"RANDOMISED CONTROL TRIAL TO STUDY ANTIBACTERIAL EFFECT OF LOW LEVEL LASER THERAPY IN INFECTIVE DIABETIC FOOT ULCERS ADMITTED TO KLES DR. PRABHAKAR KORE HOSPITAL AND MEDICAL RESEARCH CENTRE, BELGAUM"

By

Dr. PRASHANT TUBACHI

Dissertation submitted to the
KLE University, Belgaum, Karnataka

In Partial Fulfillment
of the requirements for the degree of

MASTER OF SURGERY (M.S.)
IN
GENERAL SURGERY

Under the Guidance of

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MAY - 2010

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I hereby declare that this dissertation entitled **"RANDOMISED CONTROL TRIAL TO STUDY ANTIBACTERIAL EFFECT OF LOW LEVEL LASER THERAPY IN INFECTIVE DIABETIC FOOT ULCERS ADMITTED TO KLES DR. PRABHAKAR KORE HOSPITAL AND MEDICAL RESEARCH CENTRE, BELGAUM"** is a bonafide and genuine research work carried out by me under the guidance of **Dr. A. S. GODHI** MS, FICS, Professor and Head, Department of Surgery, Jawaharlal Nehru Medical College, Belgaum-590010.

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LIST OF ABBREVIATIONS USED

CBC	-	Complete blood count
CDC	-	Centre for Disease Control and Prevention
CVD	-	Cardiovascular disease
DM	-	Diabetes mellitus
DOA	-	Date of admission
DOD	-	Date of discharge
FBS	-	Fasting blood sugar
HDL	-	High density lipoprotein
He-Ne	-	Helium – Neon
HNF	-	Hepatocyte nuclear transcription factor
IP	-	In patient
IPF	-	Insulin promoter factor
J/cm^2	-	Joules/centimeter^2
KHz	-	Kilo Hertz
LASER	-	Light amplification stimulated emission of radiation
LDL	-	Low density lipoprotein
LLLT	-	Low level laser therapy
MODY	-	Maturity onset diabetes in young
mW	-	Milli watt
No.	-	Number
OPD	-	Out patient department
PAD	-	Peripheral arterial disease
PAI	-	Plasminogen activating factor
UKB	-	Urinary ketone body

ABSTRACT

Background and objectives

Foot infections are frequent complication of patients with diabetes mellitus, accounting up to 20% of diabetes-related hospital admissions. Major changes can be seen in wounds treated with low level laser therapy (LLLT) (Increased granulation tissue, fibroblast proliferation, collagen synthesis, early epithelialization and enhanced neovascularisation). The objective of study was to assess anti-infective property of LLLT in diabetic foot infections.

Methodology

The present randomized controlled trial was conducted in Department of Surgery, KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belgaum on 60 patients with infected diabetic foot ulcers during January-2008 to December-2008. Patients were divided into two groups using computerized randomization chart. Group I received conventional dressing with betadine and group II received LLLT along with conventional dressing.

Results

In this study control group had 25 males and 5 female and study group had 23 males and 7 females. Maximum number of patients (40) were in the age group of 51 to 65 years. 43.33% of patients had history of DM for a period of 6 to 10 years and 36.66% had diabetes for 10 years or more. Neuropathy was present in 35% patients whereas vasculopathy was present in 10% of the patients. Twenty four (80%) patients had positive ulcer culture in control group and culture

negative ulcer were 6 (20%) on tenth day. In study group culture positive ulcers were 20 (66.66%) and culture negative ulcers were 10 (33.33%) (p=0.3811).

Conclusions

The wound subjected to LLLT with conventional therapy showed decreased infection rate at 10^{th} day of culture as compared to conventional dressing group alone but it was statistically not significant (p=0.3811). Large scale trials required to know the anti-infective property of LLLT. Multicentre trials are required to know the anti-infective property of LLLT.

Key word

Diabetic foot infection; Diabetes mellitus; Low level laser therapy (LLLT).

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INTRODUCTION

Foot infections are a frequent complication of patients with diabetes mellitus, accounting for up to 20% of diabetes-related hospital admissions.[1] 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%.[2] 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%.[3] 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.[4] 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.

The fundamental pathophysiological factors leading to diabetic ulcer remain incompletely understood. The triad of neuropathy, ischaemia and infection is commonly considered as most important. These diabetic ulcers are known to be resistant to conventional treatment and may herald severe complication if not treated wisely. Research findings to date based upon animal, human and invitro cellular studies have shown beneficial effect of low level laser therapy (LLLT) on wound healing and LLLT can play major, adverse effect free, useful role in healing chronic diabetic ulcers resistant to conventional mode of treatment.

Reports of LLLT applied to soft tissue invitro and invivo suggest stimulation of specific healing processes. Major changes seen in the wounds treated with the LLLT include increased granulation tissue, increased fibroblast proliferation, increased collagen synthesis, early epithelialization and enhanced neovascularisation.

In view of no study regarding the anti-infective property of LLLT in diabetic infections the present study was undertaken to know whether Low Level Laser Therapy given daily with clustered probe at 60 mW at 5 KHz frequency, for two minutes at a dose of two to four J/cm^2 along with conventional treatment compared to conventional therapy alone, is effective in reducing the infection.

OBJECTIVES

The objective of the present study was to know the antibacterial property of LLLT in infected diabetic foot ulcers.

REVIEW OF LITERATURE

DIABETES MELLITUS

Diabetes mellitus (DM) refers to a group of common metabolic disorders that share the phenotype of hyperglycemia.[5] Depending on the etiology of the DM, factors contributing to hyperglycemia include

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

The metabolic dysregulation associated with DM causes secondary pathophysiologic changes in multiple organ systems that impose a 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.[6] It also predisposes to cardiovascular diseases. With an increasing incidence worldwide, DM will be a leading cause of morbidity and mortality for the foreseeable 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.[7] Unlike patients with type 1 diabetes mellitus, patients with type 2 are not absolutely dependent upon insulin for life, even though many of them are ultimately treated with insulin.

Historical aspects

Diabetes mellitus is a disease that was recognized in antiquity, but its history has been characterized by numerous cycles of discovery, neglect and rediscovery. Its history may be divided into four major periods that reflect different phases in the understanding and management of the disease. 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.[8]

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. [9]

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.

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 then hypothetical hormone was named 'insulin' by Jean de Meyer in 1909, over a decade before its discovery.

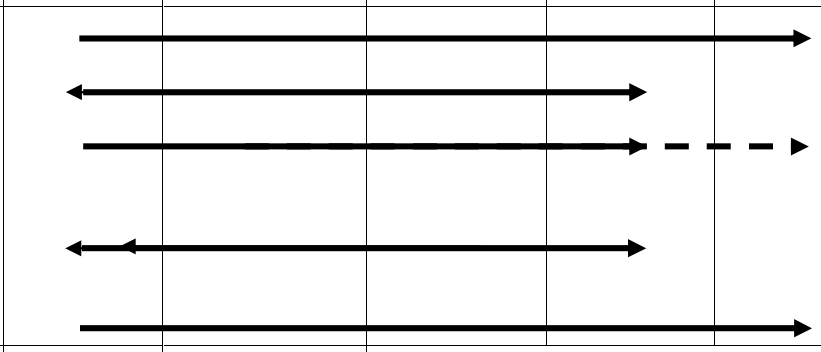
Insulin was discovered at the University of Toronto in 1921, through collaboration between Frederick G. Banting, Charles H. Best, James B. Collip and J.J.R. Macleod.

Definitive proof from the diabetes control and complications trial (DCCT) published in 1993, that strict glycemic control could slow or prevent the development of diabetic microvascular complications.[10]

Classification

DM 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.[6] 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.[11] The two broad categories of DM are designated type 1 and type 2. Both types of diabetes are preceded by a phase of abnormal glucose homeostasis as the pathogenic process progresses. Type 1 diabetes is the result of complete or near-total insulin deficiency. Type 2 DM is a heterogeneous group of disorders characterized by variable degrees of insulin resistance, impaired insulin secretion, and increased glucose production. Distinct genetic and metabolic defects in insulin action and/or secretion give rise to the common phenotype of hyperglycemia in type 2 DM and have important potential therapeutic implications now that pharmacologic agents are available to target specific metabolic derangements. Type 2 DM is preceded by a period of abnormal glucose homeostasis classified as impaired fasting glucose (IFG) or impaired glucose tolerance (IGT).

Table 1: Spectrum of glucose homeostasis and diabetes mellitus[5]

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		

Etiologic Classification of Diabetes Mellitus[5]

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

Type 2 diabetes mellitus is less common in non-Western countries where the diet contains fewer calories and caloric expenditure on a daily basis is higher. However, as people in these countries adopt Western lifestyles, weight gain and type 2 diabetes mellitus are becoming virtually epidemic.

In the United State of America, 10 to 20% of diabetic children and adolescents now have type 2 DM with ethnic minorities, African Americans, Mexicans and Pima Indians having the highest prevalence. 85% of children with type 2 DM are obese.[12]

The worldwide prevalence of DM has risen dramatically over the past two decades, from an estimated 30 million cases in 1985 to 177 million in 2000. Based on current trends, >360 million individuals will have diabetes by the year 2030.[13] Although the prevalence of both type 1 and type 2 DM is increasing worldwide, the prevalence of type 2 DM is rising much more rapidly because of increasing obesity and reduced activity levels as countries become more industrialized. This is true in most countries, and 6 of the top 10 countries with the highest rates are in Asia.[14] In the United States, the Centers for Disease Control and Prevention (CDC) estimated that 20.8 million persons, or 7% of the population, had diabetes in 2005 (~30% of individuals with diabetes were undiagnosed). Approximately 1.5 million individuals (>20 years) were newly diagnosed with diabetes in 2005. In 2005, the prevalence of DM in the United Sates was estimated to be 0.22% in those <20 years and 9.6% in

those >20 years. In individuals >60 years, the prevalence of DM was 20.9%. The prevalence is similar in men and women throughout most age ranges (10.5% and 8.8% in individuals >20 years) but is slightly greater in men >60 years.[14] Worldwide estimates project that in 2030 the greatest number of individuals with diabetes will be 45–64 years of age. The estimated numbers of persons with diabetes in India in the year 2000 were 31.7 million and the number is expected to be 79.4 million by 2030.[15]

More than 220 million people worldwide have diabetes. In 2005, an estimated 1.1 million people died from diabetes. The actual number is likely to be much larger, because although people may live for years with diabetes, their cause of death is often recorded as heart disease or kidney failure. Almost 80% of diabetes deaths occur in low- and middle-income countries. Almost half of diabetes deaths occur in people under the age of 70 years; 55% of diabetes deaths are in women. WHO projects that diabetes deaths will double between 2005 and 2030. Healthy diet, regular physical activity, maintaining a normal body weight and avoiding tobacco use can prevent or delay the onset of diabetes.

Race

The prevalence of type 2 diabetes mellitus varies widely among various racial and ethnic groups. Type 2 diabetes mellitus is becoming virtually 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 recognized increasingly in younger persons, particularly in highly susceptible racial and ethnic groups and the obese. In some areas, more type 2 than type 1 diabetes mellitus is being diagnosed in prepubertal children, teenagers, and young adults. Virtually all cases of diabetes mellitus in older individuals are type 2.

Indian problem

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.[16] The prevalence of diabetes is four to six fold lower in rural areas, which is probably attributed to a conventional lifestyle which has beneficial effect on glucose tolerance (IGT). National Urban Diabetes Survey done in six cities, found age standardized prevalence rates of 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 five percent for DM and 13% prevalence of impaired glucose tolerance.

Causes for diabetic pandemic

The type 2 DM epidemic is tightly and consistently linked to that of obesity, both geographically and chronologically. Many factors like, urbanization and mechanization, together with globalized pattern of western pattern of lifestyle, together with poverty, lack of education and low socio-economic status and inner city

deprivation are emerging as significant risk factors for DM. Lack of breast feeding, low birth weight is associated with insulin resistance and type 2 DM in adult life (especially in subjects who become obese) due to long term metabolic response during poor fetal nutrition.[17]

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,[18] leads to decreased glucose transport in muscle, elevated hepatic glucose production, and increased breakdown of fat.

The genetics of type 2 diabetes are complex and not completely understood, but presumably this disease is related to multiple genes (with the exception of maturity-onset diabetes of the young [MODY]). Evidence supports inherited components for pancreatic beta-cell failure and insulin resistance. Considerable debate exists regarding the primary defect in type 2 diabetes mellitus. Most patients have insulin resistance and some degree of insulin deficiency. However, insulin resistance per se is not the sine qua non for type 2 diabetes mellitus because many people with insulin resistance (particularly those who are obese) do not develop glucose intolerance. Therefore, insulin deficiency is necessary for the development of hyperglycemia. Insulin concentrations may be high, yet inappropriately low for the level of glycemia.

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 excess weight increases risk for diabetes 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.[19] A simplified scheme for the pathophysiology of abnormal glucose metabolism in type 2 diabetes mellitus is depicted in the image below.

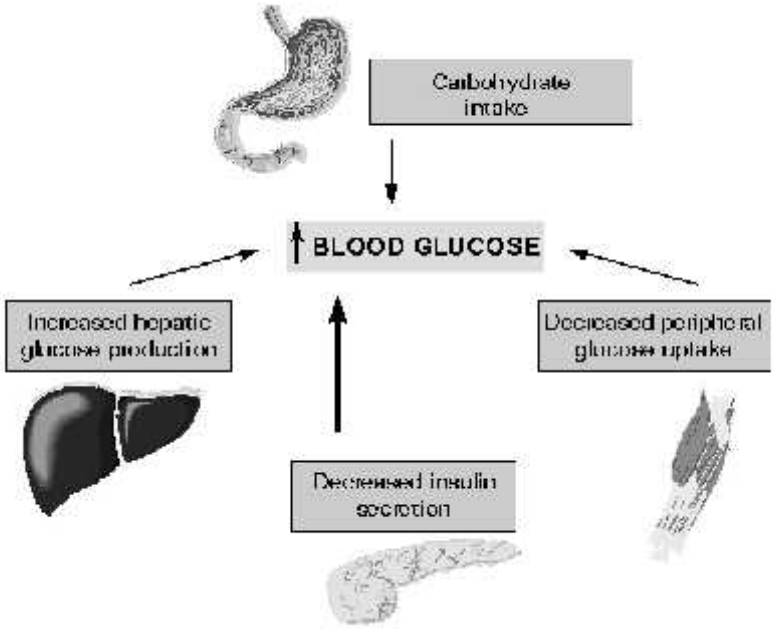


Figure 1: Pathophysiology of type 2 diabetes mellitus

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 (ie, 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 (eg, family history, smoking, hypertension, elevated low-density lipoprotein-cholesterol [LDL-C], low HDL-C), determine cardiovascular risk.

Diagnosis

The National Diabetes Data Group and World Health Organization have issued diagnostic criteria for DM based on the following premises:[6]

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.[6] They are:

Acute

- Diabetic ketoacidosis
- Hyperglycemic Hyperosmolar state
- Hypoglycemia

Long term:

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

Others

- ***Infections***
  - UTI
  - Tuberculosis

- Candidiasis – oral / vulvovaginal
- Mucor mycosis
- Necrotising fasciitis
- Periodontitis

- Dupuytrens contracture
- Pseudogout

Neuropathy and diabetes mellitus[20-26]

- The prevalence of diabetic neuropathy in patients with type 2 diabetes is 32 percent overall and more than 50 percent in patients over 60 years of age.
- Diabetic neuropathy correlates with the duration of diabetes and glycemic control) type1 & 2 DM.
- May manifest as
    1. Polyneuropathy
    2. Mono-neuropathy
    3. Autonomic Neuropathy
- Both myelinated and unmyelinated nerve fibers are affected.
- Because 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 etiologies 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 following, which begins in the feet & spreads proximally.
    1. Numbness,
    2. Tingling
    3. Sharpness
    4. Burning

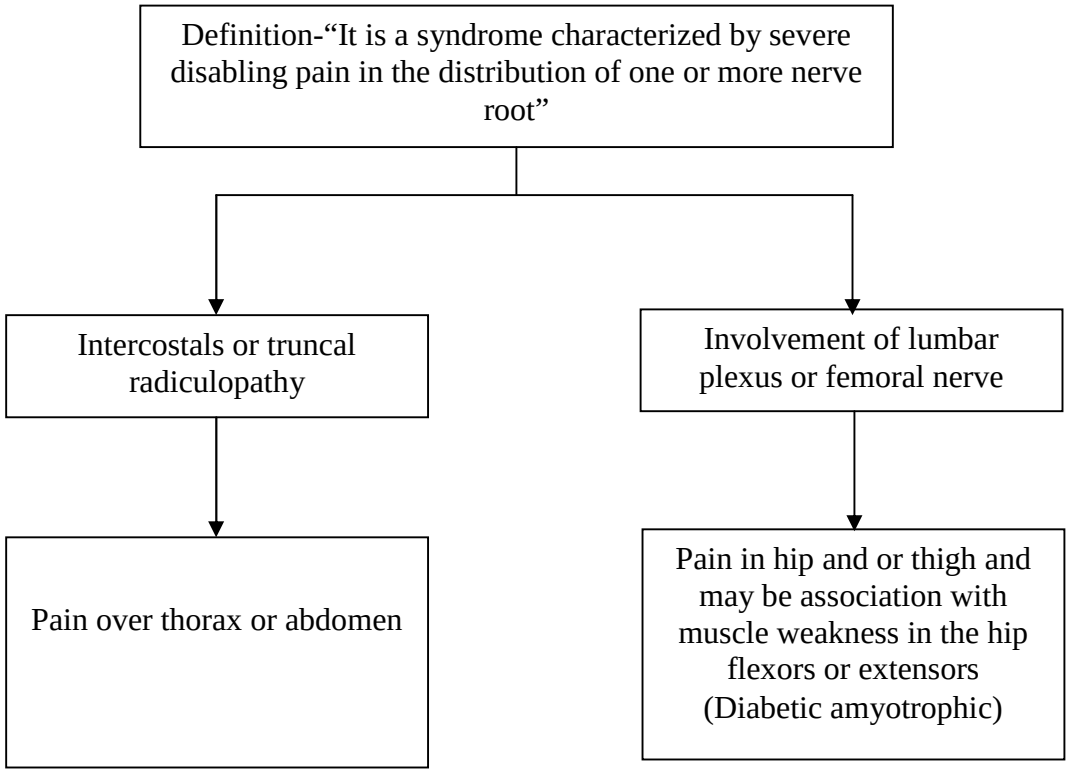
Any combination of these symptoms may develop as neuropathy progresses

- 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 worsen 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 & eventually disappears, but a sensory deficit in the lower extremities persists.
- Neuropathic pain develops in some of these individuals, occasionally preceded by improvement in their glycemic control.

Diabetic Neuropathy

It may be accompanied by - Motor weakness

Figure 2: Diabetic neuropathy



Treatment of diabetic neuropathy

- Improved glycemic control should be pursued and will improve nerve conduction velocity, but the symptoms of diabetic neuropathy may not necessarily improve.
- Avoidance of neurotoxins (alcohol), supplementation with vitamins for possible deficiencies (B12, B6, folate).
- Symptomatic treatment.

- Since pain of acute diabetic neuropathy may resolve over the first year, analgesics may be discontinued as progressive neuronal damage from DM occurs.
- Chronic, painful diabetic retinopathy is difficult to treat but may respond to
    1. Tricyclic antidepressants - Amitryptiline, desipramine, nortriptyline
    2. Gabapentin
    3. NSAIDs (Avoid in renal dysfunctions)
    4. Others (Mexilitine, Phenytoin, Carbamazepine, Capsaicin cream)
- Referral to pain management center may be necessary.

Neuropathy

Neuropathy is present in over 80% of patients with foot ulcers.[27-28]

Peripheral sensory neuropathy

Interferes with normal protective mechanisms and allows the patient to sustain major or repeated minor trauma to the foot, often 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

Lower Extremity Complications

DM is the leading cause of nontraumatic lower extremity amputation in the United States.[6] Foot ulcers and infections are also a major source of morbidity in individuals with DM. The reasons for the increased incidence of these disorders in DM involve the interaction of several pathogenic factors: neuropathy, abnormal foot biomechanics, PAD, and poor wound healing. 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

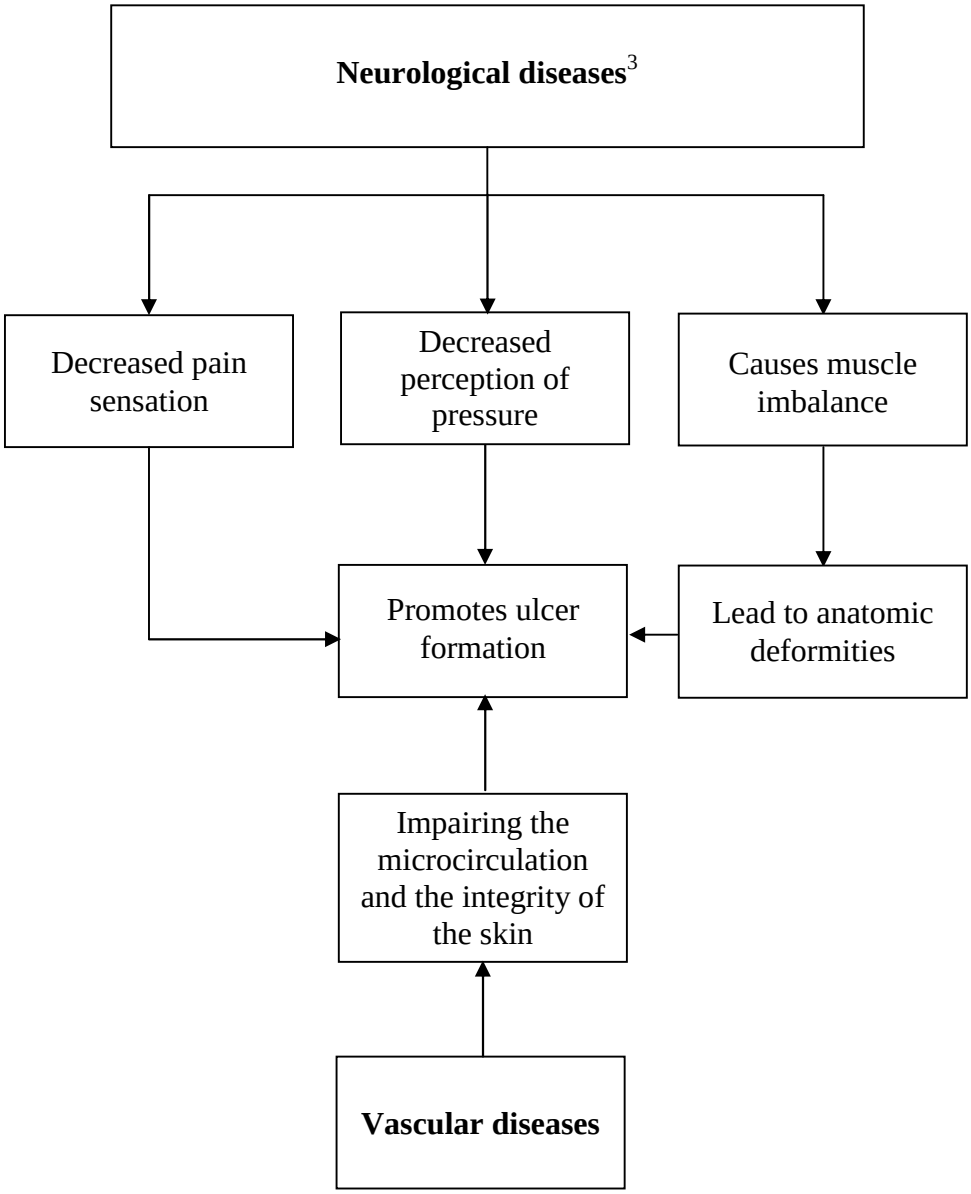
- History of previous ulcer or amputation
- Poor glycemic control.

Table 2: Wagner Ulcer Classification System

Grade	***Lesion***
0	No open lesions; may have deformity or cellulitis
1	Superficial diabetic ulcer (partial or full thickness)
2	Ulcer extension to ligament, tendon, joint capsule, or deep fascia without abscess or osteomyelitis
3	Deep ulcer with abscess, osteomyelitis, or joint sepsis
4	Gangrene localized to portion of forefoot or heel
5	Extensive gangrenous involvement of the entire foot

Large callouses are often precursors to or overlie ulcerations. Foot problems are an important cause of morbidity in patients with diabetes mellitus.[27-28]

Figure 3: Pathogenesis of Diabetic Foot



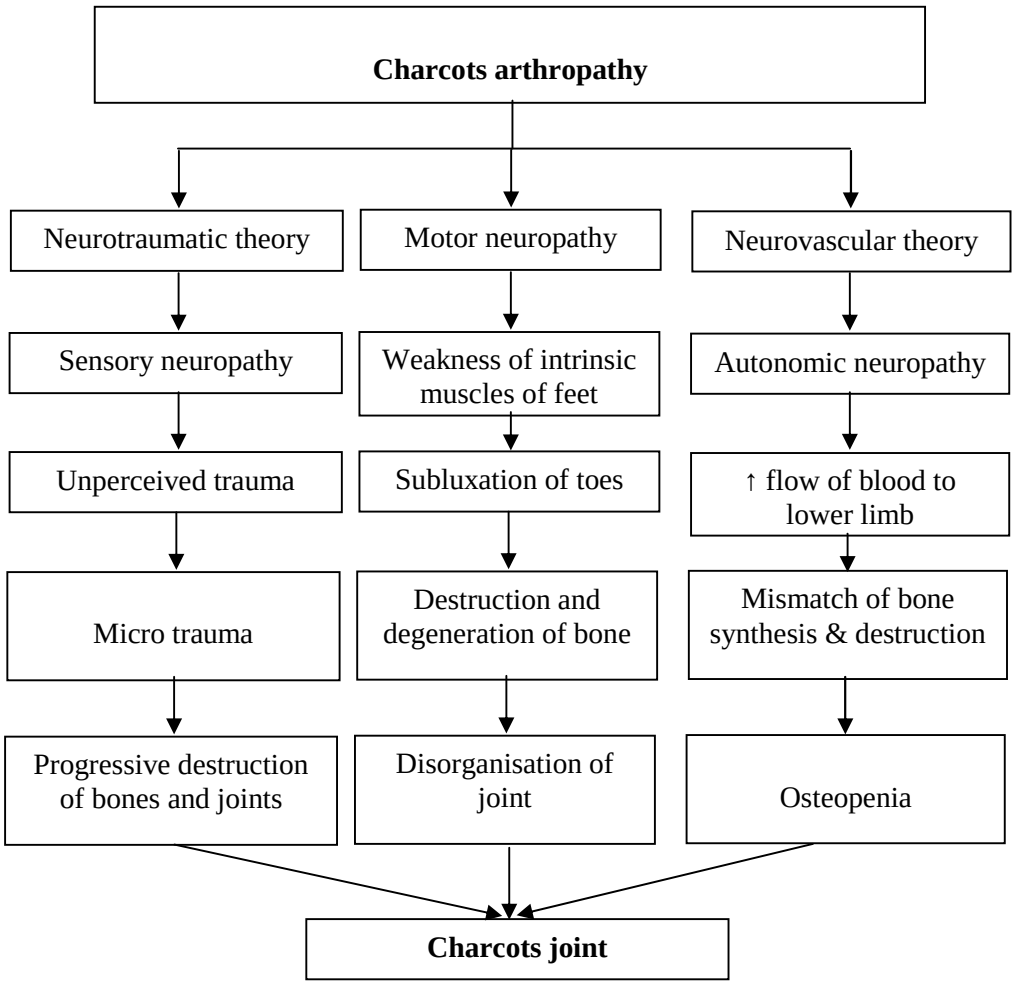
Physical signs resulting from diabetic neuropathy

The physical examination may reveal several abnormalities that result from neuropathy, such as

- Crowding of toes.
- Cockup toes

- Hallux valgus
- Hallux rigidus
- Clawing of toes
- Charcot arthropathy (also called diabetic neuropathic arthropathy).

Figure 4: Pathophysiology of charcot arthropathy[29]



VASCULAR CHANGES IN DIABETES

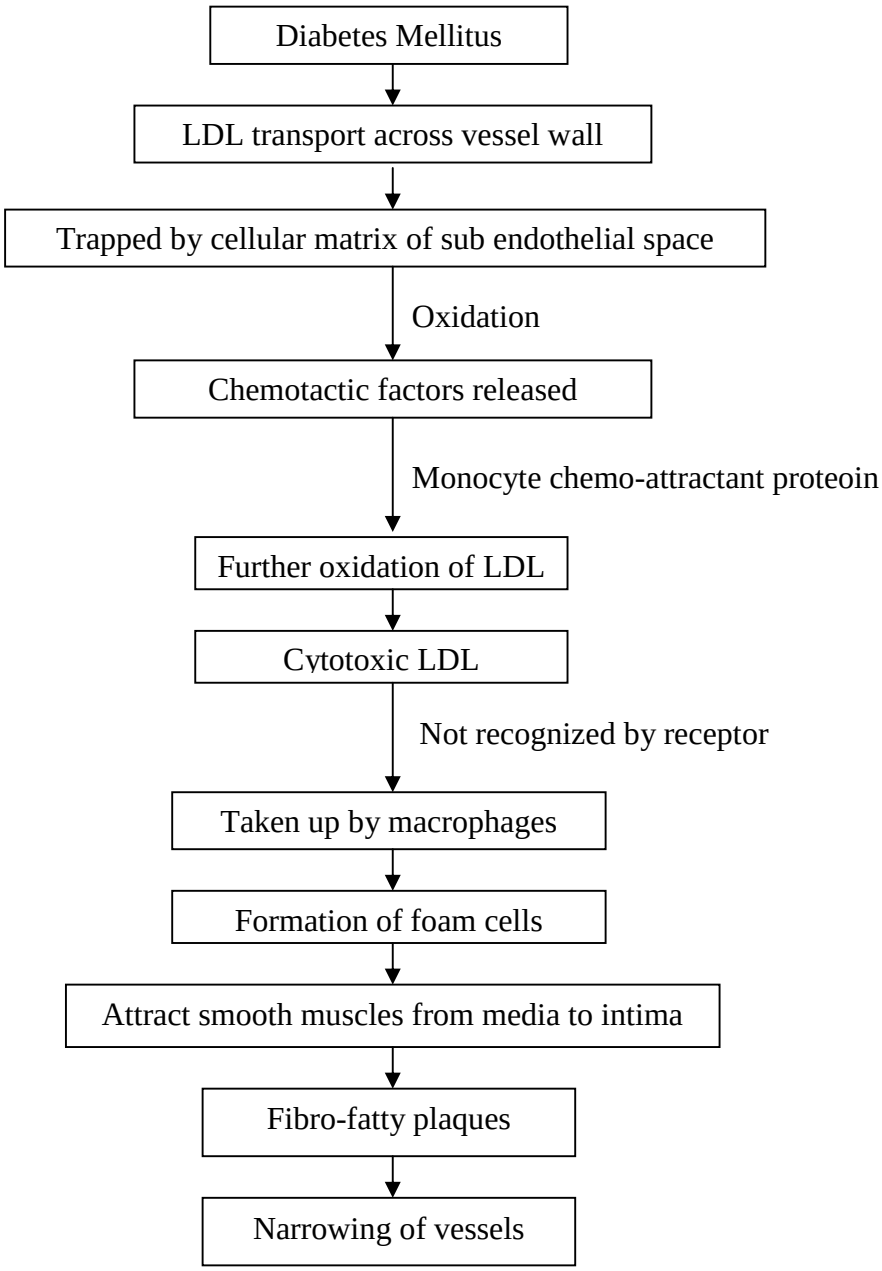
Atherosclerosis

Chronic inflammatory process that can be converted into acute clinical event by plaque rupture. (Berliner). 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.

Lipoproteins Pathogenesis

Figure 5: Pathophysiology of diabetic vasculopathy



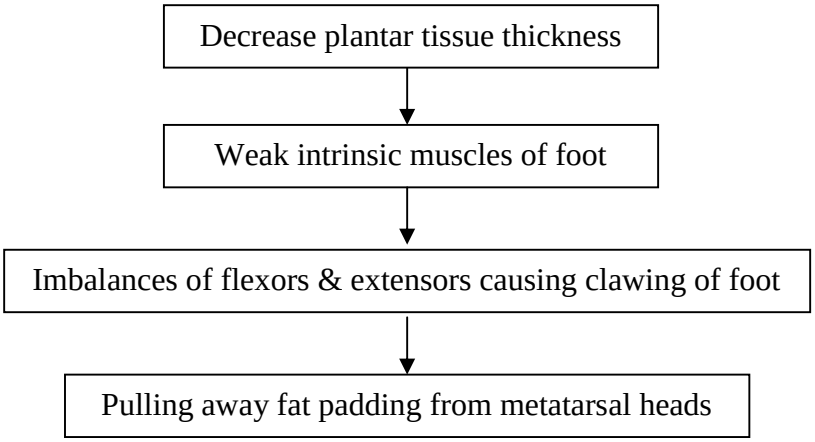
Changes in foot caused by diabetes

1. Peripheral neuropathy :

    A. Dryness of skin.

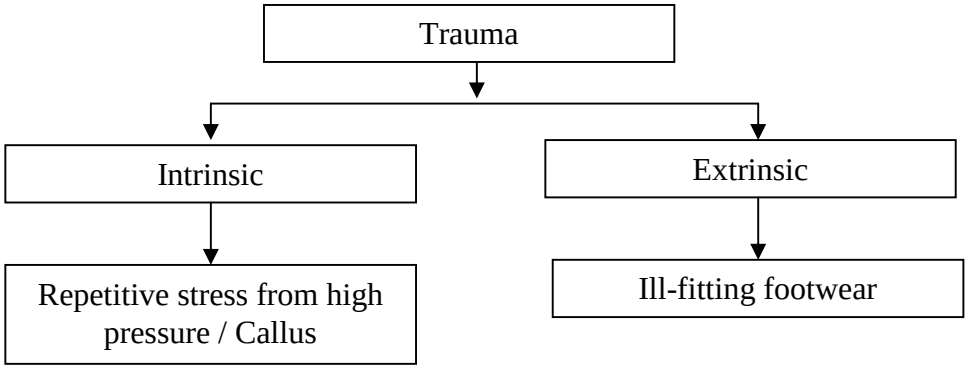
    B. Callus formation.

2. High pressure at bony prominences :



3. Limited joint mobility

4. Trauma :



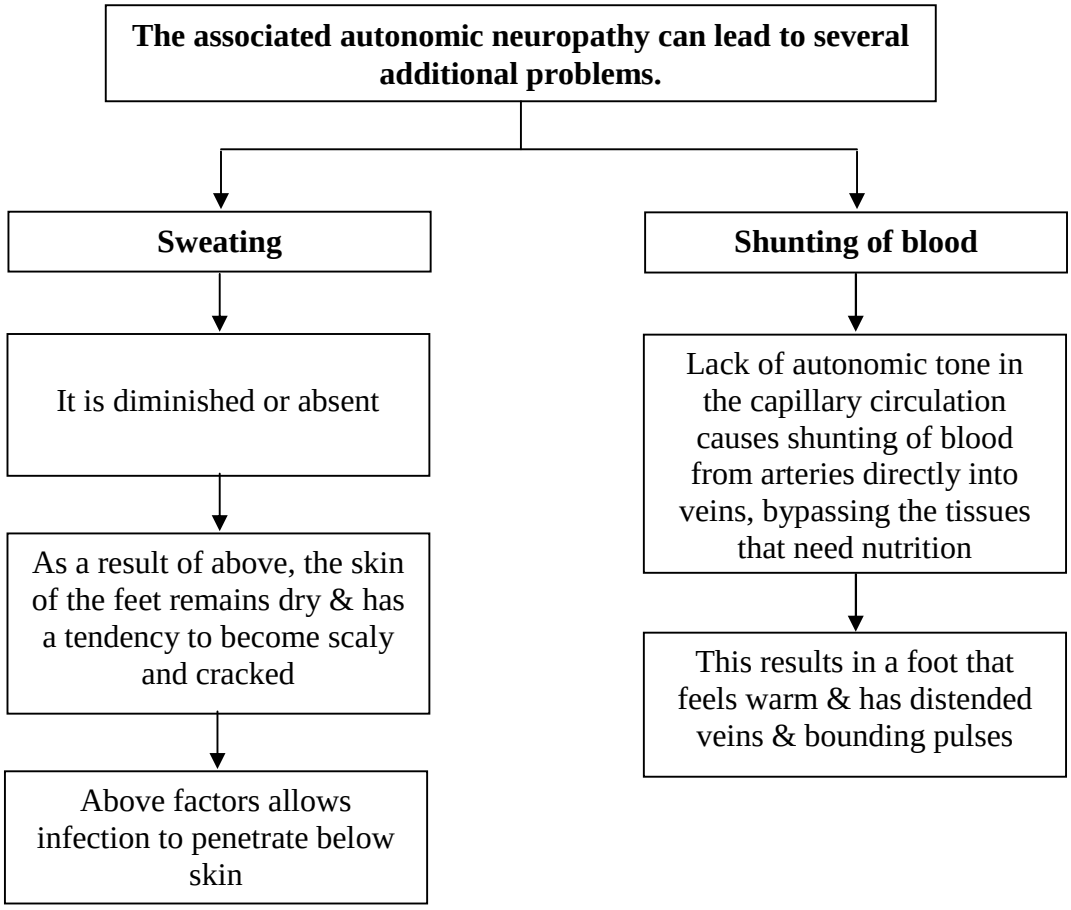
Later complication

Charcot arthropathy

- Characterized by collapse of the arch of the mid foot and bony prominences in peculiar places.
- Caused by- the triad of
    1. Small muscle wasting
    2. Decreased sensation
    3. Abnormal distribution of weight when standing.

The associated autonomic neuropathy can lead to several additional problems.

Figure 6: Pathophysiology of autonomic neuropathy in diabetics



Despite these apparent signs of adequate perfusion, the foot is vulnerable to local "microvascular" gangrene, will heal very poorly and slowly, and will be less able to resist infection.

Recommendations[30]

- The feet should be examined at least annually in patients with Type-2 diabetes and in those with Type-1 diabetes for more than 5 years.
- A detailed neurological examination & assessment for Peripheral Vascular Disease should be performed.

Patients should be considered at particularly high risk for future plantar ulceration if they have[31]

- A previous h/o foot ulceration or amputation.
- Neuropathic foot deformities, especially with overlying bunions or calluses.

Prophylactic foot care

It is important that prophylactic advice on foot care be given to any patient whose feet are at high risk. The recommendations for prophylactic foot care are -

Avoid

- Smoking.
- Walking barefoot.
- The use of heating pads or hot water bottles.
- Stepping into a bath without checking the temperature.

The feet should be

- Washed daily in tepid water.
- Mild soap should be used and the feet should be dried by gentle patting.
- A moisturizing cream or lotion should then be applied.

Toe Nails

The toe nails should be:

- Trimmed to the shape of the toe.
- Filed to remove sharp edges.

Shoes

- The patient's shoes should be snug, not tight.
- Patients who have mis-shaped feet or have had a previous foot ulcer may benefit from the use of special customized shoes.
- A prospective study found that shoe variables other than the recommendation for customized shoes (e.g. style, width, length or type of shoe) had no preventive effect.

The use of customized shoes, however, reduced the development of new foot ulcers from 58 to 28 percent over one year of follow-up in a second report.[8]

Socks

Socks should be:

- Cotton
- Loose fitting

- Should be changed every day.

Inspection of feet

The feet should be inspected daily, looking between and underneath the toes and at pressure areas for skin breaks, blisters, swelling, or redness. The patient may need to use a mirror or, if vision is impaired, have someone else perform the examination.

Examination of foot by medical person

A particularly effective strategy is to make specific recommendations to the patient in the form of a "contract'. And to advise the patient to request that his or her feet be examined at every visit to the doctor or nurse.[32]

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.[33]

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.[34]

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 uneradicated 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.[35]

In general, people with diabetes have infections that are more severe and take longer to cure than equivalent 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.[36] 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 institute the appropriate antibacterial therapy and to avoid further complications.[37,38]

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.

LOW LEVEL LASER THERAPY

Historical background of low energy helium-neon (He-Ne) LASER[39]

Light Amplification by Stimulated Emission of Radiation (LASER)

Table 3: Historical Background of Therapeutic Laser

Scientist	Year	Contribution made in development of laser
Albert Einstein	1916	Principle on which lasers are based were postulated. Credit for the development of laser theory is generally given to Albert Einstein. In his theory "Zur Quantum Theories der Strahlung", published in 1916, he first used the name "stimulated emission".
Mester et al	1985	Non invasive lasers were introduced into medicine in 1980's, since then, have gained wide application in many areas of health care.
Lam TA et al	1986	Although the beneficial effects of laser photostimulation are now generally accepted, the mechanism by which laser facilitates the wound healing & tissue repair, is yet to be clearly understood.
Lysons RF	1987	
Kert et al	1989	The non invasive low power lasers up to 500 Mw have been reported to have stimulatory, anti-inflammatory & analgesic effects.
Meller MM	1990	The beam of laser energy is coherent, polarised, focussed & monochromatic.
Schindl A et al	1998 ; 2000	Low energy lasers are effective as analgesics & accelerates the healing of injured tissues.

Wound healing and repair

Principle of Arndt – Schultz

- If the quantity of energy is too small to stimulate the absorbing tissues, NO significant reaction will take place.

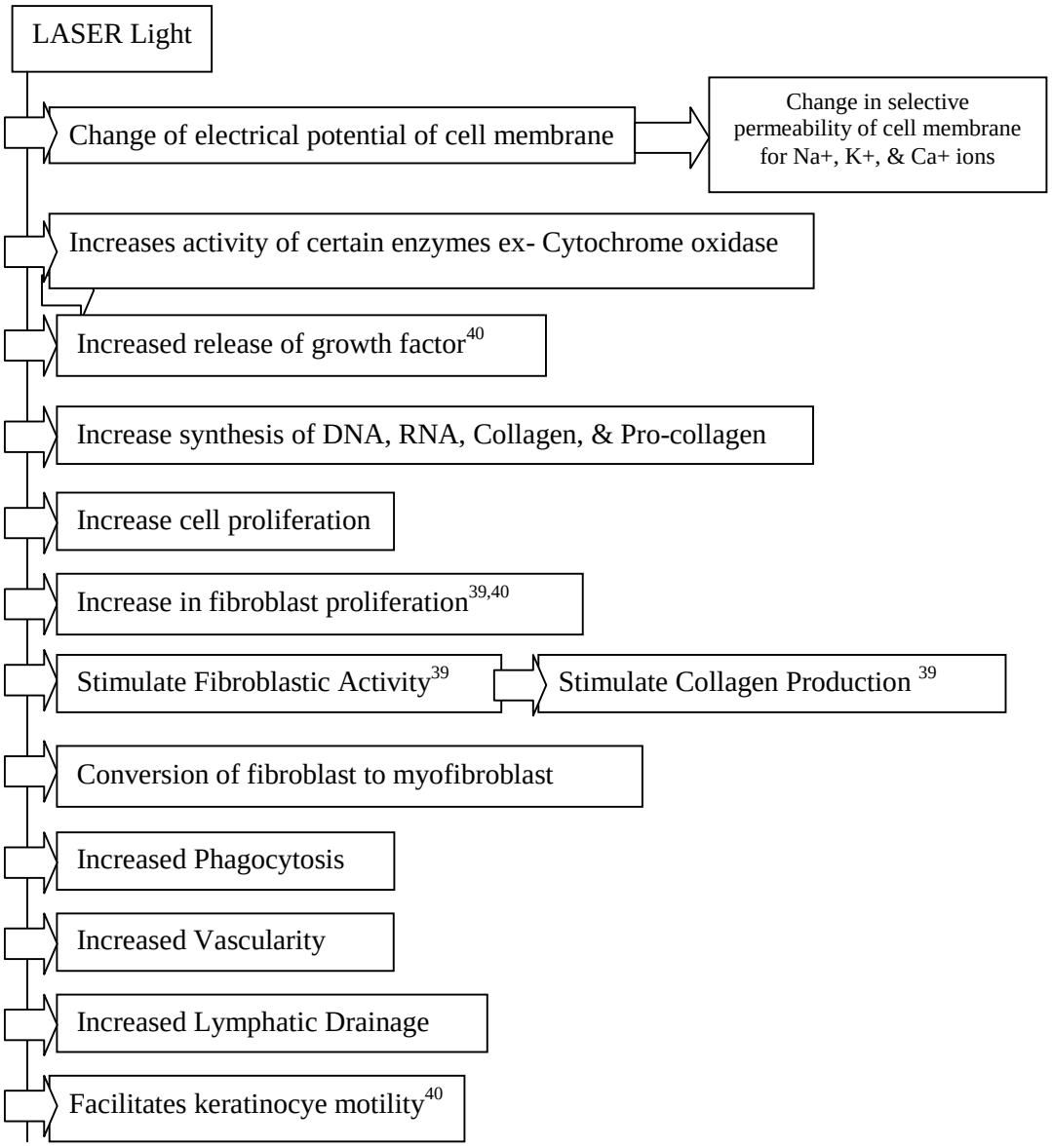
- If the quantity of energy absorbed per unit of time is adequate to stimulate, the absorbing tissue will perform its normal function.

- If the quantity of energy is too great, per unit time, the absorbing tissue will be disrupted and cannot perform its normal function.

- Local tissue temperatures SHOULD NOT be elevated above 45°C otherwise tissue destruction is likely to occur.

- Therefore proper selection of laser dose is necessary to prevent photo-inhibitory effects (As it works on the principle of ARNDT – SCHULTZ).

Figure 7: Mechanism of Action of Laser at Cellular Level



The word **LASER** is an acronym for **L**ight **A**mplification by **S**timulated **E**mission of **R**adiation

- According to the European Standard IEC 601, the definition of LASER is "Any device which can be made to produce or amplify electromagnetic radiation in the wavelength range from 180 nm to 1 mm, primarily by the process of controlled stimulated emission".

- Strictly speaking, a laser is a LIGHT AMPLIFIER if the radiation produced is within the visible range or a RADIATION AMPLIFIER if the radiation produced is within the infrared or ultraviolet radiation ranges.
- As the point of interest, the first name suggested for the laser was "Light Oscillation by Stimulated Emission of Radiation" but this resulted in acronym LOSER.
- A predecessor of the laser was MASER (Microwave Amplification by Stimulated Emission of Radiation). It works on the same principle as the laser, but within the micro-wave range.
- The LASER is the latest & most advanced of our light sources.

Properties of helium-neon LASER[41]

Monochromaticity

The lasers are of single specific wavelength and hence of defined frequency.

Coherence

- The laser radiation is not only of the same wavelength but also in phase (The peaks and troughs of the electric and magnetic fields are occurring at the same time).
- Coherence generally means order, or synchronicity.
- Coherence means that the photons are well ordered i.e. they be connected and build light waves that remain synchronized with one another over long distance. When a rough surface is illuminated with visible laser light, a kind of grainy quality can be observed in the light. These grainy elements, or granules, are called "laser speckles" and occur because of interference between different light

beams. This is because light with a sufficiently high coherence length can be "combined" in the same way that waves of water combine when they meet.

- Waves of coherent light stay in phase in long trains of waves. The length of these trains of waves, the coherence length, may vary from one light source to another .An ordinary light bulb has a low coherence length, a matter of thousandths of a millimeter. A He-Ne laser, on the other hand, may have a very high coherence length -decimeters or even several meters.

Collimation

- If a collimating lens or lens system is placed in front of the diode, the **light's parallelity can be increased**. This is called collimation. A collimated beam is quite parallel, and with such a beam, we can irradiate from a distance and still achieve high power density over a small area.
- Because of spatial coherence lasers remain in a parallel beam. It is a common misconception that light from a laser is always in the form of a parallel beam.

Gas and diode LASER

- **Gas laser**

  The light from a gas laser is usually parallel (typically in the order of milli-radian).

- **Diode laser**

  The light from diode laser (without collimating optics) is ordinarily much more divergent, with an angle of "spread" of around 30-90 degrees. This is mainly due to diffraction when the light is emitted from the resonator mirror on the semiconductor crystal.

Characteristics, which differentiate laser from "normal" light

The laser light has essential characteristics, which differentiate it from "normal" light:

1. Narrow bandwidth
2. Its high level of coherence

Principal components of LASER system[41]

A laser, whether large or small, always includes the following parts:-

1. Energy source = Power supply.
2. Lasing (amplifying) medium.
3. Resonating cavity.

Energy source or power supply

The energy source may be an electrical current, optical radiation from a flash lamp or another (pumping) laser, radio waves or microwaves, or a chemical reaction.

Lasing medium

- The lasing medium must be able to store the energy supplied, which it does by a process called "Population Inversion". This stored energy can then be emitted in an organized fashion, known as stimulated emission of radiation.
- When a photon with the right photon energy enters the electromagnetic field of the excited atom with energy stored in this way, some of the energy is emitted by the creation of identical photon. The first photon is not absorbed. Hence, both the new & old photon can then stimulate other atoms in the lasing

medium to emit their stored energy. It is like chain reaction, causing an "avalanche" of light in which all the photons have exactly the same photon energy.

- Hence, the radiation from laser always has a certain fixed wavelength, which is determined by the structure of the lasing medium & does not, like the radiation, from other light sources, consist of a wide range of wavelengths. There are concurrently number of different types of lasers, and these produce light, UV, or IR radiation of various wavelengths. Usually a laser has one characteristic wavelength, though it is sometimes possible to choose within a range of wavelength, it is common among semiconductor lasers. There are a few lasers designed in such a fashion that the wavelength could be changed, even during operation - Tunable lasers.
- Stimulated emission occurs when the incident photon has exactly the same energy as the released photon; the first photon causes release of the second photon simultaneously, i.e. the second photon must follow the oscillations of the first photon, so that the two photons oscillate in phase. This phase-locked, wavelength specific, photon chain reaction results in the monochromatic laser light.

Resonating cavity[41]

The lasing medium is generally elongated in shape, often in the form of a channel (gas lasers) or a narrow rod (solid state lasers) or doped channel (semiconductor lasers), & is fitted with mirrors at its ends. These mirrors form the resonating cavity. The light produced in the lasing medium is reflected back into the lasing medium several times & stimulates new light production.

Importance of resonating cavity

Increases the lasing medium's amplification and makes the light more coherent. The elongation and mirror arrangement also make it possible for the light to emerge as a parallel beam (that may be focused to an extremely small point). To extract the light from the laser, one mirror is made somewhat transparent – most often 1-20% transparency, so that between 99% and 80% of the light is reflected back. Hence, the light intensity inside a laser is always higher than the output power.

LASER physics[41]

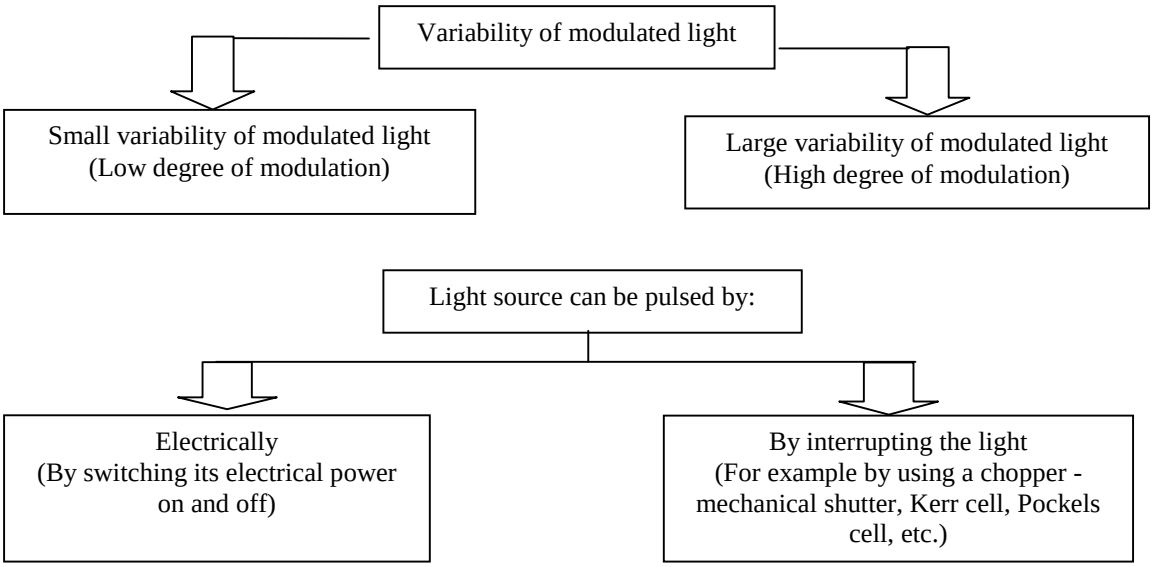
Output power = strength (W / mW)[41]

- Power is measured in watts (W). The Strength or Power output of a laser is thus measured in watts or miliwatts (mW = A thousandth of a watt).
- Higher output power means higher power density, which is often desirable.
- In addition, a higher output power means that a certain desired dose (input energy, measured in joules per cm^2 of skin) is more quickly reached because energy is the same as power multiplied by time.

Continuous and pulsed LASERS[41]

- õ A light source usually emits light at a constant intensity. This is known as CONTINUOUS WAVE (cw) EMISSION. However, it is also possible to make lasers & other lamps with varying intensity, known as AMPLITUDE MODULATED EMISSION.

õ The variability of modulated light may be small (Low degree of modulation) or large (High degree of modulation). In the extreme cases (100% modulation), the intensity periodically reaches zero.



- If the light intensity only varies between a maximum & zero, then the modulation is known as SQUARE WAVE AMPLITUDE MODULATION and the light source is said to be pulsed –the same as switching it on & off.

How a light source is pulsed?[41]

- A pulsed light source can be pulsed either electrically, by switching its electrical power on and off, or by interrupting the light, for example by using a chopper (mechanical shutter, Kerr cell, Pockels cell, etc.).

- It may be pulsed at a high or a low frequency, i.e. with many or few pulsed per second.

- If the lamp (or laser) emits visible light and is pulsed at a low frequency (up to about 30 pulses per second), we can see that the light is blinking.

- If the lengths of the periods of light are equal to the lengths of the periods of zero intensity, the "duty cycle" is 50%, as light is emitted during 50% of the overall time.

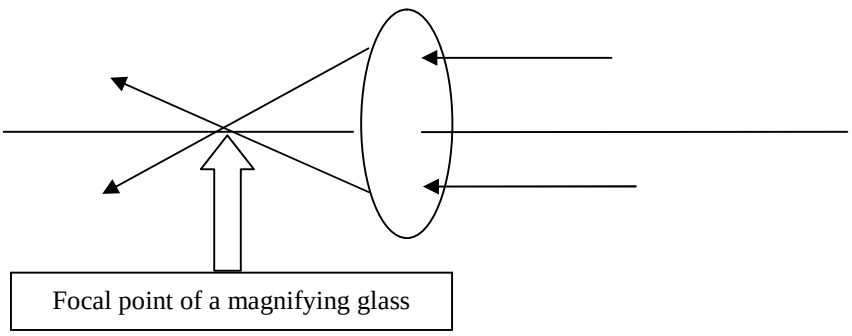
Average power output[41]

For pulsed lasers, the output power value to be used for dose calculation is the laser's average power. When a laser is chopped, the pulsing can have different duty cycles. If the light is "on" for 30% of the time and "off' for 70% of the time, the duty cycle is 30%. Then the average power is 30% of the peak power (if the switching is momentary i.e. a true square wave).

Power density[41]

- Power density is another way of saying "light intensity" or "light concentration", and is the “light output power per unit area of the target being illuminated by the laser light”
- This is usuallly measured in watts per square centimeter (W/cm2).
- High power density is achieved, for instance, at the focal point of a magnifying glass.

Figure 8: Focal Point for High Power Density



Risk of eye injury[41]

Even in the infancy of laser technology there was an awareness of the increased risk of eye damage compared to conventional light sources. Hence, new rules introduced to handle this hazard. Lasers were divided into five categories (Class 1,2, 3A, 3B and 4) according to their potential to damage the eye.

Risk of Eye Injury

Class	Safety with regard to eye damage
Classes 1-3A	Are considered safe
Class 3B	Involves a certain risk
Class 4	• Definite risk • Comprises strong industrial and surgical lasers capable of Burning and cutting.

Note that this classification has nothing to do with the medical use, efficiency or quality of lasers but refers solely to the possible risk of eye injury.

Lasers in medicine and surgery[41]

In the table below, the names, wavelengths, working modes, and uses of the most common laser systems used in medicine are listed :

Table 4: Classification of Therapeutic Lasers

Laser name	Wavelength	Pulsed or cont.	Medical use
Crystalline laser medium			
KTP/532	532 nm	p/c	Leg vein treatment
Ruby	694	P	Tattoo & hair removal
Alexandrite	755	P	Bone cut, Hair removal
Nd:Yag	1064	P	Coagulation of tumours
Ho:Yag	2130	P	Surgery, Root canal, Lithotripsy
Er:Yag	2940	P	Dental drill, Laser peeling
Ti: Sapphire	Tunable	P	Two photon PDT
Semiconductor lasers			
InGaAIP	630-700	C	Biostimulation
GaAIAs	780-820-870	C	Biostimulation & Surgery
GaAs	904-905	P	Biostimulation
Liquid laser medium			
Dye laser	Tuneable	P/C	Kidney stones
Rhodamine	560-650	P/C	PDT, Dermatology
Gas lasers			
Excimer	193, 248, 308	P	Eye, Vascular surgery
Argon	350-514	C	Dermatology, Retinopathy
Copper	578	P/C	Dermatology
HeNe	633, 3390	C	Biostimulation
CO2	1064	P/C	Dermatology, Surgery

Production of LASER radiation by helium-neon (He-Ne) units[41]

- The Helium-Neon laser is in the gaseous form and its wavelength is fixed at 632.8nm.
- The light beam produced by the He-Ne lasing medium is released as bright, intense, monochromatic (same wave length), coherent (in same phase) and collimated (parallel) laser beam.
- It consists of a long tube containing these natural gases at low pressure surrounded by the flashgun tube.
- Excitation of these atoms leads to different energy levels between them and the transfer of energy giving of a photon of wavelength equivalent to the energy gap. The photons are reflected back and forth along the tube giving rise to further photon emission and emerging as narrow beam (Of about 1 millimeter diameter) from transparent end.
- Helium- Neon laser gives radiation in the red visible region at 632.8 nm.
- Once the unit is activated, electrical energy is delivered to the lasing medium. Initially this energy will result in excitation of the neon atoms, neon being the active medium with in He-Ne units. Within such laser, the helium merely acts as a buffer assisting with excitation of the neon atoms and cooling processes.

  He-Ne unit : Function

  Neon atoms : The active medium

  Helium atoms : Acts as a buffer assisting with excitation of the neon atoms and cooling processes

- Spontaneous emission of radiation from the neon atoms occurs readily as the excited states are inherently unstable. However the rate of supply of energy to

the neon atoms within the medium is far greater than that dissipated through the process of spontaneous emission by these atoms. Consequently, after a short period the majority of the neon atoms will be in the higher energy states. This condition is known as **population inversion**; instead of the majority of neon's atoms being at resting states and relatively few at higher energy levels, as is normally the case the reverse situation occurs.

- The He-Ne laser was the first laser system to be used in clinical and research applications principally in Eastern Europe and china in 1970s.
- The He-Ne laser has the fibre optic applicators, it enable the operator to direct the output onto the target area more easily. The technique is called contact technique and it has greater therapeutic effect on the tissue.
- The laser generally, works continuously but can be pulsed by switching the electric power supply or by means of a beam chopper. In that case, half the output is lost (if the duty cycle is 50%) compared to the continuous working mode.
- Typical output is 1-10 Mw, sent directly (parallel beams) or via fiber optics to a treatment probe (usually divergent light). The He Ne laser is the most coherent of the different therapeutic types, having a coherence length of centimeters to meters. This is an important factor for the biological result.

METHODOLOGY

The present study was conducted in the Department of Surgery, KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belgaum on patients with infected diabetic foot ulcers during the period of January 2008 to December 2008.

Study design

One year randomized controlled trial.

Study period

The present study was conducted during January 2008 to December 2008.

Method of collection of data

Source of Data

Patients with infected diabetes foot admitted at KLES, Dr. Prabhakar Kore Hospital and Medical Research Centre, Belgaum.

Sample size

A sample size of 60 cases divided into two groups.

Sampling procedure

The sample size was calculated based on patient data for the last three consecutive years that is 2005, 2006, 2007 of KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belgaum.

Selection criteria

Inclusion Criteria

- Type 1 and 2 diabetes patient.
- Diabetics between 18 to 65 years.
- Culture positive ulcer on admission.
- Duration of the ulcer of more than four weeks.

Exclusion Criteria

- Pulseless limb.
- Immunocompromised patients.
- Associated osteomyelitis.
- Cardiac conductivity disorders.
- Skin malignancy.
- Diabetic ketoacidosis.
- Cellulites.
- Serum creatinine > 2.
- Diabetic gangrene.

Procedure

The study was conducted in Department of Surgery at KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belgaum during one year duration. The study was approved by the Ethical and Research Committee of Jawaharlal Nehru Medical College, Belgaum.

After finding the suitability as per inclusion and exclusion criteria patients were selected for the study and briefed about the nature of the study, the interventions used and written informed consent was obtained (Annexure–I). Further, descriptive data of the participants like name, age, sex, detailed history, were obtained by interviewing the participants and clinical examination and necessary investigations like complete blood count, blood urea and serum creatinine and culture of the ulcer on admission were recorded on predesigned and pretested proforma (Annexure-II). Further the patients were divided into two groups using a computerized randomization chart.

Methodology

Both the groups were given empirical antibiotic (Tab. Ciprofloxacin-Tinidazole) for two days and culture sensitive antibiotics after two days. Group I received standard treatment that is conventional dressing with betadine. In group II patients received LLLT along with conventional dressing.

Specification for LLLT

- Duration – 10 days.
- Frequency – 5 KHz.
- Cluster probe – 60 mW.
- Dose – 12 J/cm^2 daily.

The wound culture was repeated on fifth day and tenth day of treatment.

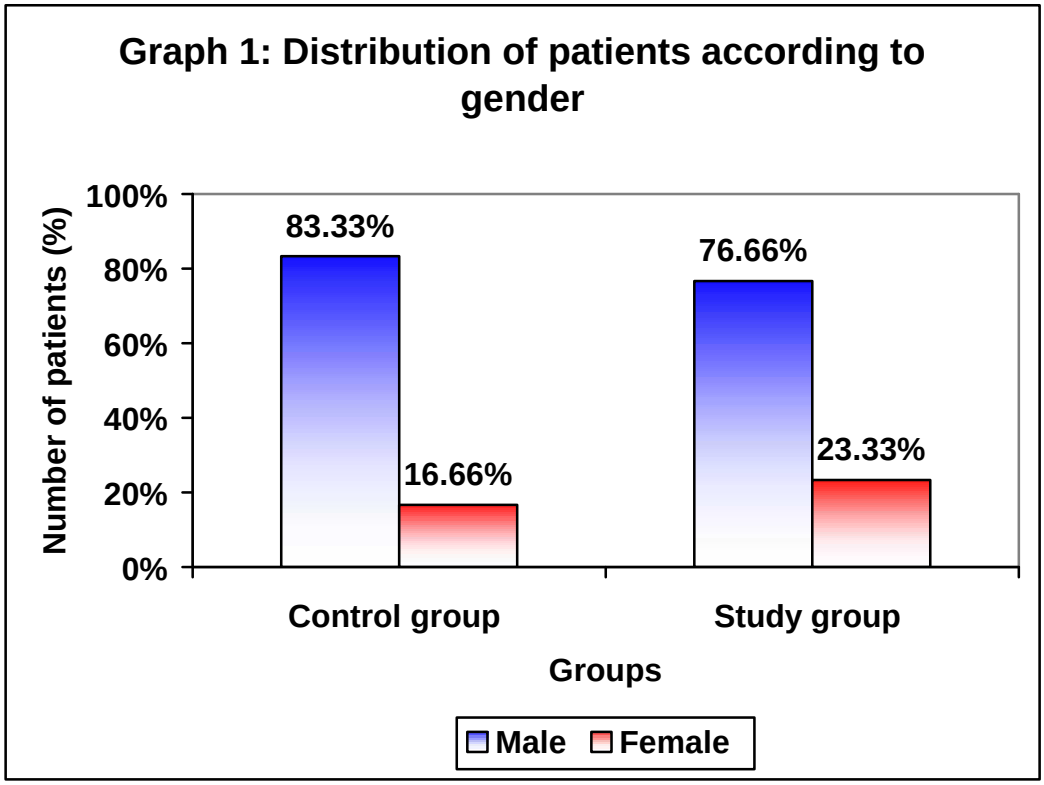
Statistical Methods

At the end of the study the data was tabulated and analysed using chi-square test.

RESULTS

Table 5: Distribution of patients according to gender

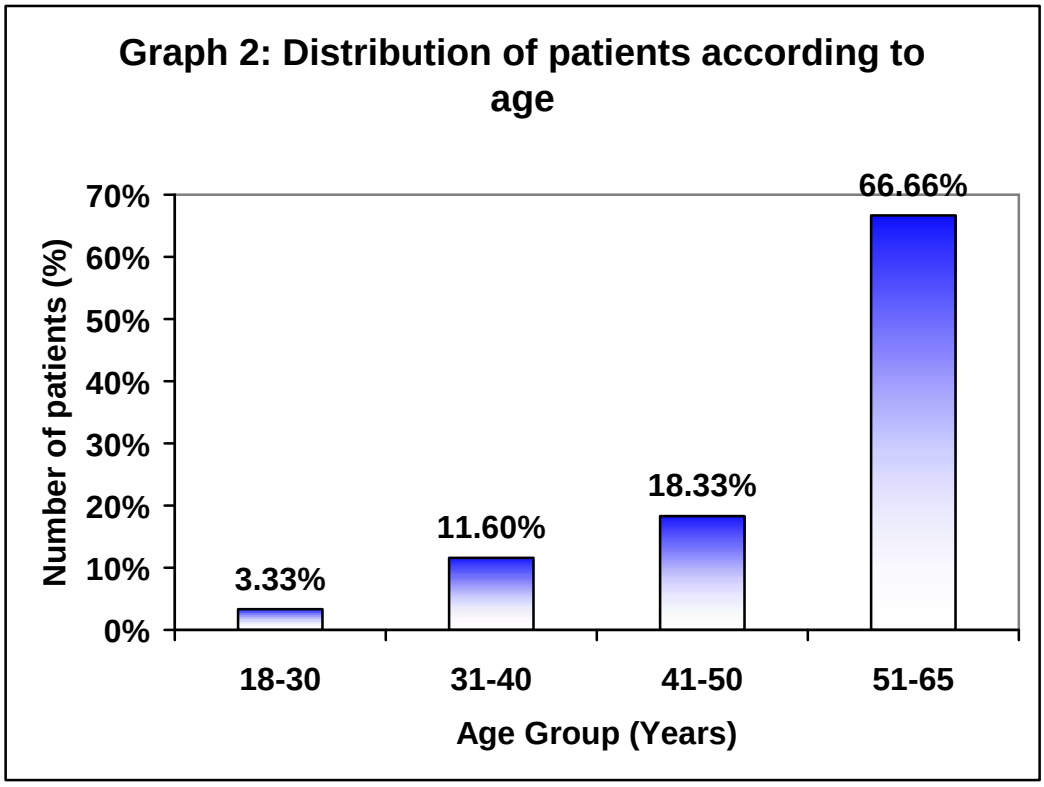
Sex	Control group		Study group	
	No.	Percentage	No.	Percentage
Male	25	83.33%	23	76.66%
Female	5	16.6%	7	23.33%
Total	30	100%	30	100%



In this study control group had 25 males and 5 female with M: F of 5:1. study group had 23 males and 7 females with M : F ratio 3 : 1.

Table 6: Distribution of patients according to age

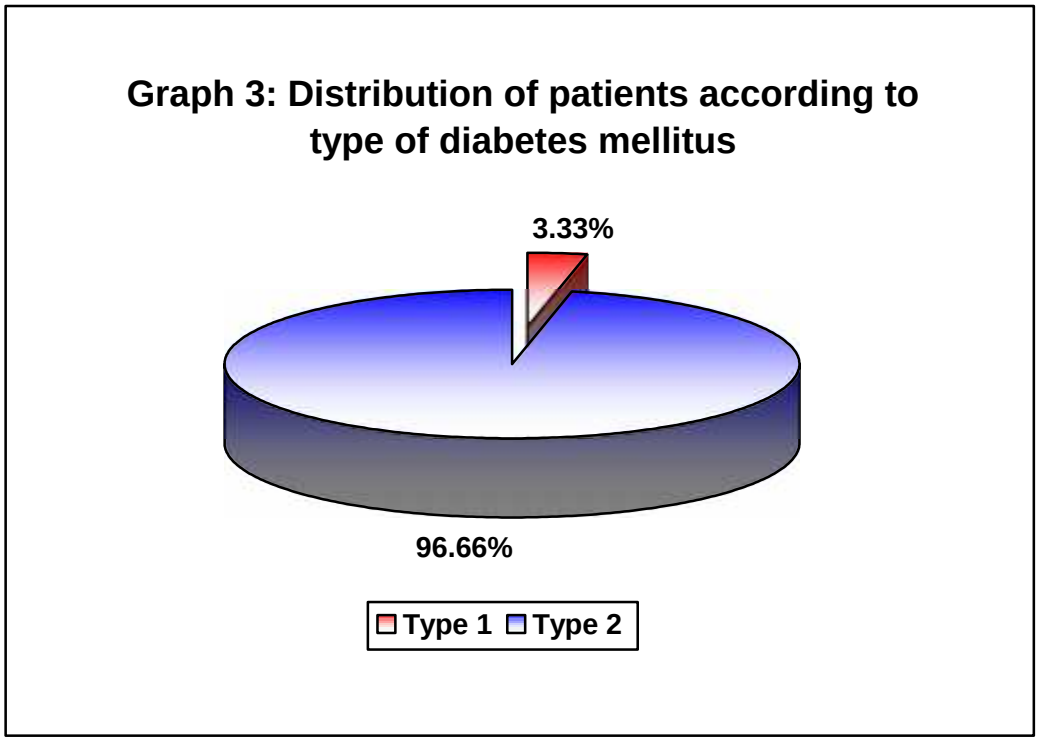
Age (Years)	Number of patients	
	No.	Percentage
18 – 30	2	3.33%
31 – 40	7	11.6%
41 – 50	11	18.33%
51 – 65	40	66.66%
Total	60	100%



In this study more number of patient were within age group of 51 – 65 years (66.66%).

Table 7: Distribution of patients according to type of diabetes mellitus

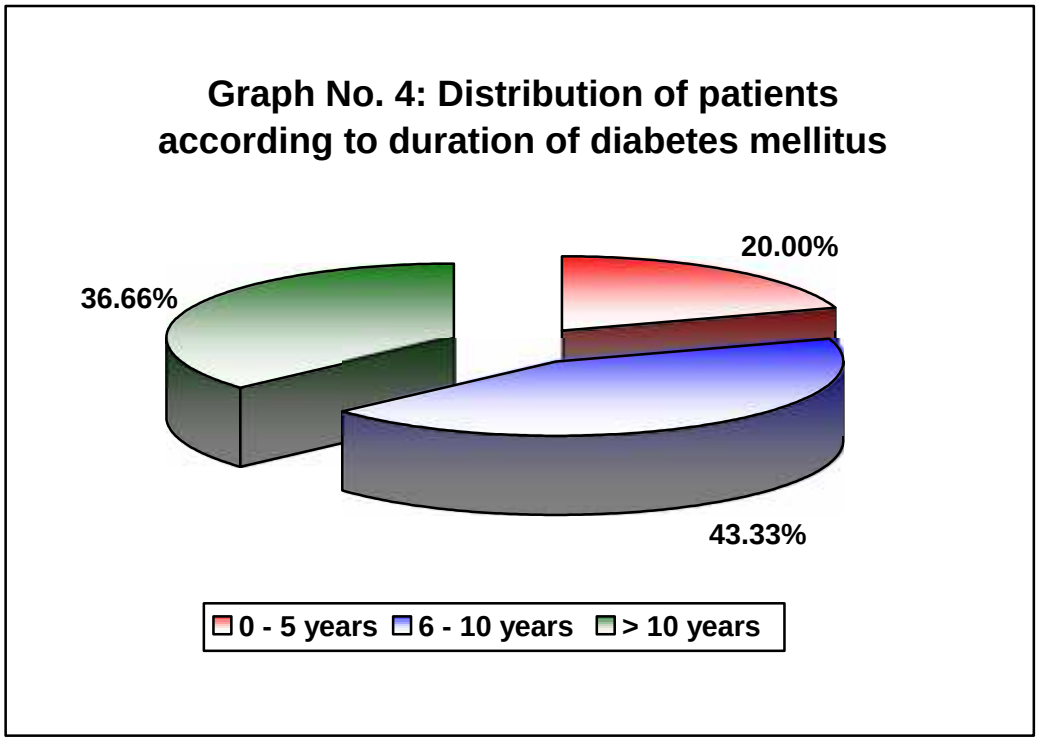
Type	Number of patients	
	No.	Percentage
Type 1	02	3.33%
Type 2	58	96.66%
Total	60	100%



Only two patients were of Type I DM and rest had Type II DM.

Table 8: Distribution of patients according to duration of diabetes mellitus

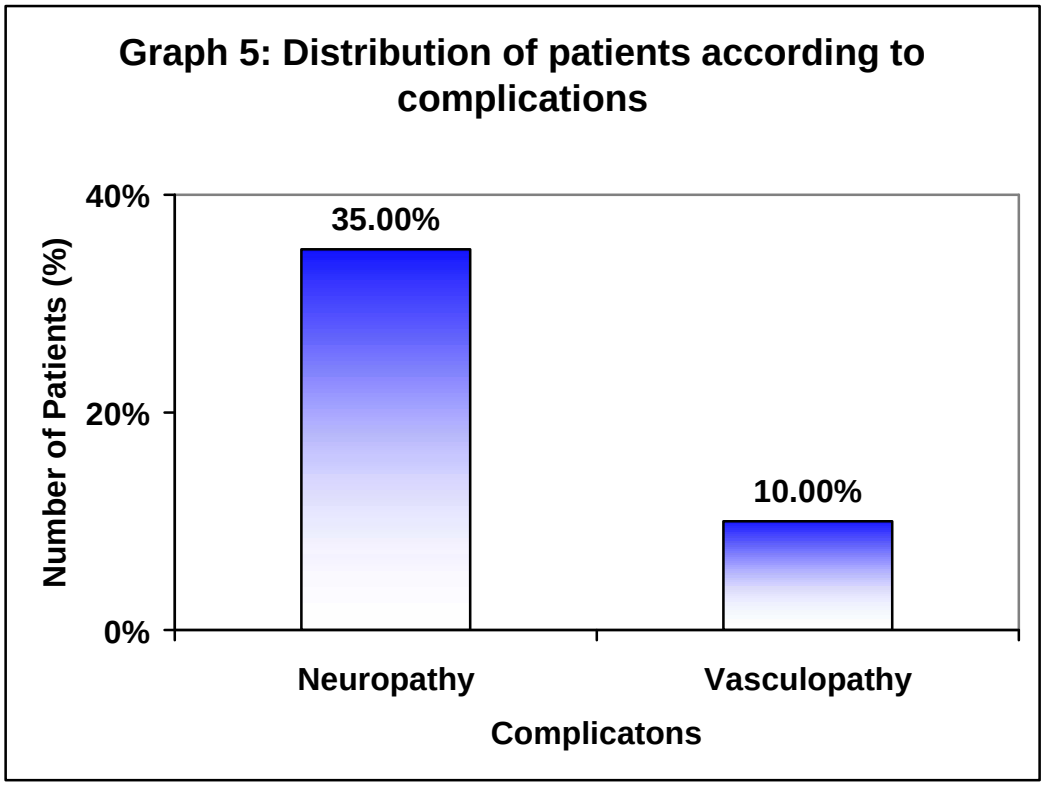
Duration (Years)	Number of patients	
	No.	Percentage
0 – 5	12	20%
6 – 10	26	43.33%
> 10	22	36.66%
Total	60	100%



In the present study 43.33% of patients had history of DM for a period of 6 to 10 years and 36.66% had diabetes for 10 years or more.

Table 9: Distribution of patients according to complications

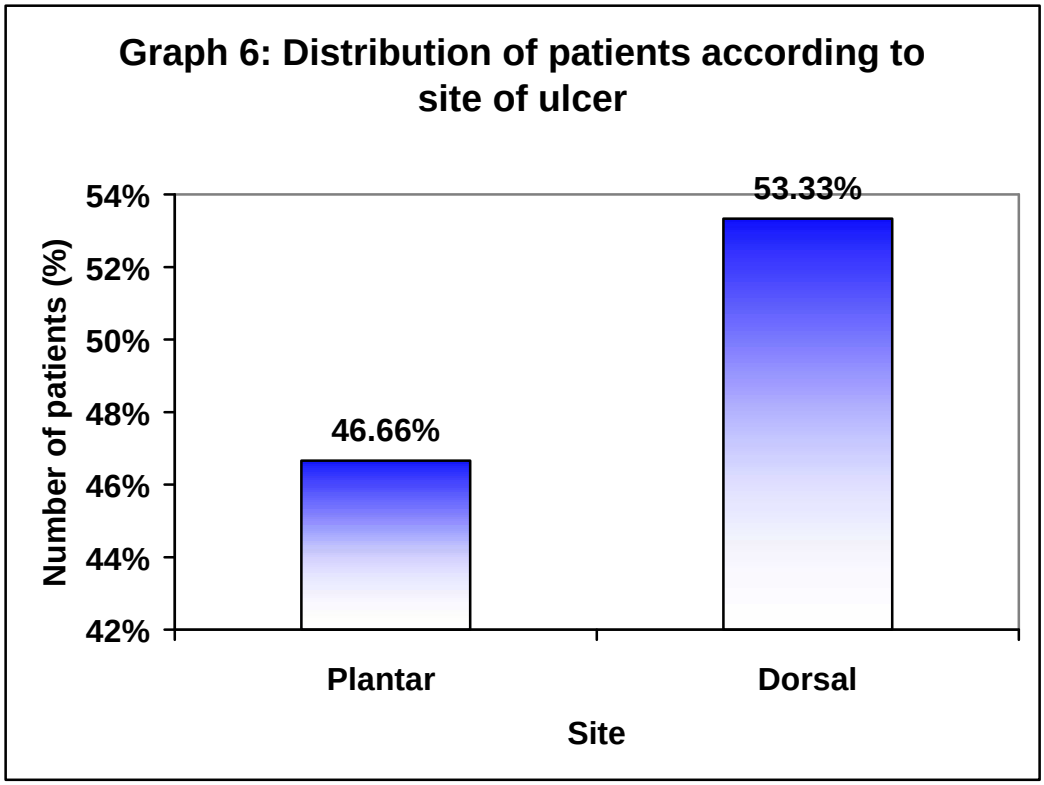
Complications	Number of patients	
	No.	Percentage
Neuropathy	21	35.00%
Vasculopathy	06	10.00%



In the present study number of patients with neuropathy were 21 (35%) whereas vasculopathy was present in 10% of the patients.

Table 10: Distribution of patients according to site of ulcer

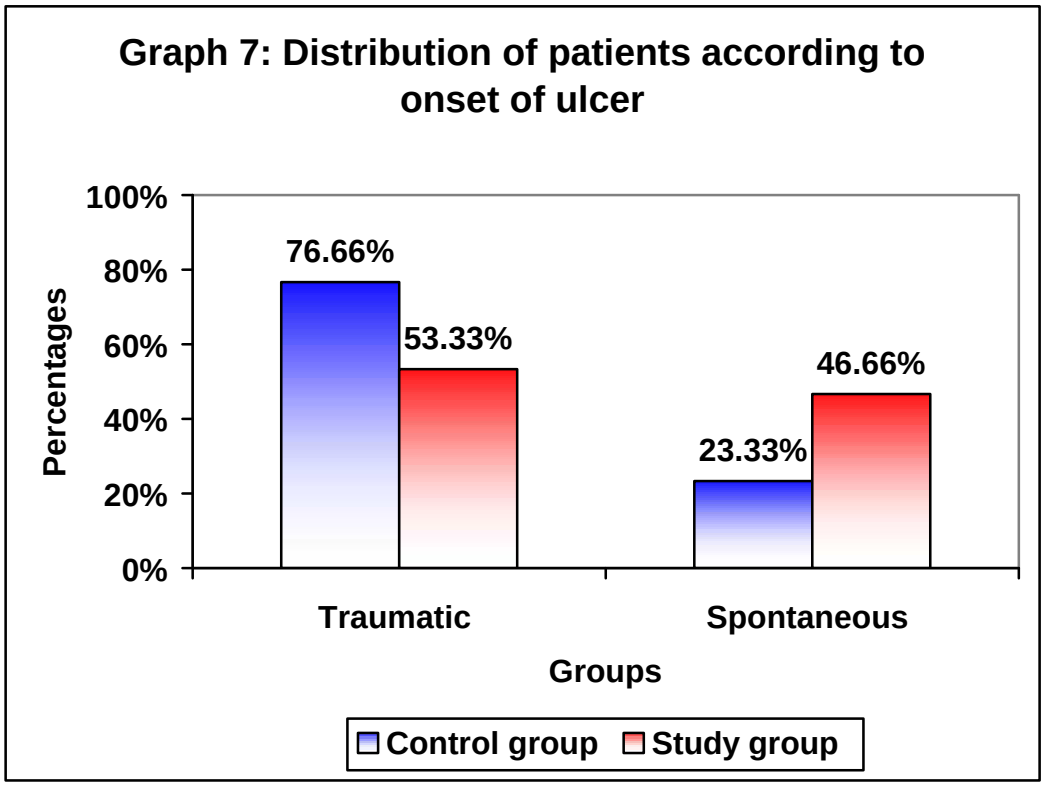
Complications	Number of patients	
	No.	Percentage
Plantar	28	46.66%
Dorsal	32	53.33%
Total	60	100%



In our study it was observed that 46.66% of patients had ulcer on plantar aspect and 53.33% had ulcer on dorsum of foot.

Table 11: Distribution of patients according to onset of ulcer

Sex	Control group		Study group	
	No.	Percentage	No.	Percentage
Traumatic	23	76.66%	16	53.33%
Spontaneous	7	23.33%	14	46.66%
Total	30	100%	30	100%

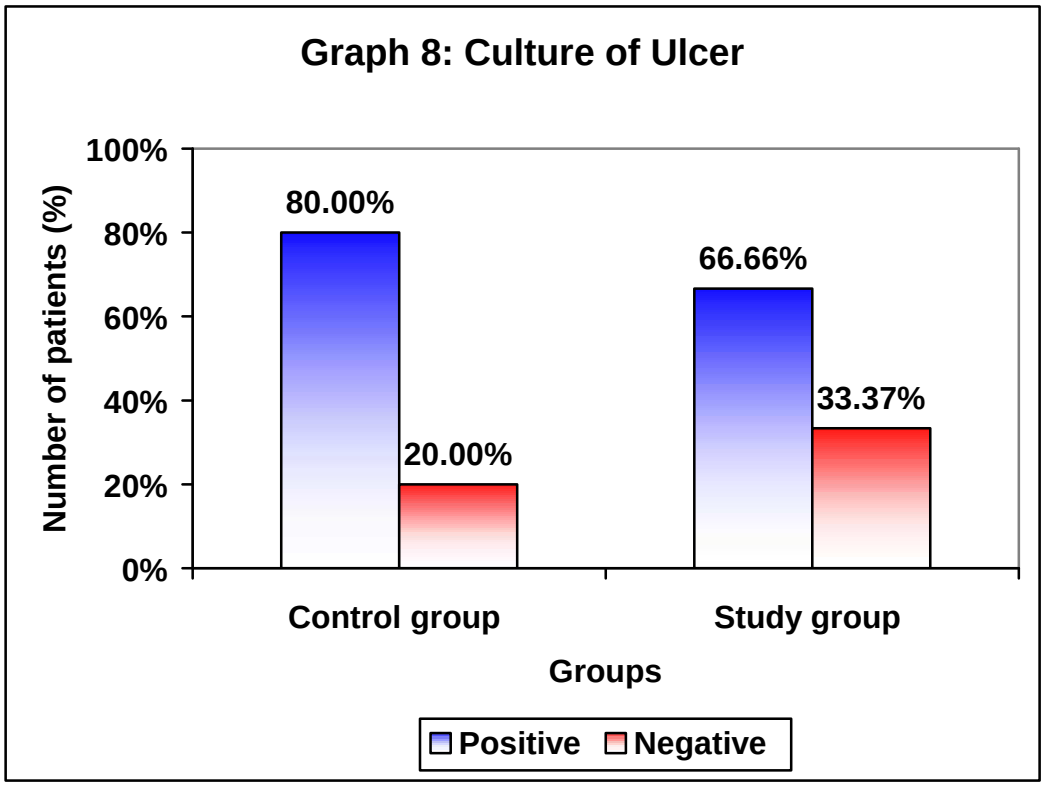


Trauma was the most common cause of origin of diabetic foot ulcer (65%) while only 35% had spontaneous ulcer origin.

Table 12: Culture of Ulcer

Sex	Control group		Study group	
	No.	Percentage	No.	Percentage
+	24	80%	20	66.66%
-	6	20%	10	33.33%
Total	30	100%	30	100%

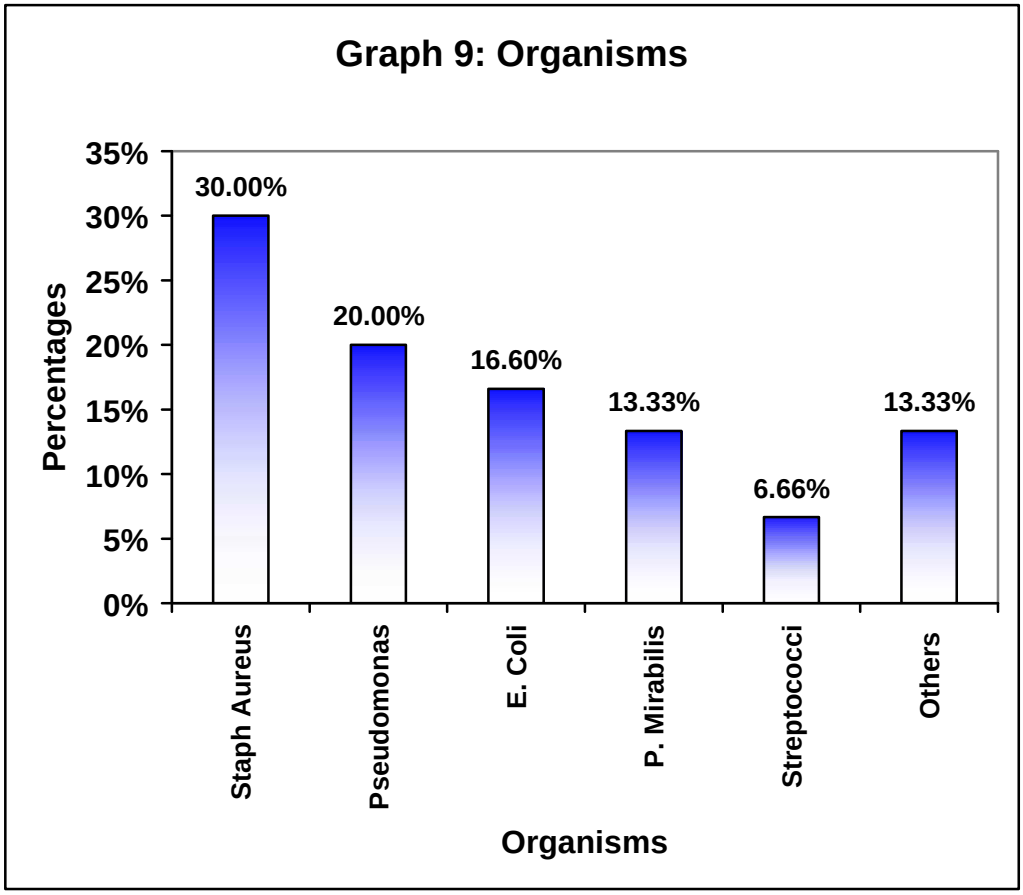
p=0.3811 (Chi square test)



In our study total number of patient with positive ulcer culture in control group were 24 (80%) and culture negative ulcer were 6 (20%) on tenth day. In study group culture positive ulcers were 20 (66.66%) and culture (-) ulcers were 10 (33.33%). LLLT was useful in reducing infection in study group. However this effect was not statistically significant (p=0.3811)

Table 13: Organisms

Organisms	Number of patients	
	No.	**Percentage**
Staph aureus	18	30.00%
Pseudomanas	12	20.00%
E. Coli	10	16.60%
P. Mirabils	08	13.33%
Strytococci	04	6.66%
Others	08	13.33%



Staph aureus and pseudomas were most common infections in diabetic foot ulcers (30% and 20% respectively).

DISCUSSION

Foot ulceration is a complication caused by diabetic disease and is commonly infected. The source of infection is usually the contamination of the break in the skin, which may be imperceptible like cracks or fissures. Most commonly infection is polymicrobial. The most common organisms are staphylococcus aureus, pseudomonas and beta haemolytic streptococci. The diabetic state, needs to be well controlled and infection should be effectively treated to achieve healing of the ulcer.

Control of infection in diabetic foot ulcers is an unbeatable challenge.Infection results in microthrombi in smaller vessels of foot. This impairs blood flow leading to ischaemia which makes eradication of bacteria difficult by antibiotics.

Low level laser therapy has shown great promise as a procedure for healing of chronic wounds. The beneficial effect of LLLT on wound healing in diabetic foot ulcers was observed in the study conducted in our institution.[42] LLLT increases hydroxyprolene level at wound site leading to faster healing as compared to conventional dressing alone.

This beneficial effect of LLLT on diabetic foot ulcers was due to its effect on wound healing dynamics, but to know whether LLLT has antibacterial effect also was the purpose of this study.

Rationale of conducting this study was to know the antibacterial effect of LLLT in diabetic foot patients. Effect of LLLT in controlling wound infections has not been studied. However LLLT has been found to increase the bacterial growth in in-vitro studies.[43] It has also been shown that LLLT improves tissue permeability resulting in increase in local antibiotic concentration.[44] This study explores the antibacterial effect of LLLT on infected diabetic foot ulcers.

The present study was conducted in JNMC and KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belgaum to know the antibacterial effect of LLLT (Helium Neon laser media) in infected diabetic foot ulcers.

In the present study it was seen that incidence of diabetic foot ulcer was more in males (80%) as compared to females (20%).The national data source, NHDS documented higher hospital rates in males suffering from diabetic foot ulcer.

Diabetic foot ulcers are most commonly seen in 6^{th} decade(66.66%),while 18.33% were affected in 4^{th} decade and 11.6% were in 3^{rd} decade, we had 2 patients (3.33%) in 2^{nd} decade. Older the patient, more the chances of diabetic foot ulcer and more chances of infections.

In our study only 2 patients (3.33%) were of type 1 DM and rest had type 2 DM. In our study 12 (20%) patients were diagnosed with DM of less than five years duration. Twenty six (43.33%) patients were suffering from DM for the duration of 6 to 10 years. It was observed that 22 patients had history of DM for more than 10 years.

In this study 35% patients had neuropathy (21) and 10% had vasculopathy (6). More than half of the patients (53.3%) had ulcer on dorsal surface of foot, remaining (46.66) had ulcer on plantar surface. Trauma was most common cause of foot ulcer in our study (65%). Thirty five percent patients had ulcers which were spontaneous in origin secondary to blister rupture or unnoticed trivial trauma.

All culture positive diabetic foot ulcer patients were included in the study. At the end of tenth day out of 30 patients in control group 24 (80%) patients had positive cultures and 6 (20%) had negative culture. Where as in study group out of 30 patients 10 (33.3%) had negative culture, in remaining 20 (66.6%) patients culture remained positive. Although numerically study group had better infection control (10 Vs 6), it was not statistically significant, as the p value of 0.3811 derived by Chi-squire test.

It was also observed that staph aureus was commonest organism accounting for 30% of wound infection, second commonest organism cultured was pseudomonas (20%).

Limitation of study

- Short follow up period to derive conclusion on long term anti-infective property of laser.
- Cost involved was not analysed.

Scope for further study

- Assessment of colony count of bacteria would be a better indicator of antibacterial property of LLLT.
- Use of different lasing media to asses the antibacterial property.
- Use of LLLT with ultraviolet waves in wound healing and their anti-infective property.

CONCLUSION

The wound subjected to LLLT with conventional therapy showed decreased infection rate at 10^{th} day of culture as compared to conventional dressing group alone. But this finding was not statistically significant. ($p<0.038$)

Large scale trials are required to know the anti-infective property of LLLT. Multicentre trials are required to know the anti-infective property of LLLT.

SUMMARY

Incidence of diabetes on the rise globally. Diabetic foot being one of the most common complications, where 15% of all diabetics develop diabetes foot ulcers. Infection of diabetic foot ulcer is a consequence of ulcer not the cause of ulcer.

Various modalities of treatment have been developed to aid faster healing of foot ulcers. Different types of dressings are used to control the infection of diabetic foot ulcers.

LLLT increases the hydroxy prolene level and help for faster healing of diabetic foot ulcers.

- All culture positive diabetic foot patients were included in study. Total 60 patients with infected diabetic foot ulcers were studied. They were divided in two groups of 30 each.

- One group received conventional dressing with antibiotic (Ciprofloxacin + Tinidazole) as a control group and study group received LLLT along with conventional dressing with same antibiotic. A comparative study was done in both groups regarding wound culture on fifth and tenth day.

- Males were more affected than females. 80% were males and 20% were females.

- 66.6% patients were within age group of 51 – 60 years of age.

- Only two patients had type I diabetes mellitus and rest had type II diabetes mellitus.

- 46.66% patient had ulcer over planter area and 53.33% patients had ulcer over dorsum of foot.

- Trauma was most common cause of diabetic foot ulcers, in 65% of patients. Rest had spontaneous onset of ulcer.

- Patient with stress factors and osteomyelitis were excluded from the study.

- In our study 24 (80%) patients in control group had positive wound culture on day 10 and six (20%) had negative would culture. Whereas in study group 20 (66.66%) patients had positive wound culture on day 10^{th} day and 10 patients had culture negative wound. Those were analysed statistically and P value was 0.038 by Chi-square test which was statistically insignificant suggesting that anti infection property of LLLT was not statistically significant.

- In our study it was also observed that mot common infective organism was staph aureus (30%) second most common was pseudomonas (20%).

- Thus LLLT with conventional dressing in the treatment of diabetic foot ulcer was effective in reducing wound infection but it was not statistically significant as compared to conventional dressing alone. Further large scale, multicentres trial are required for better comparison.

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ANNEXURE I - CONSENT FORM

Introduction

Mr./Miss/Mrs_____

your are invited to participate in our research study that is **"RANDOMISED CONTROL TRIAL TO STUDY ANTIBACTERIAL EFFECT OF LOW LEVEL LASER THERAPY IN INFECTIVE DIABETIC FOOT ULCERS ADMITTED TO KLES PRABHAKAR KORE HOSPITAL AND MEDICAL RESEARCH CENTRE, BELGAUM".**

Since you are suffering from Diabetes and the foot ulcer, which is not healing since a long time and will be requiring treatment for the same, you are eligible to be part of the study and hence asked to participate. This research is about the beneficial effects of Low level laser therapy on your foot ulcer and the result of this research will help in a better treatment of similar participants in the future.

If you agree to be part of this research, we would ask you some relevant clinical history. You are free to not to answer to which ever questions you think are not relevant. A clinical examination will be done and then culture will be taken from wound. On the first day Empirical antibiotic will be given and regular betadine dressings will be done. After culture sensitivity report the effective antibiotic will be started and low level laser therapy will be given at the ulcer site. Culture will be repeated on 5^{th} and 10^{th} day.

There are chances you may have a speedy and better recovery with this therapy and it will also help in the treatment if participants with similar complaints in the future. Your decision of whether or not to participate in this study will not effect the quality of treatment you receive. Further you may withdraw from the study at any time.

All the new information collected about you during this course of study will be kept confidential to the extent permitted by law. Any information which identifies you personally, will not be released without your written consent.

This study does not have any damaging aspect and there are no chances of injury during the course of the study, but if injured the investigator is not responsible. There will be no extra cost incurred by you. However you will have to pay for the routine investigations, which are a part of the existing management protocol for the treatment of diabetes. There is no commitment for any reimbursement or any compensation for the participant. The participation in this study is entirely voluntary and you may withdraw from the study at any time. At any time during or after the study, for any information you may contact the researcher.

Dr. Prashant Tubachi Room No.126, Chanakya Hospital, J.N. Medical College, Nehru Nagar, Belgaum. Karnataka. **Contact No: 9886914650**	**Chairman,** Institutional Ethics Committee, Dr. V.D. Patil, Phone: 0831-2471350.

Signature of the participant or legally authorized representative:

Subject Name : _____

Signature or the Left Thumb Print of Subject : _____

Witness Name: _____ Signature : _____

Investigators Name: _____ Signature: _____

Date : _____ Place : _____

ANNEXURE II – PROFOMA

I) Patient identification data

Name: IP/OPD No :

Age: DOA :

Sex: DOD :

Occupation:

Address:

II) Chief complaints

Medical history

Peripheral Neuropathy ( )

Nephropathy ( )

Retinopathy ( )

PVD ( )

CVD ( )

Diabetic status

Type :

Duration :

Méditation :	Oral Hypoglycemics	Insulin
	( )	( )
Complication	Neuropathy	Vasculopathy
	( )	( )

Ulcer details

1. Mode of Onset — Traumatic ( )
   Spontaneous ( )
   Pressure ( )
   Others ( )
2. Duration
3. Progress

Wound observation

1. Site
2. Size
3. Shape
4. Edge
5. Margin
6. Floor
7. Base
8. Discharge
9. Surrounding Skin

Neurological examination

VASCULAR EXAMINATION :	Left	Right
Popliteal a.	( )	( )
Ant. Tibial	( )	( )
Post Tibial	( )	( )
Dorsalis Pedis	( )	( )

Any foot deformity present

Toe deformity

Bunion

Charcots foot

Foot drop

If amputation has been done specify

Date :

Side :

Level :

Cause for amputation:

Foot wear assessment

Does patient wear appropriate shoes

Does patient require contact cast immobilization

Investigation

CBC

FBS 1st _____ Date :_____ Time :_____

2nd (24 hr apart) ___ Date :_____ Time : _____

Culture from the ulcer site on admission, on 5th and 10th day

Sr. Creatinine

UKB

Urine

Routine

Microscopy

X-ray Foot

AP View

Lat. View

Type of Dressing

Saline ( )

Saline + Betadiene ( )

LLLT Specifications (if applicable)

Duration :

Power :

Frequency :

Cluster Probe :

Dose :

Daily / Alternate Days:

ANNEXURE III – PHOTOGRAPHS

Photo 1: Low Level Laser Equipment

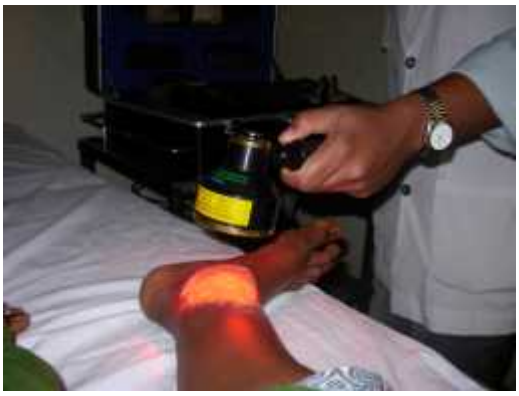

Photo 2: Low Level Laser Therapy Being Administered

Photo 3: Low Level Laser Therapy Being Administered

Photo 4: Low Level Laser Therapy Being Administered

ANNEXURE IV MASTER CHART – CONTROL GROUP

STUDY GROUP

Sl. No.	IP No.	Gender	Age (Years)	History		Diabetic status			Complications		Ulcer details				Investigations												Outcome
												Onset			CBC				FBS				Culture			
				Peripheral neuropathy	PVD	Type	Duration (Years)	Medication	Neuropathy	Vasculopathy	Site	Traumatic	Spontaneous	Duration	Haemoglobin (gm%)	PCV	TLC	Platelet Count (Lakhs)	Day 1	Day 2	UKB	Sr. Creat (mg/dL)	Day 1	Day 5	Day 10	
1	262663	M	27	-	-	I	7	IN	-	-	P	+	-	2.0	10.6	32	10800	2.3	170	130	-	1.2	STP	STP	STP	+
2	259398	F	46	-	-	II	10	OR	-	-	D	+	-	1.5	13.0	36	8300	2.2	190	145	-	1.4	STP	STP	STP	+
3	256124	M	37	+	-	I	12	IN/OR	-	-	P	+	-	1.5	10.0	30	8900	1.6	202	175	-	1.6	PSU	PSU	PSU	+
4	255419	M	36	-	-	II	5	IN	-	-	D	+	-	2.0	12.4	35	10600	1.7	230	210	-	1.6	STP	STP	STP	+
5	291243	M	35	-	-	II	7	OR	-	-	D	-	+	2.2	14.0	39	13200	2.6	135	157	-	1.4	ECL	ECL	-	-
6	299207	M	39	-	-	II	5	OR	-	-	P	-	+	3.0	9.0	30	14000	2.0	136	163	-	0.9	PSU	PSU	PSU	+
7	247311	F	50	+	-	II	15	OR	+	+	D	+	-	1.0	12.0	34	12300	1.6	190	178	-	1.9	STR	STR	-	-
8	247398	M	43	-	-	II	10	OR	-	-	D	+	-	1.5	11.0	33	11066	1.7	132	100	-	1.6	PSU	PSU	PSU	+
9	259383	M	42	+	-	II	2	IN	+	-	P	+	-	2.0	11.5	36	12300	2.2	113	110	-	1.3	STR	STR	-	-
10	259691	F	47	-	-	II	10	IN	-	-	D	-	+	1.0	13.0	37	11300	2.5	170	110	-	0.8	STP	STP	STP	+
11	250562	M	49	-	-	II	12	OR	+	+	P	-	+	1.0	12.0	35	12350	1.7	135	110	-	1.4	PMR	PMR	-	-
12	238998	M	60	+	-	II	15	IN	-	+	D	+	-	1.5	12.0	34	13500	1.6	138	170	-	1.2	ECL	ECL	ECL	+
13	252958	M	60	+	-	II	17	IN/OR	+	-	P	-	+	2.0	13.0	39	12500	1.7	130	180	-	1.2	STP	STP	STP	+
14	254624	M	52	-	-	II	16	IN/OR	+	-	D	+	-	2.5	10.5	29	10500	1.6	190	135	-	1.3	PMR	PMR	PMR	+
15	262373	M	54	-	-	II	17	IN/OR	-	-	D	+	-	1.0	9.0	26	10200	1.5	200	160	-	1.2	STP	STP	STP	+
16	254906	F	56	+	-	II	19	IN/OR	+	-	P	+	-	1.0	10.5	30	11320	1.6	210	97	-	0.9	STR	STR	STR	+

Sl. No.	IP No.	Gender	Age (Years)	History		Diabetic status			Complications		Ulcer details				Investigations												Outcome
												Onset			CBC				FBS				Culture			
				Peripheral neuropathy	PVD	Type	Duration (Years)	Medication	Neuropathy	Vasculopathy	Site	Traumatic	Spontaneous	Duration	Haemoglobin (gm%)	PCV	TLC	Platelet Count (Lakhs)	Day 1	Day 2	UKB	Sr. Creat (mg/dL)	Day 1	Day 5	Day 10	
17	259632	M	54	-	-	II	14	OR	+	-	D	+	-	1.5	10.6	32	10500	1.9	160	192	-	1.1	ECL	ECL	ECL	+
18	264562	M	55	-	-	II	7	IN	-	-	D	+	-	1.0	10.6	31	11500	1.6	190	145	-	1.4	STP	STP	STP	+
19	273252	M	55	+	-	II	10	IN	-	-	P	+	-	1.0	16.0	45	9800	1.2	176	128	-	1.8	ECL	ECL	ECL	+
20	276860	M	56	-	-	II	11	IN/OR	+	-	D	-	+	1.5	10.0	30	12500	1.6	172	185	-	0.5	OTH	OTH	OTH	+
21	263488	M	57	-	-	II	15	IN/OR	-	-	D	+	-	2.0	12.0	35	13200	1.2	130	123	-	1.2	PMR	PMR	PMR	+
22	271248	M	57	+	-	II	3	OR	-	-	P	+	-	1.0	9.0	26	13000	1.1	179	127	-	1.4	PSU	PSU	PSU	+
23	295693	F	55	-	-	II	5	OR	+	-	D	+	-	1.5	9.5	30	12300	1.2	123	130	-	1.1	OTH	OTH	OTH	+
24	249791	M	57	+	-	II	7	IN	-	-	D	-	+	1.5	13.0	36	11500	1.3	127	130	-	1	STP	STP	STP	+
25	253906	M	56	-	-	II	10	IN/OR	-	-	P	+	-	1.0	14.0	39	12500	1.5	160	120	-	1.7	OTH	OTH	OTH	+
26	254644	M	59	+	-	II	13	IN	+	-	D	+	-	2.0	16.0	45	11500	2.6	175	157	-	1	STP	STP	-	-
27	257189	M	57	-	-	II	10	IN	-	-	D	+	-	2.5	12.0	34	12500	2.7	200	174	-	1.7	OTH	OTH	OTH	+
28	258228	M	60	+	-	II	11	IN	+	-	P	+	-	2.5	10.0	28	13000	2.8	210	160	-	1.9	STP	STP	-	-
29	259383	M	56	-	-	II	14	OR	-	-	D	+	-	1.0	11.5	34	11500	2.8	175	97	-	1.2	PMR	PMR	PMR	+
30	278525	M	58	+	-	II	13	IN/OR	-	-	D	+	-	2.5	12.0	36	11250	1.7	137	105	-	0.9	ECL	PMR	ECL	+

Sl. No.	IP No.	Gender	Age (Years)	History		Diabetic status			Complications		Ulcer details				Investigations													Outcome
												Onset			CBC				FBS				Culture				
				Peripheral neuropathy	PVD	Type	Duration (Years)	Medication	Neuropathy	Vasculopathy	Site	Traumatic	Spontaneous	Duration	Haemoglobin (gm%)	PCV	TLC	Platelet Count (Lakhs)	Day 1	Day 2	UKB	Sr. Creat (mg/dL)	Day 1	Day 5	Day 10	
1	273416	F	24	+	-	II	2	IN	-	-	P	+	-	1.0	14.0	40	13000	1.6	170	123	-	0.5	STP	STP	STP	+
2	272134	M	40	-	-	II	8	IN	-	-	P	-	+	1.5	10.0	30	12200	1.7	127	130	-	1.2	STR	STR	-	-
3	253921	M	40	-	-	II	10	IN	-	-	D	+	-	1.0	13.0	39	11500	1.2	170	115	-	1.6	PSU	PSU	PSU	+
4	282529	F	35	-	-	II	14	OR	-	+	P	-	+	1.5	9.3	27	15300	1.6	176	110	-	1.7	PSU	PSU	-	-
5	253906	M	50	+	-	II	3	IN/OR	+	-	D	+	-	2.0	9.7	26	14500	2.5	120	145	-	1.4	STP	STP	STP	+
6	264539	M	50	+	-	II	10	IN	-	-	P	+	-	1.0	11.2	32	14200	2.2	135	110	-	1.6	ECL	ECL	ECL	+
7	268196	M	50	-	-	II	11	IN/OR	-	-	D	-	+	2.0	13.4	39	12200	2.6	123	126	-	1.2	STP	STP	STP	+
8	250562	M	49	+	-	II	15	IN/OR	-	-	P	+	-	1.5	11.0	42	10500	2.4	137	200	-	1.1	PSU	PSU	PSU	+
9	252223	M	42	-	-	II	6	OR	-	-	P	-	+	1.0	13.0	39	16500	2.5	190	139	-	1.6	ECL	ECL	ECL	+
10	246510	F	60	+	-	II	7	OR	+	-	P	+	-	1.5	10.0	30	12000	1.2	207	176	-	1.4	STP	STP	-	-
11	258061	M	51	+	-	II	9	IN	+	-	D	+	-	1.0	12.0	35	11500	1.3	205	170	-	1.9	PSU	PSU	PSU	+
12	266756	M	52	+	-	II	10	OR	-	+	D	+	-	2.0	13.0	39	12500	1.4	210	205	-	1.8	STP	STP	STP	+
13	266753	M	52	+	-	II	4	OR	-	-	P	-	+	2.5	11.0	32	11750	1.5	185	140	-	1.2	OTH	OTH	-	-
14	273416	F	39	-	-	II	6	OR	-	-	P	+	-	2.0	12.0	36	11500	1.7	160	130	-	0.7	STP	STP	STP	+
15	258567	M	60	+	-	II	10	IN	+	-	P	-	+	1.5	14.0	37	12500	1.2	155	110	-	0.8	PSU	PSU	PSU	+
16	266031	M	57	-	-	II	7	OR	+	-	D	+	-	2.0	15.0	42	12600	1.7	190	170	-	1.2	PMR	PMR	PMR	+

Sl. No.	IP No.	Gender	Age (Years)	History		Diabetic status			Complications		Ulcer details				Investigations													Outcome
												Onset			CBC				FBS				Culture				
				Peripheral neuropathy	PVD	Type	Duration (Years)	Medication	Neuropathy	Vasculopathy	Site	Traumatic	Spontaneous	Duration	Haemoglobin (gm%)	PCV	TLC	Platelet Count (Lakhs)	Day 1	Day 2	UKB	Sr. Creat (mg/dL)	Day 1	Day 5	Day 10	
17	262373	M	54	+	-	II	4	IN	-	-	P	-	+	2.0	10.0	30	1120	1.8	155	130	-	1.6	STP	STP	STP	+
18	271766	M	56	+	-	II	7	OR	+	-	D	+	-	2.0	10.5	32	10200	1.9	185	123	-	1.5	ECL	ECL	ECL	+
19	275988	M	58	+	-	II	10	IN	-	-	P	+	-		12.0	35	15600	1.7	120	115	-	1.2	PMR	PMR	PMR	+
20	277631	F	55	+	-	II	12	IN/OR	-	-	D	-	+		13.4	38	12300	1.8	146	150	-	1.8	PSU	PSU	-	-
21	276128	M	54	-	-	II	4	OR	+	-	P	+	-		15.0	45	10000	1.9	100	95	-	1.4	ECL	ECL	ECL	+
22	278800	F	60	+	-	II	3	OR	+	-	P	-	+		11.5	33	11500	1.7	126	155	-	1.6	OTH	OTH	-	-
23	252958	M	60	+	-	II	2	IN	-	-	D	+	-		10.0	30	9500	2.3	201	150	-	1.7	PMR	PMR	-	-
24	258507	M	58	-	-	II	10	IN	-	-	P	-	+		12.0	35	19000	2.5	170	160	-	1.8	STP	STP	-	-
25	272862	M	58	-	-	II	15	IN/OR	-	-	D	+	-		11.5	30	12200	1.7	160	110	-	1.6	PSU	PSU	PSU	+
26	274283	M	60	+	-	II	14	IN/OR	-	-	P	-	+		9.4	28	10500	1.2	100	140	-	1.7	OTH	OTH	-	-
27	277809	M	57	-	-	II	12	IN/OR	-	-	D	-	+		10.5	30	11500	1.8	120	134	-	1.2	PSU	PSU	PSU	+
28	280700	M	52	-	-	II	10	IN	-	-	P	-	+		11.4	30	11750	1.6	150	156	-	1.6	ECL	ECL	-	-
29	293999	M	58	+	-	II	8	IN	+	-	D	+	-		10.0	30	10500	1.7	156	125	-	0.8	PMR	PMR	PMR	+
30	295693	F	55	-	-	II	7	OR	+	-	P	-	+		15.0	45	11500	2.5	190	129	-	0.9	OTH	OTH	OTH	+

ANNEXURE IV

KEY TO MASTER CHART

-	-	Absent
+	-	Present
CBC	-	Complete blood count
D	-	Dorsum
ECL	-	Escherchia coli
F	-	Female
FBS	-	Fasting blood sugar
Hb%	-	Haemoglobin
IN	-	Insulin
IP No.	-	Inpatient Number
M	-	Male
Mg/dL	-	Milligram per decilitre
OR	-	Oral
OTH	-	Others
P	-	Plantar
PCV	-	Packed cell volume
PMR	-	Proteus mirabilis
PSU	-	Pseudomonas
PVD	-	Peripheral vascular disease
Sl. No	-	Serial Number
Sr.	-	Serum

STP	-	Staphylococcus aureus
STR	-	Streptococci
TLC	-	Total leucocytic count
UKB	-	Urine ketone body