tendons in the neurovascular plane. Medial plantar artery is the smaller terminal branch of the posterior tibial artery, it divides into a superficial branch that supplies skin on the medial side of the sole and deep branch that supplies muscles of great toe. Lateral planter artery, the larger terminal

branch broadly supplies the sole of the foot. Its deep branch completes the planter arch by anastomosing with the deep planter artery, a branch of dorsalis pedis artery. The deep planter arch is located in the distal part of the middle third of the foot. The deep planter artery is predominant in 72% and the lateral planter in 22 percent, or equal contribution in 6 %. C Gabrielli et al. Anat 2001 [6]

The inferior surface of each toe from the head of the metatarsal bone to the base of the distal phalanx is provided with a strong fibrous sheath which is attached to the side of the phalanges. The distal end of the sheath is closed and is attached to the base of the distal phalanx. The sheath together with the inferior surfaces of the phalanges and the interphalangeal joints forms a blind tunnel in which lies the flexor tendons of toes.

The skin of the dorsum of the foot is thin hairy and freely mobile on the underlying tendons and bones. Sensory supply is derived from the superficial peroneal nerve assisted by deep peroneal, saphenous and sural nerves. The superficial peroneal nerve emerges from between the peronious bravis and extensor digitorum longus muscle in the lower part of the leg, It divides into medial and lateral cutaneous branches that supply the skin on the dorsum of the foot, the medial side of big toe and adjacent sides of the second, third, fourth and fifth toes. The deep peroneal nerve supplies the skin of the adjacent sides of the big and second toes.

Saphenous nerve passes on the dorsum of the foot infront of the medial malleolus. It supplies the skin along the medial side of the foot as forward as the head of the first metatarsal bone. Sural nerve enters the foot behind the lateral malleolus and supplies the skin along the lateral margin of the foot and the lateral side of the little toe. The nail beds and the skin covering the dorsal surface of the terminal phalanges are supplied by the medial and lateral planter nerve. (Richard S Snells clinical Anatomy).

The dorsal venous arch lies in the subcutaneous tissue over the heads of the metatarsal bones and drains on the medial side into the great saphenous vein, which leaves the dorsum of foot in front of the medial malleolus and on the lateral side in to the small saphenous vein.

Pathophysiology

Diabetic foot is one of the disastrous complications of DM. This devastating complication is initiated by the sensory, motor, autonomic neuropathy and ischemia due to micro and macro angiopathy, emboli, arterial spasm in cold season, dehydration and hypotension. Lower limb edema also decreases capillary flow causing superficial ischemic ulceration. Major vascular occlusion leading foot ischemia needs vascular intervention by the vascular surgeon or by the interventional radiologist accordingly to improve the circulation which will help rapid wound healing. Conte MS et,al (Society for vascular surgery lower extremity guidelines writing group) [7].

According to the study by Wrobel et al entitled “Diabetic Foot Biomechanics and Gait Dysfunction” [8], diabetic patients exhibit a conservative gait where patients have a slower walking speed, wider base of gait, and prolonged double support time that promote repetitive trauma due to walking activities which exposes the foot to moderate or high pressure and shear forces. This coupled with neuropathy, soft tissue changes such as glycosylation of foot connective tissue, thinner fat pads, and stiffer joints put diabetic patients at risk of diabetic foot ulceration.

There are many risk factors for DFU, and its pathogenesis is very complex, it can be divided into three categories: peripheral neuropathy, peripheral arterial disease, and infection. Major risk factors mentioned in literature are as under.

(i) Neuropathy
(ii) Angiopathy.
(iii) Deranged renal function
(iv) Repeated Infections
(v) Aging
(vi) Uncontrolled body mass index
(vii) Impaired vision
(viii) Uncontrolled hyperglycemia and hypercholesterolemia.
(ix) Trauma
(x) Disturbances in joint movement due to uric acid deposition or callus formation

Combination of sensory and motor damage results in an unequal foot load and insecure gait with pain insensitivity and deformed joints leads ulcer or hyperkeratosis. Autonomic system dysfunction is responsible for anhydrosis, overheating of the skin through increased deeper blood perfusion resulting in core heating, fissured skin and break of dermal barrier causing microbial invasion. The minor damage will continue to accumulate and result in progressive damage and difficulty in wound healing.

Motor neuropathy causes atrophy of foot muscles secondary to denervation. Small muscles of the foot like extensor digitorum brevis, lumbrical and interosseous muscles are paralyzed gradually. The anatomy of foot arch changes and metatarsophanangeal joints are hyperextended, interphalengeal joints show flexion leading to foot deformities. It results in unequal foot load and insecure gait with pain insensitivity. Wrobel JS [8].

Soft tissue is crushed between the prominent head of metatarsal joint and ground leading soft tissue necrosis and ulcer formation which acts as a nidus of infection. A compromised immune system initiates the process of infection and ulceration. If timely surgical intervention is not taken destruction of the foot will progress very rapidly.

Infection

DFU acts as a portal of entry for infection. The bacteria most often associated with DFU include not only gram-positive bacteria such as *S. aureus* (MSSA—methicillin-susceptible *Staphylococcus aureus*, and MRSA—methicillin-resistant Staphylococcus aureus), Streptococcus β-hemolytic *and* Corynebacterium striatum but also gram-negative bacteria such as Pseudomonas aeruginosa, E. coli, Acinetobacter baumannii, *Proteus* spp., and Enterobacter spp. and some anaerobic bacteria that reside more deeply in the wounds, such as Bacteroides spp., *Prevotella* spp., Clostridium spp., and Peptostreptococcus spp.

Microorganisms gather in specific areas within DFU wounds, where they grow and multiply, wrapping themselves with extracellular polymers containing polysaccharides and lipids. The extracellular polymeric substances (EPS) secreted by the cells embedded in the ulcer aggregate with bacteria and fungi to form biofilms.

A biofilm is defined as a community of microorganisms attached to an inert or living surface by a self-produced polymeric matrix or an assemblage of polymicrobial cells associated with a surface and enclosed in a matrix of primarily polysaccherid material. These films give bacteria the ability to adhere to both biotic and abiotic surfaces. Because biofilms are resistant to antimicrobial agents and immune to chemical attacks, they delay wound healing and cause chronic inflammation and repeated infections if wound care and timely debridement is not accomplished. Osteomyelitis occurs in many diabetic patients with a foot wound and can be difficult to diagnose (optimally defined by bone culture and histology) and treatment require resection or surgical debridement or prolonged antibiotic therapy. Lipsky BA et al. [9]

RESULTS

Out of 1oo patients, all had one or more associated co morbidities like HTN, IHD or CKD, post CVA, morbid obesity, limited mobility, local trauma, pressure sores or anemia. Male to female ratio in patients was of 2:1. 80 percent pts included in the present study had foot deformities ranging from toes deformities as hyperextension or fixed flexion of interphalangeal joints, mal-positioning, adduction of big toe and loss of foot arches leading to flat broad foot or charcot foot. Results showed that IHD, hypertension, CKD, duration of diabetes, and ambulation status were significantly associated with major amputations.

Comorbid conditions

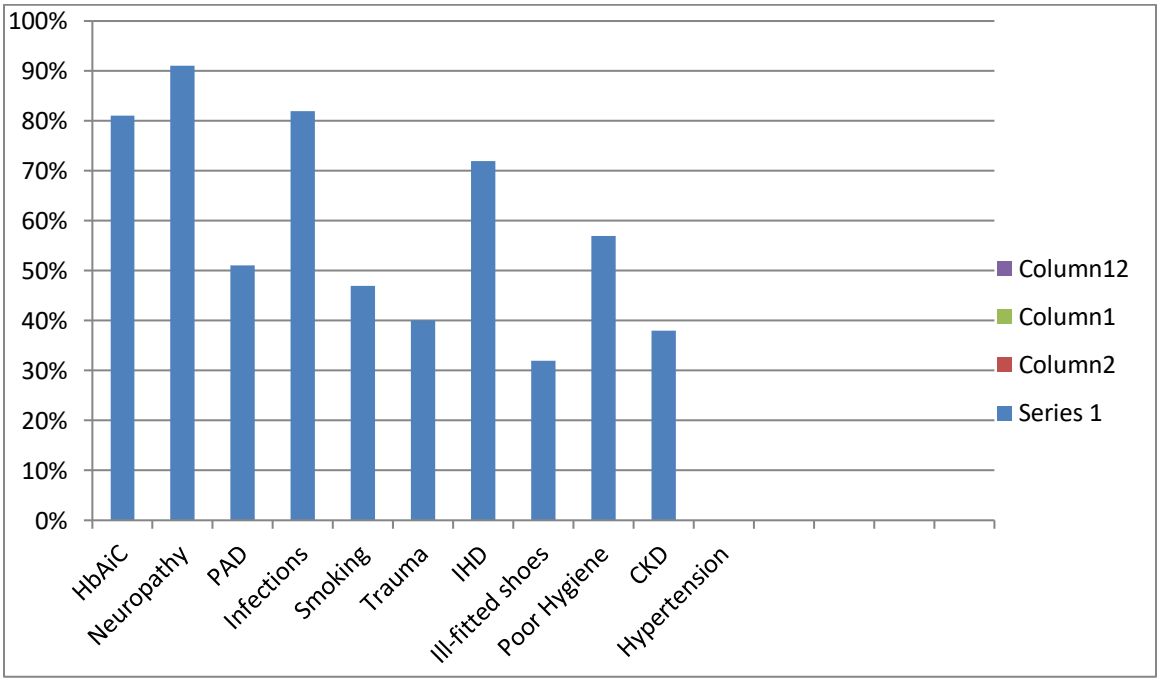

Figure 1. Types of amputations performed.

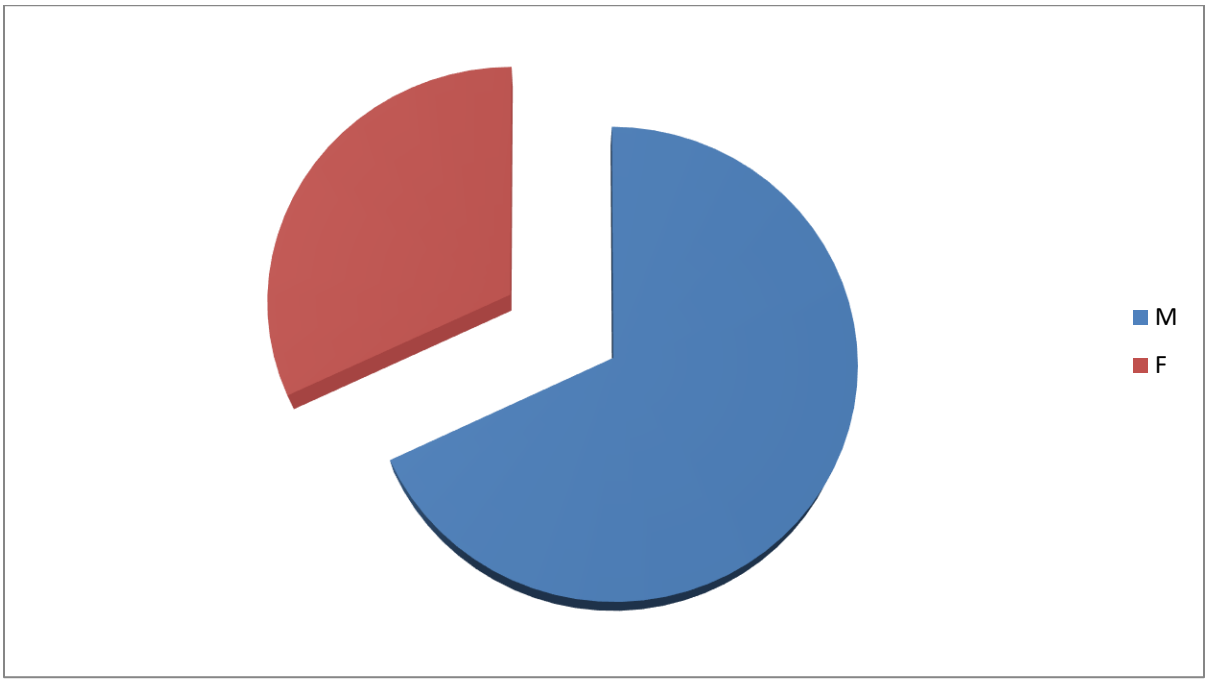

Figure 2

More than 90 percent of patients had polyneuropathy involving both feet. 50% percent had impaired foot circulation from macro or micro-angiopathy. 40% of patients were obese with BMI more than 30%. 39 % of patients had bilateral diabetic foot. Anemia with Hb lower than 10 g/dl was noted in 10 percent patients. The duration of diabetes mellitus showed a significant association with major amputation. Majority (63.49%) of participants in this study were diabetic for more than 10 years.

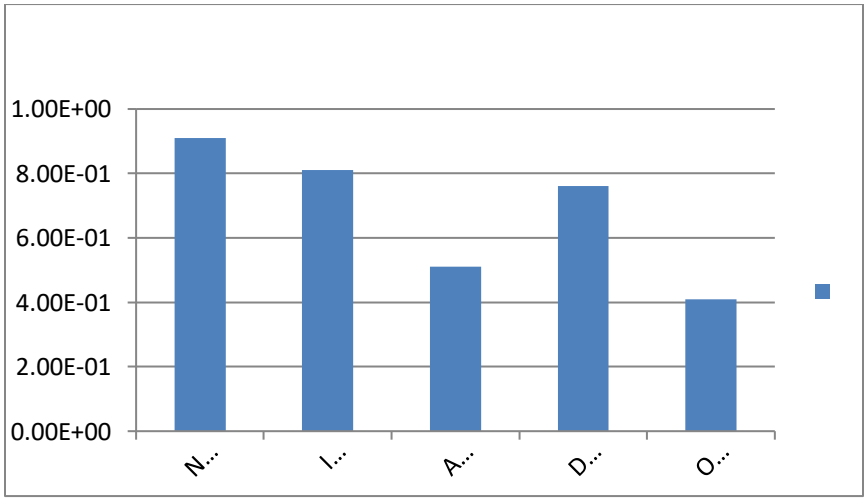

Figure 3

Neuropathic, ischemic and infected foot complications.

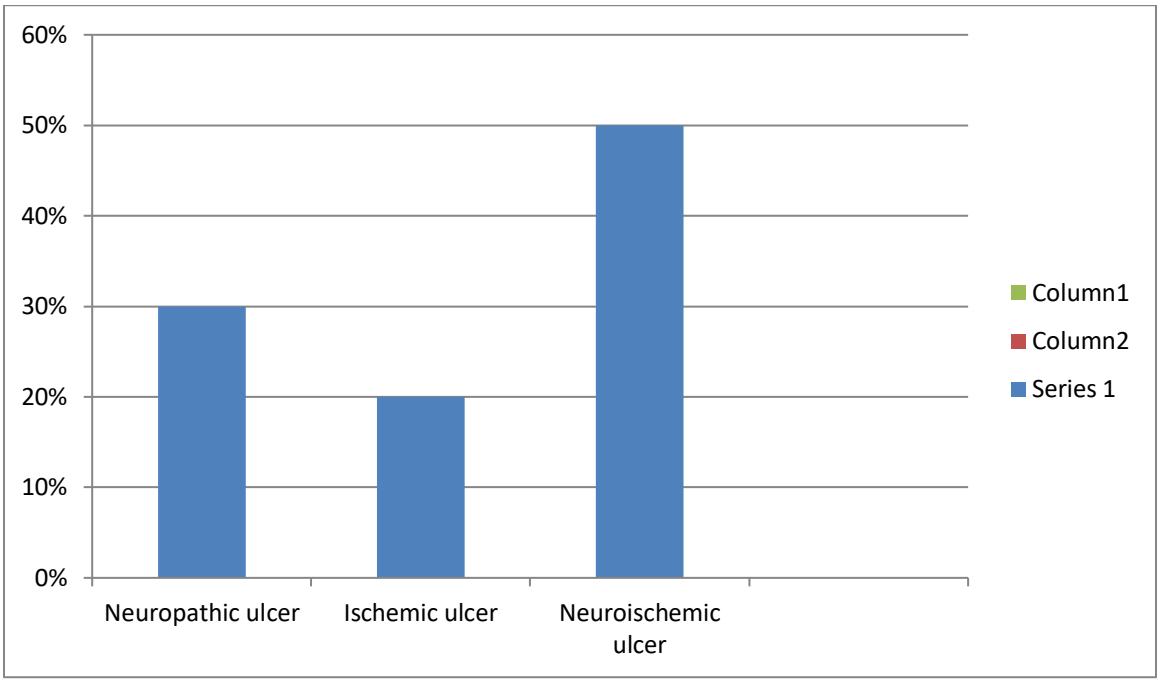

Figure 4

Location of diabetic foot ulcers.

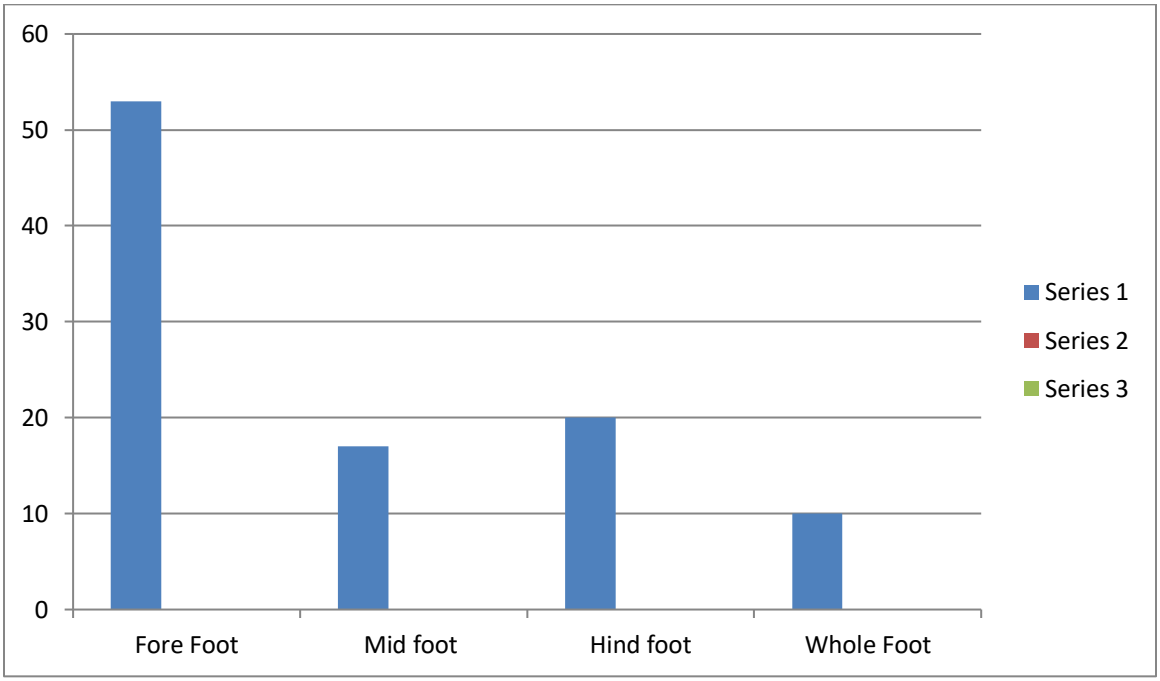

Figure 5

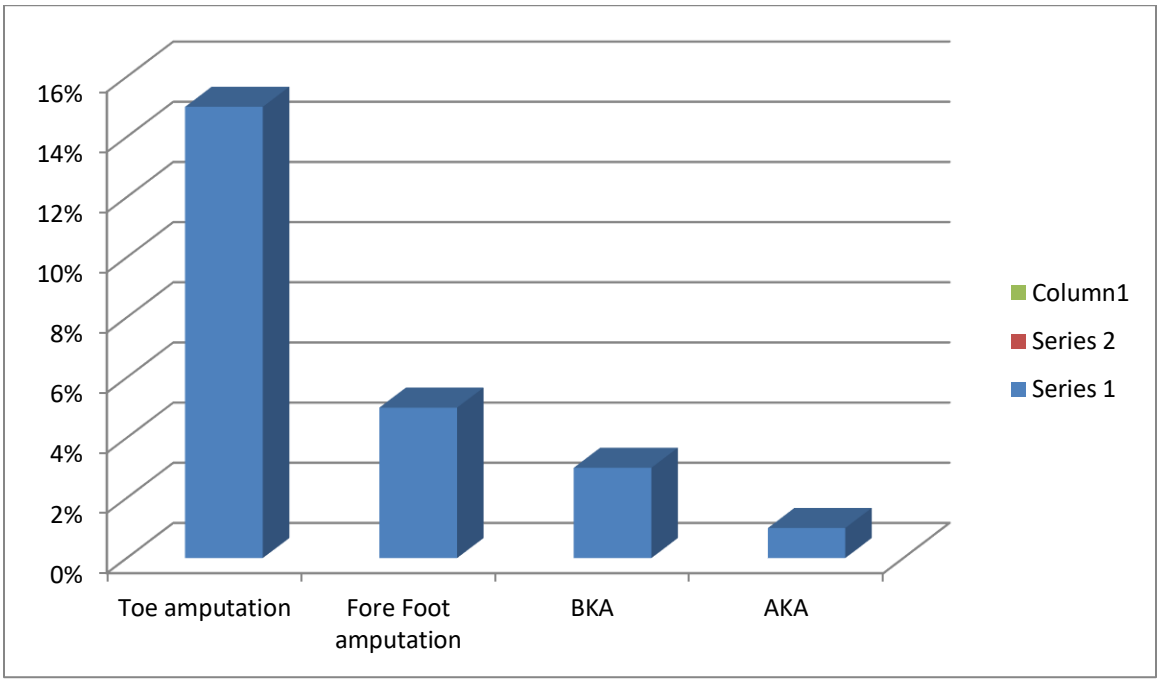

Figure 6

Patients were followed for 8 months. 7 patients returned with new wound infections or ulcers within 6 months follow up. 6 patients were referred to plastic surgery. Six patients did not return for follow-up after initial proper debridement. Three patients died, one due to delayed presentation secondary to gas gangrene, other due to Myocardial Infarction, one presented e sepsis and hypo glycaemia. 5 patients referred to vascular surgery after initial debridement and treatment of infection.

DISCUSSION

We noticed a sharp increase in the incidence of Diabetic foot ulcer in patients returning from Hajj or Umrah as many factors affected the pathophysiology of diabetic foot during the hajj. Long walk with bare feet or wearing ill-fitted and uncomfortable shoes or sit on wheel chair for long periods in the same position caused foot ulceration. Some patients developed friction burns, tissue ischemia and secondary bacterial infection over the sole after walking bare foot during hot season. Two third patients in this study were male and above 50. Age and male gender were associated with 1.6-fold increase in foot ulcer risk [10].

A smoking history of more than 20 years was one key factor that was shown to lead to a more proximal amputation in 40% of cases in one study. Results of a systematic review and meta-analysis by Liu et al, 2018, suggested that smoking increased the risk of diabetic foot amputation [11].

50% patients included in the study were hypertensive. Hypertension and CKD are also a strong etiological factor in Diabtic foot [12]. 9% patients presented with fracture of the head of metatarsal or other short bones of the foot specially in morbidly obese patients. Preventive measures and individual care could have decreased the incidence of these conditions and the morbidity and mortality of DM foot.

- Paralysis of the short muscles of the foot leading to foot deformities and unequal weight distribution and ulcer formation over pressure areas secondary to motor neuropathy
- Prolonged standing with hammer toe deformity accidental trauma and burns causing planter ulcer at the tip of distal phalanx of toes and under the metatarsal heads. Cross leg pressure sore or heel or malleolar ulcer in bed ridden patients.
- Sensory, motor, autonomic neuropathy.
- Micro and macro angiopathy.
- Poor hygienic condition and compromised immune system leading to fungal infection of the inter-digital clefts or paronychia.
- Extreme cold causing arterial spasm of already diseased vessel and impaired distal tissue perfusion leading to ischemia, ulceration or toe gangrene.
- Micro emboli from heart in patients with atrial fibrillation etc.
- Lower limb DVT.
- Self inflicted local burns around ankle for treatment of sciatica. Local custom of applying hot iron rod for the treatment of pain.

The single most important intervention in the management of diabetic foot was debridement. Debridement is the surgical removal of the nonviable, necrotic or gangrenous tissue from the wound bed and decompression by drainage of close compartment and off loading. Thorough wound irrigation with normal saline was very effective way of complete drainage of infected fluid. Counter dependent incision proximally instead of long skin incision to irrigate the cavity and self drainage of pus out of deep wound instead of spreading infection proximally in the deeper planes was very effective to treat foot infection.

Surgical debridement was used to remove the biofilm and convert the chronic inflammatory phase to acute phase and finally to healing phase. It promoted wound healing by accelerating granulation tissue formation and re-epithelialization. Debridement also played an important role in the control of infection as necrotic tissue provides breeding ground for bacterial proliferation and spread. Debridement was performed in the minor OR room. As diabetic patients had neuropathy, it was possible to do debridement without local anesthesia in most of the cases. This is very economical way of diabetic foot treatment. We used povidone iodine for dirty wounds and saline soaked gauze dressing over granulating wounds. Twice a day dressing was required in grossly infected and pouring wounds initially then once a day dressing. When healthy granulation tissue covered the wound alternate day dressing was enough. Patient family is educated and explained about the procedure of dressings at home to avoid unnecessary hospital visit daily after the granulation tissue covered the wound. Patients were then followed once a week till wounds healed completely. We preferred amputation of metatarsal heads or release of flexor tendon to prevent pressure over the ulcer on the plantar aspect of foot below the metatarsal head or the tip of distal phalanx which helped rapid healing of ulcers.

Early surgical intervention had dramatic effect in the control of infection, wound healing, and improvement of general health and wellbeing of the patient. Late presentation with deep seated infections and osteomyelitis of short

bones of the foot and spread of infection in the tendinous planes is difficult to treat and leads to increased morbidity. Involvement of the patient and family in the treatment plan has a very effective role in patient satisfaction and compliance. Nursing care is very important in bed-ridden patients to decrease aggravation of the bedsores or ulcers

Patients with extensive gangrene, deep extensive infected wounds, subcutaneous crepitus, systemic signs of infection or other multi-medical problems needed hospitalization for medical treatment and planned extensive debridement. Initially broad spectrum IV antibiotics were prescribed after taking culture swab and when the culture result were available antibiotics switched accordingly to C/S. Dead and fractured short bones acting as foreign body and cause of injures to the surrounding soft tissue of the foot were included in the debridement. Bone biopsy is the gold standard to diagnose osteomyelitis. MRI is considered as the best imaging modality for bone infection in diabetic patient. Younes NA et al. [13]

Debridement is recommended by the Scottish Intercollegiate Guidelines Network (SIGN) diabetic foot guidelines alongside antibiotic therapy for infection and pressure relief as a treatment for patients who have developed ulceration or gangrene [14]. We observed that discharge of pus and exudate from diabetic foot wound causes maceration of skin. Dressing, by acting as an absorbent not only controls maceration of surrounding skin but also acts as a barrier to surrounding microorganisms and minimizes the foul smell of the wound as well.

Effective method of debridement is by sharp surgical excision of dead infected gangrenous tissue, local drainage and decompression of tight closed compartment to avoid pressure over the vessels and nerves and to prevent extension of venous thrombosis or cellulitis proximally. Other methods of debridement are autolytic e.g. Hydrogel, Foam, alginates, Hydrocolloids semi-permeable polymeric membranes, silver containing, hydrotherapy and maggot debridement.

Medicinal maggots have the ability to remove non viable tissue selectively and may reduce the risk of secondary super infection as resistant strains of bacteria are also included in their meal. It promotes granulation tissue growth which may fasten the process of wound healing. Maggots eat out the dead tissue even from the deeper areas where surgical approach is not possible. "Larvae have the ability to eat bacteria including antibiotic resistant strains e.g. MRSA and dead necrotic tissue and liquefy infected dead tissue. Beasley Wd. et al." [15]. Maggots are capable of consuming all bacteria and believed to produce antimicrobial secretions. Raja JM [16].

One morbidly obese 55 years old diabetic neglected patient with Rt. diabetic foot developed maggots in his wound which proved to be a blessing for him. House or fruit flies deposited their eggs in the wound, causing wound myiasis. To our surprise, his wound was having only healthy granulation tissue and all dead necrotic tissue was consumed by the maggots even in the depth of the wound. About 150 maggots were removed and wound was irrigated with saline and pyodine soaked gauze dressing was done. Recovery was rapid and wound healed in about 3 weeks.

Povidone Iodine solution was used for aseptic measures. Povidone iodine is not only bactericidal for gram positive and gram negative bacteria but it also kills resistant strains like MRSA, including pathogens within biofilms, several viruses, fungi, spores, protozoa and amoebic cysts and yeasts. Bigliardi PL et,al. [17]

Dressings have very important role in the process of wound healing. Jiang P[18]. Ideal dressing should have the property of providing the environment to keep the wound moist and at the same time absorbing the excessive

exudates. Ideally it should be bacteriostatic and local debriding activity to promote granulation tissue growth and wound healing effect. Dressings should be biocompatible and should not cause allergic or immune reactions. We used povidone soaked gauze or saline soaked gauze for packing deep dirty wounds after debridement, with good results in the form of reduction in wound infection, decrease in inflammatory response and early healing of wounds. Dressings prevent secondary wound infection. Because there are different types of wounds and the characteristics of each phase of wound healing differ, there is no single dressing that meets all requirements for use with diabetic foot ulcer that can be effectively applied in all cases. Each dressing has its own characteristics. Appropriate application of dressings increases the rate of healing, thereby reducing healing time, and reducing the cost of treatment. Saline soaked gauze dressings are cost effective and are effective, economical and can be used in almost all diabetic foot wounds.

Wound type, patient requirements, and cost should be considered when selecting a dressing. Presently available dressings for diabetic foot ulcers can be divided into two categories: traditional dressings and current dressings. Dressing with the gauze, cotton pads and bandages are still most widely used in clinical practice due to their low cost and simple manufacturing used in the treatment of diabetic foot wounds to provide cushioning that reduces pressure, prevents abrasion, protects the wound, absorbs exudates and prevents direct contact between the wound and contaminants. Dry dressings tend to adhere to the wound, causing secondary damage, pealing-off the newly grown epithelium when the dressing is changed. Adherence can be avoided by use of saline soaked gauze dressing and soaking gauze with saline before removing it from the wound. There are many types of dressings with different mode of action but some are not cost effective. Some types of dressings are enlisted below.

- Basic Dressing.
- Absorbent Dressings
- Moisturizing dressings
- Bacteriostatic dressings. By adding honey, antibiotics, metals and metal oxides are the common pharmaceutical ingredients.
- Hydrogel dressing. These are insoluble hydrophilic polyurethane polymers, have moisturizing properties biocompatibility and similarity to living tissue, properties that allow hydrogel to produce the best wound healing effect.
- Anti-inflamatory Hydrogels
- Anti-oxidant Hydrogels
- Proangiogenic Hydrogels
- Silver Ion hydrogel antibacterial dressings used to eradicate wound infection without effecting healthy tissues(Biological Dressings) [30]

- Classification of dressings on the basis of mode of action:
- Chitosan Dressings, hemostatic, bio-adhesive, antibacterial, antifungal, anti-viral, anti inflammatory dressings promote cellular and tissue regeneration.

- Composite Dressings. Biomaterials come in many forms and structures including thin films, hydrogels, sponges, nanofibers and other type of structures. Yang L et.al [19].
- Bioactive dressings contains epidermal growth factor, vascular endothelial growth factor promotes regeneration of blood vessels
- Scaffold Dressings with stem cells.
- Acellular wound matrix dECM (Decellularized Extracellular Matrix)
- Cytokine Dressings
- Exosome Dressings can deliver lipids, proteins and nucleic acid to recipient cells when circulating in to extracellular space.
- Autologus, platelets rich plasma dressings.
- NPWT can improve the wound microenvironment, control wound infection, promotes endothelial cell regeneration. Wound edema and bleeding are the adverse effect. The negative pressure value should be appropriately maintained and adjusted to avoid bleeding tendency of the wound when applying this new modality. Lin Chin et al. NPWT can effectively accelerate wound healing. The mechanism is to keep the wound moist and inhibit bacterial growth. The number of bacteria in the wound particularly gram negative bacteria are significantly reduced. NPWT improves wound blood perfusion and promoting wound healing, promoting cell proliferation, angiogenesis and wound tissue repair, modulate cytokine expression.

- Some other novel modalities for diabetic foot wound management are:
- Hyperbaric Oxygen Therapy. HBOT is effective in the healing of foot ulcer and reduction of major amputation but there is no significant reduction in mortality with this treatment,
- Ultrasound Therapy
- Transcutaneous Electrical Nerve Stimulation
- Shockwave Therapy.
- Photobiomodulation.
- Nanomedicine
- Growth factors include platelet-derived growth factors (PDGFs), β, TGF-FGFs, and connective tissue growth factor (CTGF). These factors stimulate cell proliferation, angiogenesis, and tissue remodeling, promoting the formation of new blood vessels and the recruitment of immune cells to the wound site. Liu Y et al. [20]
- TENS, nanomedicine, shockwave therapy, HBOT also have an effective role in poorly healing DM foot wounds. Olajumoke Arinola Oyebode et al. [21]
- These modalities and dressings may well augment wound healing but are not a substitute for debridement. Infected necrotic and gangrenous tissue should be debrided before opting for them.
- Saline soaked gauze dressing is still the gold standard, economical and effective way in the treatment of diabetic foot ulcer. In dry wound, saline soaked gauze can be used which has bacteriostatic activity as well.

Infected tissue is debrided daily and wound is irrigated with saline till healthy granulation develops locally which not only acts as a barrier for the entry of infection but also provide base for the growth of epithelium over it.

Total contact cast or non-removable walker (TCC), Removable cast walker (RCW), bed rest, use of wheel chair, crutches and surgical decompression reduce local pressure over the affected area of the foot by redistribution of loads. RCW was better as it is removable and light. Wound inspection and dressing is easy but TCC is permanent and heavy and wound cleaning and dressing is difficult. POP cutter to cut TCC is required but the results in the form of ulcer healing and safety from repeated trauma and reduction in foot swelling and inflammatory process is better in both TCC and RCW.

Gas gangrene, a potentially lethal complication of diabetic foot should be considered if there is crepitus over wound by palpation or evident pockets of air extending proximally on X-Rays foot. Timely surgical treatment may safe life. Immunocompromization also contributes in the development of foot infection. X Rays foot, presence of elevated ESR and C reactive protein support the diagnosis of soft tissue and bone infection and can be used to monitor the response to antibiotic therapy.

Simple preventive measures like foot care education and regular inspection of foot by the patient, caregiver, nursing care, avoiding local foot pressure, injuries, wearing special medical shoes, weight reduction in obese patients and control of DM improve treatment outcome and wound healing. Control of DM enhances wound healing. Wu SC et al. [22]. Surgical interventions like debridement, dressings, physiotherapy, TCC or RCW for neuropathic ulcer and charcot foot have dramatic effect on wound healing. David Dayya et al [14]. Reduction in Hb A1C has a definitive role in wound healing, readmission and amputation reduction rates. Regular exercise increases claudication distance and slow the process of neuropathy as well. Insoles have good result to reduce shear or side to side stress on the planter surface which is important single factor in DFU healing. Combination of felted foam with other therapeutic foot wear is considered to have a role in ulcer healing. Neuropathy results in unequal distribution of weight and bio-mechanic is disturbed. Wrobel JS et al. [23]

Foot infection is difficult to treat especially when it extends proximally in the deep tendinous planes. Early diabetic foot infection CS results noted in this study were gram positive microorganisms only but those pts presented late or had previous H/O hospitalization had poly microbial infection including MRSA or gram negative infection. Same pattern of culture was seen in the previous studies e.g. By Dowd SE et al. [24] Poor circulation causes skin and soft tissue ischemia, necrosis and fibrosis. Moreover as immune system is compromised, repeated use of antibiotics before the patient consult surgeon; chronic infected wounds have resistant strains of microorganisms. These patients need repeated debridement and dressings. Some patients presented with systemic manifestations of sepsis and co-morbid condition. These pts needed medical evaluation and appropriate medical intervention to control DM, hydration keeping in view cardiovascular status, renal function etc. and antibiotics according to CS results.

Biofilm formation prevent the entry of antibiotics in the infected tissue, promotes the emergence of multidrug resistance strains and therapeutic rejection. Orfal R et al. [25]. Biofilm destruction by debridement, NPWT and ultrasound therapy contributes to wound healing [29].

Epithelialization is challenging especially when the surface area is significant, the development of novel therapies have gained increased interest with a particular focus on biological dressings and their effect on key wound healing pathways. Jordan Holl J et al [26].

Screening exams should be performed every 3-6 months for high risk patients. Increased foot temperature is a pre ulcerative sign. Temp sensing mats and socks have been successful in identifying pre ulcer or DFU. Pressure sensing modalities including insoles may help for early offloading before or during DFU episodes. Schaper NC et al. IWGDF 2020 [27].

Effective medical therapies for patients with PAD should be prescribed to prevent major adverse cardiovascular events for patients with PAD, including anti-platelets, antithrombotic therapy, lipid-lowering and antihypertensive medications. Gerhard-Herman MD et al. Preventive measures to avoid foot injuries and control of DM, lipid level, avoiding smoking and revascularization had positive impact on wound healing. Local management of DFUs such as debridement, dressing, revascularization, stem cell decompression, oxygen therapy, revascularization, wound repair, offloading, stem cell transplantation, and anti- infection treatment and use of latest technologies have promising out come. Yang L et al. [28] Nano therapy technique is used nowadays to achieve different objectives in the management of DM foot ulcer, which is expense and not available everywhere.

Improving the general condition of the patient, and psychotherapy also have very effective role in wound healing and pts compliance to treatment. Regular physiotherapy especially bed side exercises improve glycaemic control, general well being, muscle power and local circulation and minimize disuse atrophy of muscles. Flexibility of limb joints is increased, gait and balance is also improved and osteoporosis is decreased. High protein diet, hydration, vitamin supplement and improving the hygienic condition of the patient has much better effect on the overall health and fitness of the patient.

Professional guidelines for screening and management of patients at risk for DFU by the society for vascular surgery 2016, international working group on the Diabatic foot 2019 and American Diabetes association 2022 are very helpful in planning preventive measures by regular foot examination. Frequent foot exams are fundamental to detect early complications. Inadequate foot care is associated with higher rates of DFU and its complications.

CONCLUSION

Early surgical consultation and intervention in the form of drainage of pus, decompression of effected close compartments, surgical debridement of gangrenous and necrotic tissue, loose dressings and off loading the pressure areas are imperative for better outcome in the treatment of Diabetic foot. Control of DM and treatment of associated medical conditions and antibiotics according to CS can steer to rapid wound healing and recovery. Repeated follow up debridement and dressings are necessary till the complete wound healing. Use of povidon iodine solution as an local antiseptic, saline wound irrigation and saline soaked gauze dressings are very effective and cheaper way to control local wound infection, to promote granulation tissue development and in wound healing.

Simple measures to protect the foot from injuries can delay or prohibit the development of DM foot. Avoiding wearing tight shoes, bare foot walk, observing good hygiene and regular feet examination is very important. Even minor trauma can trigger the worsening course of DM foot which spread very rapidly if timely preventive measures

are not taken. Some time pt is not anticipating so rapid spread of infection and soft tissue destruction leading to increased morbidity and mortality.

Poly neuropathy, macro and micro angiopathy, smoking, IHD and other organ system involvement undermine the healing process. Vascular interventions like stenting of the atherosclerotic segments of lower limb vessel or bypass procedures improve foot circulation that helps in the healing process. Exposure to cold can cause arterial spasm which instigate foot ischemia is also avoidable. Adequate hydration is mandatory which can improve tissue perfusion and healing of foot ulcers.

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