A Dissertation on

“A COMPARATIVE STUDY BETWEEN WAGNERS AND UNIVERSITY OF TEXAS SCORING SYSTEM IN DIABETIC FOOT ULCER MANAGEMENT”

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BONAFIDE CERTIFICATE

Certified that this dissertation is the bonafide work of **Dr. PRABHAKARAN. U** on **"A COMPARATIVE STUDY BETWEEN WAGNERS AND UNIVERSITY OF TEXAS SCORING SYSTEM IN DIABETIC FOOT ULCER MANAGEMENT"** during his M.S. (General Surgery) course from March 2016 to September 2016 at the Madras Medical College and Rajiv Gandhi Government General Hospital, Chennai – 600003.

Prof.Dr.P.RAGUMANI. M.S.
Director,
Institute of General Surgery,
Madras Medical College &
Rajiv Gandhi Government
General Hospital,
Chennai – 600 003.

Prof.Dr.K.RAMASUBRAMANIAN, M.S.,
Professor of General Surgery,
Institute of General Surgery,
Madras Medical College &
Rajiv Gandhi Government
General Hospital,
Chennai – 600 003.

Prof.Dr.MK.MURALITHARAN M.S,M.Ch,
DEAN,
Madras Medical College &
Rajiv Gandhi Government General Hospital,
Chennai – 600 003.

DECLARATION

I, certainly declare that this dissertation titled, "**A COMPARATIVE STUDY BETWEEN WAGNERS AND UNIVERSITY OF TEXAS SCORING SYSTEM IN DIABETIC FOOT ULCER MANAGEMENT**", represent a genuine work of mine . The contribution of any supervisors to the research are consistant with normal supervisory practice, and are acknowledged.

I, also affirm that this bonafide work or part of this work was not submitted by me or any others for any award, degree or diploma to any other university board, neither in India or abroad. This is submitted to The Tamil Nadu Dr.MGR Medical University, Chennai in partial fulfilment of the rules and regulation for the award of Master of Surgery Degree Branch 1 (General Surgery).

Date :

Place: **Dr.PRABHAKARAN. U**

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ABSTRACT

BACKGROUND AND OBJECTIVE

Diabetes is one of the most common co-morbid illness in our community. Objective of this study is that the following two ulcer classification systems were applied to new foot ulcers to compare them as predictors of outcome: the Wagner (grade) and the University of Texas (UT) (grade and stage) wound classification systems

To describe the lesions we treat study and compare outcomes and to identify measures to decrease morbidity and mortality due to diabetic foot disease

METHODS

Between July 2016 and September 2016, 50 patients with diabetic foot who got admitted to Institute of General Surgery,Rajiv Gandhi Government General Hospital,Chennaiwere subjected to surgical treatment depending upon the Wagner's classification and university of texas classification sytem. Data was collected and analyzed.

RESULTS

Majority of the patients came with poor glycemic control at the time of presentation. Conservative management with antibiotics was useful in some patients. Most number of patients needed surgical treatment either in the form of debridement or amputation.

INTERPRETATION AND CONCLUSION

Patient education and strict glycemic control can reduce the burden of diabetic foot. Early diagnosis and hospitalization, appropriate treatment including medical and surgical treatment according to the grade can reduce the morbidity mortality and improve the outcome of the disease. Increasing stage, regardless of grade, is associated with increaseed risk of amputation and prolonged ulcer healing time. The UT system’s inclusion of stage makes it a better predictor of outcom

KEY WORDS: Antibiotics; Amputation; Wagner classification; Complications; Glycemic control.

“Wagner’s Classification for diabetic foot disease (adopted from Levin and O’Neals)”

Grade	**Description**
Grade 0	High risk foot and no ulceration
Grade 1	Superficial Ulcer; Total destruction of the thickness of the skin
Grade 2	Deep Ulcer (cellulitis); Penetrates through skin,fat,ligaments not affecting bone
Grade 3	Osteomyelitis with Ulceration or abscess
Grade 4	Gangrenous patches limited to toes or part of the foot
Grade 5	Gangrene of the entire foot

Table 6 **University of Texas Classification System**

Stage	Grade			
	O	I	II	III
A	Pre- or post-ulcerative lesions completely epithelized	Superficial wound not involving tendon, capsule, or bone	Wound penetrating to tendon or capsule	Wound penetrating to bone or joint
B	Infected	Infected	Infected	Infected
C	Ischemic	Ischemic	Ischemic	Ischemic
D	Infected and ischemic	Infected and ischemic	Infected and ischemic	Infected and ischemic

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Introduction

INTRODUCTION

Four categories of diabetes are recognized. Type 1, formerly insulin-dependent diabetes mellitus (IDDM), is an autoimmune disease affecting the pancreas. Individuals with type 1 diabetes are prone to ketosis and unable to pro- duce endogenous insulin. Type 2, formerly non-insulin dependent diabetes mellitus (NIDDM), accounts for 90% to 95% of cases diagnosed. Type 2 diabetes is characterized by hyperglycemia in the presence of hyperinsulinemia due to peripheral insulin resistance. Gestational as well as genetic defects and endocrinopathies are recognized as other types of diabetes (11). Diabetes is associated with numerous complications related to microvascular, macrovascular, and metabolic etiologies. These include cerebrovascular, cardio- vascular, and peripheral arterial disease; retinopathy; neu- ropathy; and nephropathy. Currently, cardiovascular com- plications are the most common cause of premature death. Diabetes continues to de one of the most common underlying cause of non-traumatic lower extremity amputations (LEAs)

Epidemiology (*INT. J. DIAB. DEV. COUNTRIES (1994), VOL. 14)*

"Mean age at diagnosis of diabetic foot and mean age at major amputation was significantly lower as compared to Western literature. This should be the sole reason to explain favourable results seen in our series specially in reference to survival at 2 years after major amputation, contralateral limb amputation rate, above knee to below knee amputation rate. Older patients reported in Western literature are more likely to have advanced atherosclerotic disease involving heart, cerebral circulation, peripheral circulation and renal circulation thus adversely affecting mortality and contralateral limb amputation rate. Above knee amputation was common in Western population and above knee to below knee amputation ratio was 1:2 vs. 1:17 in Western vs. our series."

"Majority of our patients have infection as a dominant feature in non-neuroischemic foot. In such cases local debridement, control of infection and diabetes, certainly improves the limb salvage. If the infection is fulminant, minor or at the most below knee amputation is enough to stop the advancing infective process. As against this in Western patients, where old age and neuroischemic limbs are common, advanced atherosclerosis, and multi- system involvement makes above knee amputation perhaps the right choice to reduce the overall mortality."

"In one population-based study in Sweden (1) the cost of treating foot ulcer was US$ 14,627 as compared to US$ 500 in our patients. The cost of treatment in-patients undergoing amputation was US$ 73,702 in Sweden as compared to US$ 2000 in our patients. This difference in cost of treatment is obviously due to marked economic disparity in two populations. Although cost of private treatment in India is less, majority of our patients have to bear the entire cost of the treatment as they are not medically insured and for them even this cost is substantial."

"Although present study shows favourable results in Indian patients as compared to Western, it will not be surprising if one sees the change in scenario in next ten to thirty years. In India the number of amputation in diabetic patients is bound to increase due to several factors like increasing prevalence of diabetes, longer survival, more ageing population, continued use of tobacco, barefoot walking, careless home surgical attempt, late reporting to medical centre and poor hygienic conditions. Unless urgent steps are taken, India might emerge as a country with highest rate of amputations for diabetic foot."

AIMS AND OBJECTIVES

The purpose of this dissertation is to compare wagners and university of texas scoring system in diabetic foot management at Institute of General surgery, Rajiv Gandhi Government General Hospital, Chennai.

The study period is between March 2016 to September 2016.

1. To evaluate and manage the different lesions of diabetic foot according to Wagner classification and university of texas scoring system.

2. To describe the lesions we treat study and compare outcomes.

3. To identify measures to decrease morbidity and mortality due to diabetic foot disease.

Review Of Literature

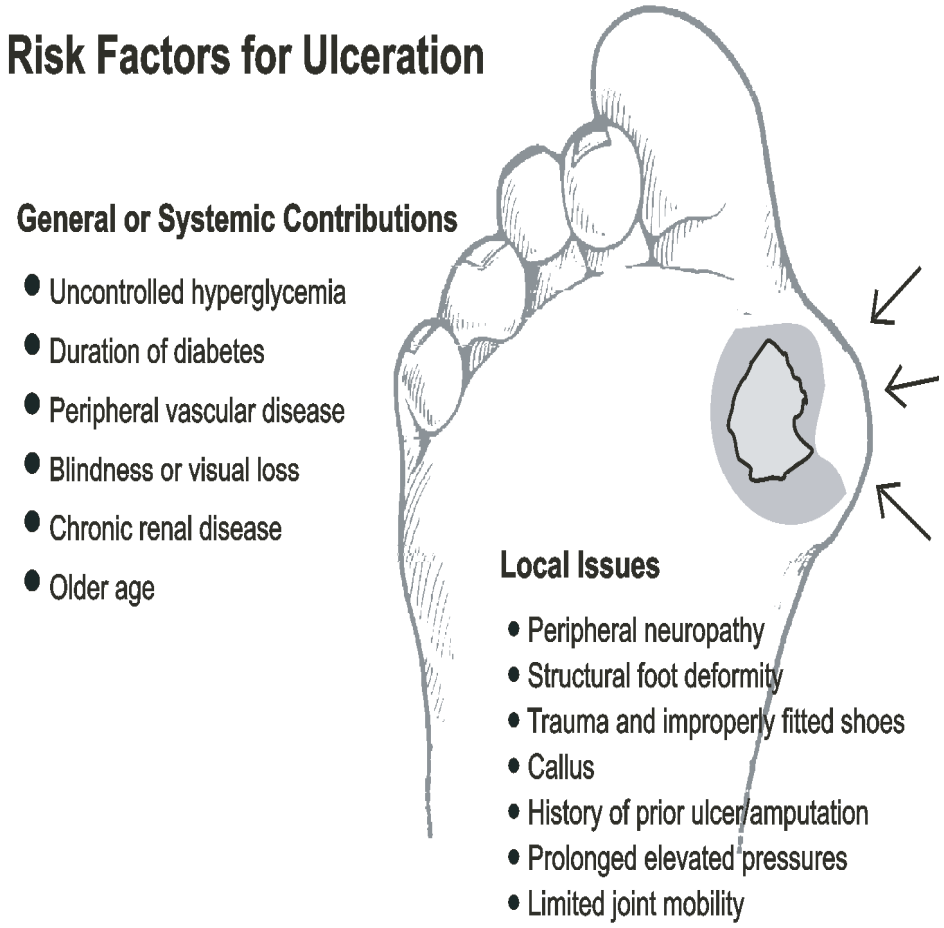
REVIEW OF LITERATURE

`“Diabetic foot ulcers occur as a result of various factors, such as mechanical changes in conformation of the bony architecture of the foot, peripheral neuropathy, and atherosclerotic peripheral arterial disease, all of which occur with higher frequency and intensity in the diabetic population.”

Risk for Ulceration

“Foot ulceration is the most common single precursor to lower extremity amputations among persons with diabetes (28-30). Treatment of infected foot wounds comprises up to one quarter of all diabetic hospital admissions , making this the most common reason for diabetes-related hospitalization in these countries (41-43). The multifactorial nature of diabetic foot ulceration has been elucidated by numerous observational studies (16, 22, 24, 26, 27, 44-48). Risk factors identified include peripheral neuropathy, vascular disease, limited joint mobility, foot deformi- ties, abnormal foot pressures, minor trauma, a history of ulceration or amputation, and impaired visual acuity (25, 49, 50). These and other putative causative factors are shown in Figure 1**.”**

Figure 1 The risk factors for ulceration may be distinguished by general or systemic considerations versus those localized to the foot and its pathology.



"Peripheral sensory neuropathy in the face of unperceived trauma is the primary factor leading to diabetic foot ulcerations (24, 27, 46, 49). Approximately 45% to 60% of all diabetic ulcerations are purely neuropathic, while up to 45% have neuropathic and ischemic components (24, 51). According to an important prospective multicenter study, sensory neuropathy was the most frequent component in the causal sequence to ulceration in diabetic patients (24)."

"Other forms of neuropathy may also play a role in foot ulceration. Motor neuropathy resulting in anterior crural muscle atrophy or intrinsic muscle wasting can lead to foot deformities such as foot drop, equinus, hammertoe, and prominent plantar metatarsal heads (25, 26, 52-54). Ankle equinus with restricted dorsiflexory range of motion is fairly common in patients with diabetic neuropathy and can be a consequence of anterior crural muscle atrophy (55-60). The decreased ankle motion, which confers higher-than- normal plantar pressures at the forefoot, has been implicated as a contributory cause of ulceration as well as recurrence or recalcitrance of existing ulcers (57, 58, 60, 61)."

“Autonomic neuropathy often results in dry skin with cracking and fissuring, creating a portal of entry for bacteria (42, 63). Auto-sympathectomy with attendant sympathetic failure, arteriovenous shunting, and microvascular thermoregulatory dysfunction impairs normal tissue perfusion and microvascular responses to injury. These alterations can subsequently be implicated in the pathogenesis of ulceration (63-67).”

“Foot deformities resulting from neuropathy, abnormal biomechanics, congenital disorders, or prior surgical inter- vention may result in high focal foot pressures and increased risk of ulceration (24, 48, 50, 57, 68-71). The effects of motor neuropathy occur relatively early and lead to foot muscle atrophy with consequent development of hammertoes, fat pad displacement, and associated increases in plantar forefoot pressures (53, 72-75). Although most deformities cause high plantar pressures and plantar foot ulcerations, medial and dorsal ulcerations may develop as a result of footwear irritation. Common deformities might include prior partial foot amputations, prominent metatarsal heads, hammertoes, Charcot arthropathy, or hallux

valgus (69, 76-79). A large prospective population-based study found that elevated plantar foot pressures are significantly associated with neuropathic ulceration and amputation (80). The study also revealed a trend for increased foot pressures as the number of pedal deformities increased."

Trauma to the foot in the presence of sensory neuropathy is an important component cause of ulceration (24). While trauma may include puncture wounds and blunt injury, a common injury leading to ulceration is moderate repetitive stress associated with walking or day-to-day activity (69, 76, 81). This is often manifested by callus formation under the metatarsal heads (48, 82, 83). A recent report suggests that even with moderate activity, ulceration may be precipitated by a higher degree of variability in activity or period- ic "bursts" of activity (84). Shoe-related trauma has also been identified as a frequent precursor to foot ulceration (28, 51, 54, 85, 86).

"Peripheral arterial disease (PAD) rarely leads to foot ulcerations directly. However, once ulceration develops, arterial insufficiency will result in prolonged healing, imparting an elevated risk of amputation (28, 87, 88). Additionally, attempts to resolve any infection will be impaired due to lack of oxygenation and difficulty in delivering antibiotics to the

infection site. Therefore, early recognition and aggressive treatment of lower extremity ischemia are vital to lower limb salvage (30, 52, 89-91)."

"Limited joint mobility has also been described as a potential risk factor for ulceration (92-94). Glycosylation of collagen as a result of longstanding diabetes may lead to stiffening of capsular structures and ligaments (cheiroarthropa- thy) (95).

The subsequent reduction in ankle, subtalar, and first metatarsophalangeal (MTP) joint mobility has been shown to result in high focal plantar pressures with increased ulceration risk in patients with neuropathy (92, 96, 97). Several reports also attribute glycosylation and altered arrangement of Achilles tendon collagen to the propensity for diabetic patients to develop ankle equinus (98, 99)."

Other factors frequently associated with heightened ulceration risk include nephropathy, poor diabetes control, duration of diabetes, visual loss, and advanced age (48, 69,

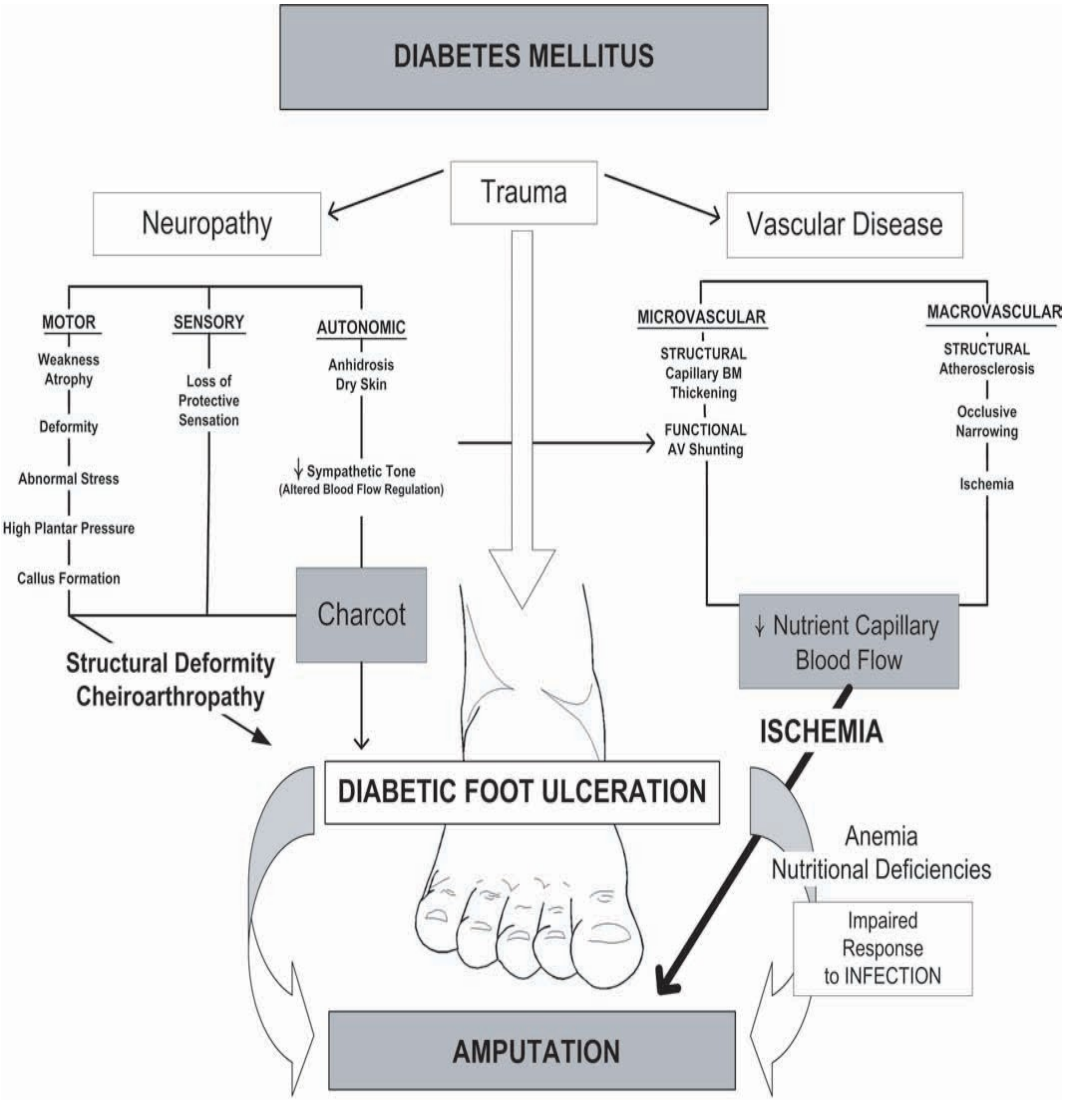


Figure 2 Diabetes mellitus is responsible for a variety of foot pathologies contributing to the complications of ulceration and amputation. Multiple pathologies may be implicated, from vascular disease to neuropathy tomechanical trauma.

93, 100). Soft tissue changes (other than cheiro arthropathy) in the feet of diabetic patients might also contribute to ulceration through the pathway of altered pressure distributions through the sole of the foot. Such alterations include a reported increased thickness of the plantar fascia with associated limitation of hallux dorsiflexion, decreased thickness of plantar soft tissue, accentuated hardness/stiffness of the skin, and a propensity to develop calluses (82, 96, 101-105). While these changes are presumably caused by glycosylation of collagen, their sum effect is to enhance plantar pressures in gait. In the presence of neuropathy, the accentuated plantar pressures can be implicated in the development of ulceration (70, 80, 92, 106).

Mechanisms of Injury

"The multifactorial etiology of diabetic foot ulcers is evidenced by the numerous pathophysiologic pathways that can potentially lead to this disorder (24, 43, 54, 62, 90, 107). Among these are two common mechanisms by which foot deformity and neuropathy may induce skin breakdown in persons with diabetes (69, 108, 109).

The first mechanism of injury refers to prolonged low pressure over a bony prominence (ie, bunion or hammertoe deformity). This generally ca uses wounds over the medial, lateral, and dorsal aspects of the forefoot and is associated with tight or ill-fitting shoes. Shoe trauma, in concert with loss of protective sensation and concomitant foot deformity, is the leading event precipitating foot ulceration in persons with diabetes (24, 28, 57, 85)."

Regions of high pedal pressure are frequently associated with foot deformity (68, 73, 76, 77, 106, 107). When an abnormal focus of pressure is coupled with lack of protective sensation, the result can be development of a callus, blister, and ulcer (110). The other common mechanism of ulceration involves prolonged repetitive moderate stress (108). This normally occurs on the sole of the foot and is related to prominent metatarsal heads, atrophied or anterior- ly displaced fat pads, structural deformity of the lower extremity, and prolonged walking. Rigid deformities such as hallux valgus, hallux rigidus, hammertoe, Charcot arthropathy, and limited range of motion of the ankle (equi- nus), subtalar, and MTP joints have been linked to the development of diabetic foot ulcers (27, 57, 71, 80, 94, 96).

Numerous studies support the significant association between high plantar pressures and foot ulceration (26, 70, 80, 92, 106, 111, 112). Other biomechanical perturbations, including partial foot amputations, have the same adverse effects (57, 68, 80, 113).

Figure 2 summarizes the various pathways and contribut- ing factors leading to diabetic foot complications.

Risk for Infection

"Infections are common in diabetic patients and are often more severe than infections found in nondiabetic patients. Persons with diabetes have an increased risk for developing an infection of any kind and a several-fold risk for develop- ing osteomyelitis (114). With an incidence of 36.5 per 1,000 persons per year, foot infections are among the most com- mon lower extremity complications in the diabetic population (excluding neuropathy), second only to foot ulcers in frequency (115)."

"It is well documented that diabetic foot infections are frequently polymicrobial in nature (30, 116-121). Hyperglycemia, impaired immunologic responses, neuropathy, and peripheral arterial disease are the major predisposing factors leading to limb-threatening diabetic foot infections (122-124). Uncontrolled diabetes results in impaired ability of host leukocytes to fight bacterial pathogens, and ischemia also affects the ability to fight infections because delivery of antibiotics to the site of infection is impaired.

Consequently, infection can develop, spread rapidly, and produce significant and irreversible tissue damage (125). Even in the presence of adequate arterial perfusion, under- lying peripheral sensory neuropathy will often allow the progression of infection through continued walking or delay in recognition (126, 127)."

Risk for Charcot Joint Disease

"It has been estimated that less than 1% of persons with diabetes will develop Charcot joint disease (128-130). Data on the true incidence of neuroarthropathy in diabetes are limited by the paucity of prospective or population-based studies in the literature. One large population-based

prospective study found an incidence of about 8.5 per 1,000 persons with diabetes per year (115); this equates to 0.85% per year and is probably the most reliable figure currently available. Much of the data clinicians rely upon have been extracted from retrospective studies of small, single-center cohorts. The incidence of reported Charcot cases is likely to be underestimated because many cases go undetected, espe- cially in the early stages (131-134)."

"Primary risk factors for this potentially limb-threatening deformity are the presence of dense peripheral sensory neu- ropathy, normal circulation, and history of preceding trau- ma (often minor in nature) (50, 135, 136). Trauma is not limited to injuries such as sprains or contusions. Foot deformities, prior amputations, joint infections, or surgical trauma may result in sufficient stress that can lead to Charcot joint disease (137-140)."

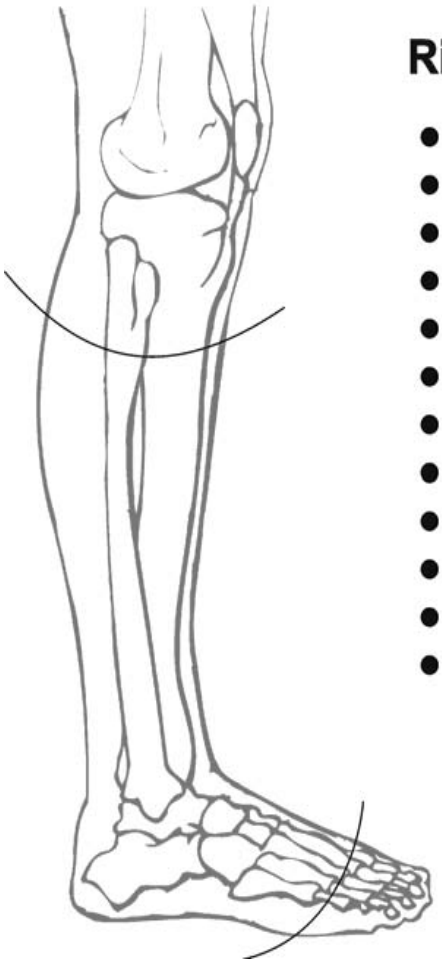
Risk for Amputation

"The reported risk of lower extremity amputations in dia- betic patients ranges from 2% to 16%, depending on study design and the populations studied (19, 21, 32, 115, 141- 144). LEA rates can be 15 to 40 times higher among the diabetic versus nondiabetic populations (8, 16, 34, 35). Although one author suggests that amputation may be a marker not only for disease severity but also for disease management, it is clear that amputation remains a global problem for all persons with diabetes (32, 143). The same risk factors that predispose to ulceration can also generally be considered contributing causes of amputation, albeit with several modifications (Fig 3)."

"While peripheral arterial disease may not always be an independent risk factor for ulceration when controlling for neuropathy, it can be a significant risk factor for amputation (24, 28, 88, 142, 145, 146). PAD affecting the feet and legs is present in 8% of adult diabetic patients at diagnosis and in 45 % after 20 years (147, 148). The incidence of ampu- tation is 4 to 7 times greater for diabetic men and women than for their nondiabetic counterparts.

Impairment of arte- rial perfusion may be an isolated cause for amputation and a predisposing factor for gangrene. Early diagnosis, control of risk factors, and medical management as well as timely revascularization may aid in avoiding limb loss (30, 52, 77, 88, 149).”

Figure 3 The risk factors for amputation are multifactorial and similar to those for ulceration.



Risk Factors for Amputation

- Neuropathy LOPS
- Peripheral arterial disease (PAD)
- Infection
- History of prior foot ulcer or amputation
- Structural foot deformity
- Trauma
- Charcot foot
- Impaired vision
- Poor glycemic control
- Older age
- Male Sex
- Ethnicity (greatest rates in blacks & Hispanics)

“While infection is not often implicated in the pathway leading to ulceration, it is a significant risk factor in the causal pathway to amputation (24, 28). Lack of wound heal- ing, systemic sepsis, or unresolved infection can lead to extensive tissue necrosis and gangrene, requiring amputa- tion to prevent more proximal limb loss. This includes soft tissue infection with severe tissue destruction, deep space abscess, or osteomyelitis. Adequate debridement may require amputation at some level as a means of removing all infected material (77, 123, 150, 151).”

“Another frequently described risk factor for amputation is chronic hyperglycemia. Results of the Diabetes Control and Complications Trial (DCCT) and the United Kingdom Prospective Diabetes Study (UKPDS) support the long-held theory that chronic poor control of diabetes is associated with a host of systemic complications (152, 153). The link between degree of glucose control and incidence or pro- gression of numerous diabetic complications has been well established by these and other studies (154, 155). Such complications include peripheral neuropathy, microan- giopathy, microcirculatory disturbances, impaired leuko- cyte phagocytosis, and glycosylation of tissue proteins. Each has adverse effects on the diabetic foot: They can con- tribute to the etiology of foot ulceration, delay normal wound healing, and subsequently lead to

amputation (25, 30, 48, 50, 72). Several studies have reported a significant correlation between elevated glucose and LEA (21, 141,"

156-161). Amputation has also been associated with other diabetes-related comorbidities such as nephropathy, retinopathy, and cardiovascular disease (21, 48, 144). Aggressive glucose control, management of associated comorbidities, and appropriate lower extremity care coordi- nated in a team environment may indeed lower overall risk for amputation (30, 90, 162-166).

"The best predictor of amputation is a history of previous amputation. A past history of a lower extremity ulceration or amputation increases the risk for further ulceration, infection, and subsequent amputation (29, 142, 157, 167). It may also be inferred that patients with previous ulceration possess all the risk factors for developing another ulcera- tion, having demonstrated that they already have the com- ponent elements in the causal pathway (24, 27, 28, 57). Up to 34% of patients develop another ulcer within 1 year after healing an index wound, and the 5-year rate of developing a new ulcer is 70% (164, 168). The recurrence rate is high- er for patients with a previous amputation because of abnor- mal distribution of plantar pressures and altered osseous architecture. The cumulative risks of neuropathy, deformity, high plantar pressure, poor glucose control, and male gen- der are all additive factors for pedal

ulceration in these dia- betic patients (26, 46, 50, 57, 111). Re-amputation can be attributed to disease progression, nonhealing wounds, and additional risk factors for limb loss that develop as a result of the first amputation."

History

"A thorough medical and foot history must be obtained from the patient. The history should address several specific diabetic foot issues (Table 2)."

Physical Examination

"All patients with diabetes require a pedal inspection whenever they present to any health care practitioner, and

Table 2 **Medical History**

Global History	Foot Specific History	
	General	Wound / Ulcer History
• Diabetes - duration • Glycemic management/control • Cardiovascular, renal and opthalmic evaluations • Other comorbidities • Treating physicians • Nutritional status • Social habits: alcohol, tobacco, drugs • Current medications • Allergies • Previous hospitalizations/surgery	• Daily activities, including work • Footwear • Chemical exposures • Callus formation • Foot deformites • Previous foot infections, surgery • Neuropathic symptoms • Claudication or rest pain	• Location • Duration • Inciting event or trauma • Recurrence • Infection • Hospitalization • Wound care • Off-loading techniques • Wound response • Patient compliance • Interference with wound care (Family or social problems for patient) • Previous foot trauma or surgery • Presence of edema - unilateral vs bilateral • Charcot foot - previous or active • Charcot treatment

they should receive a thorough lower extremity examina- tion at least once annually (175). Patients with complaints relating to the diabetic foot require more frequent detailed evaluations. The examination should be performed system- atically so that important aspects are not overlooked (62). It begins with a gross evaluation of the patient and extremi- ties. Any obvious problem can then receive closer scrutiny. Key components of the foot examination are presented in Table 3. Although not specifically mentioned in this sec- tion, it is assumed that a general medical assessment (including vital sign measurements) will be obtained."

Diagnostic Procedures

"Diagnostic procedures may be indicated in the assess- ment and care of the diabetic foot. Consideration should be given to the following tests in concert with those suggested by members of the consulting team. It should be noted that many of the following tests lack the ability to impart a definitive diagnosis, necessitating clinical correlation."

Laboratory Tests

"Clinical laboratory tests that may be needed in appropri- ate clinical situations include fasting or random blood glu- cose, glycohemoglobin (HbA1c), complete blood count (CBC) with or without differential, erythrocyte sedimenta- tion rate (ESR), serum chemistries, C-reactive protein, alka- line phosphatase, wound and blood cultures, and urinalysis. Caution must be exercised in the interpretation of laborato- ry tests in these patients, because several reports have doc- umented the absence of leukocytosis in the presence of severe foot infections (117, 122, 151, 176-178). A common sign of persistent infection is recalcitrant hyperglycemia despite usual antihyperglycemic regimens (150)."

Imaging Studies

"The diabetic foot may be predisposed to both common and unusual infectious or noninfectious processes, partially because of the complex nature of diabetes and its associat- ed vascular and neuropathic complications. As a result, imaging presentations will vary due to lack of specificity in complex clinical circumstances (179-181). Such variability creates a challenge in the interpretation of imaging studies. Therefore,

imaging studies should only be ordered to estab- lish or confirm a suspected diagnosis and/or direct patient management. Distinguishing osteomyelitis from aseptic neuropathic arthropathy is not easy, and all imaging studies (Fig 4) must be interpreted in conjunction with the clinical findings (123, 151)."

"Plain radiographs should be the initial imaging study in diabetic patients with signs and symptoms of a diabetic foot disorder (180, 182)."

"Radiographs can detect osteomyelitis, osteolysis, fractures, dislocations seen in neuropathic arthropathy, medial arterial calcification, soft tissue gas, and foreign bodies as well as structural foot deformities, pres- ence of arthritis, and biomechanical alterations (183). Acute osteomyelitis might not demonstrate osseous changes for up to 14 days. Serial radiographs should be obtained in the face of an initial negative radiographic image and a high clinical suspicion of osseous disease (117, 123)."

“Technetium-99 methylene diphosphonate (Tc-99 MDP) bone scans are often used in diabetic foot infection to deter- mine the presence of osteomyelitis. Although highly sensi- tive, this modality lacks specificity in the neuropathic foot (184, 185). Osteomyelitis, fractures, arthritis, and neuro- pathic arthropathy will all demonstrate increased radiotrac- er uptake. However, a negative bone scan is strong evidence against the presence of infection. To improve the specifici- ty of nuclear imaging, white blood cells can be labeled with Tc-99 hexamethylpropyleneamineoxime (Tc-99 HMPAO), indium-111 oxime, or gallium-67 citrate (179, 186-189).”

“Indium-111 selectively labels polymorphonuclear leuko- cytes and is more specific for acute infections than Tc-99 MDP scanning. Chronic infections and inflammation are not well imaged with indium-111, because chronic inflam- matory cells (ie, lymphocytes) predominate and are not well labeled with indium. Combining Tc-99 MDP and indium- 111 increases the specificity of diagnosing osteomyelitis (190). This combined technique is useful, because the Tc-99 MDP scan localizes the anatomic site of inflammation and the indium-111 labels the infected bone (180, 191).

The indium-111 scan is not typically positive in aseptic neuropathic arthropathy, although false-positive indium scans can occur (192-194). A 100% sensitivity and 89% specificity have been reported with the combined technique in evaluat- ing diabetic infections (190, 191, 195)."

Table 3 **Lower Extremity Diabetic Foot Exam**

Vascular Examination

- Palpation of pulses
  Common femoral, popliteal
  Dorsalis pedis, posterior tibial
- Handheld Doppler examination
- Skin / limb color changes
  Cyanosis, erythema
  Elevation pallor, dependent rubor
- Presence of edema
- Temperature gradient
  (ipsilateral and contralateral extremity)
- Dermal thermometry
- Integementary changes
  Skin atrophy - thin, smooth, parchment-like skin
  Abnormal wrinkling
  Absence of hair growth
  Onychodystrophy
- Previous hospitalizations/surgery

Neurologic Examination

- Vibration perception
  Tuning fork 128 cps
  Measurement of vibration perception threshold (biothesiometer)
- Light pressure:
  Semmes-Weinstein 10 gram monofilament
- Light touch: cotton wool
- Two point discrimination
- Pain: pinprick (sterile needle)
- Temperature perception: hot and cold
- Deep tendon reflexes: patella, Achilles
- Clonus testing
- Babinski test
- Romberg test

Footwear Examination

- Type of shoe (athletic, oxford, comfort, etc.)
- Fit
- Depth of toe box
- Shoewear, patterns of wear
- Lining wear
- Foreign bodies
- Insoles, orthoses

Dermatologic Examination

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

Musculoskeletal Examination

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

Vascular Evaluation

"The lower extremity must be assessed for vascular and neuropathic risk factors. Although positive findings in the neurologic examination rarely require further evaluation, positive findings of vascular insufficiency may require fur- ther consultation. The indications for vascular consultation include an ankle brachial index of less than 0.7, toe blood pressures less than 40 mmHg, or transcutaneous oxygen tension ($TcPO_2$) levels less than 30 mmHg, since these measures of arterial perfusion are associated with impaired wound healing (27, 47, 87, 90, 212, 213)."

"If the history and physical examination suggest ischemia (ie, absent pedal pulses) or if a nonhealing ulcer is present, further evaluation in the form of noninvasive testing is war- ranted."

"Noninvasive arterial studies should be performed to determine lower extremity perfusion. Such studies may include Doppler segmental arterial pressures and waveform analysis, ankle-brachial indices (ABI), toe blood pressures, and $TcPO_2$ (89, 214, 215). Ankle-brachial indices may be misleading, because ankle pressures can be falsely elevated due to medial arterial calcinosis and noncompressibility of affected arteries (52, 216, 217).

A growing body evidence suggests that toe blood pressures in diabetic patients may have a role in predicting foot ulceration risk as well as pre- dicting successful wound healing (213, 218, 219). $TcPO_2$ measurements have received similar support in the litera- ture (47, 87, 212). Although not consistently predictive of wound healing outcomes, these physiologic measures of tis- sue oxygenation are highly predictive of wound healing failure at levels below 25 mmHg (87, 212, 220). Both tests can be performed distally on the foot regardless of arterial calcification in the major pedal arteries, and they are both favorable at pressures in the range of 40 mmHg (90, 212, 213)."

"Laser Doppler velocimetry and measurement of skin perfusion pressure (SPP) have primarily been used in research settings, but can accurately assess blood flow and oxygen tension in the superficial arterioles and capillaries of the skin (220-225). Several recent reports indicate that laser Doppler measurement of SPP can be highly predictive of critical limb ischemia and wound healing failure at levels less than 30 mmHg (223, 224)."

"Vascular consultation should be considered in the presence of abnormal noninvasive arterial studies or a nonheal- ing ulceration (30, 54, 173, 215, 226). Arteriography with clearly visualized distal runoff allows appropriate assess- ment for potential revascularization (227-229). Magnetic resonance angiography (230) or CT angiogram are alterna- tives for evaluation of distal arterial perfusion (229, 231).

Neurologic Evaluation

"Peripheral sensory neuropathy is the major risk factor for diabetic foot ulceration (24, 26, 27, 46, 50). The patient his- tory and physical examination utilizing the 5.07 Semmes- Weinstein monofilament (10-g) wire are sufficient to identi- fy individuals at risk for ulceration (26, 232-235)."

"Vibration perception threshold assessment with the bioth- esiometer is also useful in identifying patients at high risk for ulceration (44, 57, 236). More sophisticated studies such as nerve conduction studies are rarely necessary to diagnose peripheral sensory neuropathy. Patients with neu- ropathic ulcerations usually have such profound sensory neuropathy that these studies add little to their clinical management (49)."

Plantar Foot Pressure Assessment

"High plantar foot pressure is a significant risk factor for ulceration (26, 45, 59, 70, 76, 80, 237). Measurement of high plantar foot pressure is possible utilizing a variety of modalities. Several computerized systems can provide quantitative measurement of plantar foot pressure (76, 81, 238-241). While these measurements may be important in identifying areas of the foot at risk for ulceration and possi- bly in evaluating orthotic adjustments (57, 59), they are pri- marily used in diabetic foot research. The Harris mat, while not as sophisticated, can provide a qualitative measurement of plantar foot pressures and can identify potentially vulner- able areas for ulceration.(242)."

Evaluation of Ulcers

"The initial evaluation of the diabetic foot ulcer must be comprehensive and systematic to ascertain the parameters that might have led to its onset as well as determine the presence of factors that can impair wound healing (25, 52, 54). Critical in this regard are assessments for vascular per- fusion (ischemia), infection/osteomyelitis, and neuropathy. As previously discussed, a thorough vascular evaluation must be performed; this includes palpation of pulses, clini- cal evaluation of capillary filling time, venous filling time, pallor on elevation, and

dependent rubor (283). If pulses are not palpable or if clinical findings suggest ischemia, nonin- vasive arterial evaluation (eg, segmental Doppler pressures with waveforms, ankle brachial indices, toe pressures, $TcPO_2$ measurements) and vascular surgical consultation are warranted. When required, these physiologic and anatomic data can be supplemented with the use of magnet- ic resonance angiography (230) or CT angiography (CTA) and subsequent use of arteriography with digital subtraction angiography (DSA) as necessary (77, 89, 284)."

Classification of Diabetic Ulcers

"Appropriate classification of the foot wound is based on a thorough assessment. Classification should facilitate treat- ment and be generally predictive of expected outcomes. Several systems of ulcer classification are currently in use in the US and abroad to describe these lesions and commu- nicate severity (62, 90, 288-292). Perhaps the easiest system is to classify lesions as neuropathic, ischemic, or neuro-ischemic, with descriptors of wound size, depth, and infec- tion (90). Regardless of which system is used, the clinician must be able to easily categorize the wound and, once clas- sified, the ensuing treatment should be directed by the underlying severity of pathology."

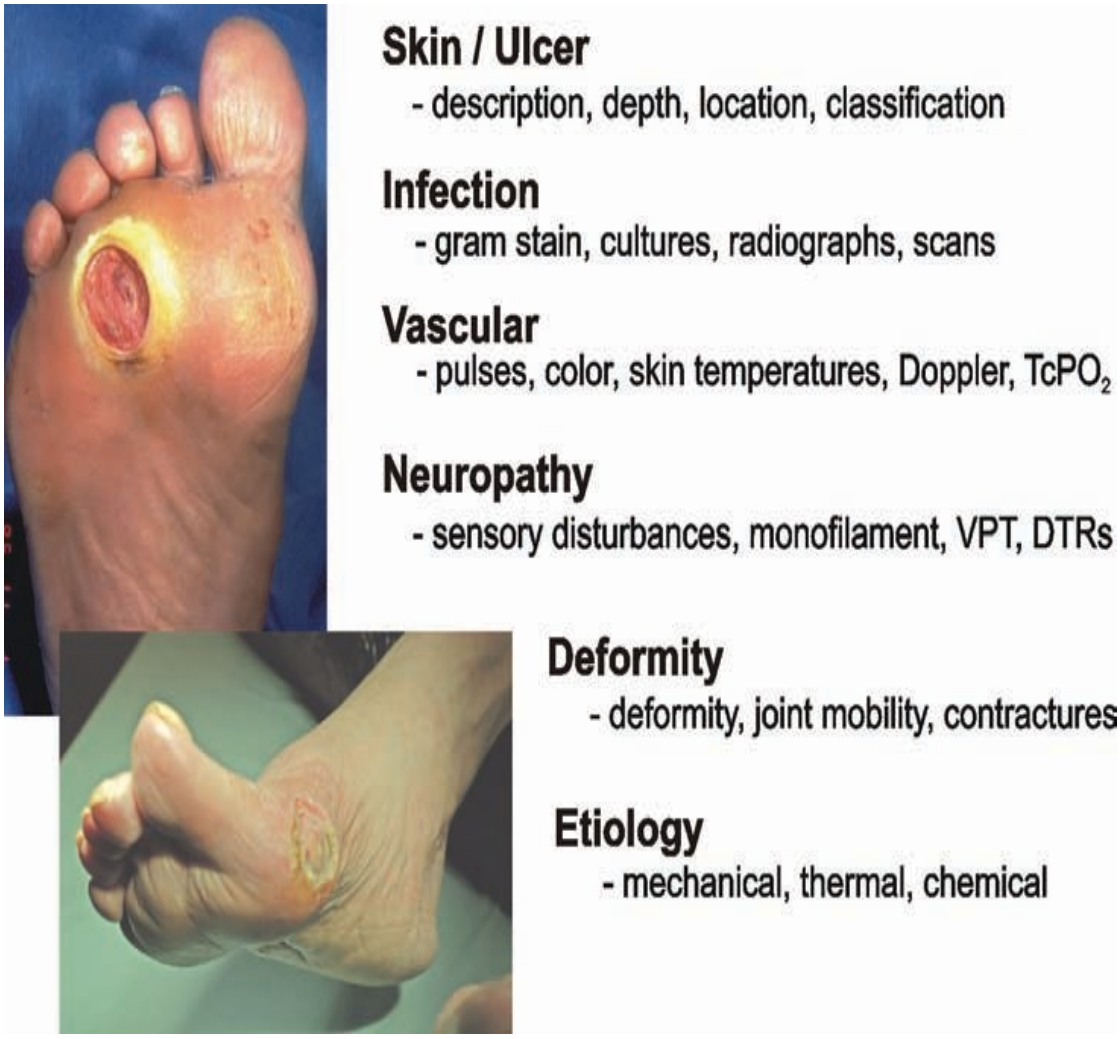
“Although no single system has been universally adopted, the classification system most often used was described and popularized by Wagner (292). In the Wagner system foot lesions are divided into six grades based on the depth of the wound and extent of tissue necrosis

the University of Texas San Antonio (UTSA) sys- tem associates lesion depth with both ischemia and infection (290). This system has been validated and is generally predictive of outcome, since increasing grade and stage of wounds are less likely to heal without revascular- ization or amputation (290, 293). The UTSA system is now widely used in many clinical trials and diabetic foot centers.”

Table 6 **University of Texas Classification System**

Stage	Grade			
	0	I	II	III
A	Pre- or post-ulcerative lesions completely epithelized	Superficial wound not involving tendon, capsule, or bone	Wound penetrating to tendon or capsule	Wound penetrating to bone or joint
B	Infected	Infected	Infected	Infected
C	Ischemic	Ischemic	Ischemic	Ischemic
D	Infected and ischemic	Infected and ischemic	Infected and ischemic	Infected and ischemic

Figure Assessment of a diabetic foot ulcer includes not only a description of the skin lesion but also the find- ings necessary for accurate assessment of thecontributing factors and etiology.



Tissue Management / Wound Bed Preparation

Debridement.

“Debridement of necrotic tissue is an inte- gral component in the treatment of chronic wounds since they will not heal in the presence of unviable tissue, debris, or critical colonization (314, 315). Undermined tissue or closed wound spaces will otherwise harbor bacterial growth (312, 316, 317). Debridement serves various functions: removal of necrotic tissue and callus; reduction of pressure; evaluation of the wound bed; evaluation of tracking and tunneling; and reduction of bacterial burden (318, 319). Debridement facilitates drainage and stimulates healing (320). However, debridement may be contraindicated in arterial ulcers (321). Additionally, except in avascular cases, adequate debridement must always precede the application of topical wound healing agents, dressings, or wound clo- sure procedures (30, 288, 322, 323). Of the five types of debridement (surgical, enzymatic, autolytic, mechanical, biological), only surgical debridement has been proven to be efficacious in clinical trials (323).”

Surgical debridement. “Surgical debridement is the cor- nerstone of management of diabetic foot ulcers. Thorough sharp debridement of all nonviable soft tissue and bone from the open wound is accomplished primarily with a scalpel, tissue nippers, curettes, and curved scissors (324). Excision of necrotic tissue extends as deeply and proximally as

necessary until healthy, bleeding soft tissue and bone are encountered. Any callus tissue surrounding the ulcer must also be removed. The main purpose of surgical debridement is to turn a chronic ulcer into an acute, healing wound (325). A diabetic ulcer associated with a deep abscess requires hospital admission and immediate incision and drainage (178). Joint resection or partial amputation of the foot is necessary if osteomyelitis, joint infection, or gan- grene are present (41, 100, 123, 151, 180, 271).When surgical or sharp debridement is not indicated, other types of debridement can be used. For example, vas- cular wounds may benefit from enzymatic debridement, while an extremely painful wound may benefit from autolytic debridement. Mechanical debridement is often used to cleanse wounds prior to surgical or sharp debride- ment. In areas where the medical staff is not trained in sur- gical or sharp debridement, these other forms of debride- ment may be useful (325)."

Table 8 **Wound Care Products**

Category	Indications	Contraindications
Dressings		
Gauze pads (312, 338, 352) - sterile gauze - sterile cotton	- Low to heavily draining wounds or surgical wounds - Wet to dry debridement	- Undefined
Transparent films (312, 352) - polyurethane film with drainage adhesive layer, semipermeable	- Dry to minimally draining wounds - Promote tissue hydration	- Infection - Significant drainage - Over prominence or friction
Hydrogels (312, 352) - gel, sheet, gauze (95% water or glycerin)	- Dry to minimally draining wounds	- Moderate or heavy drainage
Foam (312, 352) - polyurethane foam (open cell, absorbent)	- Moderate, large exudate - Clean wound surface - Super absorbent and conformable to topography	- Dry wounds
Hydrocolloids (312, 352) - wafer with adhesion, (carboxymethylcellulose,pectin, gelatin) impermeable to oxygen	- Low to moderate drainage * Prevents tissue hydration	- Heavy drainage - Sinus tract
Calcium alginates (312, 352) - fiber pad derived form seaweed (may be combined with silver or collagen)	- Heavy exudative wounds	- Minimal drainage or dry wounds
Collagen dressings (302, 312, 325, 352) - particles or composite pads with collagen component (derived from bovine collagen)	- Low to heavily draining wounds	- Dry wounds
Antimicrobial dressings (312, 334, 352) - contain silver, iodine in various forms preparations (eg, cadoxemer iodine)	- Infected or clean wounds to prevent infection	- Allergies to components
Topical Therapies / Agents		
Saline (302, 352) **Amorphous hydrogels** **Skin cleansers** - isotonic solutions for irrigation, hydrating dressings	- Clean or infected wounds	- Undefined
Detergents/Antiseptics (302, 352) - povidone-iodine, - chlorhexidine - chloroxylenol - hypochlorite - benzethonium chloride	- Contaminated or infected wounds	- Healthy granulating wounds
Topical Antibiotics (302, 320, 352) - bacitracin, neomycin - mupirocin, polymyxin B - silver sulfadiazine - mafenide (creams, ointments)	- Contaminated or infected wounds	- Healthy granulating wounds
Enzymes (302, 312, 319, 328, 332-335) - collagenase - papain-urea	- Necrotic tissue - Escharotic wounds	- Healthy or infected wounds

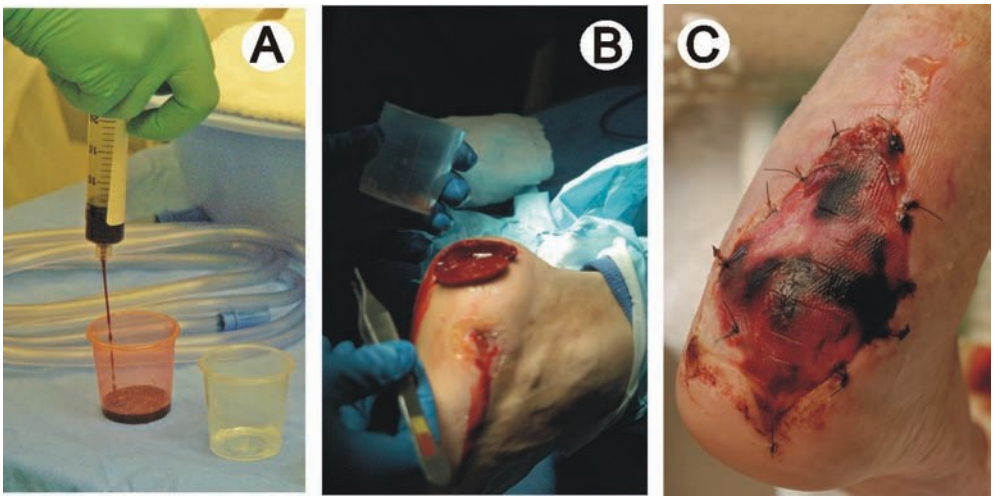


Figure **"**New technologies have been developed that have proved useful for management of diabetic ulcerations. *(A)*Platelet-rich plasma (PRP) involves use of the patient's blood, which is collected and then fractionated through centrifuga- tion. A platelet-rich and platelet-poor supernatant remains. *(B)* This case involved use of autologous platelet-rich plasma gel activated with thrombin and placed onto a healthy wound bed. *(C)* The platelet gel or clot may also be covered with a synthetic skin graft substitute."

An Infra Malleolar infection occurring in a Diabetic patient is characterised as a Diabetic foot Infection. (1)

Every 30 second, a leg is lost because of DM. These ulcers tend to heal slowly, need intensive care and healing can be complicated by infection and gangrene, leading to long-term in-hospital treatment and/or amputation. (4)

Moreover, foot ulcers have major negative effects on quality of life, due to loss of mobility, loss of work and reduction of social activities (4), one study has demonstrated that quality of life of diabetic foot ulcer patients was comparable to that of patients with recurrent breast cancer(5).

Despite these poor outcomes, the feet of diabetic patients have traditionally received relatively little attention from health care workers and scientists. (6)

However, in recent decades our knowledge on diabetic foot ulcers has increased, with a rise in the number of scientific publications and the production of guidelines on prevention and management. (6, 7, 8)

Hospitalization, surgical procedures, and prolonged/broad-spectrum antibiotic therapy may predispose diabetic patients to become colonized and/or infected with antibiotic-resistant organisms such as Methicillin-Resistant Staphylococcus aureus (MRSA) or vancomycin-resistant enterococci (VRE). (1)

CLASSIFICATION

IWGDF (PEDIS) and IDSA. IWGDF developed a system for classifying diabetic foot wounds that uses the acronym PEDIS, which stands for perfusion, extent (size), depth (tissue loss), infection, sensation (neuropathy). While originally developed as a research tool (11), it offers a semiquantitative gradation for the severity of each of the categories. The infection part of the classification differs only in small details from the classification developed by IDSA. Major advantages of both classifications are clear definitions and a relatively small number of categories, making them more user-friendly for clinicians having less experience with diabetic foot management. Importantly, the IDSA classification has been prospectively validated (12, 13, 14) as predicting the need for hospitalization (in one study, 0 for no infection, 4% for mild, 52% for moderate, and 89% for severe infection) and for limb amputation (3% for no infection, 3% for mild, 46% for moderate, and 70% for severe infection) (13).

Wagner—Wagner, in collaboration with Meggitt, developed perhaps the first, and still among the most widely used, classification schemes for diabetic foot wounds (2, 15). It assesses ulcer depth and the presence of infection and gangrene with grades ranging from 0 (pre- or post-ulcerative) to 5 (gangrene of the entire foot). The system only deals

explicitly with infections of all types (deep wound abscess, joint sepsis, or osteomyelitis) in grade 3.

S(AD)/SAD—This is an acronym for 5 key points of foot ulcers: size, (area, depth), sepsis (infection), arteriopathy, and denervation (3). Each point has 4 grades, thus creating a semi-quantitative scale. Infection is graded as none, surface only, cellulitis, and osteomyelitis; these are not further defined. One study reported good inter- observer agreement. Unlike the other key points, studies have not shown infection to be related to outcome of the foot ulcer (3, 16).

The SINBAD ulcer classification is a simplified version of the S(AD)/SAD system with a decreased number of grades of infection (present or absent) (17).

University of Texas (UT) ulcer classification (18)—This system has a combined matrix of 4 grades (related to the depth of the wound) and 4 stages (related to the presence or absence of infection or ischemia). The classification successfully predicted a correlation of the likelihood of complications in patients with higher score.

Ulcer Severity Index [49]—This index measures 20 clinical parameters and allows determination of an infection score by combining the scores for erythema, edema, and purulence, while counting exposed bone

separately. In 1 study, presence or absence of infection in this index was not associated with a difference in wound healing (19).

Diabetic Ulcer Severity Score (DUSS) and MAID (20, 21)—These scoring systems are based on specific wound characteristics associated with stages of wound repair. Studies have found no significant correlation between soft tissue infections and wound healing, although there was a trend toward more infection in the higher-risk groups (20, 21).

DFI Wound Score (22)—Lipsky et al developed this 10- item scoring system to measure outcomes in studies of various antimicrobial treatments for DFIs. The score consists of a semi quantitative assessment of the presence of signs of inflammation, combined with measurements of wound size and depth. Explicit definitions allow numerical scoring of wound parameters. An evaluation of the wound score calculated for 371 patients with DFI demonstrated that it significantly correlated with the clinical response and that scores demonstrated good internal consistency (22). Patients with more severe wounds had higher scores; clinical response was favourable at the follow-up assessment in 94.8% with a baseline score <12 compared with 77.0% with a score >19. Surprisingly, excluding scores for wound discharge (purulent and non-purulent), leaving an 8-item score, provided better measurement statistics (22).

The DFI Wound Score appears to be a useful tool for predicting clinical outcomes in treatment trials, but its complexity requires clinicians to use a scoring sheet (22).

Table 2. Infectious Diseases Society of America and International Working Group on the Diabetic Foot Classifications of Diabetic Foot Infection

Clinical Manifestation of Infection	PEDIS Grade	IDSA Infection Severity
No symptoms or signs of infection	1	Uninfected
Infection present, as defined by the presence of at least 2 of the following items: • Local swelling or induration • Erythema • Local tenderness or pain • Local warmth • Purulent discharge (thick, opaque to white or sanguineous secretion)		
Local infection involving only the skin and the subcutaneous tissue (without involvement of deeper tissues and without systemic signs as described below). If erythema, must be >0.5 cm to ≤2 cm around the ulcer. Exclude other causes of an inflammatory response of the skin (eg, trauma, gout, acute Charcot neuro-osteoarthropathy, fracture, thrombosis, venous stasis).	2	Mild
Local infection (as described above) with erythema > 2 cm, or involving structures deeper than skin and subcutaneous tissues (eg, abscess, osteomyelitis, septic arthritis, fasciitis), **and** No systemic inflammatory response signs (as described below)	3	Moderate
Local infection (as described above) with the signs of SIRS, as manifested by ≥2 of the following: • Temperature >38°C or <36°C • Heart rate >90 beats/min • Respiratory rate >20 breaths/min or $PaCO_2$ <32 mm Hg • White blood cell count >12 000 or <4000 cells/μL or ≥10% immature (band) forms	4	Severe[a]

Abbreviations: IDSA, Infectious Diseases Society of America; $PaCO_2$, partial pressure of arterial carbon dioxide; PEDIS, perfusion, extent/size, depth/tissue loss, infection, and sensation; SIRS, systemic inflammatory response syndrome.

[a] Ischemia may increase the severity of any infection, and the presence of critical ischemia often makes the infection severe. Systemic infection may sometimes manifest with other clinical findings, such as hypotension, confusion, vomiting, or evidence of metabolic disturbances, such as acidosis, severe hyperglycemia, and new-onset azotemia

Table 3: DFI Wound Score

Item	Assessment	Scoring
Wound parameters[a]		
Purulent discharge	Absent	0
	Present	3
Other signs and symptoms of inflammation[a]	Absent	0
Nonpurulent discharge	Mild	1
Erythema	Moderate	2
Induration		
Tenderness		
Pain	Severe	3
Local warmth		
Range of wound parameters (10-item) subtotal		0–21
Range of wound parameters (8-item) subtotal		0–15
Wound measurements[a]		
Size (cm^2)	<1 1–2 >2–5 >5–10 >10–30 >30	0 1 3 6 8 10
Depth (mm)	<5 5–9 10–20 >20	0 3 7 10
Undermining (mm)	<2 2–5 >5	3 5 8
Range of wound measurements subtotal		3–28
Range of total 10-item[b] DFI wound score		3–49
Range of total 8-item[b] DFI wound score		3–43

The 10-item score: purulent discharge, nonpurulent discharge, erythema, induration, tenderness, pain, warmth, size, depth, undermining. The 8-item score leaves out purulent and nonpurulent secretions.

Abbreviation: DFI, diabetic foot infection.

[a] Definitions for wound parameters and wound measurement can be found in the original article

[b] Each assessed and placed in one of the preassigned categories.

BACTERIOLOGY

Aerobic, Gram-positive cocci are the predominant organisms responsible for acute DFI, with Staphylococcus aureus the most commonly isolated pathogen (1, 9, and 10).

In wounds that are chronic, especially in patients who have recently been treated with antimicrobial therapy, infections are more frequently polymicrobial and the causative pathogens are more diverse, often including aerobic gram-negative bacilli and obligate anaerobic bacteria (1, 10)

- **Staphylococcus aureus**

  They are gram positive, non-motile, non sporing facultative anaerobes arranged in clusters. They grow readily in ordinary culture media under aerobic or anaerobic conditions with a temperature range of 10-42 C. (23) On Nutrient Agar, the colonies are circular convex, smooth, opaque and Golden Yellow.

- On Blood Agar, the colonies are similar and most strains are Beta Haemolytic.
- On Mac Conkey's medium, colonies are smaller and pink due to lactose fermentation. (24)
- They produce toxins like haemolysins, leucocidins, enterotoxins, exfoliative toxin and coagulase. (25, 26)

- Direct microscopy shows gram positive cocci in clusters Culture is diagnostic and colonies are smooth, circular and vary in size from 1 to 4 mm depending on the strain and medium used. (23) Most isolates are resistant to Benzyl Penicillin by production of Pencillinase. For broad spectrum penicillins, 20% of strains show resistance by Beta Lactamase production, by the presence of mecA gene. Newer drugs like Vancomycin and Teicoplanin are often needed. (27)

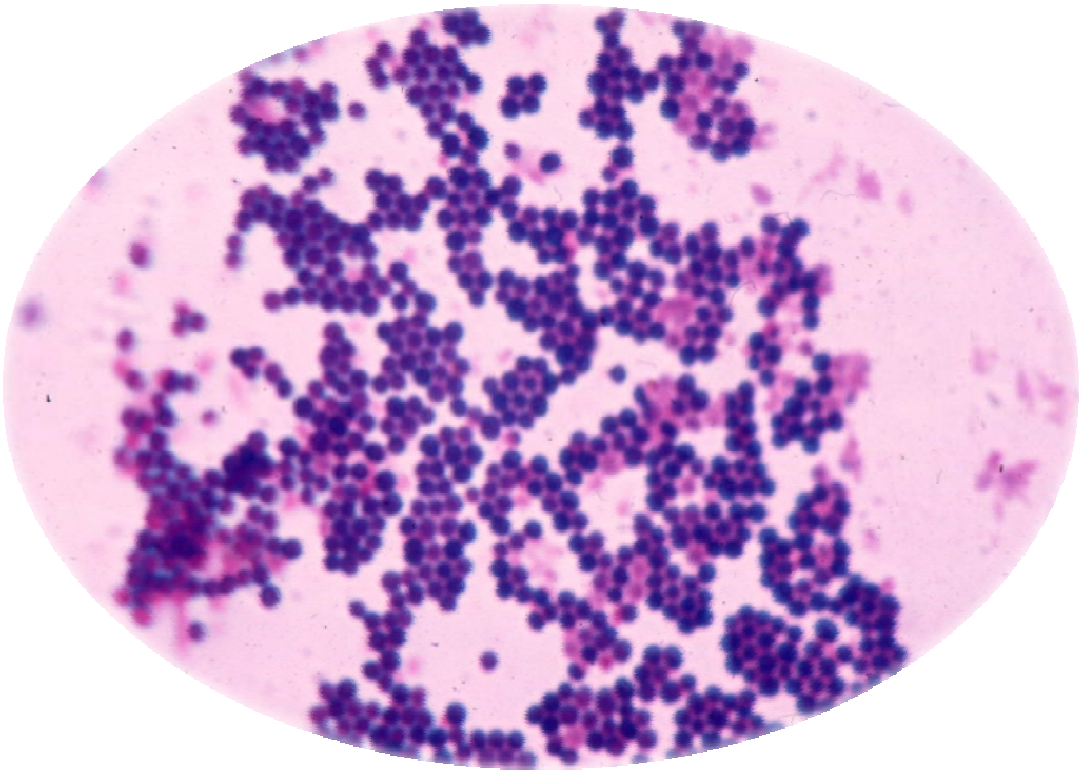


Fig 1: Staphylococci- Gram Staining

METHODOLOGY

AND MATERIALS

METHODOLOGY AND MATERIALS

This study was conducted in the Institute of General Surgery, RGGGH. The Institute receives large number of diabetic foot patients. In that 50 patients were included in the study between July 2016 to September 2016. Patients with Chronic Diabetic Foot and previous amputations were also included in the study. Patients were recruited from the surgical OPD and admitted. Data were collected by detailed history, clinical examination, wound or ulcer and were recorded in the pre-designed profoma. Age, sex, socioeconomic status, duration and type of diabetes, wagner's classification,university of texas scoring examination findings, blood investigations, renal function test, swab of the wound. X-ray and treatment provided were collected. At presentation, the site of the ulcer was noted, and a photograph was taken. After wound debridement, the area of each ulcer was measured using a wound-mapping chart . Each ulcer was graded using both classification systems and staged using the UT system. Ulcers were labeled infected if a purulent discharge was present with two other local signs (warmth, erythema, lymphangitis, lymphadenopathy, oedema, pain). Wound depth was evaluated using a sterile blunt probe. The ability to probe to bone (20) with the presence of local or systemic infection and suggestive radiological features provided a clinical diagnosis of osteomyelitis. The diagnosis of lowerextremity

vascular insufficiency was made clinically on the basis of absence of both pedal pulses of the involved foot and/or an ankle-brachial pressure index of 0.9 (21). Patients initially were seen in the diabetic foot clinic on a weekly basis and were provided with the best possible care for their ulcers at each visit. To remove extensive callus and necrotic tissue, wound debridement was performed. After wound dressing, pressure relief was provided with either a scotchcast boot or a total contact cast. Broad spectrum antibiotics were prescribed if ulcers showed clinical signs of infection (growth factors were not used to enhance healing in this study). Patients with cl clinical evidence of ischemia had noninvasive ultrasound vascular studies and were seen by the vascular surgeon if necessary. Patient follow-up was part of the normal treatment. Unhealed ulcers were followed up for a minimum period of 6 months. Once a patient's ulcer had healed completely or a lower-limb amputation was performed, the outcome was noted and the patient was deemed to have completed the study

Statistical analysis;

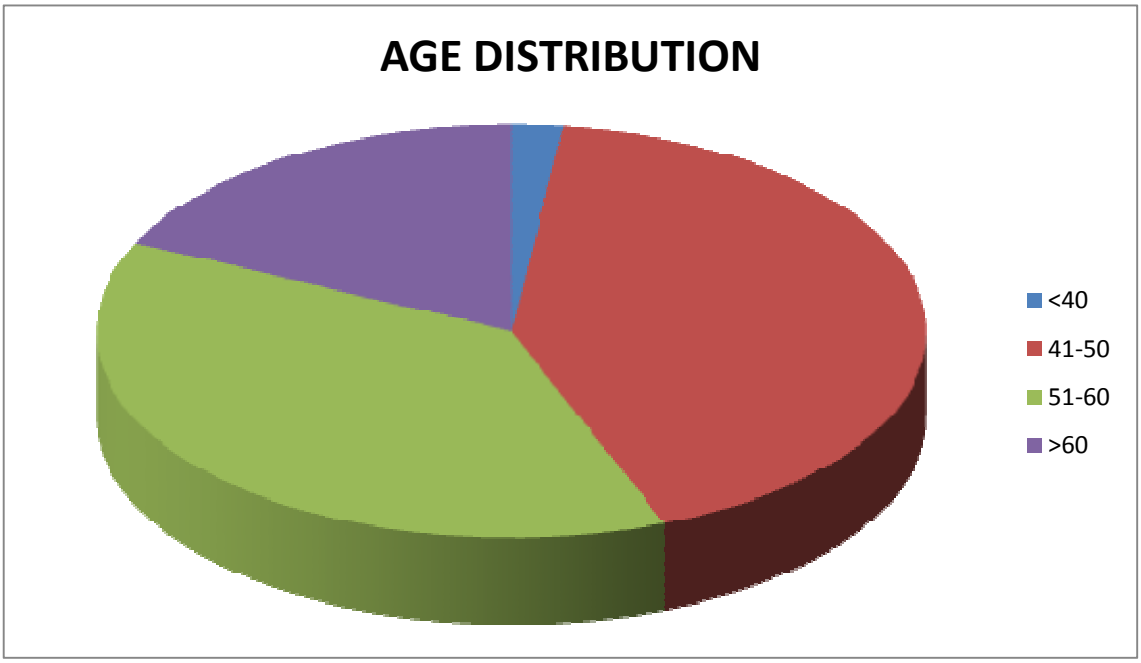
A chi square test was used to assess the trend association between increasing grade or stage and the prevalence of lower-extremity amputation (25,25a). To assess the potential association between stage and the number of amputations performed by the end of the study period, chi square analysis with odds ratio (OR) was performed. Kaplan-Meier survival analysis was used to estimate median healing times, and a log-rank test was used to compare healing times for different levels of grade or stage. Cox regression analysis was used to assess the ability of grade and stage to predict healing within the study period (25,25a). The 95% CI was calculated whenever appropriate, and statistical significance was defined as a P value 0.05. Statistical analysis was performed using SPSS for Windows, version 9.0 (SPSS, Chicago).

RESULTS

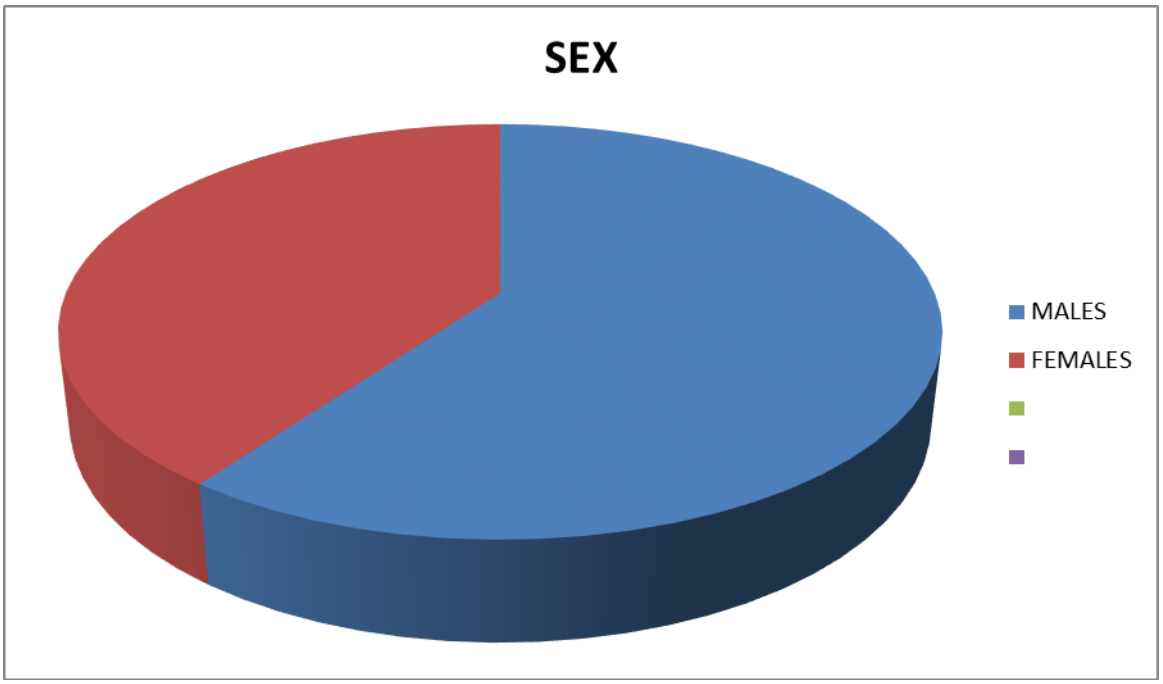
RESULTS

Table 1 – Sociodemographic characteristics of the patients

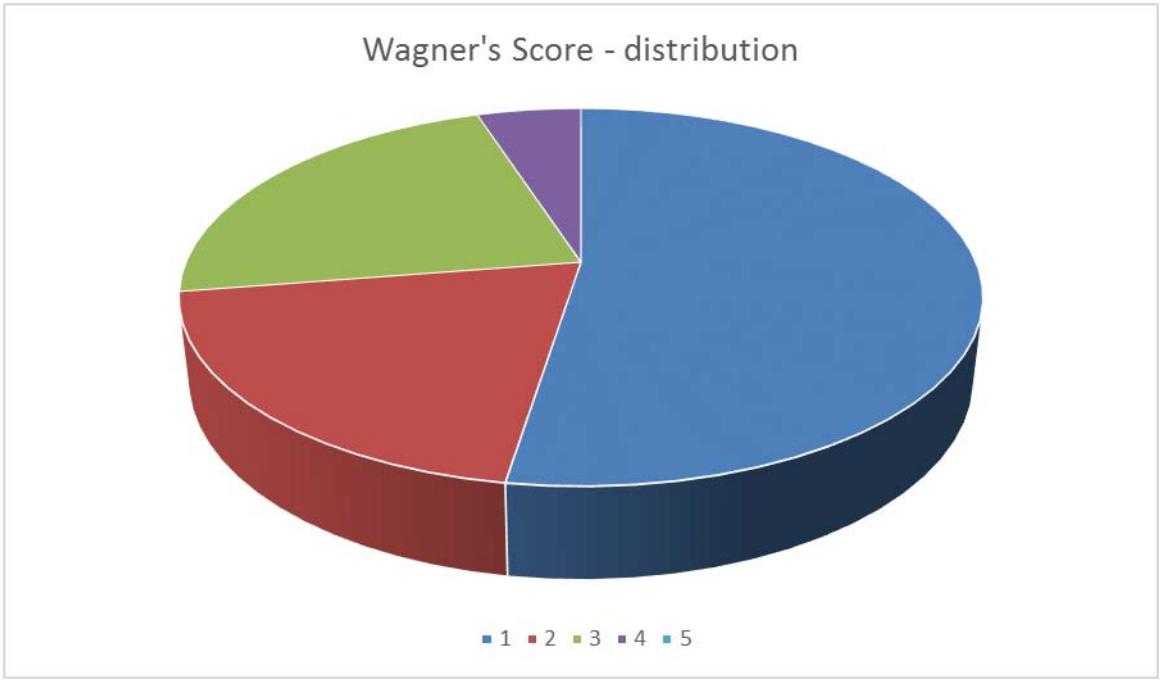
Characteristics	Number	%age
Age/years		
<40	1	2%
41-50	21	42%
51-60	19	38%
>60	9	19%

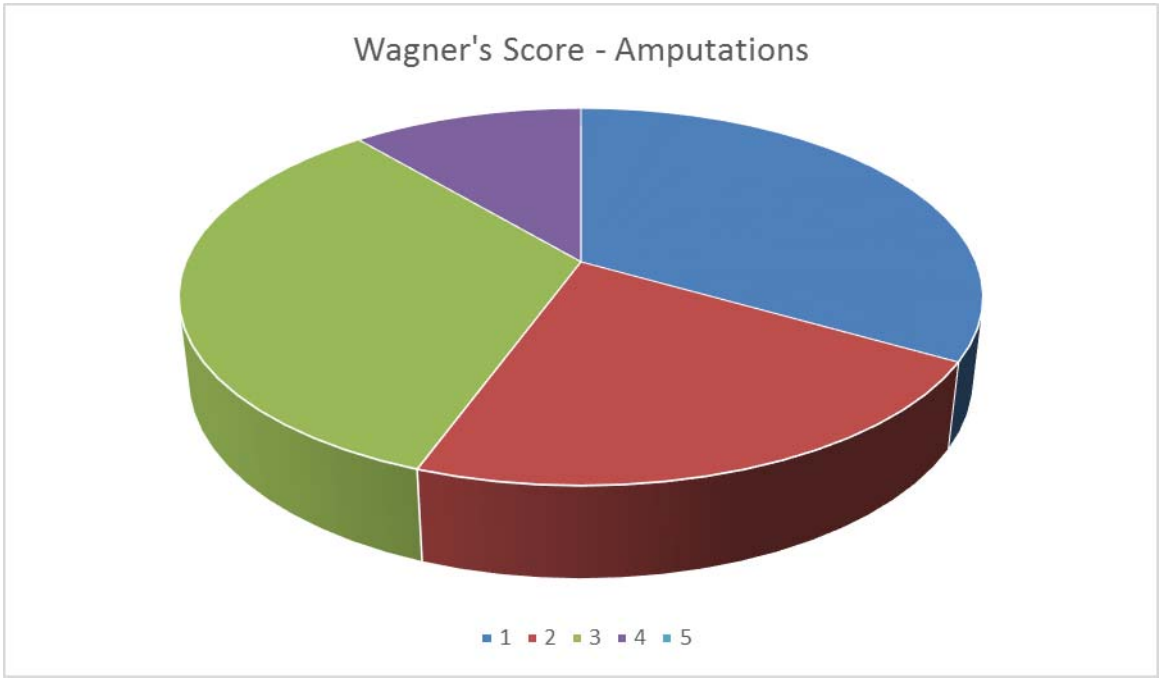

AGE DISTRIBUTION
<40
41-50
51-60
>60

Sex	Number	%
Male	33	66
Female	17	34



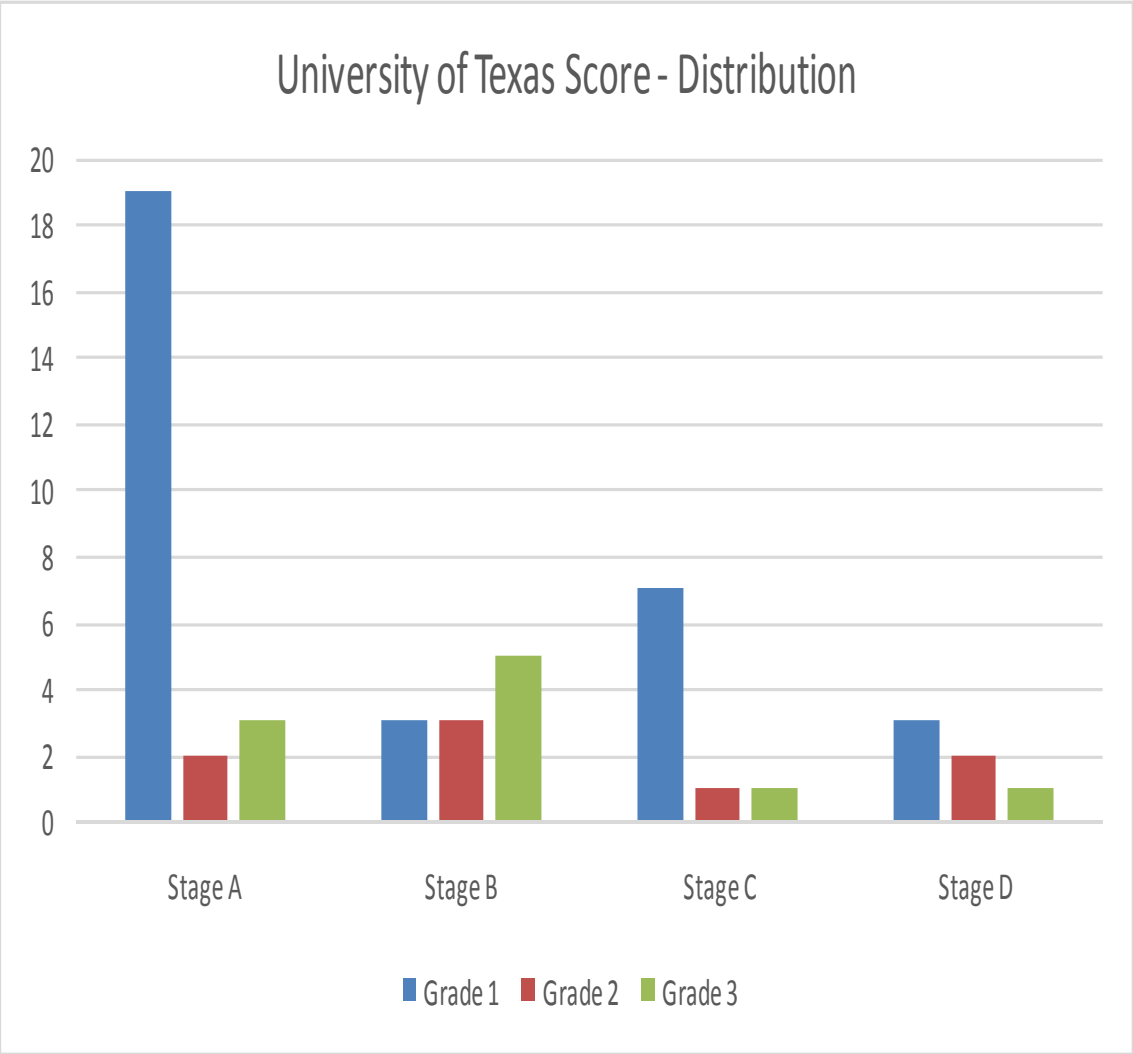
Characteristics	N**umber**	**%**
Type of diabetes		
Type I	0	0
Type II	50	100
Socioeconomic status		
Lower	7	14
Middle	30	60
Upper	13	26


Wagner's Score - distribution
1
2
3
4
5


Wagner's Score - Amputations
1
2
3
4
5

UNIVERSITY OF TEXAS CLASSIFICATION SYSTEM

Stage	Grade	n	No of Amputations
A	1	19	0
A	2	2	0
A	3	3	0
B	1	3	0
B	2	3	1
B	3	5	2
C	1	7	1
C	2	1	0
C	3	1	1
D	1	3	1
D	2	2	1
D	3	1	1


University of Texas Score - Distribution
20
18
16
14
12
10
8
6
4
2
0
Stage A
Stage B
Stage C
Stage D
Grade 1
Grade 2
Grade 3

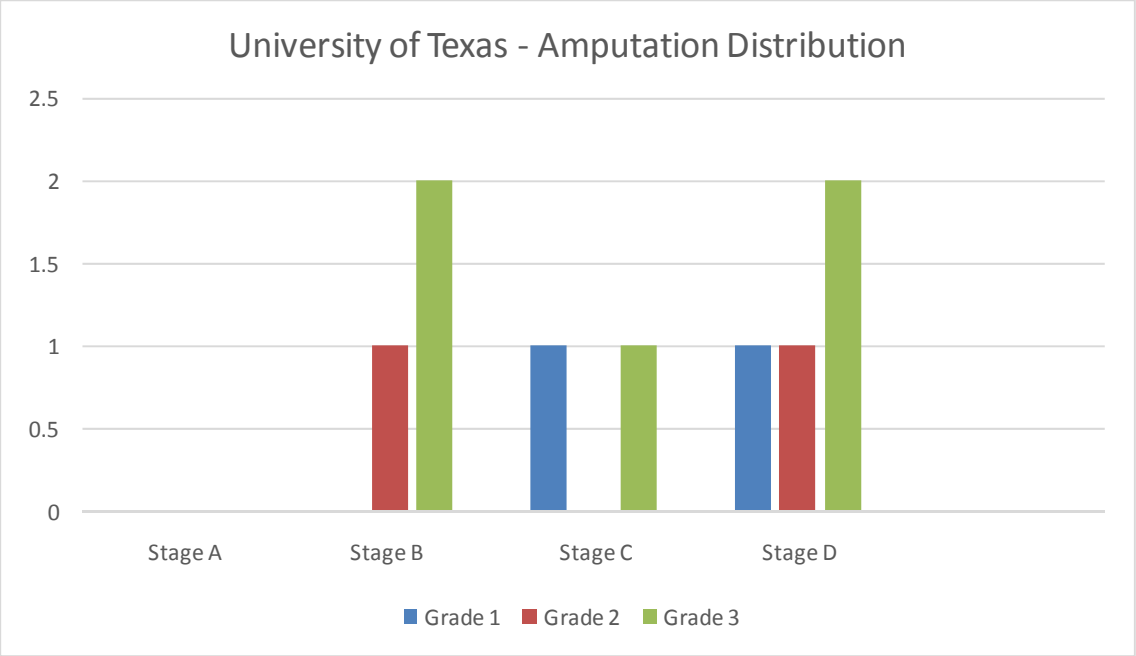

University of Texas - Amputation Distribution
2.5
2
1.5
1
0.5
0
Stage A
Stage B
Stage C
Stage D
Grade 1
Grade 2
Grade 3

Table 5- Culture report

Investigations	**No.of patients**	**%**
Culture		
Staph.aureus	11	22
Mixed	19	38
Others	20	40

Diabetes is associated with complications in its long run. Foot infection and subsequent amputation of a lower extremity are one of the most common reason for hospitalisation. As observed in our study, it is more common in males (n 33). More common age group is between 40-60 in our study. The hallmark of diabetic foot is its gross infection and major contributing factors for late presentation are poor knowledge about the disease, undetected diabetes, trust in faith healers, bare foot gait.

Peripheral neuropathy and infection are common risk factors diabetic foot. In our study mixred infection(n 19), includes aerobes, anaerobes, is common.

Four patients died of sepsis(n=3) and chronic renal failure(n=1)

Of all patients, 15% had lower-limb amputations as a result of their nonhealing ulcers, 65% had ulcers that healed completely, 4% (three patients) died, and the remaining 16% had ulcers that still had not healed at study termination, despite a minimum follow-up period of 6 months Wagner grade showed a significant positive trend with increased number of amputations (x2 trend = 21.0, P 0.0001). This was also true for both grade (x2 trend = 23.7, P 0.0001) and stage (x2 trend = 15.1, P = 0.0001) of the UT system. Using the UT stage, patients were 11 times more likely to undergo a lower-limb amputation if their ulcers were infected (stage B)

when compared with clean nonischemic ulcers (stage A) (27.5 vs. 3.3%, P 0.0001, OR = 11.1, 95% CI 3.0–41.0). Patients with noninfected ischemic ulcers (stage C) were five times more likely to undergo a lower-limb amputation when compared with stage A ulcers, but this did not reach statistical significance (13.6 vs. 3.3%, P = 0.09, OR = 4.6, 95% CI 0.9–24.7). However, when ischemic ulcers (with or without infection) were combined, patients with ischemic ulcers (stages C and D) were three times more likely to undergo amputation when compared with patients with nonischemic (stages A and B) ulcers (32.5 vs. 14.7%, P 0.05, OR = 2.8, 2 = 6.1, 95% CI 1.2–6.5). Patients with a combination of infection and ischemia (stage D) were 15 times more likely to undergo a lower-limb amputation when compared with patients with clean nonischemic ulcers (stage A) (33.3 vs. 3.3%, P 0.0001, x2 = 21.2, OR = 14.7, 95% CI 3.7–58.2). Grade for the Wagner (r = 0.26, P 0.01) and UT (r = 0.26, P 0.01) systems both showed a weak positive correlation with ulcer healing time for the 65% of patients whose ulcers healed completely, but stage did not (r = 0.06, P = 0.48).

Kaplan-Meier survival analysis showed no significant difference between median healing times in grades 1, 2, and 3 of the Wagner system (8, 16, and 11 weeks, respectively) ($x^2 = 5.68$, $df = 3$, $P = 0.13$) or median healing times in grades 1, 2, and 3 of the UT system (8, 12 and 16 weeks, respectively) ($x^2 = 5.47$, $df = 2$, $P = 0.07$). However, analysis showed that the median healing times (7, 11, 16, and 20 weeks) increased with each stage of the UT system ($x^2 = 10.24$, $df = 3$, $P = 0.02$).

DISCUSSION

DISCUSSION

Data on the burden of diabetes-related complications from developing countries are relatively rare and comparisons between them are made difficult by differing degrees of population selection and by the use of different clinical methods (23). Despite a number of local initiatives to improve access to foot care, the vast majority of people with diabetes in developing countries do not have access The report by the Centers for Disease Control and Prevention (CDC) on geographic disparities in diabetes-related amputations on the Texas–Mexico border reported that incidence of diabetes-related amputations of lower extremity in this area was nearly double the rate of non-border countries (25). Wide differences between other centres have also been reported (10). In order to identify the reasons for such differences, it is necessary to compare the outcomes of clinical care in different populations, both between centres and between countries, and this requires careful definition of the populations selected for the study.

“Wagner’s Classification for diabetic foot disease (adopted from Levin and O’Neals)”

Grade	**Description**
Grade 0	High risk foot and no ulceration
Grade 1	Superficial Ulcer; Total destruction of the thickness of the skin
Grade 2	Deep Ulcer (cellulitis); Penetrates through skin,fat,ligaments not affecting bone
Grade 3	Osteomyelitis with Ulceration or abscess
Grade 4	Gangrenous patches limited to toes or part of the foot
Grade 5	Gangrene of the entire foot

A robust system of ulcer classification is necessary for this purpose. A number of groups have used classification schemes to seek associations between baseline variables and clinical outcome, but the results have been inconsistent. Armstrong and colleagues (16) reported a significant association between outcome and ulcers of increasing depth (UT grade), and especially in the presence of ischaemia, infection or both (UT stage). This study was limited, however, in using just a linear-by-linear association for analysis, and by the use of amputation (which should properly be regarded as a treatment rather than a clinical endpoint), as the single outcome measure. This could have influenced the results if, for instance, amputation was established as the treatment of choice for certain types of lesion, such as osteomyelitis (10).

A close association was later shown between the results of the UT system and the earlier Wagner classification (26). In contrast to the report by Armstrong *et al.*(16), however, Treece and colleagues (18) found no association between infection and any outcome measure (healing, non-healing, amputation, death), although differences were observed between ulcer area, depth and the presence or not of peripheral arterial disease (PAD). The same group has recently confirmed these findings, reporting that the dominant factors influencing healing in a UK population were ulcer area and the presence of ischaemia (27).

Few longitudinal studies have assessed the power of a foot ulcer classification system in predicting clinical outcome. The results of the study revealed that grade and stage affect the outcome of diabetic foot ulcers. The higher the grade, the greater the number of amputations performed. The trend for the UT grade was slightly greater than that for the Wagner grade. As for stage, the presence of infection and/or ischemia increased the risk of amputation. Because of small numbers of patients in each group, the increased amputation risk seen with stage C did not reach statistical significance, but when regrouped, patients with ischemia (stages C and D) had higher risk of amputation compared with patients without ischemia (stages A and B). Previous studies have shown that infection and peripheral vascular disease are associated with an increased risk of amputation (26,27). In addition, only stage both showed a positive relationship with time to healing and predicted healing within the study period. It should be noted, however, that grading and staging were done at presentation only. Some patients may have had recurrent wound infection, which would prolong wound healing, and a few patients had revascularization procedures, which enhance wound healing.

The HbA1c level was not measured for all patients at presentation or at the same time point and therefore was not used for analysis. Additionally, only 6% of patients underwent revascularization before the end of the study. These confounding factors may have altered or undermined the expected effects of grade and stage at baseline on amputation rates and healing time Wagner grade 4 and 5 ulcers were poorly represented in this study group, making it impossible to say if grades 4 and 5 add extra predictive power to the wound classification system. Gangrene is present in grades 4 and 5 and is usually due to a combination of ischemia and infection; these grades will, in most cases, have a similar outcome. Further studies are necessary to compare clinical outcomes of Wagner grade 4 and 5 ulcers with that of UT grade 3, stage D—an argument that makes the UT system appear simpler and more practical. An infected ischemic ulcer that penetrates to tendon (grade 2, stage D, or, simply, grade 2D of the UT system) alternatively will be grade 2 of the Wagner system. A labeling of grade 2 of the Wagner system thus will not alert other members of the foot care team of the presence of infection and ischemia, which can prolong wound healing and increase the risk of lower-limb amputation. The addition of stage to grade improves the descriptive and predictive power of a wound classification system, especially for ulcers within the same grade.

The UT system, which combines grade and stage, is more descriptive and shows a greater association with increased risk of amputation and prediction of ulcer healing when compared with the Wagner system. Therefore, for groups rather than individual patients, the UT system, which is simple and easy to use, is a better predictor of clinical outcome.,

CONCLUSION

CONCLUSION

Diabetic foot and its complications are troublesome, source consuming and producing disability, morbidity and mortality. — Increasing stage, regardless of grade, is associated with increased risk of amputation and prolonged ulcer healing time. The UT system's inclusion of stage makes it a better predictor of outcome

Prevention is the best treatment

Grading of the diabetec foot lesions according to Wagner's classification helps in choosing appropriate treatment to the grade. Patient education and strict glycemic control can reduce the burden of diabetic foot. Early diagnosis and hospitalization, appropriate treatment including medical and surgical treatment according to the grade can reduce the morbidity mortality and improve the outcome of the disease

Key to master chart

Sex – M-Male; F-female

Type - type of diabetes – I;II

Culture- Mx-mixed organisms; S- stap.aureus alone

"Wagner's Classification for diabetic foot disease (adopted from Levin and O'Neals)"-

Grade	**Description**
Grade 0	High risk foot and no ulceration
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Grade 4	Gangrenous patches limited to toes or part of the foot
Grade 5	Gangrene of the entire foot

Treatment- A-antibiotics;Amp-amputation (of any type);D-debridment;I&D- incision and drainage

Rft- renal function test ; Ab-abnormal; N-normal

Mortality- yes;no Cause- Sep-Sepsis; DKA-Diabetic Ketoacidosis; CRF- Chronic Renal Failure

Table 6 **University of Texas Classification System**

Stage	Grade			
	O	I	II	III
A	Pre- or post-ulcerative lesions completely epithelized	Superficial wound not involving tendon, capsule, or bone	Wound penetrating to tendon or capsule	Wound penetrating to bone or joint
B	Infected	Infected	Infected	Infected
C	Ischemic	Ischemic	Ischemic	Ischemic
D	Infected and ischemic	Infected and ischemic	Infected and ischemic	Infected and ischemic

DATA COLLECTION SHEET

I.Patient particulars:

Name	DOA	Case No.
Age	DOS	I.p.No.
Sex	DOD	Address

Occupation:

II.Diagnosis

III.Chief complaints (with duration)

A.Ulcer

B.Discharge

C.Other complaints

PAST HISTORY:

HISTORY OF PREVIOUS OPERATION -

DURATION OF DIABETES -

OTHER COMPLICATIONS OF DIABETES

PERSONAL HISTORY:

EXAMINATION:

INVESTIGATIONS:

MANAGEMENT:

Operated /Non operated-

POST OPERATIVE COURSE:

Recovery -

Complications -

FOLLOW UP:

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Master chart

S.No	Name	Age	Sex	Type	Culture	Wagner Score	Univ of Texas Score	Treatment	RFT	Mortality/ Cause
1	RAVI	40	M	2	MX	1	A1	DEB	N	NO
2	RAJESH	56	M	2	S	2	B2	DEB	N	NO
3	ELUMALAI	67	M	2		3	B3	AMP	N	NO
4	MURUGAN	45	M	2		2	D2	DEB	N	NO
5	GOBINATH	55	M	2	MX	3	B3	DEB	N	NO
6	NAGENDRAN	51	M	2		1	D1	DEB	N	NO
7	MARIMUTHU	47	M	2	S	1	D1	DEB	N	NO
8	NATARJAN	49	M	2	S	1	B1	DEB	N	NO
9	JOSEPH	76	M	2	S	4	D3	AMP	A	Yes/Sepsis
10	GABRIEL	65	M	2		3	B3	DEB	N	NO
11	MOOSA	44	M	2	MX	1	C1	AMP	N	NO
12	MOHAMMED	54	M	2	MX	1	B1	DEB	N	NO
13	FAROOQ	48	M	2	MX	1	B1	DEB	N	NO
14	GOPALAN	43	M	2		2	B2	DEB	N	NO
15	RAJARAJAN	55	M	2	MX	1	C1	DEB	N	NO
16	RAJENDRAN	58	M	2	S	3	A3	DEB	N	NO
17	KANGEYAN	41	M	2	MX	1	C1	DEB	N	NO
18	KESAVAN	42	M	2		2	C2	A	N	NO
19	KATHIRAVAN	52	M	2	S	1	A1	DEB	N	NO
20	IYYAPAN	51	M	2	MX	1	C1	DEB	N	NO
21	SILAMBARASAN	60	M	2	MX	3	B3	DEB	N	NO
22	MAARI	46	M	2		1	C1	DEB	N	NO
23	SONAMUTHU	44	M	2	S	1	A1	DEB	N	NO
24	MALAIYANDI	39	M	2		1	C1	A	N	NO
25	MARUDHAMALAI	65	M	2		3	A3	DEB	N	NO
26	UTHIRAKUMAR	59	M	2	MX	3	B3	AMP	A	YES/SEPSIS

27	THIRUSELVAN	55	M	2		2	B2	AMP	A	NO
28	KUBERAN	44	M	2		1	A1	A	N	NO
29	MADHAN	49	M	2	MX	2	A2	DEB	N	NO
30	SATHYAMOORTHY	48	M	2		1	A1	A	N	NO
31	ROSAIYA	55	M	2		1	C1	DEB	N	NO
32	KULASEKARAN	58	M	2	MX	3	A3	DEB	N	NO
33	VEERAIYAN	43	M	2		1	A1	A	N	NO
34	RAJALAKSHMI	56	F	2		1	A1	DEB	N	NO
35	ESWARI	67	F	2	MX	3	C3	AMP	N	YES/CRF
36	RAJI	45	F	2	S	1	A1	A	N	NO
37	SULTHANA	56	F	2		2	A2	DEB	N	NO
38	MARIYAMMAL	54	F	2	MX	1	A1	A	N	NO
39	ELLAMAL	43	F	2	MX	1	A1	DEB	N	NO
40	ANJALAI	41	F	2		1	A1	A	N	NO
41	MARY	65	F	2	MX	2	D2	AMP	N	NO
42	LAKSHMI	78	F	2	S	1	B1	AMP	A	NO
43	NACHIYAR	55	F	2		1	A1	A	N	NO
44	RAKKAMAL	54	F	2	MX	1	A1	DEB	N	NO
45	ROSY	46	F	2	S	1	A1	A	N	NO
46	PARAMESHWARI	55	F	2		1	A1	DEB	N	NO
47	SUNDARI	43	F	2	S	1	A1	A	N	NO
48	VISALATCHI	45	F	2		1	A1	DEB	N	NO
49	MURUGESHWARI	56	F	2	MX	1	A1	DEB	N	NO
50	REKHA	77	F		MX	1	D1	AMP	A	YES/SEPSIS

Key to master chart

Sex – M-Male; F-female

Type - type of diabetes – I;II

Culture- Mx-mixed organisms; S- stap.aureus alone

"Wagner's Classification for diabetic foot disease (adopted from Levin and O'Neals)"-

Grade	**Description**
Grade 0	High risk foot and no ulceration
Grade 1	Superficial Ulcer; Total destruction of the thickness of the skin
Grade 2	Deep Ulcer (cellulitis); Penetrates through skin,fat,ligaments not affecting bone
Grade 3	Osteomyelitis with Ulceration or abscess
Grade 4	Gangrenous patches limited to toes or part of the foot
Grade 5	Gangrene of the entire foot

Treatment- A-antibiotics;Amp-amputation (of any type);D-debridment;I&D- incision and drainage

Rft- renal function test ; Ab-abnormal; N-normal

Mortality- yes;no Cause- Sep-Sepsis; DKA-Diabetic Ketoacidosis; CRF- Chronic Renal Failure

Table 6 **University of Texas Classification System**

Stage	Grade			
	O	I	II	III
A	Pre- or post-ulcerative lesions completely epithelized	Superficial wound not involving tendon, capsule, or bone	Wound penetrating to tendon or capsule	Wound penetrating to bone or joint
B	Infected	Infected	Infected	Infected
C	Ischemic	Ischemic	Ischemic	Ischemic
D	Infected and ischemic	Infected and ischemic	Infected and ischemic	Infected and ischemic