`A DISSERTATION ON

"EVALUATION AND MANAGEMENT OF DIABETIC FOOT ACCORDING TO WAGNER'S CLASSIFICATION AT RGGGH"

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BONAFIDE CERTIFICATE

Certified that this dissertation is the bonafide work of **Dr. J.ANAND PRASATH** on **"EVALUATION AND MANAGEMENT OF DIABETIC FOOT ACCORDING TO WAGNER'S CLASSIFICATION AT RGGGH"** during his M.S. (General Surgery) course from July 2015 to September 2015 at the Madras Medical College and Rajiv Gandhi Government General Hospital, Chennai – 600003.

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DECLARATION

I, certainly declare that this dissertation titled, **"EVALUATION AND MANAGEMENT OF DIABETIC FOOT ACCORDING TO WAGNER'S CLASSIFICATION AT RGGGH",** represent a genuine work of mine. The contribution of any supervisors to the research are consistent 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 fulfillment of the rules and regulation for the award of Master of Surgery Degree Branch 1 (General Surgery).

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Date :
Place:

ABSTRACT

BACKGROUND AND OBJECTIVE

Diabetes is one of the most common co-morbid illness in our community.

One of its complication in long course is diabetic foot. Morbidity and mortality due to this complication is a major health issue. This study is aimed to evaluate and manage the different lesions of diabetic foot according to Wagner classification.

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 2015 and September 2015, 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. Data was collected and analyzed.

RESULTS

Majority of the patients presented with higher grade and with poor glycemic control at the time of presentation. Conservative

management with antibiotics was useful in a small subset of the patients. Majority of the patients needed surgical treatment in the form of debridement to amputations.

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.

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

CONTENTS

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; neuropathy; and nephropathy. Currently, cardiovascular complications 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 4A

“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 Indian 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. Indian series."

"Majority of Indian 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."

OBJECTIVES

The purpose of this dissertation is to evaluate and manage diabetic foot according to Wagner's classification at Institute of General surgery, Rajiv Gandhi Government General Hospital, Chennai.

The study period is between July 2015 to September 2015.

1) To evaluate and manage the different lesions of diabetic foot according to Wagner classification.

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

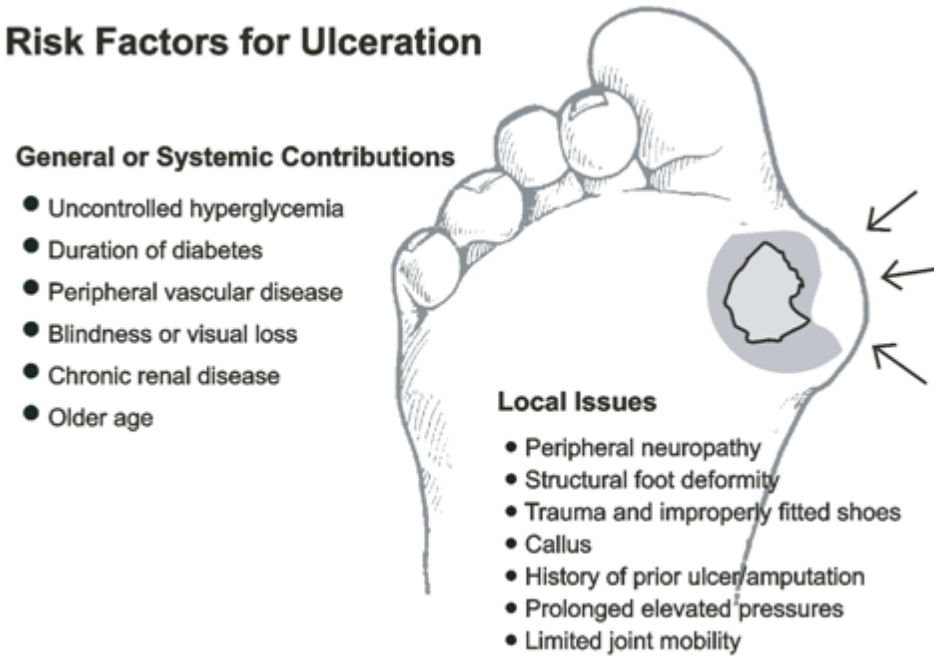
REVIEW OF LITERATURE

"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, 93, 100).

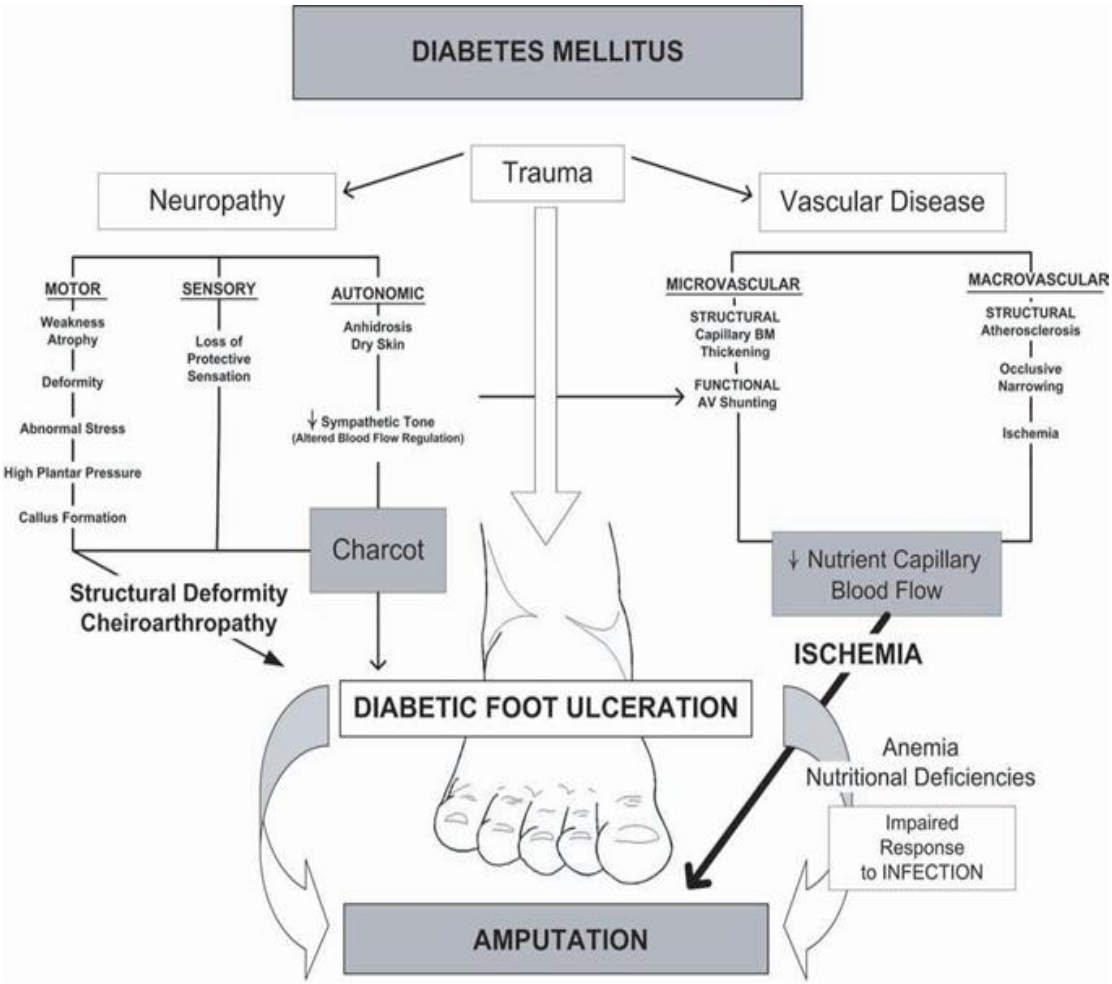


Figure 2 Diabetes mellitus is responsible for a variety of foot pathologies contributing to the complications of ulceration and amputation. Multiple pathologies may be implicated, from vascular disease to neuropathy tomechanical trauma.

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).”

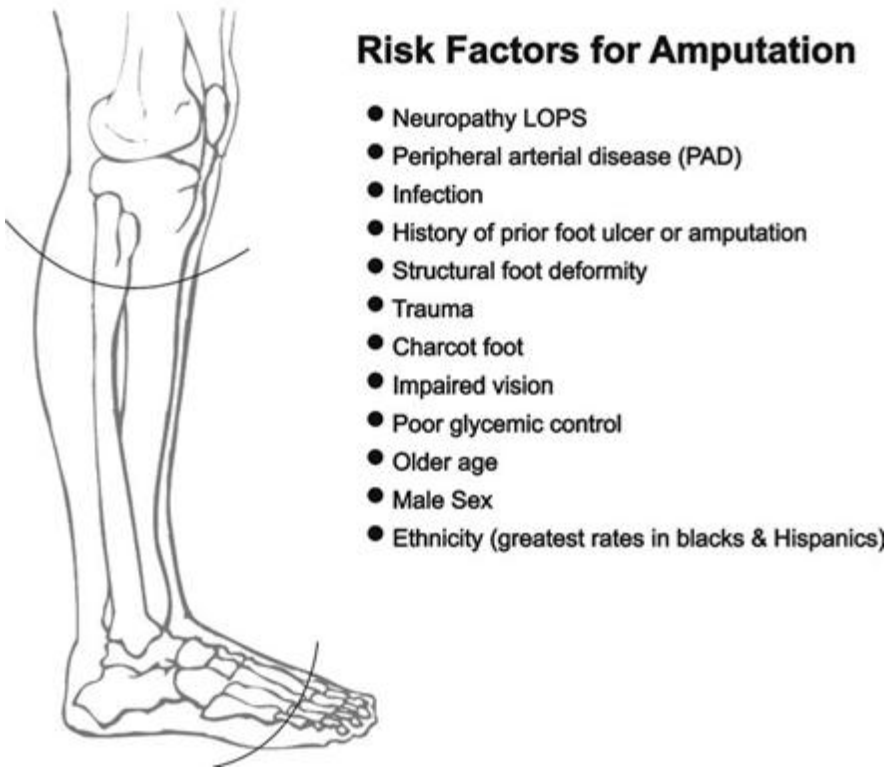
RISK FOR AMPUTATION

“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.



"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 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 section, it is assumed that a general medical assessment (including vital sign measurements) will be obtained."

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

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 neuro- pathic 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 further 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 non healing 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 predicting successful wound healing (213, 218, 219). $TcPO_2$ measurements have received similar support in the literature (47, 87, 212). Although not consistently predictive of wound healing outcomes, these physiologic measures of tissue 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 assessment for potential revascularization (227-229). Magnetic resonance angiography (230) or CT angiogram are alternatives 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 history 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 biothesiometer 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 neuropathic 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 possibly in evaluating orthotic adjustments (57, 59), they are primarily 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 vulnerable 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, clinical 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, noninvasive 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			
	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

Figure Assessment of a diabetic foot ulcer includes not only a description of the skin lesion but also the find- ings necessary for accu- rate 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 cornerstone 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

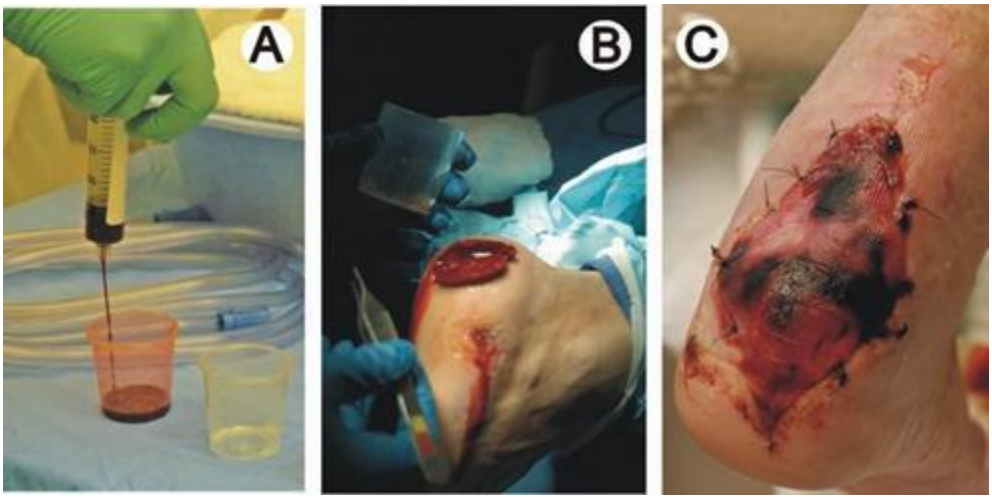


Figure **"**New technologies have been developed that have proved useful for management of diabetic ulcerations. *(A)*Platelet-rich plasma (PRP) involves use of the patient's blood, which is collected and then fractionated through centrifuga- tion. A platelet-rich and platelet-poor supernatant remains. *(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."

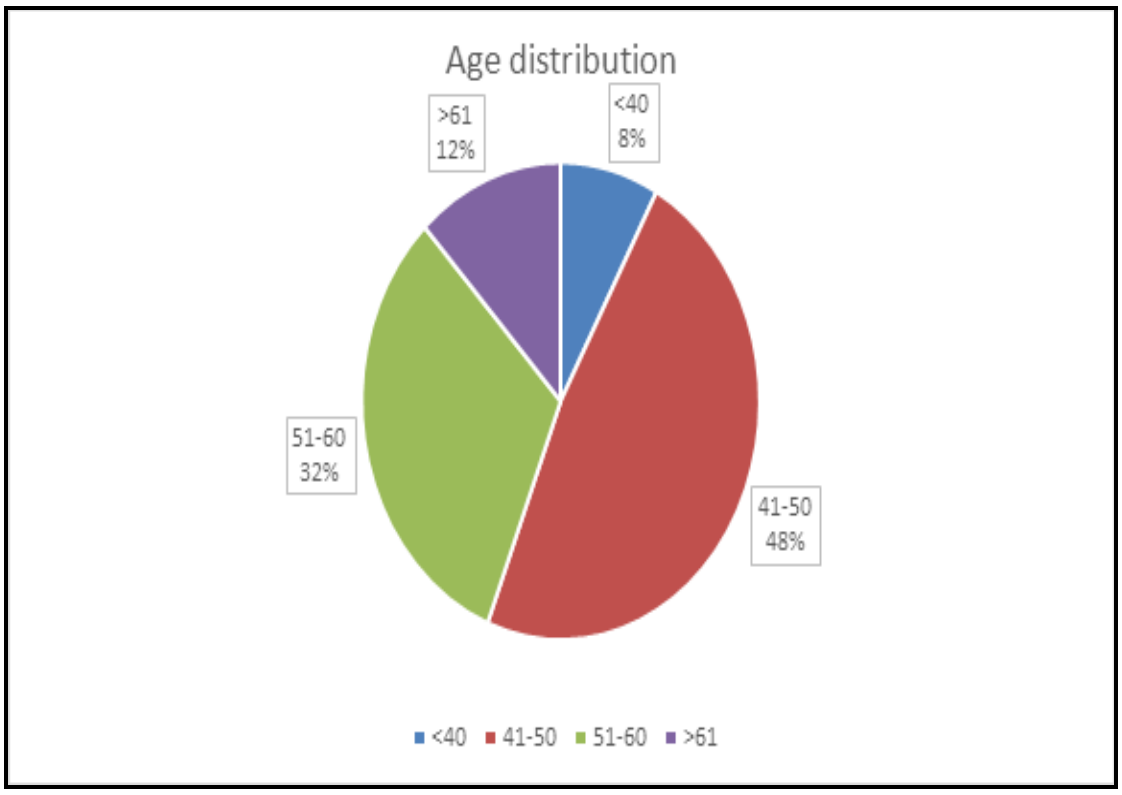
METHODOLOGY AND MATERIALS

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 2015 to September 2015. 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, examination findings, blood investigations, renal function test, swab of the wound. X-ray and treatment provided were collected. Treatment was carried out in both medical and surgical means. Antibiotics – aminoglycosides, cephalosporins, penicillin derivatives were used.

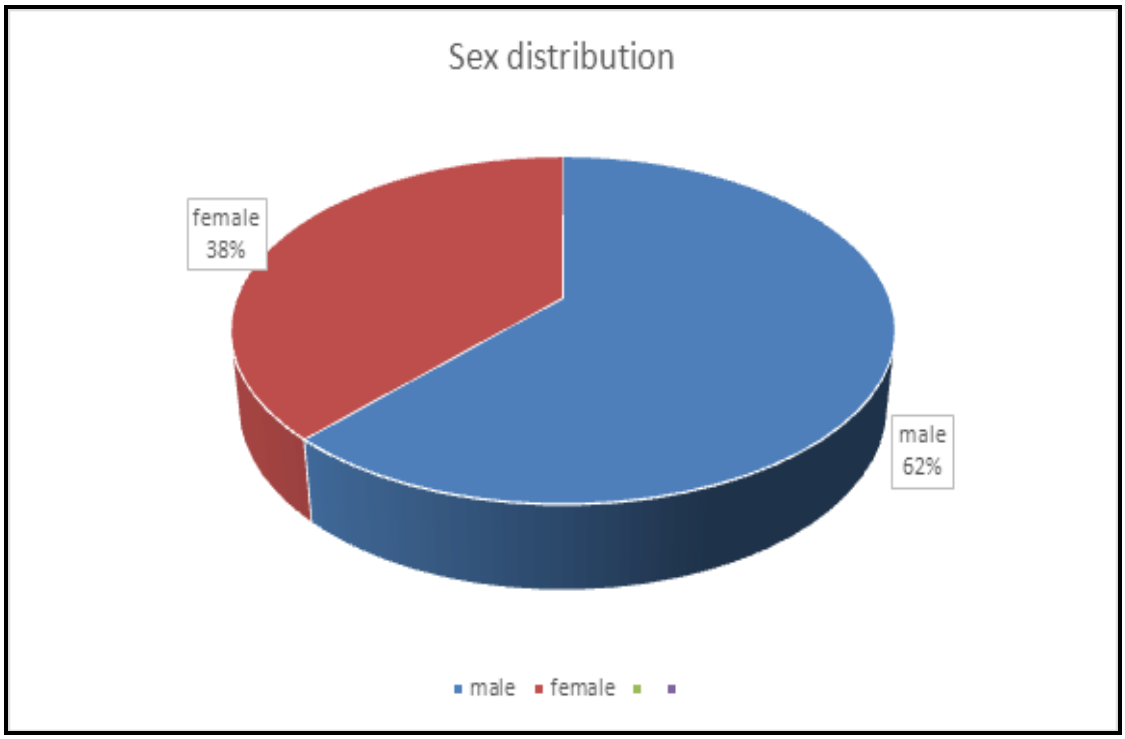
RESULTS

Table-1: Sociodemographic characteristics of the patients

Characteristics	Number	%age
Age/years		
<40	4	8
41-50	24	48
51-60	16	32
>60	6	12



Sex	Number	%
Male	31	62
Female	19	38



Characteristics	Number	%
Type of diabetes		
Type I	1	2
Type II	49	98
Socioeconomic status		
Lower	7	14
Middle	30	60
Upper	13	26

Table 2- Number of patients according to Wagner's classification (n=50)

Grade	No.of Patients	%
0	3	6
1	7	14
2	12	24
3	15	30
4	11	22
5	2	4

Distribution according to Wagner's Classification

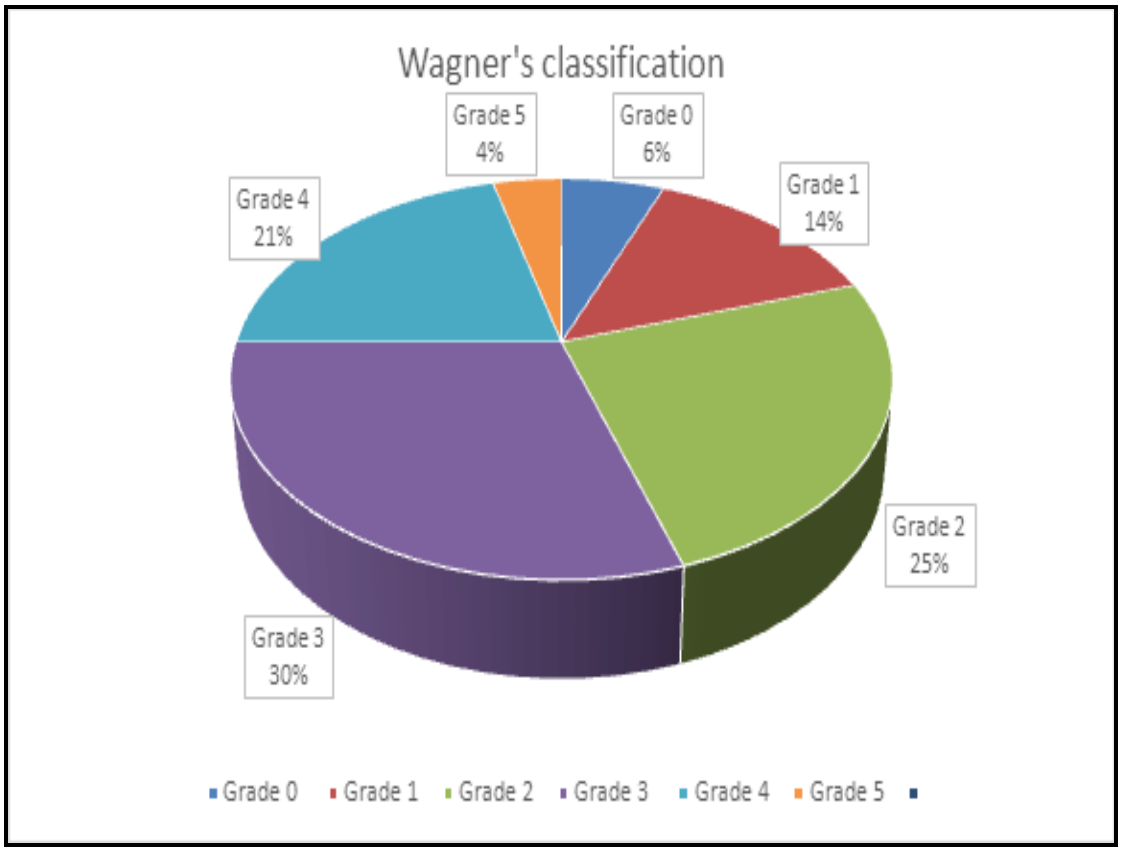
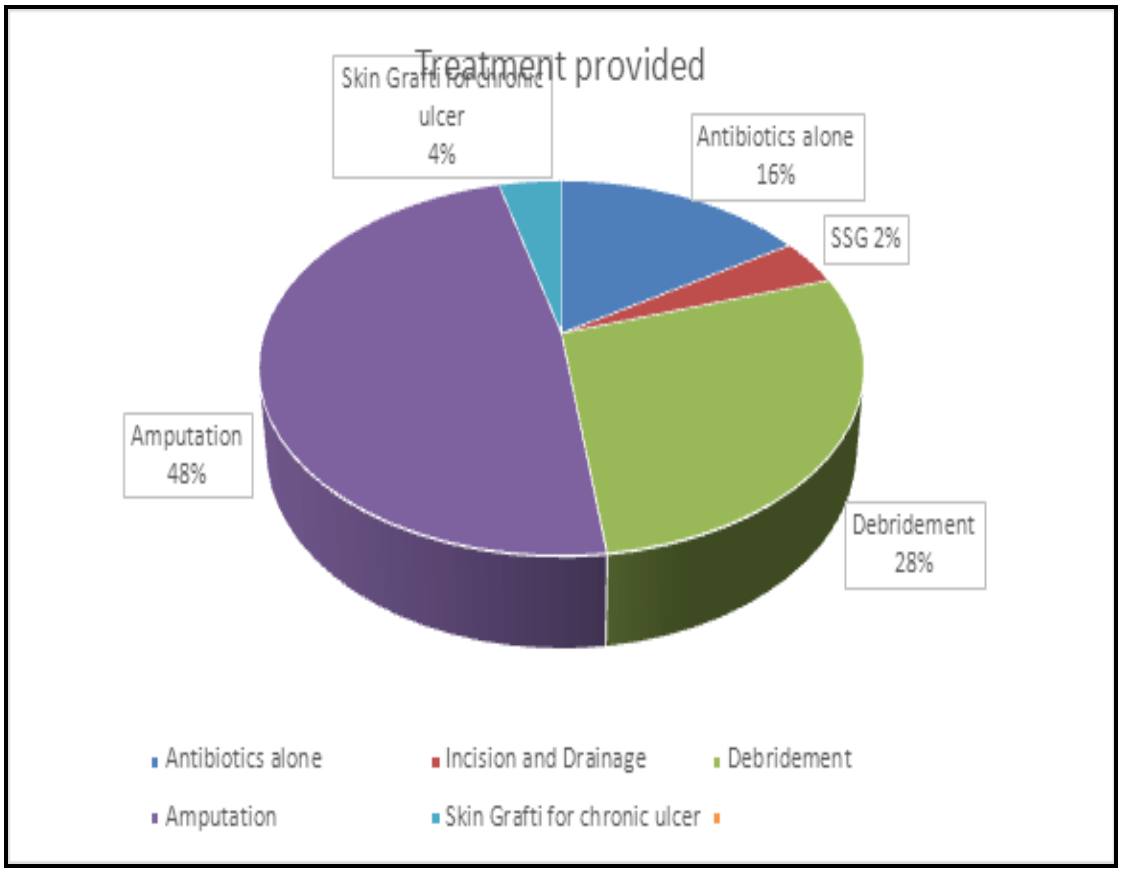


Table 3- Treatment provided

Type of Treatment	No. of Patients	%
Antibiotics alone	8	16
Incision and drainage	2	4
Debridement	14	28
Amputation	25	50
Skin Graft for chronic ulcer	1	2



Type of amputation	No.of patient
Rye's/toe	16
Syme's	4
Below knee	5

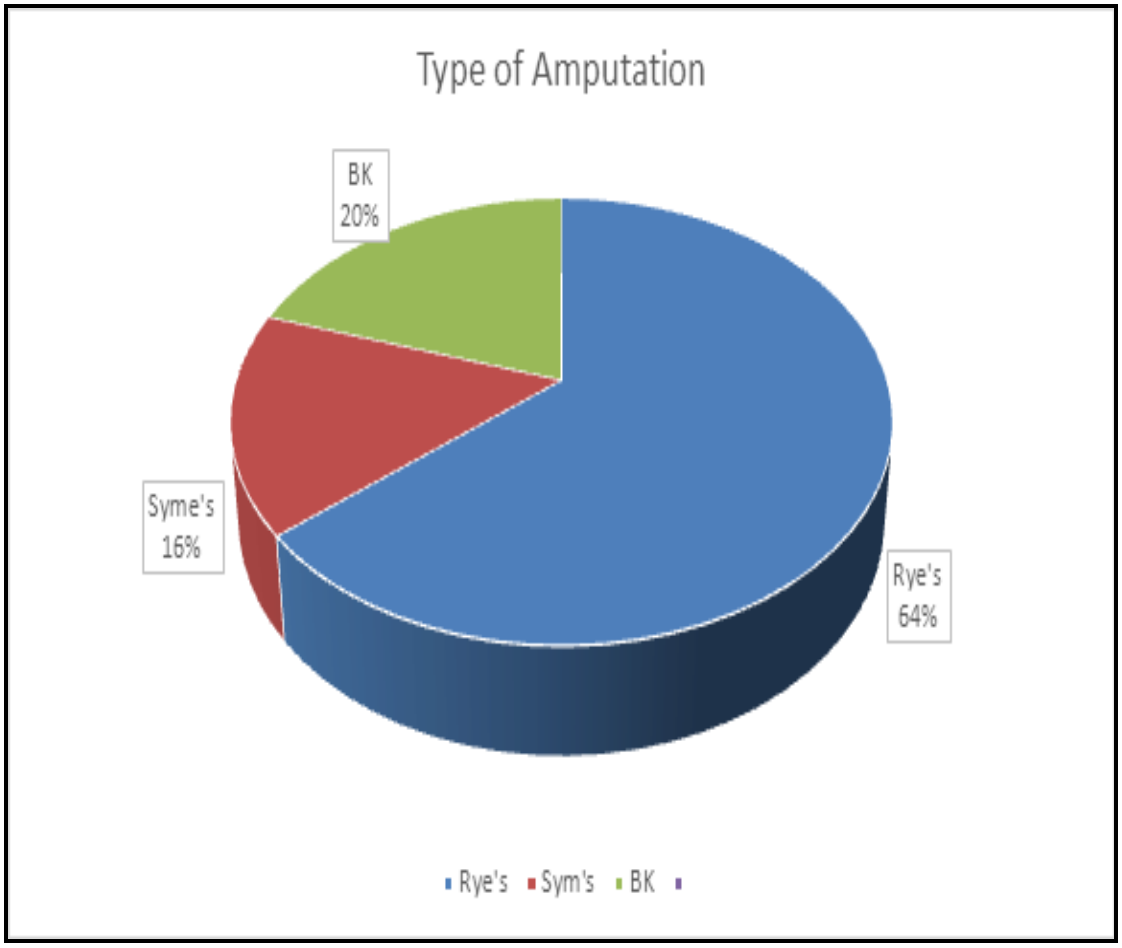


Table 4– Cause of Mortality in Diabetic foot disease (n=3)

Cause	Number of patients
Septicaemia	2
Ketoacidosis	1
Chronic renal failure	1

Table 5- Culture report

Investigations	No.of patients	%
Culture		
Staph.aureus Isolated	13	26
Mixed	25	50

From the above observed data , most of the patients presented with advanced grade, grade 2 – 24%, grade 3-30%, grade 4 – 22%. Henceforth surgical management was required in most of the patients. Amputation in half of the Ptients and debridement in 28% of patients highlighting the advanced disease at presentation.

Wagner's classification may be different for a surgeon as compared to physician because patients come to a surgeon with advanced disease hence the greater grade of patients were in our study in more percentage.

DISCUSSION

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. 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, includes aerobes, anaerobes, is common

The standard treatment for diabetic foot according to Wagner's classification is

1. Grade 0 - Prevention

2. Grade 1 - Antibiotics and good glycemic control

3. Grade 2 – hospitalisation as they need surgical management alonmg with antibiotics and glycemic control

4. Grade 3 – requires some sort of amputation

5. Grade 4 – wide debridement along with amputation

6. Grade 5 – preferred treatment is below knee amputation

There were 4 mortalities in our study, al had high Wagner's score.of these 2 was due to septicemia, 1 due to ketoacidosis, 1 due to chronic renal failure.

Prevention strategy including patient education in foot care, prophylactic skin and nail care and foot wear reduces the risk of foot ulceration and amputation rates.

CONCLUSION

Diabetic foot and its complications are troublesome, source consuming and producing disability, morbidity and mortality.

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

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PATIENT CONSENT FORM

STUDY TITLE: "EVALUATION AND MANAGEMENT OF DIABETIC FOOT ACCORDING TO WAGNER'S CLASSIFICATION"

STUDY CENTRE

Rajiv Gandhi Government General hospital and Madras Medical College.

PARTICIPANT NAME: AGE: SEX:
I.P. NO :

I confirm that I have understood the purpose of treatment and procedure for the above study. I have the opportunity to ask the question and all my questions and doubts have been answered to my satisfaction.

I have been explained about the possible complications that may occur during the interventional procedure. I understand that my participation in the study is voluntary and that I am free to withdraw at any time without giving any reason.

I understand that the investigator, regulatory authorities and the ethics committee will not need my permission to look at my health records both in respect to the current study and any further research that may be conducted in relation to it, even if I withdraw from the study. I understand that my identity will not be revealed in any information released to third parties or published, unless as required under the law. I agree not to restrict the use of any data or results that arise from the study.

I hereby consent to participate in this study of the "EVALUATION AND MANAGEMENT OF DIABETIC FOOT ACCORDING TO WAGNER'S CLASSIFICATION"

Date: signature / thumb impression of patient

Place:

Patient's name:

Signature of the Investigator: _____

Name of the investigator:

INFORMATION SHEET

We are conducting a study on **"EVALUATION AND MANAGEMENT OF DIABETIC FOOT ACCORDING TO WAGNER'S CLASSIFICATION"** among patients attending Rajiv Gandhi Government General Hospital, Chennai and for that your information is valuable to us.

The purpose of this study is to assess the magnitude of problem and evaluate and manage the different lesions of diabetic foot according to Wagner classification at RGGGH,Chennai.

We are selecting certain cases and if you are found eligible, we may be using your information which in any way do not affect your final report or management.

The privacy of the patients in the research will be maintained throughout the study. In the event of any publication or presentation resulting from the research, no personally identifiable information will be shared.

Taking part in this study is voluntary. You are free to decide whether to participate in this study or to withdraw at any time; your decision will not result in any loss of benefits to which you are otherwise entitled.

The results of the special study may be intimated to you at the end of the study period or during the study if anything is found abnormal which may aid in the management or treatment.

Signature of the Participant

Signature of the Investigator

Date

Place

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:

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

MASTER CHART

S.No	Name	Age	Sex	Type	Culture	Wagner's	Treatment	Rft	Mortality/Cause
1	Elumalai	50	M	II	Mx	3	D	N	No
2	Murugan	52	M	II		1	A	N	No
3	Murugesan	47	M	II	S	2	D	N	No
4	Marimuthu	65	M	II	Mx	4	Amp	Ab	Yes/Sep
5	Natesan	43	M	II		2	D	N	No
6	Nagendran	44	M	II		1	I&D	N	No
7	Natarajan	46	M	II	Mx	3	Amp	Ab	No
8	Muthu	42	M	II	Mx	3	Amp	N	No
9	Rajesh	37	M	II	Mx	4	Amp	N	No
10	Raju	36	M	I		0	A	N	No
11	Ravi	54	M	II	Mx	3	Amp	N	No
12	Mahesh	52	M	II	S	3	Amp	N	No
13	Mani	51	M	II	Mx	3	Amp	N	No
14	Manikandan	47	M	II	S	2	D	N	No
15	George	68	M	II	Mx	3	D	Ab	Yes/CRF
16	Christian	47	M	II	Mx	2	D	N	No
17	Nagesh	56	M	II	S	1	I&D	Ab	No
18	Karthik	39	M	II		0	A	N	No
19	Balaji	57	M	II	Mx	3	Amp	Ab	No
20	Balamurugan	59	M	II	Mx	4	Amp	Ab	No
21	Subramani	58	M	II	S	4	Amp	N	No
22	Silambarasan	47	M	II		1	Ssg	N	No
23	Arasu	46	M	II	Mx	3	Amp	N	No
24	Arapuli	65	M	II	S	2	D	N	No
25	Kangeyan	61	M	II	Mx	5	Amp	Ab	Yes/Sep
26	Dharmalingam	48	M	II	Mx	3	Amp	N	No
27	Kesavan	56	M	II	Mx	4	Amp	N	No

S.No	Name	Age	Sex	Type	Culture	Wagner's	Treatment	Rft	Mortality/Cause
28	Gajendran	49	M	II		0	A	N	NO
29	Iqbal	52	M	II	S	2	D	N	No
30	Ismail	47	M	II	Mx	4	Amp	N	No
31	Mohammed	57	M	II	Mx	3	Amp	N	No
32	Rajeshwari	58	F	II	S	2	D	N	No
33	Lakshmi	46	F	II	Mx	3	Amp	Ab	No
34	Mahalaksmi	47	F	II	S	2	D	N	No
35	Muniyammal	48	F	II	Mx	4	Amp	Ab	No
36	Ellamal	49	F	II	S	2	D	N	No
37	Muthulakshmi	39	F	II		1	A	N	No
38	Pothumpon	48	F	II	Mx	4	Amp	N	No
39	Shakthi	49	F	II	S	3	Amp	N	No
40	Mani	47	F	II		1	A	N	No
41	Maheshwari	46	F	II	Mx	4	Amp	N	No
42	Eshwari	47	F	II	Mx	4	Amp	N	No
43	Sulthana	53	F	II	Mx	3	Amp	N	No
44	Indira	56	F	II		2	D	N	No
45	Raji	54	F	II		2	A	N	No
46	Anjalai	55	F	II	Mx	4	Amp	N	No
47	Nageshwari	47	F	II	S	2	D	N	No
48	Patchaiyammal	70	F	II	Mx	5	Amp	Ab	Yes/DKA
49	Murugeshwari	47	F	II		1	A	N	No
50	Rekha	50	F	II	S	3	D	N	No

INSTITUTIONAL ETHICS COMMITTEE
MADRAS MEDICAL COLLEGE, CHENNAI-3

EC Reg No.ECR/270/Inst./TN/2013
Telephone No. 044 25305301
Fax : 044 25363970

CERTIFICATE OF APPROVAL

To
Dr.J.Anand Prasath
Postgraduate M.S.(General Surgery)
Madras Medical College
Chennai 600 003

Dear Dr.J.Anand Prasath,

The Institutional Ethics Committee has considered your request and approved your study titled **"Evaluation and Management of Diabetic Foot according to Wagner's Score" No.29072015.**

The following members of Ethics Committee were present in the meeting held on 07.07.2015 conducted at Madras Medical College, Chennai-3.

1. Prof.C.Rajendran, M.D.,	: Chairperson
2. Prof.R.Vimala, M.D., Dean, MMC, Ch-3	: Deputy Chairperson
3. Prof.Sudha Seshayyan, M.D., Vice-Principal, MMC, Ch-3	: Member Secretary
4. Prof.B.Vasanthi, M.D., Professor Pharmacology, MMC	: Member
5. Prof.P.Ragumani, M.S., Professor, Inst.of Surgery, MMC	: Member
6. Prof.Md.Ali, M.D., D.M., Prof. & HOD of Medl.G.E., MMC	: Member
7. Prof.Baby Vasumathi, Director, Inst.of O&G, Ch-8	: Member
8. Prof.K.Ramadevi, Director, Inst.of Biochemistry, MMC	: Member
9. Prof.Saraswathy, M.D., Director, Inst. Of Pathology, MMC	: Member
10. Prof.Srinivasagalu, Director, Inst.of Inter Med. MMC	: Member
11. Thiru S.Rameshkumar, B.Com., MBA	: Lay Person
12. Thiru S.Govindasamy, B.A., B.L.,	: Lawyer
13. Tmt.Arnold Saulina, M.A., MSW.,	: Social Scientist

We approve the proposal to be conducted in its presented form.

The Institutional Ethics Committee expects to be informed about the progress of the study and SAE occurring in the course of the study, any changes in the protocol and patients information/informed consent and asks to be provided a copy of the final report.

Member Secretary, Ethics Committee

MEMBER SECRETARY
INSTITUTIONAL ETHICS COMMITTEE
MADRAS MEDICAL COLLEGE
CHENNAI-600 003

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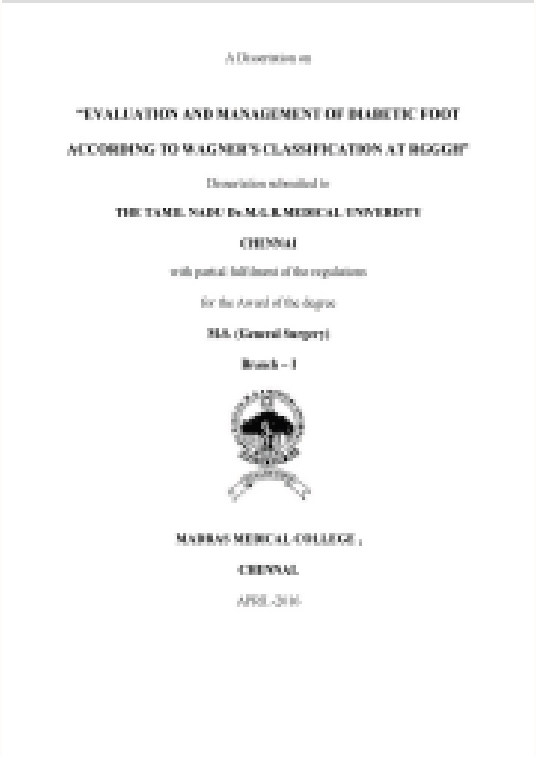


A Dissertation on

"EVALUATION AND MANAGEMENT OF DIABETIC FOOT ACCORDING TO WAGNER'S CLASSIFICATION AT RGGGH"

Dissertation submitted to

THE TAMIL NADU Dr.M.G.R.MEDICAL UNIVERSITY

CHENNAI

with partial fulfilment of the regulations

for the Award of the degree

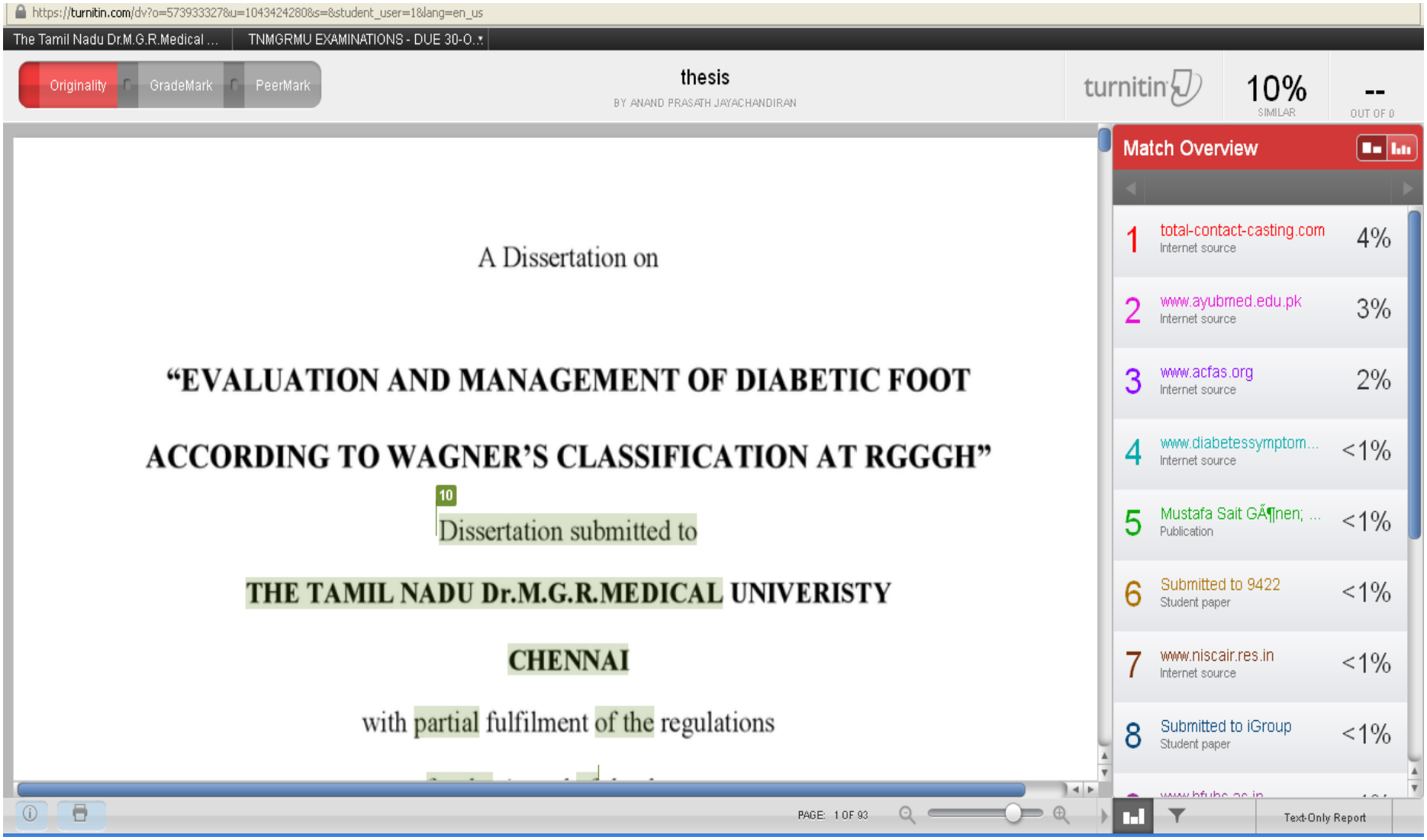
M.S. (General Surgery)

Branch – I

MADRAS MEDICAL COLLEGE,

CHENNAI

APRIL 2016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