“ROLE OF WAGNER’S CLASSIFICATION IN PREDICTING THE OUTCOME IN DIABETIC FOOT ULCER PATIENTS ADMITTED IN KLES DR.PRABHAKAR KORE HOSPITAL-A ONE YEAR CROSS SECTIONAL STUDY”

REG.NO. BH0112008

Dissertation

Submitted to the
KLE University, Belgaum, Karnataka

In Partial Fulfillment
of the requirements for the degree of

M. S.
in
GENERAL SURGERY

DEPARTMENT OF SURGERY,
JAWAHARLAL NEHRU MEDICAL COLLEGE,
BELGAUM, KARNATAKA

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ENDORSEMENT BY THE HOD/PRINCIPAL/ HEAD OF THE INSTITUTION

This is to certify that the dissertation entitled **"ROLE OF WAGNER'S CLASSIFICATION IN PREDICTING THE OUTCOME IN DIABETIC FOOT ULCER PATIENTS ADMITTED IN KLES DR. PRABHAKAR KORE HOSPITAL-A ONE YEAR CROSS SECTIONAL STUDY"** is a bonafide research work done by **CANDIDATE REG. NO. BH0112008.**

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LIST OF ABBREVIATIONS USED

ABI	-	Ankle-brachial index
AD	-	Anno Domini
AP	-	Anteroposterior
ATP	-	Adenine triphosphatase
ATPase	-	Adenine triphosphatase
BMI	-	Body mass index
CBC	-	Complete blood count
CI	-	Confidence interval
Cms	-	Centimeters
CVD	-	Cardiovascular disease
DC	-	Differential count
DM	-	Diabetes mellitus
DNA	-	Deoxyribonucleic acid
DUSS	-	Diabetic Ulcer Severity Score
e.g.	-	For example
ESRD	-	End-stage renal disease
FBS	-	Fasting blood sugar
FDA	-	Food and Drug Administration
FPG	-	Fasting plasma glucose
g	-	Grams
GDM	-	Gestational diabetes mellitus
h	-	Hour
Hb	-	Haemoglobin
HDL-C	-	High-density lipoprotein-cholesterol

HNF	-	Hepatocyte nuclear transcription factor
ID	-	Identification number
IDF	-	International Diabetes Federation
ie	-	That is
IFG	-	Impaired fasting glucose
IGT	-	Impaired glucose tolerance
IWGDF	-	International Working Group on the Diabetic Foot
kg/m^2	-	Kilograms per square meter
Kgs	-	Kilograms
Lat.	-	Lateral
LDL	-	Low-density lipoprotein
mg/dl	-	Milligram per decilitre
mg/dL	-	Milligram per decilitre
mm Hg	-	Millimeters of mercury
mm	-	Millimeters
mmol/L	-	Millimole per litre
MRI	-	Magnetic resonance imaging
MRSA	-	Methicillin resistant staphylococcus aureus
n	-	Total number
OPD	-	Out patient department
OR	-	Odds ratio
p	-	Probability
PAI	-	Plasminogen activator inhibitor
PEDIS	-	Perfusion, extent (size), depth (tissue loss), infection,
PTB	-	probe-to-bone

PVD	-	Peripheral vascular disease
SINBAD	-	Site, ischemia, neuropathy, bacterial infection, and death
Sr.	-	Serum
TLC	-	Total leukocyte count
US	-	United states
UT	-	University of Texas
UTI	-	Urinary tract infection
VRE	-	Vancomycin-resistant enterococci
vs	-	Versus
WHO	-	World Health Organization

ABSTRACT

Background and Objectives

Diabetes mellitus-related foot ulceration is very common. Several classification systems for diabetic foot ulcers have been proposed. The present was intended to assess the role of Wagner wound classification in predicting the outcome of diabetic foot ulcer.

Methodology

This present one year cross sectional study was carried out at the Department of General Surgery, KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belgaum. A total of 100 patients with diabetic foot ulcer who presented during the study period that is, from January 2013 to December 2013 were included. The diabetic foot ulcers were graded according to the Wagner's classification.

Results

In this study majority of the patients were males (79%) and the male to female ratio was 3.76:1. The mean age was noted as 55.8 ± 10.45 years. Majority of the patients had duration of ulcer less than one month (88%). Surrounding skin was inflamed in 60% of the patients, necrosis was present in 40% and slough was noted in 98% while 44% of the patients had necrotic tissue. Based on Wagner's Classification, most of the patients (48%) had Grade II diabetic foot ulcers. With regard to management, in 44% of the patients debridement was done and 36% of the patients had disarticulation or amputation in 36%. Of the 48 patients with grade II ulcer, 79% of the patients had healing without amputation. Of the 58

patients with grade I and II diabetic foot ulcers, 82.76% had healing without amputation compared to 17.24% of the patients who needed amputation. Patients with Grade III, IV and V had 3.59 times higher risk of amputation compared to patients with grade I and II. **(**$p<0.001$; 95% CI- 1.95 to 6.62).

Conclusion and interpretation

Grading of diabetic foot ulcer based on Wagner's classification affects and predicts the outcome and the risk of amputation increases with increasing grade.

Keywords

Diabetic foot ulcer; Risk of amputation; Wagner's Classification;

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INTRODUCTION

Diabetes mellitus (DM) is a metabolic disease characterized by hyperglycemia resulting from defects in insulin secretion, insulin action or both. The chronic hyperglycemia of diabetes is associated with long term damage, dysfunction and failure of various organs, especially the eyes, kidneys, nerves, heart and blood vessels.[1]

Long term complications of diabetes include retinopathy with potential loss of vision, nephropathy leading to renal failure, peripheral neuropathy with risk of foot ulcers, amputations and Charcot joints, and autonomic neuropathy causing gastro intestinal, genitourinary and cardiovascular symptoms and sexual dysfunction.[1]

Foot infections are a frequent complication of patients with diabetes mellitus, accounting for up to 20% of diabetes-related hospital admissions.[2,3] Infectious agents are associated with the worst outcomes, which may ultimately lead to amputation of the infected foot unless prompt treatment strategies are ensued.

Among persons diagnosed as having diabetes mellitus, the lifetime risk of developing a foot ulcer is estimated to be 15%.[4] Based on recent studies, the annual population-based incidence ranges from 1.0% to 4.1% and the prevalence ranges from 4% to 10%, which suggests that the lifetime incidence may be as high as 25%.[5]

Lower extremity disease, including peripheral arterial disease, peripheral neuropathy, foot ulceration, or lower extremity amputation, is twice as common in diabetic persons compared with nondiabetic persons and it affects 30% of diabetic

persons who are older than 40 years.[6] Foot ulcers cause substantial emotional, physical, productivity, and financial losses.

Diabetic foot ulcers are wounds on the feet that occur in people with diabetes, a condition where blood sugar levels are abnormally high. If a foot ulcer goes untreated and does not heal, it may become infected. Patients with diabetic neuropathy are subject to ulcerations that may be complicated by infection and gangrene, with subsequent risk of amputation. It is the job of the surgeons to identify and manage these problems early to avoid the unfortunate complication of amputation.

Because diabetic foot ulceration is a serious problem and because ulcers are heterogeneous in terms of etiology, anatomic location, depth of tissue involvement, and associated circumstances, including the presence or absence of infection, classification is needed in order to predict ulcer outcome and conduct clinical trials.[7,8]

In the literature, several classification systems for diabetic foot ulcers have been proposed. These classification systems have to comply with certain characteristics, such as precision, flexibility, specificity, and simplicity. They can be of great help for the assessment of treatment schemes. Classifications are also useful in standardization and analysis of multicenter research. The classification most frequently used analyzes one or more of the following elements: infection, neuropathy, vasculopathy, and the extent (surface and depth) of the ulcer. Further these classification schemes help in predicting outcomes.[7]

The best known and widely available classifications are Meggit/Wagner, Gibbon's, Frykberg's and Coleman's, Forrest's, Knighton's, the Texas Diabetic Wound Classification, and the Ten-Level Seattle Wound Classification System.[9-18] Each of these classifications was developed to accomplish a particular objective, utilizes different criteria, and categorizes lesions according to different rationales. Only a few of these classifications were evaluated for the assessment of the prognosis on salvage of the ulcerated or dysvascular diabetic limb.[7]

The Meggit/Wagner classification is probably the best known and the most frequently used. This system is based on three features: depth of the ulcer, the degree of infection, and the presence or absence of gangrene and its extent. Grades 1 to 3 are mainly based on neuropathy, while grade 4 and 5 represent mainly ischemic lesions. The grading system was adapted in 1988 by Calhoun, et al., in order to combine medical and surgical elements of therapy to monitor the treatment of diabetic foot infection.[19]

Nevertheless, it is described as very simple and, therefore, often considered to be inconveniently inaccurate. Also it is postulated that, the scheme provides insufficient levels to discriminate between wounds that may benefit from nonsurgical rather than surgical management. However these concepts remain controversial due to the scanty data and prompt further assessment. Classifying the ulcers and then treating them helps us to compare the outcomes better and so identify measures to reduce the morbidity and mortality due to diabetic foot disease. Considering the above facts the present study was planned to assess the role of Wagner wound classification in predicting the outcome of diabetic foot ulcer.

OBJECTIVES

1. To know the role of Wagner's classification in predicting the outcome in diabetic foot ulcer patients admitted in KLES Dr. Prabhakar Kore Hospital.

2. To know the grade of Wagner's classification to which majority of diabetic foot ulcer patients admitted in KLES Dr. Prabhakar Kore Hospital belong to.

REVIEW OF LITERATURE

DIABETES MELLITUS

Diabetes mellitus (DM) refers to a group of metabolic disorders that share the phenotype of hyperglycemia.[20] Depending on the etiology, factors causing hyperglycemia include

1. Decreased insulin secretion
2. Decreased glucose utilization
3. Increased glucose production.

The metabolic derangements associated with DM cause secondary pathophysiologic changes in multiple organ systems that cause tremendous burden on the individual with diabetes and on the health care system. Diabetes mellitus is the leading cause of end-stage renal disease (ESRD), nontraumatic lower extremity amputations, and adult blindness.[1] It also predisposes to cardiovascular diseases. With an increasing incidence worldwide, DM will be the leading cause of morbidity and mortality in the future.

Type 2 diabetes mellitus is a group of disorders characterized by hyperglycemia and associated with microvascular (Retinal, renal, possibly neuropathic), macrovascular (Coronary, peripheral vascular), and neuropathic (autonomic, peripheral) complications.[21] Unlike patients with type 1 diabetes mellitus, patients with type 2 are not absolutely dependent upon insulin, even though many of them are ultimately treated with insulin.

Historical aspects

The sweet taste of diabetic urine was noted in the 5th and 6th century AD by the Indian physicians (Sushruta and Charaka) and in the 17th century by Thomas Willis. The term 'Diabetes mellitus', an allusion to the honeyed taste of urine, was first used in the late 18th century by John Rollo and others, to distinguish it from other polyuric states in which urine was tasteless.

The 'ancient' period witnessed the first clinical descriptions of diabetes and complications. The 16th to 18th centuries have been termed the 'diagnostic' period, as diabetes mellitus was then identified as a separate disease entity, while the mid to late 19th century may be regarded as the first 'experimental' period, during which the glucoregulatory role of the pancreas became clear and the biochemical disturbances of diabetes were initially characterized.[22]

In 1893, Edovard Laguesse named the pancreatic islets after Paul Langerhans, who had described them in 1869, and suggested that they produced a glucose lowering substance. This hypothetical hormone was named 'insulin' by Jean de Meyer in 1909, over a decade before its discovery.

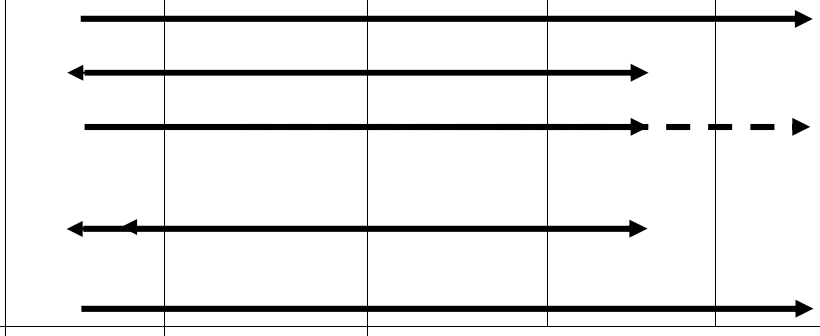
Insulin was discovered at the University of Toronto in 1921, through collaboration between Frederick G. Banting, Charles H. Best, James B. Collip and J.J.R. Macleod.

Finally, the 20th century has seen a dramatic increase in knowledge about diabetes. The discovery of insulin in 1921-22 has had profound scientific, clinical and social consequences.[23]

Classification

Diabetes mellitus is classified on the basis of the pathogenic process that leads to hyperglycemia, as opposed to earlier criteria such as age of onset or type of therapy.[24] The two broad categories of DM are designated type 1 and type 2. Both the types are preceded by a phase of disturbed glucose homeostasis, as the pathogenic process progresses. Type 1 diabetes is the result of complete or near-total insulin deficiency.

Spectrum of glucose homeostasis and diabetes mellitus[20]

Type of diabetes	**Normal glucose tolerance**	**Hyperglycemia**			
		Impaired fasting glucose or impaired glucose tolerance	**Diabetes mellitus**		
			No insulin required	**Insulin required for control**	**Insulin required for survival**
Type 1					
Type 2					
Other Specific types					
Gestational diabetes					
Time (years)					
FPG (mg/dl)	< 100	100-125	≥ 126		
2-h pg (mg/dl)	< 140	140 – 199	≥ 200		

Type 2 DM is a heterogeneous group of disorders characterized by variable degrees of insulin resistance, decreased insulin secretion, and increased glucose production. Distinct genetic and metabolic defects in insulin action and/or secretion finally lead to hyperglycemia in type 2 DM and have important therapeutic implications as pharmacologic agents that target specific metabolic derangements

are available now. Type 2 DM is preceded by a period of disturbed glucose homeostasis classified as impaired fasting glucose (IFG) or impaired glucose tolerance (IGT).

Etiologic Classification of Diabetes Mellitus[20]

I. Type 1 diabetes (β-cell destruction, usually leading to absolute insulin deficiency)
   a. Immune-mediated
   b. Idiopathic

II. Type 2 diabetes (may range from predominantly insulin resistance with relative insulin deficiency to a predominantly insulin secretory defect with insulin resistance)

III. Other specific types of diabetes.
   a. Genetic defects of β cell function characterized by mutations in:
      i. Hepatocyte nuclear transcription factor (HNF) 4 (MODY 1).
      ii. Glucokinase (MODY 2).
      iii. HNF-1 (MODY 3).
      iv. Insulin promoter factor-1 (IPF-1; MODY 4).
      v. HNF-1 (MODY 5).
      vi. NeuroD1 (MODY 6).
      vii. Mitochondrial DNA.
      viii. Subunits of ATP-sensitive potassium channel.
      ix. Proinsulin or insulin conversion.
   b. Genetic defects in insulin action
      i. Type A insulin resistance.

ii. Leprechaunism.

iii. Rabson-Mendenhall syndrome.

iv. Lipodystrophy syndromes.

c. Diseases of the exocrine pancreas-pancreatitis, pancreatectomy, neoplasia, cystic fibrosis, hemochromatosis, fibrocalculous pancreatopathy, mutations in carboxyl ester lipase.

d. Endocrinopathies-acromegaly, Cushing's syndrome, glucagonoma, pheochromocytoma, hyperthyroidism, somatostatinoma, aldosteronoma.

e. Drug or chemical induced-vacor, pentamidine, nicotinic acid, glucocorticoids, thyroid hormone, diazoxide, β-adrenergic agonists, thiazides, phenytoin, α-interferon, protease inhibitors, clozapine.

f. Infections - Congenital rubella, cytomegalovirus, coxsackie.

g. Uncommon forms of immune-mediated diabetes "stiff-person" syndrome, anti-insulin receptor antibodies.

h. Other genetic syndromes sometimes associated with diabetes—Down's syndrome, Klinefelter's syndrome, Turner's syndrome, Wolfram's syndrome, Friedreich's ataxia, Huntington's chorea, Laurence-Moon-Biedl syndrome, myotonic dystrophy, porphyria, Prader-Willi syndrome

IV. Gestational diabetes mellitus (GDM)

Epidemiology

Diabetes mellitus is fast becoming the epidemic of the 21st century. Type 2 diabetes, which is more prevalent (more than 90% of all diabetes cases) and the

main cause of the diabetes epidemic, now affects 5.9% of the world's adult population with almost 80% of the total in developing countries.[25] Nowhere is the diabetes epidemic more pronounced than in India as the World Health Organization (WHO) reports show that 32 million people had diabetes in the year 2000.[26]

World Health Organization reported that, 346 million people worldwide have diabetes. In 2004, an estimated 3.4 million people died from consequences of high blood sugar. More than 80% of diabetes deaths occur in low- and middle-income countries. WHO states that, diabetes deaths will double between 2005 and 2030.[27]

Race

The prevalence of type 2 diabetes mellitus varies widely among various racial and ethnic groups. Type 2 diabetes mellitus is becoming pandemic in some groups of Native Americans and Hispanic people. The risk of retinopathy and nephropathy appears to be greater in blacks, Native Americans, and Hispanics.

Sex

Type 2 diabetes mellitus is slightly more common in older women than men.

Age

While type 2 diabetes mellitus traditionally has been thought to affect individuals older than 40 years, it is being diagnosed increasingly in younger persons, particularly in obese and in highly susceptible racial and ethnic groups. In some areas, type 2 than type 1 diabetes mellitus is being more commonly diagnosed in prepubertal children, teenagers, and young adults. Virtually all cases of diabetes mellitus in older individuals are type 2.

Indian statistics

Several epidemiological studies in migrant Indians and India itself show that, the population has a high genetic predisposition for diabetes, which is precipitated by environmental factors such as urbanization.[28] The prevalence of diabetes is four to six fold lower in rural areas, which is probably due to the conventional lifestyle which has beneficial effect on glucose tolerance (IGT). National Urban Diabetes Survey done in six cities, found that age standardized prevalence rates were 12% for diabetes; with a slight male preponderance and 14% for impaired glucose tolerance. Subjects under the age of 40 years, had a prevalence of 5% percent for DM and 13% prevalence of impaired glucose tolerance.

The International Diabetes Federation (IDF) estimates the total number of diabetic subjects to be around 40.9 million in India and this is further set to rise to 69.9 million by the year 2025.[25] It is clear that in the last two decades, there has been a striking increase in the prevalence of diabetes among both urban as well as the rural Indians, with a implication that Southern India has seen the sharpest increase. Later studies confirmed this high prevalence of diabetes in urban south India. Although in rural India the prevalence of diabetes is much lower than in the urban population, even here the prevalence rates are rapidly rising, however more studies are needed to prove this. Variations in the prevalence rates of diabetes in different urban populations of India are expected because of the large differences in the prevalence of cardiovascular risk factors in different regions. It is evident that there is a shift in age of onset to younger age groups, which is upsetting and this could have adverse effects on the nation's economy. Hence, the early identification of at-risk individuals and appropriate intervention to boost physical activity, bring about

changes in dietary habits could to a great extent help to prevent/ delay, the onset of diabetes and thus reduce the burden due to its associated complications in India.[28]

Pathophysiology

Hyperglycemia results from lack of endogenous insulin, which is either absolute, as in type 1 diabetes mellitus, or relative, as in type 2 diabetes mellitus. Relative insulin deficiency usually occurs because of resistance to the actions of insulin in muscle, fat, and the liver and an inadequate response by the pancreatic beta cell. Insulin resistance, which has been attributed to elevated levels of free fatty acids in plasma,[29] leads to decreased glucose transport in muscle, elevated hepatic glucose production, and increased breakdown of fat.

Presumably, the defects of type 2 diabetes mellitus occur when a diabetogenic lifestyle (ie, excessive caloric intake, inadequate caloric expenditure, obesity) is superimposed upon a susceptible genotype. The body mass index at which the risk for diabetes increases varies with different racial groups. For example, compared with persons of European ancestry, persons of Asian ancestry are at increased risk for diabetes at lower levels of overweight.[30] A simplified scheme for the pathophysiology of abnormal glucose metabolism in type 2 diabetes mellitus is depicted in the image below.

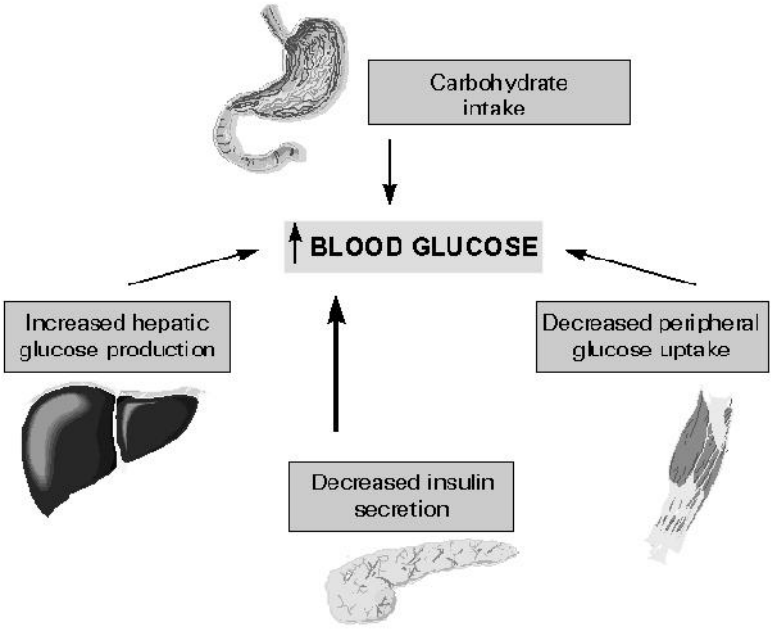


Figure 1. Pathophysiology of type 2 diabetes mellitus[1]

Hyperglycemia appears to be the determinant of microvascular and metabolic complications. However, glycemia is much less related to macrovascular disease. Insulin resistance with concomitant lipid (i.e., small dense low-density lipoprotein [LDL] particles, low high-density lipoprotein-cholesterol [HDL-C] levels, elevated triglyceride-rich remnant lipoproteins) and thrombotic (ie, elevated type-1 plasminogen activator inhibitor [PAI-1], elevated fibrinogen) abnormalities, as well as conventional atherosclerotic risk factors (e.g., family history, smoking, hypertension, elevated low-density lipoprotein-cholesterol [LDL-C], low HDL-C), determine cardiovascular risk.[1]

Diagnosis

The National Diabetes Data Group and World Health Organization have issued diagnostic criteria for DM based on the following premises:[1]

Criteria for the Diagnosis of Diabetes Mellitus

- Symptoms of diabetes plus random blood glucose concentration 11.1 mmol/L (200 mg/dL)[a] *or*
- Fasting plasma glucose 7.0 mmol/L (126 mg/dL)[b] *or*
- Two-hour plasma glucose 11.1 mmol/L (200 mg/dL) during an oral glucose tolerance test[c]

[a]Random is defined as without regard to time since the last meal.
[b]Fasting is defined as no caloric intake for at least 8 h.
[c]The test should be performed using a glucose load containing the equivalent of 75 g anhydrous glucose dissolved in water; not recommended for routine clinical use.

Risk factors for Type 2 Diabetes Mellitus

- Family history of diabetes (i.e. parent or sibling with type 2 diabetes)
- Obesity (BMI≥25 kg/m2)
- Habitual physical inactivity
- Race/ethnicity (e.g. African, American, Hispanic American, Native American, Asian American, Pacific Islander)
- Previously identified IFG or IGT
- History of GDM or delivery of baby > 4kg (>9 Ib)
- Hypertension (blood pressure ≥ 140/90 mHg)
- HDL cholesterol level ≤ 35mg/dL (0.90mmol/L) and / or a triglyceride level ≥ 250mg/dL (2.82 mol/L)
- Polycystic ovary syndrome or acanthosis nigricans.
- History of vascular disease.

Complications

Diabetes has both acute and long term complications.[1] They are:

Acute

- Diabetic ketoacidosis
- Hyperglycemic Hyperosmolar state
- Hypoglycemia

Long term:

- Retinopathy
- Neuropathy
- Nephropathy
- Ischemic heart disease
- Cerebrovascular disease
- Peripheral vascular disease
- Hypertension.

Others

- ***Infections***
  - UTI
  - Tuberculosis
  - Candidiasis – oral / vulvovaginal
  - Mucor mycosis
  - Necrotising fasciitis
  - Periodontitis
- Dupuytren's contracture

- Pseudogout

Neuropathy and diabetes mellitus[31-35]

- The prevalence of diabetic neuropathy in patients with type 2 diabetes is 32 % overall and more than 50%in patients over 60 years of age.
- Diabetic neuropathy correlates with the duration of diabetes and glycemic control) type 1 and 2 DM.
- May manifest as
    1. Polyneuropathy
    2. Mono-neuropathy
    3. Autonomic Neuropathy
- Both myelinated and unmyelinated nerve fibers are affected.
- As the clinical features of diabetic neuropathy are similar to those of other neuropathies, the diagnosis of diabetic neuropathy should be made only after other possible causes are excluded.

Poly-neuropathy / Mono-neuropathy

- The most common form of diabetic neuropathy is distal symmetric polyneuropathy.
- It presents as:
    1. Distal sensory loss - most frequent presentation
    2. Hyperesthesia
    3. Paresthesia
    4. Dysesthesia

- Symptoms includes a sensation of the following, which begins in the feet & spreads proximally.
    1. Numbness,
    2. Tingling
    3. Sharpness
    4. Burning

Any combination of these symptoms may develop as neuropathy progresses

- Physical examination reveals
    1. Sensory loss
    2. Loss of ankle reflexes
    3. Abnormal position sense.
- Pain typically involves lower extremities, is usually present at rest, and worsens at night.
- Both an acute (lasting <12 months) and a Chronic form of painful diabetic neuropathy have been described.
- As diabetic neuropathy progresses, the pain subsides & ultimately disappears, but a sensory deficit in the lower extremities remains.
- Neuropathic pain develops in some of these individuals, occasionally preceded by improvement in the glycemic control.

Diabetic Neuropathy

It may be accompanied by - Motor weakness

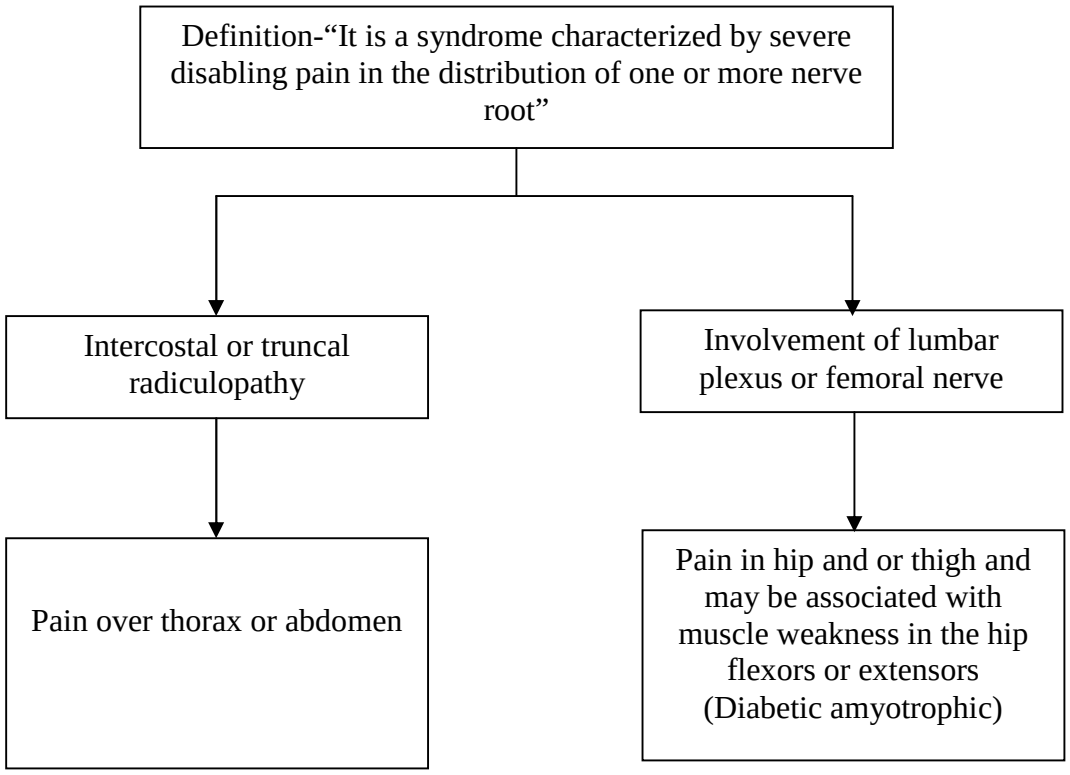


Figure 2. Diabetic neuropathy

Neuropathy

Neuropathy is present in over 80% of patients with foot ulcers.[36]

Peripheral sensory neuropathy

Interferes with normal protective mechanisms and allows the patient to sustain major or repeated minor trauma to the foot, frequently without knowledge of the injury.

Motor and sensory neuropathy

Lead to abnormal foot muscle mechanics and to structural changes in the foot (hammer toe, claw toe deformity, prominent metatarsal heads, Charcot joint).

Autonomic neuropathy

Results in anhidrosis and altered superficial blood flow in the foot, which promote drying of the skin and fissure formation.

Peripheral arterial disease and poor wound healing

Impede resolution of minor breaks in the skin, allowing them to enlarge and to become infected.

Disordered proprioception

Causes abnormal weight bearing while walking and subsequent formation of callus or ulceration.

Approximately 15% of individuals with DM develop a foot ulcer and a significant subset will ultimately undergo amputation (14 to 24%).

DIABETIC FOOT

A diabetic foot infection is most simply defined as any inframalleolar infection in a person with diabetes mellitus. These include paronychia, cellulitis, myositis, abscesses, necrotizing fascitis, septic arthritis, tendonitis, and osteomyelitis. The most common and classic lesion, however, is the infected diabetic “mal perforans” foot ulcer. Wound infection is the deposition and multiplication of

bacteria in tissue with colony count of more than 10^5 bacteria per gram of tissue with an associated host reaction.[37]

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.

Anatomy of the foot[38,39]

The human foot acts as a pliable platform to support the body weight in the upright posture and as a lever to propel the body forwards in walking, running or jumping. It has 26 bones, 29 joints, 42 intrinsic muscles, various ligaments, 4 mm thick skin, exquisite nerve supply and abundant vascularity with good collaterals. These component works together to provide the body with support, balance with mobility.

Parts

Structurally the foot has three main parts;

1. *The fore foot:* It is composed of phalanges and metatarsals. They are connected together by metatarso phalangeal joint at the balls of the foot. The fore foot bears the half of the body weight and balance pressure on the balls of the foot.
2. *The mid foot:* It is composed of five tarsals bones. It forms the foot's arch and serves as a shock absorber.

3. *The hind foot:* It links the mid foot to ankle. It is composed of two long bones of the lower leg, the tibia and the fibula which forms ankle joint with talus. This subtalar joint is formed between talus and calcaneum which is cushioned inferiorly by a fat layer.

Arches

The foot consists of three arches.

1. Medial longitudinal arch

    - It is the highest and the most important arch of the foot.
    - It is composed of calcaneum, talus, navicular, cuneiforms and first three metatarsal bones. The submit of the arch is formed by talus.
    - It acts as a shock absorber.

2. Lateral longitudinal arch

    - It is characteristically low arch.
    - It is composed of calcaneum, cuboid, fourth and fifth metatarsal bones. The submit of the arch is formed by calcaneum.
    - It transmits the body weight and thrust to the ground.

3. Transverse arch

    - It is a continuous structure formed by cuboid, three cuneiforms and the bases of the metatarsal bones.

Factors responsible for the maintenance of the arches

1. Ligaments and plantar aponeurosis.
2. Action of extrinsic and intrinsic muscles of the foot.
3. Structure of the bones.

Functions of the arches of the foot

1. They distribute body weight to the weight bearing areas of the sole mainly heel and the base of the toes (first and fifth).
2. They act as a springs chiefly the medial longitudinal arch which helps in walking and running.
3. They also act as a shock absorbers in stepping and jumping.
4. The concavity of the arches protects the soft tissue of the sole against pressure.

Sole

The skin of the sole is about 4 mm thick. It is adapted for weight bearing. There are subcutaneous concentrations of the fat over the weight bearing areas such as heel, lateral margin of the sole and across the plantar aspect of the metatarsal heads. Numerous fibrous bands between the skin and the plantar aponeurosis prevent undue movement of sole during walking.

Muscles

Intrinsic

- Origin and insertion are located within the foot.

- They include plantar flexors, dorsiflexors, abductors and adductors of the toes.
- They also support the arches of the foot.

Extrinsic

- Origin of these muscles are in the lower leg.
- They have long tendon that crosses the ankle to insert on the bones of foot except the talus.
- They are responsible for the movement at the ankle, foot and toes.
- They also support the arches of the foot.

Major joints and movements

- Ankle joint – Dorsiflexion and plantar flexion.
- Subtalar joint – Inversion and eversion.
- Midtarsal joint – Abduction and adduction.

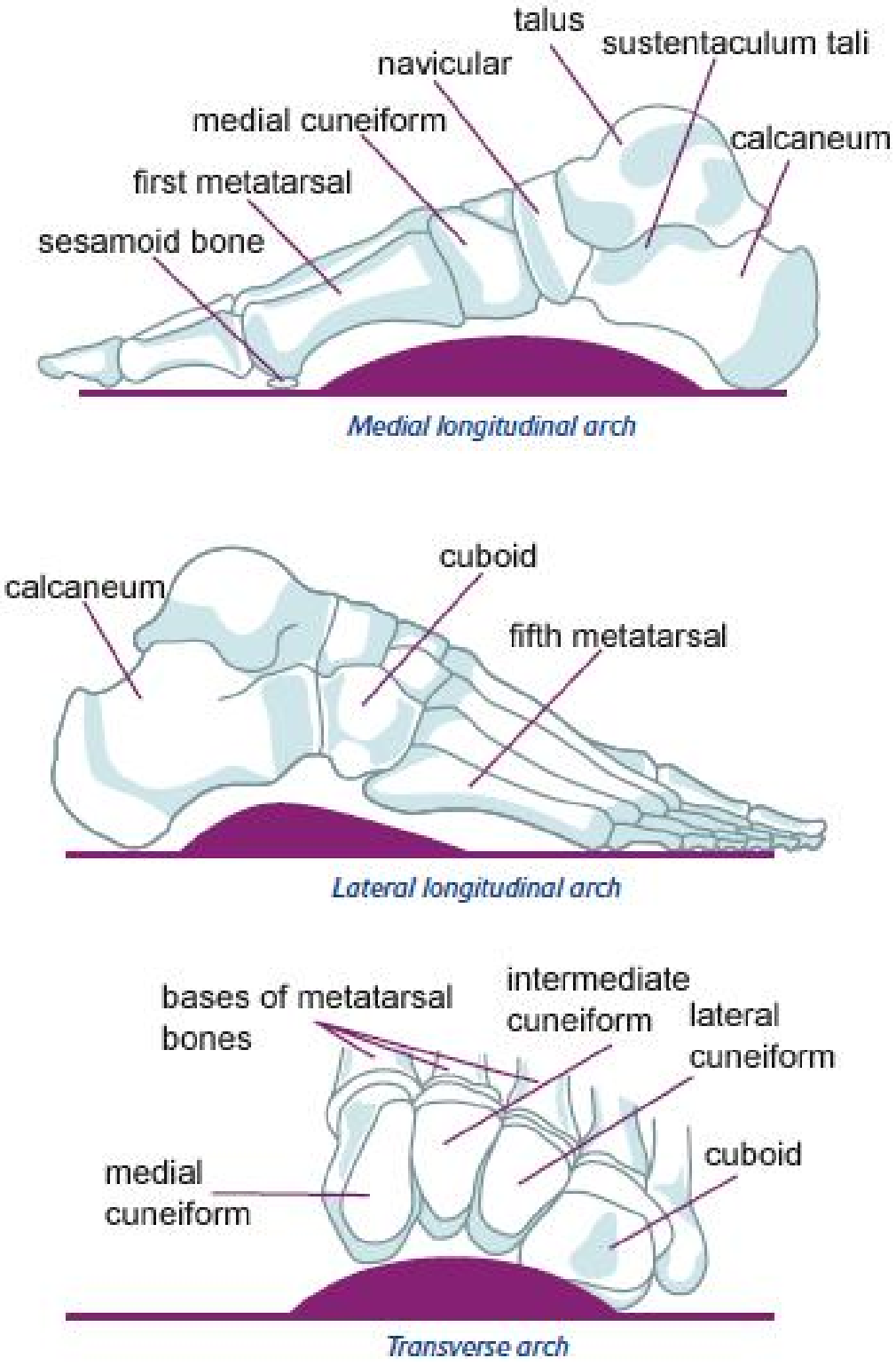


Figure 3. Arches of the foot[38,39]

Blood supply

Anterior tibial artery continues as a dorsalis pedis artery in the foot. Dorsalis pedis artery gives off a arcuate artery that along with its branches supplies the outer four toes. The dorsalis pedis artery continues down to supply the great toe. Posterior tibial artery in the sole of the foot divides into two branches, the lateral and medial plantar arteries that supplies the sole of the foot. The peroneal artery descends down and supply posterior and the outer aspect of the heel.

Nerve supply

Sensory nerve supply

Dorsum

- The saphenous nerve: It supplies the medial border of the foot upto the ball of the great toe.
- The superficial peroneal nerve: It supplies entire dorsum of the foot except the lateral border, medial border and the cleft between the first and second toe.
- The sural nerve: It supplies the lateral border of the foot upto the tip of the little toe.
- The deep peroneal nerve: It supplies the cleft between the first and the second toes.
- The digital branch of the medial and lateral plantar nerve supplies the distal part of the dorsum of the toes.

Sole

- Medial calcanean branch of tibial nerve: It supplies posterior and medial portion of the sole.
- Medial plantar nerve: It supplies the anteromedial portion of the sole and medial three and half digits.
- Lateral plantar nerve: It supplies anterolateral portion of the sole and lateral one and half digits.

Motor nerve supply

- Deep peroneal nerve.
- Superficial peroneal nerve.
- Tibial nerve - Medial plantar nerve; Lateral plantar nerve.

Epidemiology

Approximately 15% of all patients with diabetes will develop a peripheral ulcer. 20% of all patients with diabetes admitted to a hospital will have a skin ulcer. The risk of amputation in a patient with diabetes is 15–40 times higher than in a patient without diabetes. The presence of an ulcer in a diabetic patient has a deep impact on the quality of life for the patient and on the delivery of care. The cost of care for diabetic ulcers and the associated amputations is staggering. Although the prevalence of chronic ulcers has been estimated to be 120/100,000 people between 45–64 year of age, the prevalence increases to more than 800/100,000 people over the age of 75 year. Persons with diabetes have up to a 40-fold greater risk of lower extremity amputation than their nondiabetic counterparts. There were approximately

86,000 hospital discharges for diabetes-related nontraumatic amputations in the United States in 1996. The 5-year survival rate after amputation of a diabetic limb is less than 50%. These grim statistics reflect an increased prevalence of peripheral lesions in diabetes, but also delayed healing.[40]

Risk factors

Risk factors for foot ulcers or amputation include male sex, diabetes >10 years' duration, peripheral neuropathy, abnormal structure of foot (bony abnormalities, callus, thickened nails), peripheral arterial disease, smoking, history of previous ulcer or amputation and poor glycemic control.

Etiology

The etiologies of diabetic ulceration include neuropathy,[41] arterial disease,[42] pressure,[43] and foot deformity.[44] Diabetic peripheral neuropathy, present in 60% of diabetic persons and 80% of diabetic persons with foot ulcers, confers the greatest risk of foot ulceration; microvascular disease and disturbed glycemic control contribute. Sensory neuropathy involving the feet may lead to unrecognized episodes of trauma due to ill-fitting shoes. Motor neuropathy, causes intrinsic muscle weakness and splaying of the foot on weight bearing, increasing the trauma. The result is a convex foot with a rocker-bottom appearance. Multiple fractures are unnoticed until bone and joint deformities become marked. This is termed a Charcot foot (neuropathic osteoarthropathy) and most commonly is observed in diabetes mellitus, affecting about 2% of diabetic persons. If a Charcot foot is neglected, ulceration may occur at pressure points, particularly the medial aspect of the navicular bone and the inferior aspect of the cuboid bone. Sinus tracts progress from

the ulcerations into the deeper planes of the foot and into the bone. Charcot change can also affect the ankle, causing displacement of the ankle mortise and ulceration, which can lead to the need for amputation.

Pathophysiology

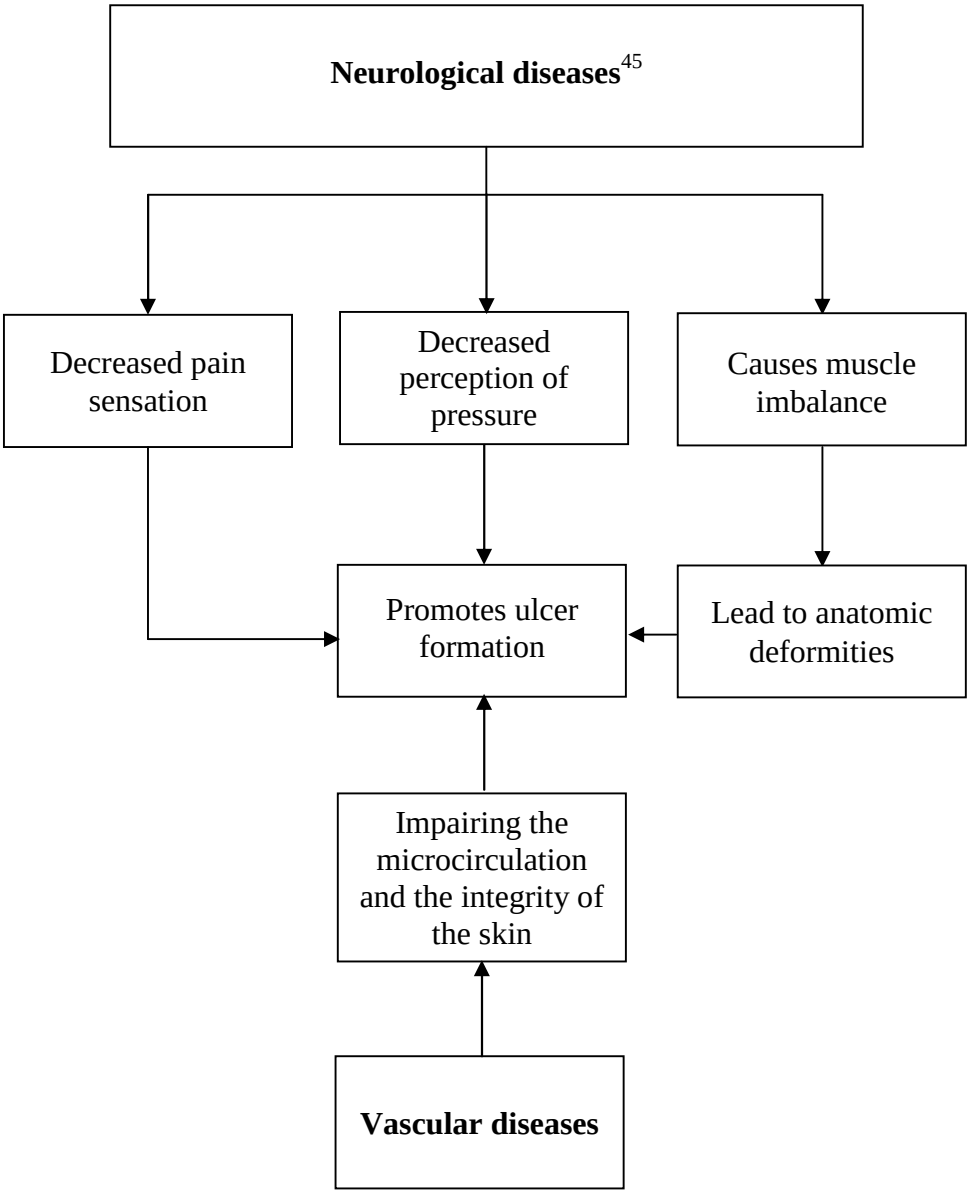


Figure 4. Pathogenesis of Diabetic Foot

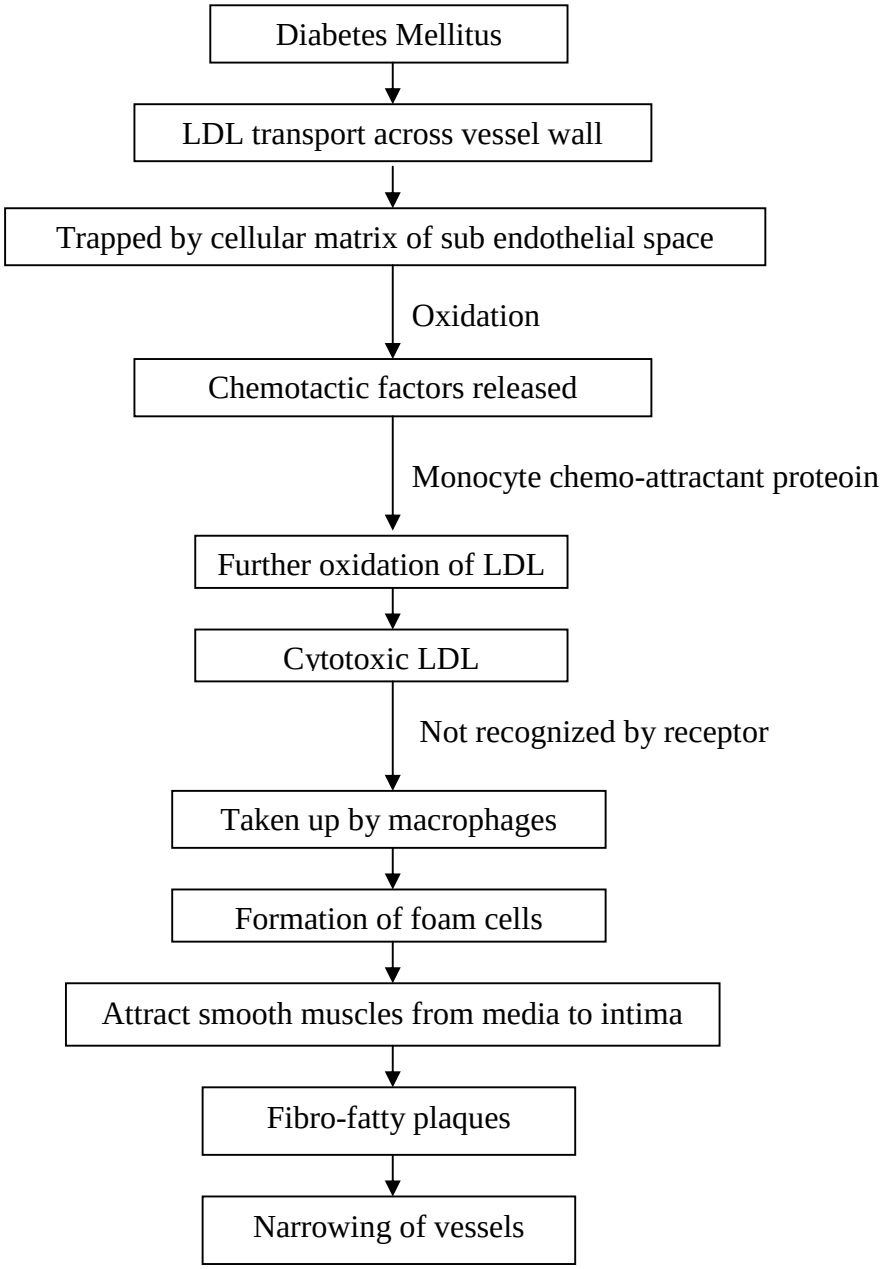


Figure 5. Pathophysiology of diabetic vasculopathy

Atherosclerosis and peripheral neuropathy occur with increased frequency in persons with DM. Development of atherosclerosis is accelerated in DM leading to increased morbidity and mortality. All the large vessels are involved in this process and clinical manifestations are apparent as a result of atherosclerotic narrowing and thrombosis of coronary, cerebral and leg vessels.

The pathophysiology of diabetic peripheral neuropathy is multifactorial and is due to vascular disease occluding the vasa nervorum; endothelial dysfunction; deficiency of myoinositol-altering myelin synthesis and diminishing sodium-potassium adenine triphosphatase (ATPase) activity; chronic hyperosmolarity, causing edema of nerve trunks; and effects of increased sorbitol and fructose.[46]

The result of loss of sensation in the foot is repetitive stress; unnoticed injuries and fractures; structural foot deformity, such as hammertoes, bunions, metatarsal deformities, or Charcot foot; further stress; and eventual tissue breakdown. Unnoticed excessive heat or cold, pressure from an ill- fitting shoe, or damage from a blunt or sharp object accidentally left in the shoe may cause blistering and ulceration. These factors, combined with poor arterial inflow, confer a high risk of limb loss on the patient with diabetes.

The infection and related issues

The source of infection is usually the contamination of the break in the skin, which may be imperceptible like cracks or fissures, puncture wounds or a major wound in a neuropathic foot due to trauma of any cause. Staphylococcus aureus and beta haemolytic streptococci rapidly colonise the break in the skin. A high frequency of anaerobic infection has also been reported.[47] The devastating developments subsequent to an infected ulcer that lead to the development of gangrene, necrotizing fascitis and life threatening situations like multi organ failure should be guarded against. The pathophysiology of these events can be constructed in the following sequence.

In persons with diabetes, infection results in microthrombi formation in the smaller vessels unlike persons without diabetes where it results in vasodilatation. This impairs blood flow in diabetes, converting the small arteries of the toes into end arteries resulting in gangrene of the toes. Osteomyelitis can be difficult to diagnose and remains a focus of untreated infection and fails to indicate to the physician the need for longer antibiotic regimen. The diagnosis of Osteomyelitis was missed in as many as two thirds of bone culture proven case. Excessive reliance on plain X rays by primary care physicians does not help. Simple probing the bone can make a diagnosis of Osteomyelitis, while scanning techniques are not always successful, some like Tc99 lack specificity, but MRI is proving helpful.

The risk of lower extremity amputation is 15 to 46 times higher in diabetics than in persons who do not have diabetes mellitus. Foot infections are the most common complications of diabetic foot and plays a main role in the development of moist gangrene.[48] In general, people with diabetes have infections that are more severe and take longer to cure than infections in other people. The infection leads to the early development of complication even after a trivial trauma, the disease progresses and becomes refractory to antibacterial therapy.[49] It is essential to assess the magnitude of bacterial infection of the lesions to avoid further complications and save the diabetic foot. Early diagnosis of microbial infections is aimed to provide the appropriate antibacterial therapy and to avoid further complications.[50]

However, these infections are difficult to treat because these patients have impaired microvascular circulation, which limits the access of phagocytic cells to the infected area and results in a poor concentration of antibiotics in the infected

tissues. Although infection is rarely implicated in the etiology of diabetic foot ulcers, the ulcers are susceptible to infection once the wound is present.

Microbiologic features of diabetic foot

Aerobic Gram-positive cocci are the predominant bacteria that colonize and acutely infect breaks in the skin. *Staph aureus* and the hemolytic streptococci (groups A, C, and G, but especially group B) are the most commonly isolated pathogens.[51] Chronic wounds develop a more complex colonizing flora, including enterococci various Enterobacteriaceae, obligate anaerobes, *Pseudomonas aeruginosa*, and nonfermentative Gram-negative rods.[52] Hospitalization, surgical procedures, and, especially, prolonged or broad-spectrum antibiotic therapy may predispose patients to colonization and/or infection with antibiotic-resistant organisms (MRSA or vancomycin-resistant enterococci [VRE]).[53] Although MRSA strains have previously been isolated mainly from hospitalized patients, community associated cases are now becoming common and are associated with poor outcomes in patients with diabetic foot infections.[54]

The impaired host defenses around necrotic soft tissue or bone may allow low-virulence colonizers, such as coagulase-negative staphylococci and *Corynebacterium* species ("diphtheroids"), to assume a pathogenic role. Acute infections in patients who have not recently received antimicrobials are often monomicrobial (almost always with an aerobic Gram-positive coccus), whereas chronic infections are often polymicrobial. Cultures of specimens obtained from patients with such mixed infections generally yield 35 isolates, including Gram-

positive and Gram-negative aerobes and anaerobes.[55] The pathogenic role of each isolate in a polymicrobial infection is often unclear.

Pathogens associated with various clinical foot-infection syndromes[56]

Foot- infection syndrome	Pathogens
Cellulitis without an open skin wound.	Beta-hemolytic streptococcus* and Staph aureus
Infected ulcer and antibiotic naïve (X).	Staph aureus and beta-hemolytic streptococcus*
Infected ulcer that is chronic or was previously treated with antibiotic therapy (Y).	Staph aureus, beta-hemolytic streptococcus, and Enterobacteriaceae
Ulcer that is macerated because of soaking (Y).	Pseudomonas aeruginosa (often in combination with other organisms)
Long-duration nonhealing wounds with (Y, Z) prolonged broad-spectrum antibiotic therapy	Aerobic gram-positive cocci (Staph aureus, coagulase-negative staphylococci, and enterococci), diphtheroids, Enterobacteriaceae, Pseudomonas species, nonfermentative gram-negative rods, and, possibly, fungi
"Fetid foot": extensive necrosis or gangrene or malodorous (Z)	Mixed aerobic gram-positive cocci, including enterococci, gangrene, malodorous Enterobacteriaceae, nonfermentative gram-negative rods, and obligate anaerobes

*Groups A, B, C, and G; X Often monomicrobial; Y Usually polymicrobial; Z Antibiotic-resistant species (eg, MRSA, vancomycin-resistant enterococci, or extended-spectrum beta-lactamase–producing gram-negative rods) are common

Risk Factors for Foot Ulceration and Infection[56]

Risk Factor	Mechanism of Injury or Impairment
Peripheral motor neuropathy	Abnormal foot anatomy and biomechanics, with clawing of toes, high arch, and subluxed metatarsophalangeal joints, leading to excess pressure, callus formation and ulcers.
Peripheral sensory neuropathy	Lack of protective sensation, leading to unattended minor injuries caused by excess pressure or mechanical or thermal injury.
Peripheral autonomic neuropathy	Deficient sweating leading to dry, cracking skin.
Neuro-osteoarthropathic deformities (i.e., Charcot disease) or limited joint mobility	Abnormal anatomy and biomechanics, leading to excess pressure, especially in the midplantar area.
Vascular (arterial) insufficiency	Impaired tissue viability, wound healing, and delivery of neutrophils.
Hyperglycemia and other metabolic derangements	Impaired immunological (especially neutrophil) function and wound healing and excess collagen cross-linking.
Patients disabilities	Patient reduced vision, limited mobility, and previous amputation(s).
Maladaptive patient behaviors	Inadequate adherence to precautionary measures and foot inspection and hygiene procedures, poor compliance with medical care, inappropriate activities, excessive weight-bearing, and poor footwear.
Health care system failures	Inadequate patient education and monitoring of glycemic control and foot care.

Infections and compromise of the foot vessels

Puncture or penetrating wounds of the plantar region or the web space infections may go up in the central non expansible plantar space. The inflammatory exudates that collects causes pressure on the small arteries in the tissues and will lead to thrombosis or obliteration. This will lead to gangrene.[57]

Recognition of wound infection[58]

The inflammatory response is a protective mechanism that aims to neutralise and destroy any toxic agents at the site of an injury and restore tissue homeostasis. The classic signs of infection include:

- Localised erythema.
- Localised pain.
- Localised heat.
- Oedema.

Further criteria include:

- Abscess.
- Discharge which may be viscous in nature, discoloured and purulent.
- Delayed healing not previously anticipated.
- Discolouration of tissues both within and at the wound margins.
- Unhealthy granulation tissue.
- Abnormal smell.
- Wound breakdown associated with wound pocketing/bridging at base of wound.

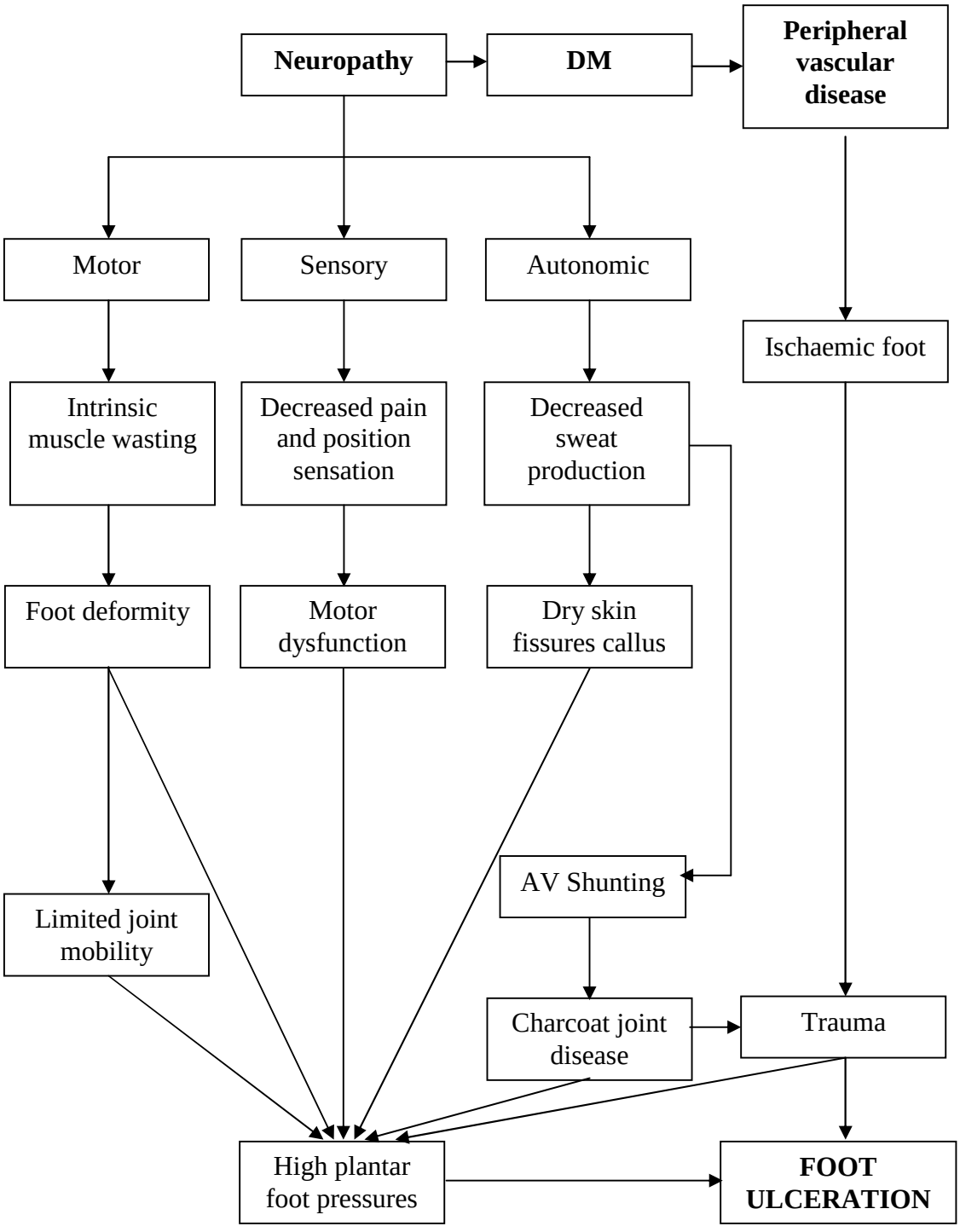


Figure 6. Clinical pathways leading to foot ulceration[59,60]

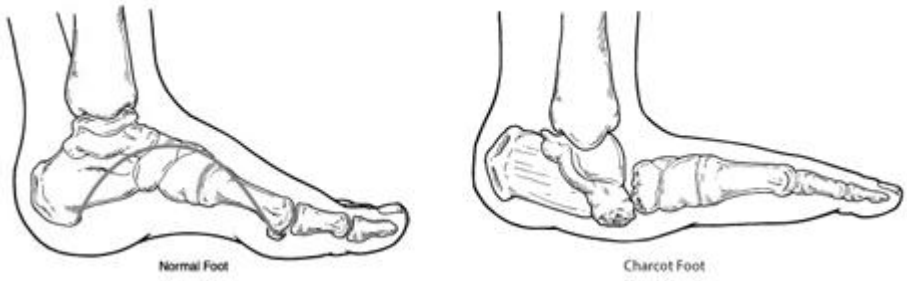


Figure 7. Charcots foot

Evaluation

- Characteristics: Size, depth, appearance, discharge and location.
- Etiological assessment: Neuropathic, ischemic, or neuro-ischemic.
- Screening for neuropathy.
    - Pressure of a 5.07 (10-g) Semmes Weinstein monofilament.
    - Vibration sensation with the use of standard tuning fork (128 cycles per second)
    - Neurologic reflex hammer.
- Probing of ulcer for underlying osteomyelitis.
- Culture sensitivity of the discharge.
- Radiograph for underlying osteomyelitis.
- Colour Doppler study for vascular pathology.
- MRI for Charcots neuropathy.

Classification

Classification systems have been developed to grade the severity of diabetic foot ulcers, provide prognosis on healing and aid in the formulation of treatment

plans. The widely used Wagner classification for diabetic foot ulcers assesses ulcer depth and the presence of osteomyelitis or gangrene.[13] The University of Texas system assesses ulcer depth, the presence of wound infection, and the presence of clinical signs of lower-extremity ischaemia.[61] This system uses a matrix of grade on the horizontal axis and stage on the vertical axis.[62]

Clinicians should consider the possibility of infection occurring in any foot wound in a patient with diabetes. Evidence of infection generally includes classic signs of inflammation (redness, warmth, swelling, tenderness, or pain) or purulent secretions but may also include additional or secondary signs (eg, nonpurulent secretions, friable or discolored granulation tissue, undermining of wound edges, foul odor).[61]

Clinicians should be aware of factors that increase the risk for DFI and especially consider infection when these factors are present; these include a wound for which the probe-to-bone (PTB) test is positive; an ulceration present for >30 days; a history of recurrent foot ulcers; a traumatic foot wound; the presence of peripheral vascular disease in the affected limb; a previous lower extremity amputation; loss of protective sensation; the presence of renal insufficiency; or a history of walking barefoot.[63]

Clinicians should select and routinely use a validated classification system, such as that developed by the International Working Group on the Diabetic Foot (IWGDF) (abbreviated with the acronym PEDIS) or IDSA, to classify infections and to help define the mix of types and severity of their cases and their outcomes. The DFI Wound Score may provide additional quantitative discrimination for research

purposes. Other validated diabetic foot classification schemes have limited value for infection, as they describe only its presence or absence.[63]

Any foot wound in a patient with diabetes may become infected. Traditional inflammatory signs of infection are redness (erythema or rubor), warmth (calor), swelling or induration (tumor), tenderness and pain (dolor), and purulent secretions. Some infected patients may not manifest these findings, especially those who have peripheral neuropathy (leading to an absence of pain or tenderness) or limb ischemia (decreasing erythema, warmth, and possibly induration). In this situation, some evidence supports the correlation of additional or secondary findings, for example, nonpurulent secretions, friable or discolored granulation tissue, undermining of the wound edges, or a foul odor, with evidence of infection. However, none of these findings, either alone or in combination, correlate with a high colony count of bacteria in a wound biopsy. Since the original IDSA DFI guidelines, we have advocated using the presence of ≥2 of the classic findings of inflammation to characterize a wound as infected. Although this definition is based only on expert consensus opinion, it has been used as the diagnostic criterion in many studies of DFI, including some used by the US Food and Drug Administration (FDA) to approve specific antibiotic agents for treating DFIs.[63]

During the systematic review of the literature we found 177 studies that identified risk factors for developing a foot infection in persons with diabetes. Identification of risk factors for DFI was the objective in only 2 studies.[64,65] In one instance, factors that were significantly associated (by multivariate analysis) with developing a foot infection included having a wound that extended to bone (based on a positive PTB test; odds ratio [OR], 6.7); a foot ulcer with a duration >30 days

(OR, 4.7); a history of recurrent foot ulcers (OR, 2.4); a wound of traumatic etiology (OR, 2.4); or peripheral vascular disease, defined as absent peripheral arterial pulsations or an ankle-brachial index (ABI) of <0.9 (OR, 1.9).[64]

Among 199 episodes of DFI, only 1 infection occurred in a patient without a previous or concomitant foot ulcer. In the second study, a retrospective review of 112 patients with a severe DFI, multivariate analysis identified 3 factors that were associated with developing a foot infection: a previous amputation (OR, 19.9); peripheral vascular disease, defined as any missing pedal pulsation or an ABI of <0.8 (OR, 5.5); or loss of protective sensation (OR, 3.4). Psychological and economic factors did not contribute significantly to infection.[64]

Several other studies examined the association between a specific medical condition and various diabetic foot complications, including infections. These types of studies lack a control group of patients without foot infection and are therefore subject to selection bias. Some studies, each of which was retrospective and reported only a small number of cases, have suggested an association between renal failure and DFI.[66,67] Finally, a report from Sri Lanka found that, compared to patients who wore shoes, those who walked barefoot for >10 hours per day had more web space and nail infections (14% vs 40%, respectively, $P < .01$).[68]

In most published classification schemes, assessing infection is a subsection of a broader wound classification. These classification systems each have somewhat different purposes, and there is no consensus on which to use.[63] Some classifications, including the Meggitt-Wagner and SINBAD (site, ischemia, neuropathy, bacterial infection, and death),[69] subjectively categorize infection only dichotomously, that is,

as present or absent, and without clear definitions. We briefly summarize the key features of commonly used diabetic foot classification schemes and wound scoring systems.

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,[70] 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, and the other two classifications. 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 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).

Other Diabetic Foot Wound Classification Schemes.

Wagner Wagner, in collaboration with Meggitt, developed perhaps the first, and still among the most widely used, classification schemes for diabetic foot wounds.[71,72] It assesses ulcer depth and the presence of infection and gangrene with grades ranging from 0 (pre- or postulcerative) 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.[73] Each point has 4 grades, thus creating a semiquantative scale. Infection is graded as none, surface only, cellulitis, and osteomyelitis; these are not further defined. One study reported good interobserver agreement.[73] Unlike the other key points, studies have not shown infection to be related to outcome of the foot ulcer.[73] 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).[69]

University of Texas (UT) ulcer classification[74]

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 stages and grades and a significantly higher amputation rate in wounds deeper than superficial ulcers.[74] A study in Brazil compared the UT and the S(AD)/SAD and SINBAD systems and found that all 3 predicted the outcomes of diabetic foot ulcers; the association of outcome with infection was stronger than that reported from the centers in Europe or North America.[75]

Ulcer Severity Index[76]

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.[76]

Diabetic Ulcer Severity Score (DUSS) and MAID[77,78]

These scoring systems are based on specific wound characteristics associated with stages of wound repair. Studies have found no significant correlation between soft tissue infection and wound healing, although there was a trend toward more infection in the higher-risk groups.

DFI Wound Score[79]

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 semiquantitative 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.[72]

Patients with more severe wounds had higher scores; clinical response was favorable 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 nonpurulent), leaving an 8-item score, provided better measurement

statistics. 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.[79]

Each of these classifications may be used in clinical practice, but they have not been compared in a large prospective trial. The PEDIS, IDSA, UT, and S(AD)SAD classification systems are fairly simple to use and appear to help predict outcomes. The DFI and DUSS wound scores are relatively complex, but each has been validated in large research trials.[79]

Clinicians should evaluate a diabetic patient presenting with a foot wound at 3 levels: the patient as a whole, the affected foot or limb, and the infected wound. Clinicians should diagnose infection based on the presence of at least 2 classic symptoms or signs of inflammation (erythema, warmth, tenderness, pain, or induration) or purulent secretions. They should then document and classify the severity of the infection based on its extent and depth and the presence of any systemic findings of infection. It is recommend assessing the affected limb and foot for arterial ischemia (strong, moderate), venous insufficiency, presence of protective sensation, and biomechanical problems. Clinicians should debride any wound that has necrotic tissue or surrounding callus; the required procedure may range from minor to extensive.[63]

Wagner Ulcer Classification System[13]

Grade	Description of ulcer
0	intact skin in patients who are at risk
I	superficial ulcers with exposed subcutaneous tissue
II	exposed tendon and deep structures
III	ulcers extend to the deep tissue and have either associated soft tissue abscess or osteomyelitis
IV	ulcers include feet with partial gangrene
V	feet ulcers with more extensive gangrenous tissue

Wagner ulcer classification system was developed for the "dysvascular" foot. It was skewed toward severe disease and contains all infections within a single grade.

Consensus is developing that the key issues in classifying a diabetic foot wound are its depth (in particular, which tissues are involved) and whether the wound is complicated by either ischemia or infection. The International Consensus on the Diabetic Foot recently published a preliminary progress report on a diabetic foot ulcer classification system for research purposes.[80] The key elements are summarized by the acronym PEDIS (perfusion, extent/size, depth/tissue loss, infection, and sensation).

MEDICAL AND SURGICAL MANAGEMENT[81]

A Baseline Approach in Managing the Acute Problem of the Diabetic Foot

1. Appraise problem
    a. Careful inspection with emphasis on webspaces and back of heels.

   b. Record pulses, venous filling time, rubor
   c. Record sensation.
2. Describe lesion
3. Describe Necrotic tissue, probe sinuses with sterile probe to determine the extent of disease.
4. Culture pus for aerobic and anaerobic organisms
5. Begin broad spectrum antibiotic until appropriate antibiotics can be given according to culture and sensitivity.
6. Medical Management of Diabetes — Blood sugar monitoring and anti diabetic measures to achieve good glycemic control, Doppler study of vessels.
7. X - ray both feet to exclude osteomyelitis.
8. No weight bearing
   a. Hospitalize with absolute bed rest when indicated.
   b. Crutches or walker when feasible.
9. Surgical Management of the Problem
   a. No soaks
   b. Antibiotics
   c. Medical Management of diabetes
   d. Dressing change atleast once daily.
   e. Surgical debridement, frequently if necessary.
   f. Consideration for possible arterial reconstruction
   g. Drainage or open amputation.
10. Rehabilitation

a. Podiatrist for patient education, preventive maintenance orthotics, healing sandals and special shoes.
b. Nutritionist to advice on diet needs.
c. Surgeon to ensure proper wound healing and proper prosthetics
d. Physician to make final decision about diabetes management.
e. Psychiatrist to return to normal activity.

Principles of Medical Management

1. Pus from ulcers sent for culture and sensitivity.
2. Careful monitoring of the blood glucose levels.
3. Appropriate antidiabetic measures either insulin preparations or oral hypoglycemic drugs.
4. Broad spectrum antibiotics to be started at the onset and change over to other antibiotics depending on the culture and sensitivity report.
5. Patients with limb threatening infections require hospitalization, it is most prudent, initially to administer antibiotics parenterally to ensure adequate serum levels.

Principles of Surgical Management

1. Early recognition and prompt intervention.
2. Control of blood glucose
3. Complete rest of injured area.
4. Careful but complete debridement and drainage of all involved areas.
5. Appropriate antibiotic coverage
6. Wound care and dressings

7. Appropriate vascular reconstructions
8. Careful follow up including podiatric appliances and modified footwear.
9. More experienced consultation as necessary.

Wagner Grade 1 foot

These are patients with superficial ulcers and cellulitis. Infection is controlled with appropriate antibiotics and debridement if required. Ulcers occur because of repetitive pressures Pressure is relieved by complete bed rest, use of total contact cast, walker, braces etc. Associated vascular insufficiency has to be corrected by vascular reconstruction.

Wagner Grade 2 and Grade 3 feet

These are patients with deep ulcers, with or without complications like abscesses and osteomyelitis. Aggressive surgical debridement, excision of the infected bone and vascular reconstruction if necessary is the mainstay of the treatment. To avoid recurrence education about foot care is essential.

Wagner Grade 4 and 5 feet

These are patients with localized or extensive gangrene. Management is by appropriate minor or major amputation followed by vascular reconstruction.

METHODOLOGY

This study was done in the Department of General Surgery, KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belgaum from January 2013 to December 2013.

Study design

The study design was a one year cross sectional study.

Study period and duration

This study was done for a period of one year from January 2013 to December 2013.

Place

This study was carried out under the Department of General Surgery, KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belgaum a teaching hospital attached to KLE University's Jawaharlal Nehru Medical College, Belgaum.

Source of Data

Patients diagnosed to diabetic foot ulcer were studied.

Sample size

A total of 100 patients with diabetic foot ulcer were studied.

Sampling procedure

The study was conducted on 100 patients fulfilling selection criteria during the study period.

Selection criteria

Inclusion

- Patients having diabetic foot ulcers secondary to diabetes mellitus

Exclusion

- Patients who had an ulcer previously at the same site.
- Foot ulcers secondary to venous disorders and arterial disorders other than diabetes mellitus

Ethical clearance

The study was approved from the Ethical and Research Committee, Jawaharlal Nehru Medical College, Belgaum.

Informed Consent

Patients with diabetic foot ulcers were evaluated and those fulfilling the selection criteria were informed about the nature of the study and a written informed consent was obtained (Annexure I).

Method of collection of data

The selected patients were interviewed and data such as age, sex, history and clinical presentation were obtained. These patients were subjected to clinical examination and the findings were noted on a predesigned and pretested proforma (Annexure II).

Investigations

The patients were subjected to the following investigations.

- Complete blood picture
- Fasting blood sugar
- Post prandial blood sugar
- Blood urea
- Serum creatinine
- X-ray of Foot
- Culture and sensitivity

Outcome variables

Grading of diabetic foot ulcer

The patients were graded according to Wagner's classification and managed with strict glycemic control, daily dressings, debridement and amputation if needed.

Outcome

The outcome was noted as either;

1. Wound healing without amputation .

2. Wound healing with amputation.

Statistical analysis

The data obtained was coded and entered in Microsoft Excel Spreadsheet. The categorical data was expressed as rates, ratios and percentages and comparison was done using Fisher's exact test. Continuous data was expressed as mean ± standard deviation. A 'p' value of less than or equal to 0.05 at 95% confidence interval was considered as statistically significant. The relative risk of amputation in different grades of diabetic foot ulcer based on Wagner classification was determined.

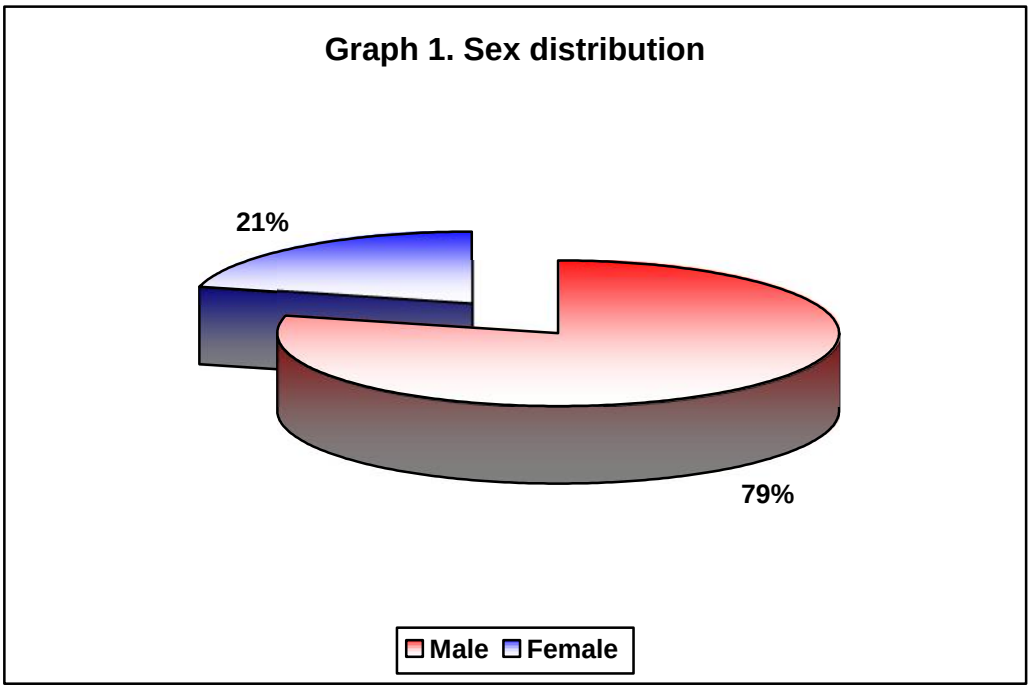
RESULTS

This one year cross sectional study was done in the Department of General Surgery, KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belgaum from January 2013 to December 2013. A total of 100 patients with diabetic foot ulcer were studied.

The data obtained was analysed and the final observations were tabulated as below.

Table 1. Sex distribution

Sex	Distribution (n=100)	
	Number	**Percentage**
Male	79	79.00
Female	21	21.00
Total	**100**	**100.00**



In the present study 79% of the patients were males and 21% were females. The male to female ratio was 3.76:1.

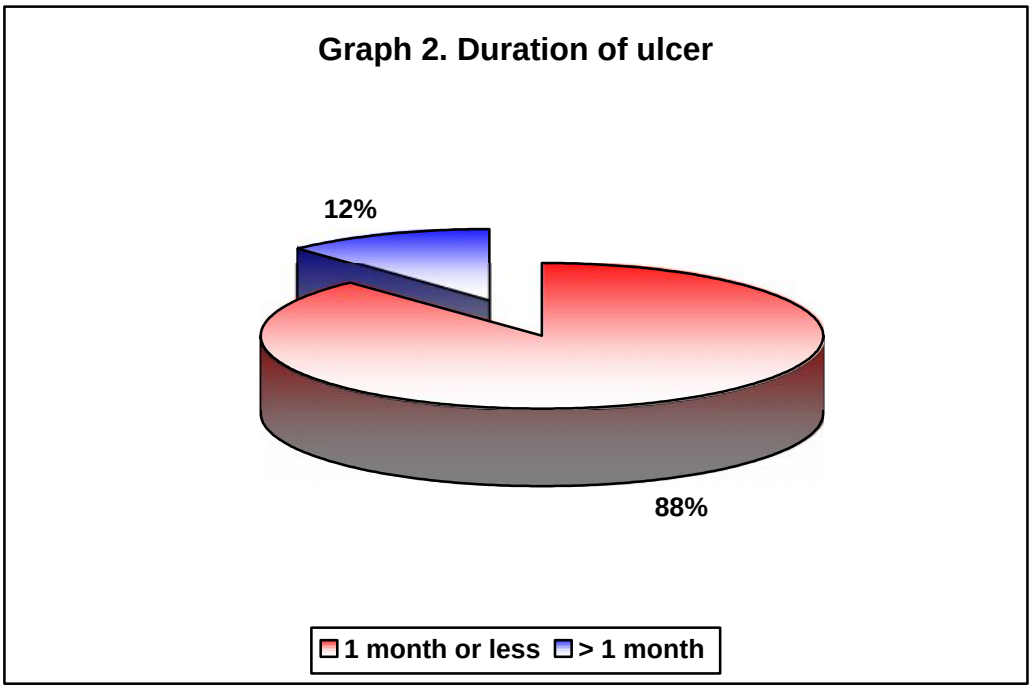
Table 2. Mean age

Sex	Total	Mean		Range	
		Mean	SD	Minimum	Maximum
Male	79	55.1	11.09	26	81
Female	21	58.6	71.84	43	70
Overall	100	55.8	10.45	26	81

In this study the mean age in males was 55.10 ± 11.09 years and in females the same was 58.6 ± 71.84 years. Overall the mean age was noted as 55.8 ± 10.45 years with range 26 being minimum and 81 being maximum.

Table 3. Duration of ulcer

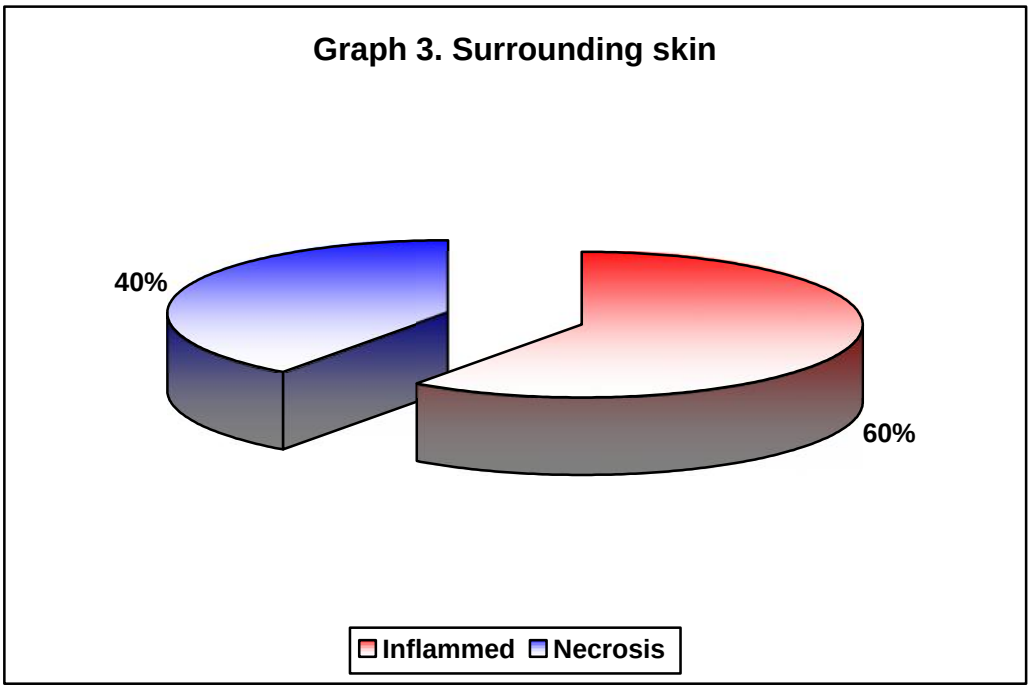
Duration	Distribution (n=100)	
	Number	Percentage
1 month or less	88	88.00
> 1 month	12	12.00
Total	**100**	**100.00**



In this study the duration of ulcer was less than or equal to one month in 88% of the patients.

Table 4. Surrounding skin

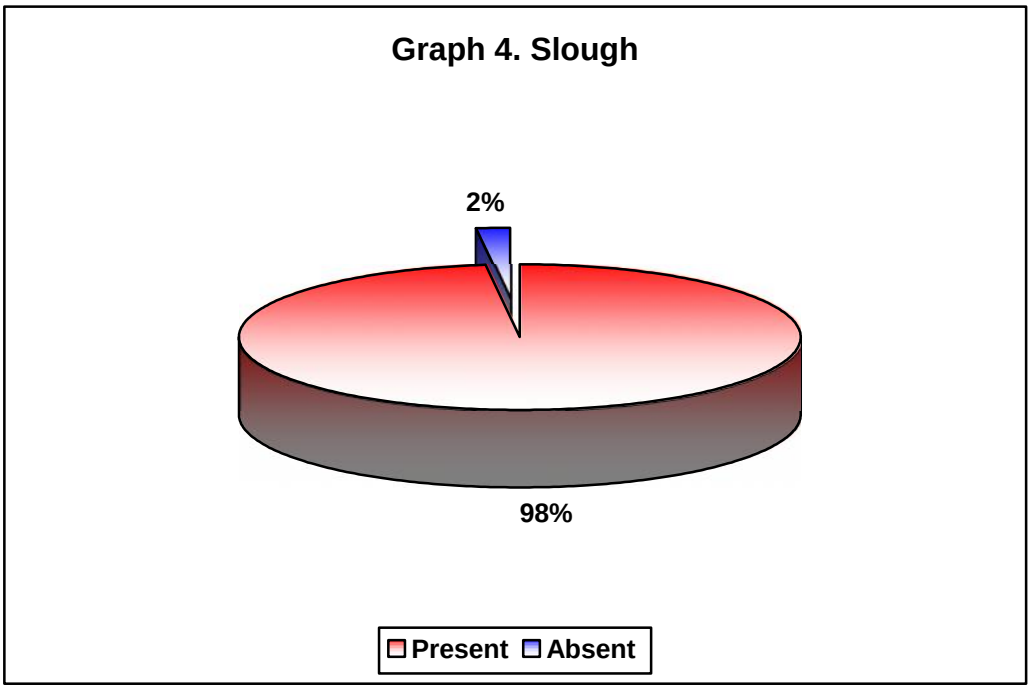
Surrounding Skin	Distribution (n=100)	
	Number	**Percentage**
Inflammed	60	60.00
Necrosis	40	40.00
Total	**100**	**100.00**



In the present study surrounding skin was found to be inflamed in 60% of the patients and necrosis was noted in 40% of the patients.

Table 5. Slough

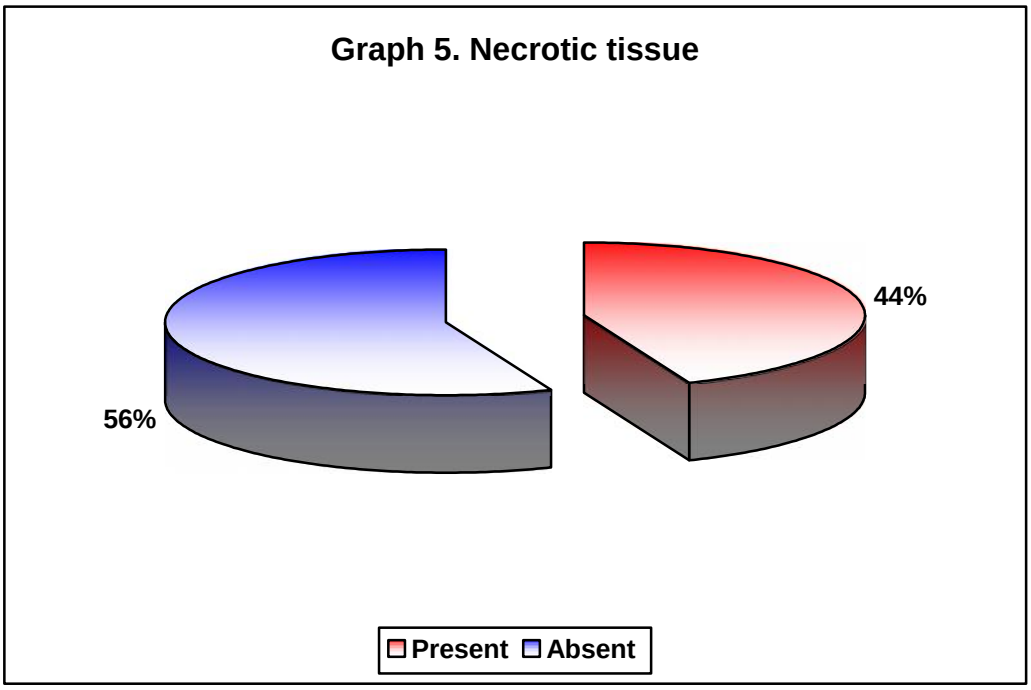
Slough	Distribution (n=100)	
	Number	**Percentage**
Present	98	98.00
Absent	2	2.00
Total	**100**	**100.00**



In the present study presence of slough was noted in 98% of the patients.

Table 6. Necrotic tissue

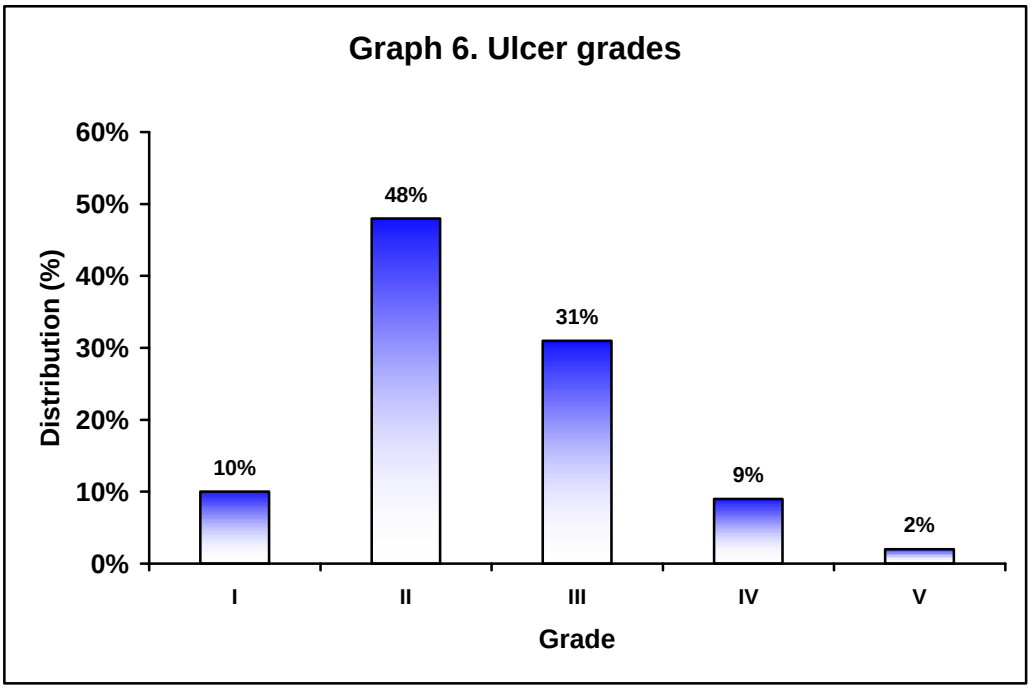
Necrotic tissue	Distribution (n=100)	
	Number	Percentage
Present	44	44.00
Absent	56	56.00
Total	**100**	**100.00**



In this study 44% of the patients had necrotic tissue.

Table 7. Ulcer grades

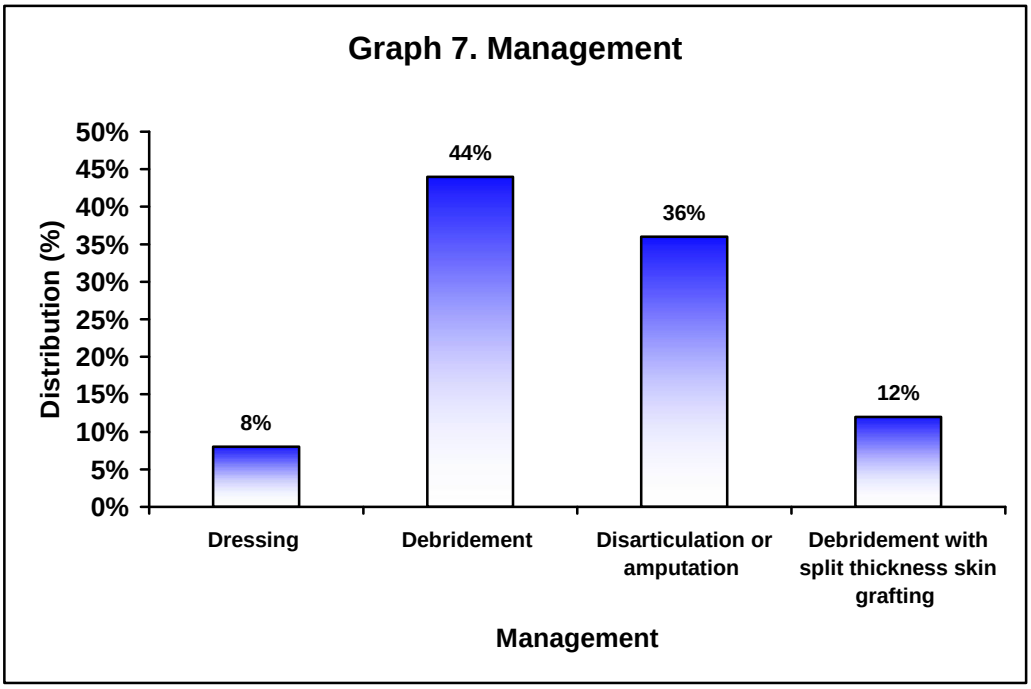
Grade	Distribution (n=100)	
	Number	**Percentage**
I	10	10.00
II	48	48.00
III	31	31.00
IV	9	9.00
V	2	2.00
Total	**100**	**100.00**



In the present study based on Wagner's Classification, most of the patients (48%) had Grade II diabetic foot ulcers followed by grade III (31%), grade I (10%), grade IV (9%) and grade V (2%).

Table 8. Management

Management	Distribution (n=100)	
	Number	**Percentage**
Dressing	8	8.00
Debridement	44	44.00
Disarticulation or amputation	36	36.00
Debridement with split thickness skin grafting	12	12.00
Total	**100**	**100.00**



In this study 44% of the patients needed debridement followed by disarticulation or amputation in 36%, debridement with split thickness skin grafting in 12% and dressing in 8%.

Table 9. Outcome and ulcer grade

Ulcer grade	Outcome			
	Healing without amputation		Healing with amputation	
	Number	Percentage	Number	Percentage
I	10	100.00	0	0.00
II	38	79.17	10	20.83
III	16	51.61	15	48.39
IV	0	0.00	9	100.00
V	0	0.00	2	100.00
Total	**64**	**64.00**	**36**	**36.00**

p<0.001

In the present study maximum patients presented with grade II ulcer (48%). Among them 79% of the patients had healing without amputation and 20.83% had healing with amputation.

As the grade of ulcer increases, the percentage of patients in each grade going for Amputation increases.

Table 10. Outcome and ulcer grade

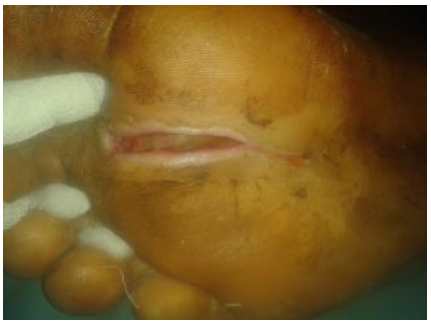
Ulcer grade	Outcome			
	Healing without amputation		Healing with amputation	
	Number	Percentage	Number	Percentage
I and II	48	82.76	10	17.24
III, IV and V	16	38.10	26	61.90
Total	**64**	**64.00**	**36**	**36.00**

p<0.001; Relative risk-3.59; 95% CI - 1.95-6.62;

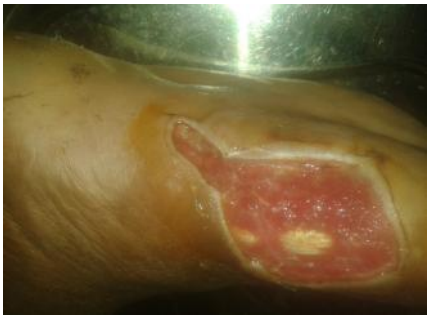
In this study 58% of the patients presented with grade I and II diabetic foot ulcers. Among these 82.76% had healing without amputation compared to 17.24% of the patients needed amputation.

Of the 42% patients with grade III, IV and V diabetic foot ulcers, 61.9% needed amputation whereas 38.1% healed without amputation.

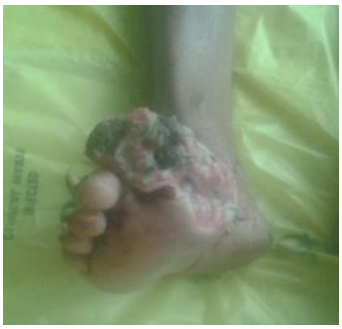
Patients belonging to Grade III, IV and V had 3.59 times higher risk of amputation compared to patients belonging to grade I and II. **(**p<0.001; Relative risk-3.59; 95% CI- 1.95 to 6.62).



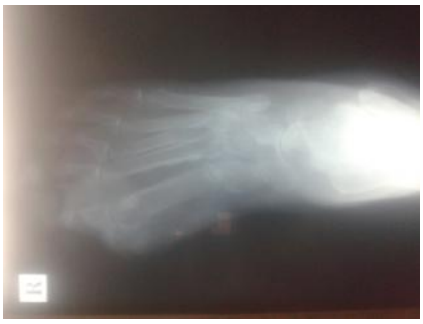
Photograph 1. Grade I ulcer



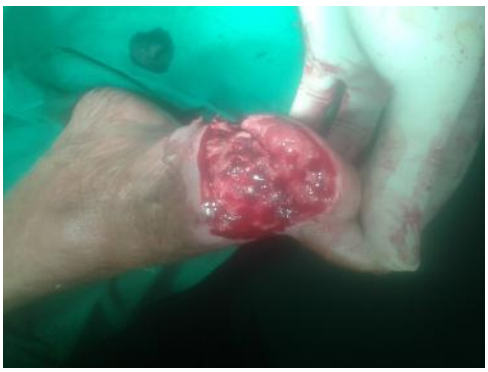
Photograph 2. Grade II ulcer with exposed tendon



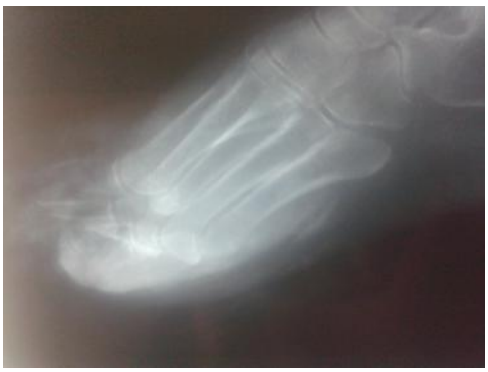
Photograph 3. Grade III ulcer with osteomyelitis



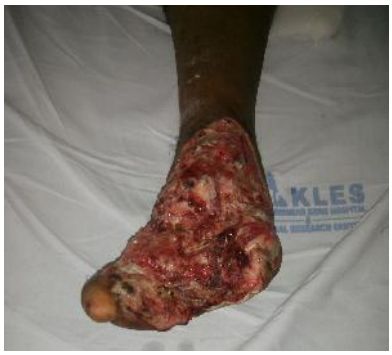
Photograph 4. X-ray foot AP view of grade III ulcer showing osteomyelitis



Photograph 5. Grade III ulcer after amputation



Photograph 6. X-ray foot lateral view



Photograph 7. Grade IV ulcer after amputation

DISCUSSION

Diabetes mellitus-related foot ulceration is very common. As a result of neuropathy, peripheral vascular disease, and infection, patients with diabetes are prone to develop diabetic foot problems that may eventually require a lower-extremity amputation. Of all individuals with diabetes mellitus, 15 percent will be affected by ulceration at least once in their lifetime.[8,9]

Because diabetic foot ulceration is a serious problem and because ulcers are heterogeneous in terms of etiology, anatomic location, depth of tissue involvement, and associated circumstances, including the presence or absence of infection, classification is needed in order to predict ulcer outcome and conduct clinical trials.[8]

Several classification systems for diabetic foot ulcers have been proposed. These classification systems have to comply with certain characteristics, such as precision, flexibility, specificity, and simplicity. They also must be applicable for education and communication between all care providers, including nurses, general practitioners, and specialists. They can be of great help for the assessment of treatment schemes. The classifications most frequently used are based on factors like infection, neuropathy, vasculopathy, and the extent (surface and depth) of the ulcer.[7]

The best known and widely available classifications are Meggit/Wagner, Gibbon's, Frykberg's and Coleman's, Forrest's, Knighton's, the Texas Diabetic Wound Classification, and the Ten-Level Seattle Wound Classification System.[8-15]

Each of these classifications were developed to accomplish a particular objective, utilizes different criteria, and categorizes lesions according to different

rationales. Only a few of these classifications were evaluated for the assessment of the prognosis on salvage of the ulcerated diabetic limb. Wagner's classification is the most widely accepted Grading system for Lesions of Diabetic foot. This study assessed the role of Wagner wound classification in predicting the outcome of diabetic foot ulcer.[7]

The present one year cross sectional study included a total of 100 patients with diabetic foot ulcer at the Department of General Surgery, KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belgaum from January 2013 to December 2013. The diabetes foot ulcers were graded according to the Wagner's classification.

The occurrence of DFUs mostly in males and middle aged subjects has been reported by several researchers.[82,85] In the present male preponderance was noted as majority of the patients were males (79%) with higher male to female ratio (3.76:1). These findings were consistent with a study from Varanasi to determine risk factors for foot ulceration where 71.13% of the patients were males and 28.86% were females.[85] The mean age in the present study was found to be 55.8 ± 10.45 years suggesting predominant involvement of elderly population. A study[85] from Varanasi to determine risk factors for foot ulceration reported mean age of the patients with diabetic foot ulcers as 55.25 years.

In this study the duration of ulcer was less than one month in majority of the patients (88%). The surrounding skin was inflamed in 60% of the patients while necrosis in 40% and of the patients. Presence of slough was noted in 98% and 44% of the patients had necrotic tissue.

In the present study most of the patients (48%) had Grade II diabetic foot ulcers followed by grade III (31%), grade I (10%), grade IV (9%) and grade V (2%) based on Wagner's Classification. Recently a study[85] to evaluate diabetic foot ulcer according to Wagner's Classification at a rural hospital in Maharashtra, India found that the commonest presentation was Wagner's Grade 2 diabetic foot.

In this study based on diabetic ulcer grade, most of the patients (44%) underwent debridement followed by disarticulation or amputation (36%), debridement with split thickness skin grafting (12%) and dressing (8%).

In the present study 48 patients had grade II diabetic foot ulcer. Of these majority of the patients (79%) had healing without amputation but a substantial number of patients (20.83%) needed amputation. However of the 10 patients who had grade I diabetic foot ulcer all (100%) had healing without amputation. Further of the 31 patients with grade III ulcer, 51.61% had healing without amputation compared to 48.39% who needed amputation. In those with grade IV and V all the patients needed amputation ($p<0.001$). Overall these findings showed an increasing trend of healing with amputation with advanced ulcer grades significantly. To evaluate the effect of Wagner classification on outcome, we calculated relative risk of amputation in patients belonging to Wagner grade III, IV and V compared to those belonging to grade I and II. It was observed that, of the 58 patients in grade I and II, 82.76% had healing without amputation and 17.24% underwent amputation ($p<0.001$).Of 42 patients in grades III, IV and V, 38.1% healed without amputation and 61.9% needed amputation. Patients with Grade III, IV and V ulceration were found to be 3.59 times higher at risk for amputation than Grade I and II ulcerations. ($p<0.001$,RR=3.59; 95% CI-1.95 to 6.62).

The findings of the present study were consistent with a study by Oyibo et al[86] who reported that the Wagner grade significantly correlate with the risk of amputation. Calhoun et al[19] reported that increased Wagner grade was associated with a higher treatment failure. Ulcers of Wagner grades 4 and 5 denote the presence of local or diffuse gangrene, which are usually due to a combination of ischemia and infection. It is thus not surprising that grade 4 and 5 ulcers were very strongly associated with amputation in our study. A study[87] conducted in Pakistan also reported that, lesser grade lesions responded well to conservative treatment with antibiotics and surgical debridement while those with higher Grades IV and V needed amputation. They also concluded that Grading diabetic foot lesions according to Wagner's classification helps in correlating appropriate treatment to Proper Grade of lesion with better outcome. Another study[88] in Karachi to know the role of wound classification in predicting the outcome of diabetic foot ulcer showed that grading diabetic foot ulcer affects and predicts the outcome and amputation rates increase with increase in the Wagner's grade. Calhoun et al.[19] found that classification of foot lesion not only enabled them to institute proper treatment regimen, but additionally, when such protocols were followed, the treatment outcome were significantly more successful than when protocols were not followed.

Overall the present study showed grading of diabetic foot ulcer based on Wagner classification affects and predicts the outcome and further, amputation rates increase with increase in grade.

CONCLUSION

In this study, we found that Grading of diabetic foot ulcer based on Wagner's classification affects and predicts the outcome and the risk of amputation increases with increasing grade. Most of the patients admitted for diabetic foot ulcers in our hospital belonged to Wagner's grade II (48%).

SUMMARY

Diabetes mellitus-related foot ulceration is very common. Several classification systems for diabetic foot ulcers have been proposed. The present was intended to assess the role of Wagner wound classification in predicting the outcome of diabetic foot ulcer.

This present one year cross sectional study was carried out at the Department of General Surgery, KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belgaum. A total of 100 patients with diabetic foot ulcer who presented during the study period that is, from January 2013 to December 2013 were included. The diabetic foot ulcers were graded according to the Wagner's classification.

In this study majority of the patients were males (79%) and the male to female ratio was 3.76:1. The mean age was noted as 55.8 ± 10.45 years with range 26 being minimum and 81 being maximum. Majority of the patients had duration of ulcer less than one month (88%). Surrounding skin was found to be inflamed in 60% of the patients, necrosis in 40% and presence of slough was noted in 98% of the patients while 44% of the patients had necrotic tissue. Based on Wagner's Classification, most of the patients (48%) had Grade II diabetic foot ulcers followed by grade III (31%), grade I (10%), grade IV (9%) and grade V (2%). With regard to management, in 44% of the patients debridement was done and 36% of the patients had disarticulation or amputation in 36%. Of the 48 patients with grade II ulcer, 79% of the patients had healing without amputation and 20.83% had healing with amputation. Of the 58 patients with grade I and II diabetic foot ulcers, 82.76% had healing without amputation compared to 17.24% of the patients who needed

amputation. Patients belonging to Grade III, IV and V had 3.59 times higher risk of amputation compared to patients belonging to grade I and II. **(**$p<0.001$; Relative risk-3.59; 95% CI- 1.95 to 6.62).

Grading of diabetic foot ulcer based on Wagner's classification affects and predicts the outcome and the risk of amputation increases with increasing grade.

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ANNEXURE I – CONSENT FORM

Mr / Mrs / Miss _____ we are requesting you to enrol yourself in study entitled, **"ROLE OF WAGNER'S CLASSIFICATION IN PREDICTING THE OUTCOME IN DIABETIC FOOT ULCER PATIENTS ADMITTED IN KLES DR. PRABHAKAR KORE HOSPITAL-A ONE YEAR CROSS SECTIONAL STUDY"** is being conducted by Dr. **** ****, Post Graduate in Surgery at Jawaharlal Nehru Medical College Belgaum, Karnataka. Under guidance of Dr. **** **** Professor, Department of Surgery, Jawaharlal Nehru Medical College, Belgaum, under KLE University, Belgaum.

Respected Sir/Madam, we request you to enrol yourself to participate in our study as you are eligible for participating in this study. During the study you will be asked some questions regarding your present complaints and your are suppose to answer to the best of your knowledge.

Your participation in research is voluntary. If you decide to participate you are free to withdraw at any time.

Purpose of study

ROLE OF WAGNER'S CLASSIFICATION IN PREDICTING THE OUTCOME IN DIABETIC FOOT ULCER PATIENTS ADMITTED IN KLES DR. PRABHAKAR KORE HOSPITAL-A ONE YEAR CROSS SECTIONAL STUDY

This study will be conducted to know the importance of classifying diabetic ulcer before giving treatment so that we can compare the outcomes better and so identify measures to reduce the morbidity and mortality due to diabetic foot disease.

Procedure involved

If you agree to enrol yourself in this study, you will be informed in detail about the procedure. You will be interviewed regarding your present, past and family history then you will be clinically examined in detail and investigated accordingly. Your lesion will be classified according to Wagner's classification. Then will be treated appropriately and the outcome measured as wound healing with granulation tissue with or without skin grafting or amputation.

Benefits and Risks

The benefits of taking part in this research are you that your foot ulcer will be graded before treatment and this will help in correlating appropriate treatment to proper grade of lesion with better outcome. There are no significant risks involved.

Voluntary participation / Withdrawal

Taking part in the study is voluntary. You may choose not to enrol yourself in this study. Your decision will not change present or future health care services offered to you at KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belgaum.

Alternatives

Even if you decline the participation in the study, you will get the routine line of management.

Privacy and confidentiality

The only people to know that you are a research subject are members of the

research team. No information about you or information provided by you during the research will be disclosed to other without your written permission except in emergency to protect your rights and welfare or if required by law.

Authorization to Publish Results

When the results of the research are published or discussed, in a conference, no information will be displayed that would disclose your identity. Any information that is obtained in connection with this study and that can be identified with you will remain confidential.

Financial Incentives for participation

No financial incentives are being offered to enrolled patients. It is purely being done with the idea of research and all the cost of the study will be borne by the investigator.

Compensation

In the event of injury, related to the study, treatment will be made available at KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belgaum. There is compensation or payment for such medical treatment by law.

Questions/Contact details

If you have any queries, in future or in case of study related injury or illness, you may contact. Dr. **** ***** at Department of Surgery, KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belgaum Phone Number **** *****.

If you have any queries about your rights as a study subject, you may call

Dr. **** ****** Professor and Head, Department of Pathology, J. N. Medical College Institutional Ethical Committee for Human Subjects Research, Ph. ***** ***** at J. N. Medical College, Belgaum.

CONSENT TO PARTICIPATE IN A RESEARCH STUDY:

I, _____ voluntarily agree to take part in this study, by signing this consent form I am not giving up my legal rights. I may withdraw at any time. I am signing after having read, or been read to me in the vernacular language including risks and the benefits and having all my questions answered.

Subject Name: _____

Signature of the participant _____ Date _____
Or Left thumb print

Witness name: _____

Signature: _____ Date _____

Investigator's name: _____

Signature: _____ Date _____

Place: _____

ANNEXURE II

DATA COLLECTION INSTRUMENT

Title: ROLE OF WAGNER'S CLASSIFICATION IN PREDICTING THE OUTCOME IN DIABETIC FOOT ULCER PATIENTS ADMITTED IN KLES DR. PRABHAKAR KORE HOSPITAL-A ONE YEAR CROSS SECTIONAL STUDY

ID Number:

OPD Number:

IPD Number:

Date:

Unit:

Patients Name: F_____ M _____ S_____

Age:

Address:

H. NO._____ Street _____ Place _____

Taluka _____ District _____

Tel Number_____

Mobile Number _____

I. SCREENING

1. Is the patient diabetic

   a. Yes b. No

2. Is there any ulcer on the foot

   a. Yes b. No

3. Did the patient have ulcer previously at the same site

   a. Yes b. No

4. Is the ulcer secondary to any venous disorder or arterial disorders

   a. Yes b. No

5. Is the patient willing to give consent

   a. Yes b. No

6. Was the consent obtained

   a. Yes b. No

7. Final result

   a. Eligible participating

   b. Eligible refusal

   c. Ineligible

   If the answer for the above question is A the person is eligible

III DEMOGRAPHIC DETAILS AND HISTORY

1. Occupation

   a. Not working

   b. Working

   c. Labourer

   d. Professional

2. Eduation

   a. Illiterate

   b. Read

   c. Write

   d. Primary

   e. Secondary

f. Graduate

g. Post graduate

3. What was the chief complaint he / she has come with ?

4. What is the mode of onset of the ulcer and how long has he/she had the ulcer ?

5. Any treatment taken previously for the ulcer

6. Any past medical history of;

Peripheral neuropathy	Yes	No
Nephropathy	Yes	No
Retinopathy	Yes	No
PVD	Yes	No
CVD	Yes	No

7. Any past history of surgery ? If yes specify

8. Diabetic status

Type Duration

Is the patient on any medication

Oral hypoglycemics

Insulin

III. Examination

1. Height (Cms)
2. Weight (Kgs)
3. BMI (Kg/m^2)
4. Pulse rate
5. Blood pressure
    1. Systolic (mm Hg)
    2. Diastolic (mm Hg)

6. Are all the peripheral pulses felt

	Right	Left
Femoral artery		
Popliteal artery		
Anterior tibial artery		
Posterior tibial artery		
Dorsalis pedis artery		

7. Is cardiovascular system normal ?

   a. Yes b. No

8. Is respiratory system normal ?

   a. Yes b. No

9. Is sensory system normal ?

   a. Yes b. No

10. Is motor system normal ?

    a. Yes b. No

11. Description of ulcer

    1. Size:
    2. Shape:
    3. Edge:
    4. Margin:
    5. Floor:
    6. Base:
    7. Discharge:
    8. Surrounding skin
    9. Slough

10. Necrotic tissue

11. Any gangrenous tissue

IV. Investigations

CBC :

Hb- ( )

TLC- ( )

DC- (N- , L- , M- ,E-)

FBS : ( ) Date Time

Blood Urea ( )

Sr. Creatinine ( )

Urine :

Routine

Microscopy

X-ray

AP view

Lat. View

Pus culture / sensitivity

V. Grading of ulcer according to Wagner Classification

VI. Treatment given to the patient

VII Outcome: Measured as either

a. Wound healing without amputation

b. Wound healing with Amputation

ANNEXURE III – KEY TO MASTER CHART

-	-	Absent
+	-	Present
a	-	Dressing
A	-	Healing without amputation
b	-	Debridement
B	-	Healing with amputation
b+stsh	-	Debridement with split thickness skin grafting
c	-	Disarticulation or amputation
d	-	Days
F	-	Female
I	-	Inflammed
M	-	Male
m	-	Months
NAD	-	No abnormality detected
NEC	-	Necrosis
w	-	Week

ANNEXURE III - MASTER CHART

Serial Number	In/Out patient number	Sex	Age (Years)	Ulcer duration	Peripheral pulses	Sensory system	Motor system	Ulcer details						Ulcer grade	Management	Outcome
								Ulcer	Discharge	Surrounding skin	Slough	Necrotic tissue	Gangrenous tissue			
1	559732	M	62	1w	+	NAD	NAD	+	+	NEC	+	+	-	II	c	B
2	561329	M	63	2m	+	NAD	NAD	+	+	NEC	+	+	-	III	b	A
3	558943	F	64	1m	+	NAD	NAD	+	+	NEC	+	+	-	II	c	B
4	566511	M	67	2m	+	NAD	NAD	+	+	NEC	+	+	-	III	b	A
5	565591	M	68	1m	+	NAD	NAD	+	+	NEC	+	+	-	II	c	B
6	561760	F	66	1.5m	+	NAD	NAD	+	+	NEC	+	+	-	II	c	B
7	566692	M	52	1m	+	NAD	NAD	+	+	I	+	-	-	II	b	A
8	569713	M	53	20d	+	NAD	NAD	+	+	I	+	+	-	III	b	A
9	570226	F	54	15d	+	NAD	NAD	+	+	I	+	-	-	II	b	A
10	570116	M	53	4m	+	NAD	NAD	+	+	NEC	+	+	-	III	b+stsh	A
11	520227	M	55	35d	+	NAD	NAD	+	+	I	+	-	-	II	b	A
12	510453	F	43	1w	+	NAD	NAD	+	+	NEC	+	+	-	III	b+stsh	A
13	510452	M	44	2w	+	NAD	NAD	+	+	I	+	-	-	II	b	A
14	511318	M	49	3w	+	NAD	NAD	+	+	I	+	+	-	III	b+stsh	A
15	513841	F	48	1w	+	NAD	NAD	+	+	NEC	+	+	-	III	b	A
16	513828	M	48	1m	+	NAD	NAD	+	+	I	+	-	-	II	b	A
17	513792	M	71	2w	+	NAD	NAD	+	+	NEC	+	+	-	III	b	A
18	515080	F	70	1m	+	NAD	NAD	+	+	I	+	-	-	II	b	A
19	516235	M	72	3w	+	NAD	NAD	+	+	NEC	+	+	-	III	b	A
20	517037	M	74	2m	+	NAD	NAD	+	+	NEC	+	+	-	III	b	A
21	535705	F	61	1w	+	NAD	NAD	+	+	I	+	-	-	I	a	A
22	538584	M	63	1w	+	NAD	NAD	+	+	NEC	+	+	-	II	c	B
23	540730	M	64	40d	+	NAD	NAD	+	+	NEC	+	+	+	IV	c	B
24	546129	M	62	15d	+	NAD	NAD	+	+	NEC	+	+	-	II	c	B
25	547286	F	65	2m	+	NAD	NAD	+	+	NEC	+	+	+	IV	c	B
26	548588	M	61	1w	+	NAD	NAD	+	+	I	+	-	-	I	a	A
27	556133	M	63	20d	+	NAD	NAD	+	+	NEC	+	+	-	II	c	B
28	561072	F	64	35d	+	NAD	NAD	+	+	NEC	+	+	-	III	b	A
29	519238	M	61	2m	+	NAD	NAD	+	+	NEC	+	+	+	IV	c	B
30	515801	M	62	15d	+	NAD	NAD	+	+	I	+	-	-	II	b	A
31	515924	M	64	15d	+	NAD	NAD	+	+	NEC	+	+	-	II	c	B
32	524145	F	68	1m	+	NAD	NAD	+	+	NEC	+	+	-	III	b	A
33	528559	M	67	1w	+	NAD	NAD	+	+	I	+	-	-	I	a	A
34	528619	M	68	15d	+	NAD	NAD	+	+	NEC	+	+	-	III	b+stsh	A
35	530672	M	67	10d	+	NAD	NAD	+	+	NEC	+	+	-	II	c	B
36	559391	F	65	15d	+	NAD	NAD	+	+	I	+	-	-	I	a	A
37	523175	M	64	1w	+	NAD	NAD	+	+	NEC	+	+	-	II	c	B

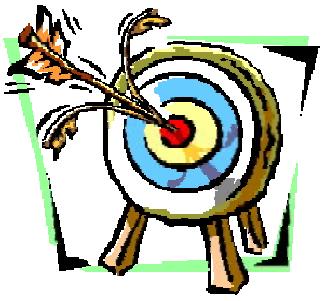
ANNEXURE III - MASTER CHART

Serial Number	In/Out patient number	Sex	Age (Years)	Ulcer duration	Peripheral pulses	Sensory system	Motor system	Ulcer details						Ulcer grade	Management	Outcome
								Ulcer	Discharge	Surrounding skin	Slough	Necrotic tissue	Gangrenous tissue			
38	517209	M	66	1m	+	NAD	NAD	+	+	NEC	+	+	-	III	b+stsh	A
39	518245	F	67	1m	+	NAD	NAD	+	+	NEC	+	+	+	IV	c	B
40	517289	M	64	35d	+	NAD	NAD	+	+	NEC	+	+	+	IV	c	B
41	517274	M	42	2w	+	NAD	NAD	+	+	I	+	-	-	III	c	B
42	519546	M	49	1w	+	NAD	NAD	+	+	I	+	-	-	II	b+stsh	A
43	519989	M	48	2w	+	NAD	NAD	+	+	I	+	-	-	II	b	A
44	520301	M	47	1m	+	NAD	NAD	+	+	NEC	+	+	+	IV	c	B
45	520674	M	47	20d	+	NAD	NAD	+	+	I	+	-	-	II	b+stsh	A
46	522002	M	48	14d	+	NAD	NAD	+	+	NEC	+	-	-	III	c	B
47	521987	M	42	1w	+	NAD	NAD	+	+	NEC	-	-	-	I	a	A
48	522108	M	45	1w	+	NAD	NAD	+	+	NEC	+	+	-	III	c	B
49	523830	M	48	2w	+	NAD	NAD	+	+	I	+	-	-	II	b+stsh	A
50	524438	M	49	1w	+	NAD	NAD	+	+	I	+	+	-	III	c	B
51	524498	M	49	1m	+	NAD	NAD	+	+	I	+	-	-	II	b	A
52	525527	M	48	10d	+	NAD	NAD	+	+	NEC	+	+	-	III	b+stsh	A
53	525421	M	48	7d	+	NAD	NAD	+	+	I	+	-	-	II	b+stsh	A
54	528927	M	48	10d	+	NAD	NAD	+	+	I	+	-	-	II	b+stsh	A
55	528878	M	48	40d	+	NAD	NAD	+	+	NEC	+	+	+	V	c	B
56	530050	M	47	10d	+	NAD	NAD	+	+	I	+	-	-	II	b	A
57	530953	M	45	1m	+	NAD	NAD	+	+	I	+	-	-	III	c	B
58	531204	M	47	7d	+	NAD	NAD	+	+	I	+	-	-	II	b	A
59	531181	M	49	1m	+	NAD	NAD	+	+	I	+	-	-	III	c	B
60	535742	M	49	8d	+	NAD	NAD	+	+	I	+	-	-	II	b	A
61	538277	M	51	1w	+	NAD	NAD	2x2	+	I	+	+	+	IV	c	B
62	538269	M	52	1m	+	NAD	NAD	1x2	+	I	+	-	-	II	b	A
63	538226	F	53	1w	+	NAD	NAD	1A	+	NEC	+	+	+	IV	c	B
64	538229	M	55	1w	+	NAD	NAD	+	+	I	+	-	-	II	b	A
65	539809	F	56	12d	+	NAD	NAD	+	+	I	+	-	-	II	b	A
66	539808	M	57	1w	+	NAD	NAD	+	+	NEC	-	-	-	I	a	A
67	539739	F	51	20d	+	NAD	NAD	+	+	NEC	+	+	+	IV	c	B
68	539716	M	54	10d	+	NAD	NAD	+	+	I	+	-	-	II	b	A
69	539676	F	52	20d	+	NAD	NAD	+	+	I	+	-	-	II	b	A
70	539776	M	52	1m	+	NAD	NAD	+	+	I	+	+	-	III	c	B
71	539758	F	55	1w	+	NAD	NAD	+	+	I	+	+	-	III	c	B
72	539984	M	53	10d	+	NAD	NAD	+	+	I	+	-	-	II	b	A
73	540029	F	56	12d	+	NAD	NAD	+	+	I	+	-	-	II	b	A
74	540809	M	54	2w	+	NAD	NAD	+	+	I	+	-	-	III	c	B
75	541142	F	57	1m	+	NAD	NAD	+	+	I	+	-	-	III	b+stsh	A
76	541072	M	55	2w	+	NAD	NAD	+	+	I	+	-	-	III	c	B

ANNEXURE III - MASTER CHART

Serial Number	In/Out patient number	Sex	Age (Years)	Ulcer duration	Peripheral pulses	Sensory system	Motor system	Ulcer details						Ulcer grade	Management	Outcome
								Ulcer	Discharge	Surrounding skin	Slough	Necrotic tissue	Gangrenous tissue			
77	540940	F	58	2w	+	NAD	NAD	+	+	I	+	-	-	III	c	B
78	541017	M	57	1w	+	NAD	NAD	+	+	I	+	-	-	II	b	A
79	541018	F	57	1w	+	NAD	NAD	+	+	I	+	-	-	II	b	A
80	536823	M	58	6d	+	NAD	NAD	+	+	I	+	-	-	II	b	A
81	539481	M	72	1w	+	NAD	NAD	+	+	I	+	+	-	III	c	B
82	542569	M	73	10d	+	NAD	NAD	+	+	NEC	+	-	-	I	b	A
83	542677	M	74	2w	+	NAD	NAD	+	+	I	+	+	-	III	c	B
84	542464	M	39	1w	+	NAD	NAD	+	+	I	+	-	-	II	b	A
85	540258	M	38	2w	+	NAD	NAD	+	+	I	+	+	-	III	c	B
86	540880	M	58	20d	+	NAD	NAD	+	+	I	+	-	-	II	b	A
87	542321	M	52	7d	+	NAD	NAD	+	+	NEC	+	-	-	I	a	A
88	543427	M	35	10d	+	NAD	NAD	+	+	I	+	-	-	II	b	A
89	547118	M	75	7d	+	NAD	NAD	+	+	I	+	-	-	II	b	A
90	547608	M	38	10d	+	NAD	NAD	+	+	I	+	-	-	II	b	A
91	547238	M	68	2w	+	NAD	NAD	+	+	I	+	-	-	II	b	A
92	548498	M	81	1w	+	NAD	NAD	+	+	I	+	-	-	II	b	A
93	550876	M	67	1m	+	NAD	NAD	+	+	NEC	+	-	-	I	a	A
94	551154	M	64	1m	+	NAD	NAD	+	+	NEC	+	+	+	V	c	B
95	551923	M	26	2w	+	NAD	NAD	+	+	I	+	-	-	II	b	A
96	551897	M	32	1w	+	NAD	NAD	+	+	I	+	-	-	II	b	A
97	551824	M	52	1w	+	NAD	NAD	+	+	I	+	-	-	I	b	A
98	554576	M	54	1m	+	NAD	NAD	+	+	I	+	+	-	III	c	B
99	554532	M	55	2w	+	NAD	NAD	+	+	I	+	-	-	II	b	A
100	554520	M	35	1m	+	NAD	NAD	+	+	I	+	-	-	II	b	A

Introduction



Objectives

Review of Literature

Methodology

Results

Discussion

Conclusion

Summary

Bibliography

Annexure-I

Annexure-II

Annexure-III