CLINICAL STUDY ON SURGICAL MANAGEMENT OF DIABETIC FOOT AND ITS COMPLICATIONS

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ABSTRACT

Diabetes is a lifelong problem, and the incidence of diabetic foot complications increases with age and duration of the disease. Infection, gangrene, ulceration, amputation are significant complications of the disease. One of the most common complications of diabetes in the lower extremity is the diabetic foot ulcer. An estimated 15% of patients with diabetes will develop a lower extremity ulcer during the course of their disease. Charcot foot, can lead to limb-threatening disorders, is one of the serious complication of long-standing diabetes. These complications frequently result in high morbidity, repeated hospitalizations, and mortality. Risk factors identified include peripheral neuropathy, vascular disorders, limitation in joint mobility, deformities of foot, change in foot pressures, minor trauma, a history of ulceration or amputation, and impaired visual acuity. Diabetic foot infections are frequently polymicrobial in nature. Hyperglycemia, impaired immunologic responses, neuropathy, and peripheral arterial disease are the major predisposing factors leading to limb-threatening diabetic foot infections. Not all foot complications can be prevented, dramatic reductions in frequently have been achieved by taking a multidisciplinary approach to patients. The emulation of the diabetic foot involves careful assimilation of the patient’s history and physical findings with the results of necessary diagnostic procedures. Early detection of foot pathology, especially in high risk patients, can lead to earlier intervention and thereby reduce the potential for hospitalization and amputation. Amputation of the foot may be indicated when neuropathy, vascular disease, and ulcerative deformity have led to soft tissues necrosis, osteomyelitis, uncontrollable infection or intractable pain. Local treatment of the ulcer consists of repeated debridement and dressing. Simple surgeries like split skin grafting or minor toe amputations

KEYWORDS: Diabetes, foot ulcers, neuropathy, ischemia.

INTRODUCTION

Diabetes mellitus is and iceberg disease. It is a worldwide problem. The incidence of diabetes mellitus is increasing globally.1 Patients with diabetes have a 12% to 25% life time risk of developing a foot ulcer.2 In the diabetic patient, the foot is prone for many pathological processes, in which almost all components of the lower extremity involved; from skin, subcutaneous tissue, muscles, bones and joints, to blood vessels and nerves. Ulceration, infection, gangrene, and amputation are significant complication of the disease, estimated to cost billions of dollars each year. Prevalence of diabetes in adults worldwide was estimated to be 4% in 1995 and is expected to rise to 5.4% by the year 2025. The number of adults with diabetes in the world will rise from 135 million in 1995 to 300 million in the year 2025. There will be a 42% increase, from 51 to 72 million, in the developed countries and a 170% increase, from 84 to 228 million, in the developing countries. The countries with the largest number of people with diabetes are, and will be in the year 2025, India, China, and the U.S.2

India alone, diabetes is expected to increase from 40.6 million in 2006 to 79.4 million by 2030.4 India presently has the largest number of diabetic patients in the world and India is thus designated to become the —diabetes capital of the world. Diabetic foot ulcers occur mostly among elderly people, and elderly diabetics have twice the risk of developing foot ulcers, three times the risk of developing a foot abscess and four times the risk of developing osteomyelitis.5

DEABETES MELLITUS

Diabetes mellitus is characterized by a chronic hyperglycemia with disturbances of carbohydrate, fat, and protein metabolism resulting from defects in insulin secretion, insulin action, or both. When fully expressed, diabetes is characterized by fasting hyperglycemia, but
the disease can also be recognized during less overt stages, most usually by the presence of glucose intolerance. The effects of diabetes mellitus include long-term damage, dysfunction, and a failure of various organs, especially the eyes, kidneys, heart, and blood vessels. Diabetes may present with characteristic symptoms such as thirst, polyuria, blurring of vision, weight loss, and polyphagia, and in its most severe forms, with ketoacidosis or hyperosmolarity, which in the absence of effective treatment, leads to stupor, coma, and death.

**DIAGNOSIS**

1. Symptoms of diabetes plus casual plasma glucose concentration is > or = to 200mg/dl (11.1 mmol/l). Casual is defined as any time of day without regard to times since last meal. The classic symptoms of diabetes include polyuria, polydipsia, and unexplained weight loss.
2. FPG > or = 126mg/dl (7.0 mmol/l). Fasting is defined as no caloric intake for at least 8 hr.
3. 2-hr post load glucose > or = to 200 mg/dl (11.1 mmol/l) during an OGTT. The test should be performed as described by WHO, using a glucose load containing the equivalent of 75g anhydrous glucose dissolved in water.

**PATHOGENESIS**

**Type 1 DM**

Type 1 DM is the result of interactions of genetic, environmental, and immunologic factors that ultimately lead to the destruction of the pancreatic beta cells and insulin deficiency. [7]

**Autoimmunity**

Type 1 diabetes is an autoimmune disease in which islet destruction is caused primarily by T lymphocytes reacting against as yet poorly defined beta-cell antigens, resulting in a reduction in beta-cell mass. T lymphocytes react against beta-cell antigens and cause cell damage. These T cells include CD4+ T cells of the Th1 subset, which cause tissue injury by activating macrophages, and CD8+ cytotoxic T lymphocytes, which directly kill beta cells and also secrete cytokines that activate macrophages. In the rare cases in which the pancreatic lesions have been examined at the early active stages of the disease, the islets show cellular necrosis and lymphocytic infiltration. This lesion is called insulitis. Locally produced cytokines damage beta cells. Among the cytokines implicated in the cell injury are TNF-γ, produced by T cells, and tumor necrosis factor and interleukin-1, produced by macrophages that are activated during the immune reaction. Autoantibody against a variety of beta-cell antigens, including insulin and glutamic acid decarboxylase, are also detected in the blood of 70% to 80% of patients and may contribute to islet damage. [8]

**Environmental Factors**

Putative environmental triggers include viruses (coxsackie and rubella most prominently), bovine milk proteins, and nitrosourea compounds. [9]

**Genetic Considerations**

Early-onset type-1 diabetes is associated with specific HLA phenotypes on chromosome 6 responsible for class 2 histocompatibility complexes, namely, DR3 and DR4. Other genes in the closely located DQ locus may confer increased risk or protection from clinical type-1 diabetes. [10]

**Type 2 DM**

Type 2 diabetes can be best understood as heterogeneous, having both genetic and environmental causes. Environmental influences, such as a sedentary lifestyle and dietary habits, clearly have a role, as will become evident when obesity is considered. Nevertheless, genetic factors are even more important than in type 1 diabetes. The two metabolic defects that characterize type 2 diabetes are:

1. Insulin resistance and
2. Beta-cell dysfunction that is manifested as inadequate insulin secretion in the face of insulin resistance and hyperglycemia. In most cases, insulin resistance is the primary event and is followed by increasing degrees of beta-cell dysfunction. [11]

**COMPLICATIONS**

**Acute Complications**

1. Diabetic Ketoacidosis: DKA is a medical emergency in patients with diabetes, with an incidence of 4-8/100 patient-years. [12] It affects type 1 patients almost exclusively.

They pathogenesis of DKA begins with insulin deficiency in the face of abnormally high concentrations of counter regulatory hormones, usually in the setting of an intercurrent illness. The hormones involved are related to sympathoadrenal activation, specifically cortisol and epinephrine, as well as glucagon, growth hormone, and thyrroxine. The underlying cause is usually infection, typically of viral etiology. Other provocative events include myocardial infraction, cerebrovascular accident, pulmonary embolus, pregnancy, and omission of insulin treatment. This results in polyuria, free water loss, and eventual dehydration. Systemic acidosis results in anorexia, abdominal pain, nausea, and vomiting, further contributing to dehydration. The respiratory compensation for ketoacidosis is often characteristic deep, labored Kussmaul respirations. Treatment is urgent and consists of vigorous fluid and electrolyte replacement as well as high doses of insulin (0.1 U/kg/h) to inhibit ketone production.

2. Hyperosmolar Hyperglycemic Nonketotic Syndrome: Hyperosmolar hyperglycemic nonketotic syndrome is an acute complication usually seen in older patients with type 2 diabetes. It results in more severe hyperglycemia...
and dehydration than in DKA, and with lesser or absent ketosis.[13]

3. Hypoglycemia: hypoglycemia is an acute complication of the pharmacologic treatments of diabetes. It can result in mental status change, seizures, and coma if sustained and prolonged. The causes are related to insulin excess that is absolute.

Chronic complications
Microvascular Complications
1. Retinopathy
2. Nephropathy
3. Diabetic neuropathy

Macrovascular Complications
1. Congestive heart failure
2. Peripheral vascular disease of the lower extremities
3. Stroke

DIABETIC FOOT
World Health Organization definition
Describes the foot of a diabetic patient that has the potential risk of pathologic consequences, including infection, ulceration, and destruction of deep tissues associated with neurologic abnormalities, various degrees of peripheral arterial disease, and metabolic complications of diabetes in the lower limb.

Mechanism of development of the diabetic foot lesions
Ulceration, infection, and Charcot arthropathy are pathologic entities of the diabetic foot.

Development of diabetic foot ulceration
Foot ulcers in diabetes result from multiple pathophysiologic mechanisms, including roles for neuropathy, peripheral vascular disease, foot deformity, higher foot pressures and diabetes severity.[14] Diabetic neuropathy and peripheral vascular disease are the main etiologic factors predisposing to foot ulceration; they may act alone, together, or in combination with other factors, such as microvascular disese biomechanical abnormalities, and an increased susceptibility to infection.[15]

The other common mechanism of ulceration involves prolonged repetitive moderate stress. This normally occurs on the sole of the foot and is related to prominent metatarsal heads, atrophied or anteriorly displaced fat pads, structural deformity of the lower extremity and prolonged walking rigid deformities such as hallux valgus, hallux rigidus, hammertoe, and limited range of motion of the ankle, and MTP joints have been linked to development of diabetic foot ulcers.[16]

Pathogenesis of a diabetic neuropathy
Proposed Hypotheses for the Pathogenesis of Diabetic Peripheral Nerve Damage.[17]

- Chronic hyperglycemia
- Nerve microvascular dysfunction
- Increased free radical formation
- Polyol pathway hyperactivity
- Protein kinase C hyperactivity
- Nonenzymatic glycation
- Abnormalities of nerve growth[18]
INVESTIGATIONS
1. URINE EXAMINATION
- Sugar to diagnose diabetes mellitus. Benedicts qualitative and quantitative tests are used.
- Ketone bodies: to know whether diabetic ketoacidosis is present or not. It can be detected by rothera’s test. It is the first sign to be detected in ketosis.
- Albumin: the earliest clinical manifestation of diabetic renal disease is the finding of small quantities of albuminuria (30-300 mg/24 hr).

2. BLOOD EXAMINATION
- Haemoglobin percentage: it is useful investigation to know about general status of patient. Reduced haemoglobin may add in addition to the other causes of non healing ulcer.
- Total WBC count: indicates defence mechanism of body.
- Differential WBC count
- ESR
- Bleeding and clotting time
- Blood grouping and Rh typing

3. BLOOD SUGARS
- FASTING BLOOD SUGAR (FBS): a fasting plasma glucose test is used to detect diabetes or pre diabetes, and measures blood glucose after the patient has gone for a maximum of 8 hrs without healing. It is estimated by Folin Wu or Somgy’s nelson method.
- POST PRANDIAL BLOOD SUGAR (PPBS): After over night fast, the patient is given breakfast of 100 grams of carbohydrates or 100 grams of glucose load then after venous blood checked for glucose level every half hour for 2 hours.
- GLUCOSE TOLERANCE TEST: the ability of the body to dispose of an additional load of glucose is known as glucose tolerance test (GTT). It can distinguish normal subject who should have good glucose tolerance from diabetic who was not.
- Glycosylated hemoglobin HbA1C.

4. RENAL FUNCTION TEST
- Blood urea and Serum creatinine.

5. LIPID PROFILE
- Total cholesterol
- HDL cholesterol
- LDL cholesterol
- VLDL
- Triglycerides

6. NEUROLOGICAL EXAMINATION
Tendon reflexes to evaluate impaired neurological status. 128 Hz tuning fork test: the graduated 128 Hz tuning fork could be used to measure vibratory sense semi quantitatively. Semmes-Weinstein monofilament: the inability to perceive the 10g monofilament (5.07) at the
toes or dorsum of the foot predicts future occurrence of a diabetic foot ulcer. The advantages of this test are its simplicity and low cost. Therefore the 10g monofilament is a useful test to determine the future risk of ulceration.

7. PERIPHERAL VASCULATURE EVALUATION

Dorsalis pedis / posterior tibial pulses: claudication is the earliest symptom of peripheral vascular disease. It often later progresses to pain at rest. Vascular assessment must include palpation of all lower extremity pulses. The femoral, popliteal, posterior tibial, and dorsalis pedis pulses may be examined. The presence of palpable pulses does not absolutely exclude peripheral vascular disease.

Hallux blood pressure: toe blood pressures less than 40mmHg, are associated with impaired wound healing. Ankle-brachial pressure index: 86 ABPI is a simple and easily reproducible method of diagnosing vascular insufficiency in the lower limbs. ABPI is obtained by dividing the ankle by brachial systolic pressures. The index is normally greater than 1. A ratio of less than 1 is considered a sign of impaired flow to the extremity. In patient with claudication the index range from 0.6 – 0.9. a value of less than 0.5 signifies severe disease, and 0.3 or below is seen in limb threatening ischemia. The blood pressure at the ankle is measures using a Doppler ultrasound machine.

Transcutaneous oxygen saturation: transcutaneous oxygen tension(normal >40mmHg) measurement has been used as a noninvasive measurement of limb perfusion. Patients with occlusive disease have significantly reduced transcutaneous oxygen tension and this has been used to determine the possibility of ulcer and optimal amputation healing. The test is performed by placing a probe over the metatarsal region of the affected foot.

Duplex ultrasound scanning of the lower limbs: duplex ultrasound scanning combines colour flow imaging with B-mode and pulsed Doppler. This provides anatomical detail, visualization of flow, and flow velocity data. It is non invasive relatively portable, and low priced. Significant stenosis is indicated by peak systolic velocity ratio greater than 2 across the arterial lesion.

Digital subtraction angiography: this is currently considered the gold standard in the assessment of occlusive disease. It is a type of fluoroscopy technique used in interventional radiology to clearly visualize blood vessels in a bony or dense soft tissue environment. Images are produced using contrast medium by subtracting a precontrast image or the mask from later images, once the contrast media has been introduced into a structure. Hence the term DSA. DSA is primarily used to image blood vessels. It is useful in the diagnosis and treatment of arterial and venous occlusions, including carotid artery stenosis, pulmonary embolisms and acute limb ischemia. Arterial stenosis, which is particularly useful for potential renal donors in detecting renal artery stenosis. Cerebral aneurysms and arterio-venous malformations.

MRA: it is an excellent imaging modality for assessing PAD. It is non invasive and avoiding contrast exposure. MRA is particularly useful in patients who are at high risk for contrast induced nephropathy, particularly elderly diabetic patients.

Culture and sensitivity of discharge: a culture can identify the etiologic agents only if specimens are appropriately collected and processed. Antibiotic susceptibility results generally help to focus antibiotic regimens. Reliable specimens are those obtained aseptically at surgery, by aspiration of pus or by biopsy of infected tissue across the intact skin. Specimen should be transported to the laboratory promptly, and in suitable transport media. Organisms isolated from reliable specimens that are the sole or predominant bacteria on both Gram stained smear and culture are likely to be the true pathogens.

MATERIALS AND METHODS

This study of prospective control trial was conducted comprising of, 100 patients of diabetic foot in the department of General surgery at M N R Medical College and Hospital SANGAREDDY, during the period of OCTOBER 2014 TO SEPTEMBER 2016.

METHOD OF COLLECTION OF DATA

- Detailed history taking.
- Thorough physical examination.
- Routine investigations.
- Relevant special investigations.
- Choosing the appropriate line of treatment.
- Assessment of patients following treatment at regular intervals in comparison to his/her pre-treatment with regards to symptoms.
- All patients are studied and clinical findings are recorded as per proforma case sheet data analyzed and necessary investigations done as per required and treatment given .predisposing factors, complications, treatment and sequel are studied, analyzed and discussed.

INCLUSION CRITERIA

- All patients with diabetes mellitus suffering from foot ulcers and infections are included in the study.
- Age group of the patients: all age groups are included in the study.
- Patients with known past history of diabetes are also included.
- Patients with gangrenous foot, complicated by diabetes are included in the study.

EXCLUSION CRITERIA

- Patients with foot infections without diabetes mellitus are excluded.
- Patients with gangrene foot of etiology other than infection of foot complicated by diabetes are excluded.
- Patients whose treatment could not be completed due to non compliance are excluded.
- Incidental diagnosis of diabetes on admission.

STUDY PLACE: - M N R MEDICAL COLLEGE AND HOSPITAL.
STUDY DURATION: - 2 YEARS (OCTOBER 2014 TO SEPTEMBER 2016).
STUDY DESIGN: - PROSPECTIVE STUDY.

DISCUSSION
Hundred cases were studied from OCTOBER 2014 TO SEPTEMBER 2016 at M N R Medical College and Hospital, SANGAREDDY. The analysis of this study as follows.

Table 02: Age.

<table>
<thead>
<tr>
<th>Age</th>
<th>Wheel, locked and Rot series 1969[^19]</th>
<th>Present Study</th>
</tr>
</thead>
<tbody>
<tr>
<td>Youngest</td>
<td>32</td>
<td>31</td>
</tr>
<tr>
<td>Oldest</td>
<td>89</td>
<td>80</td>
</tr>
</tbody>
</table>

When compared with Wheel, Lock and Root series, there is not much HR difference in youngest and oldest age group.

Table 03: Sex Wise Distribution.

<table>
<thead>
<tr>
<th>Sex</th>
<th>Jennifer A. May field et. al.,[^20]</th>
<th>Present Study</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>No. of Cases</td>
<td>Percentage</td>
</tr>
<tr>
<td>Male</td>
<td>32</td>
<td>53</td>
</tr>
<tr>
<td>Female</td>
<td>29</td>
<td>47</td>
</tr>
</tbody>
</table>

Like Jennifer A. May field et. al., study, the Present study had more number of male patients 39 (78%) suffering from diabetic foot lesions than females 11 (22%). But the proportion between the two was far greater in this study. The present study had ratio of Male: Female as 3.54: 1. The incidence is more among males probably as they are mostly working out door, which makes them more vulnerable for trauma and sequel.

Table 04: Mode of Presentation (Gangrene).

<table>
<thead>
<tr>
<th></th>
<th></th>
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</tr>
</thead>
<tbody>
<tr>
<td>No. of Cases</td>
<td>946</td>
<td>614</td>
<td>1319</td>
<td>100</td>
</tr>
<tr>
<td>No. of Cases with Gangrene</td>
<td>236</td>
<td>274</td>
<td>64</td>
<td>24</td>
</tr>
<tr>
<td>Percentage</td>
<td>24.9</td>
<td>44.78</td>
<td>5</td>
<td>24</td>
</tr>
</tbody>
</table>

Incidence of gangrene in the present series is comparatively equal to that of Bell series of 1960.

Table 05: Site of Lesions.

<table>
<thead>
<tr>
<th>Site of Lesion</th>
<th>Apelquist et al.,[314][^22]</th>
<th>Reiber et al., [n=302][^23]</th>
<th>Present Study[^100]</th>
</tr>
</thead>
<tbody>
<tr>
<td>Toes</td>
<td>51</td>
<td>52</td>
<td>44</td>
</tr>
<tr>
<td>Dorsum of foot</td>
<td>14</td>
<td>11</td>
<td>64</td>
</tr>
<tr>
<td>Plantar metatarsal heads, Mid foot &amp; Heel</td>
<td>9</td>
<td>18</td>
<td>36</td>
</tr>
<tr>
<td>Multiple ulcers</td>
<td>7</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Whole fore foot</td>
<td>0</td>
<td>0</td>
<td>56</td>
</tr>
</tbody>
</table>

Out of 100 cases studied in this series, 22 cases (44%) presented as diabetic . ulcers. Out of these, 22 cases, the most common site of occurrence was on dorsum of foot 32% where as in Apelquist et. al., and Reiber et. al., study the common site was toes which was 51% and 52% respectively. Surprisingly toes (15%) were the least common site to be involved in the present study.
Table 06: History of Trauma.

<table>
<thead>
<tr>
<th></th>
<th>Jennifer A. Mayfield et. al.[26]</th>
<th>Present Study</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>No. of Cases</td>
<td>Percentage</td>
</tr>
<tr>
<td>Trauma</td>
<td>27</td>
<td>44</td>
</tr>
<tr>
<td>New onset of Ulcer without trauma</td>
<td>24</td>
<td>39</td>
</tr>
</tbody>
</table>

60 (60%) cases in this series had a history of trauma before the onset of the lesion. In Jennifer. A. May Field et al., there was no significant percentage of cases: with respect to history of trauma prior to occurrence of diabetic foot lesion.

Table 07: Comparison of Incidence of Neuropathy.

<table>
<thead>
<tr>
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<th></th>
<th></th>
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<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>No of cases</td>
<td>3175</td>
<td>150</td>
<td>264</td>
<td>354</td>
<td>100</td>
</tr>
<tr>
<td>Neuropathy</td>
<td>1206</td>
<td>74</td>
<td>84</td>
<td>125</td>
<td>52</td>
</tr>
<tr>
<td>Percentage</td>
<td>37.99</td>
<td>44.33</td>
<td>31.81</td>
<td>35.31</td>
<td>52</td>
</tr>
</tbody>
</table>

NEUROPATHIC LESIONS (COMPPLICATIONS)
In the present study 52 (52%) cases were found to have neuropathy. The majority of the patient had history of diabetes of more than 5 years. This shows that peripheral neuropathy is common in long standing diabetic patients.

Table 08: Culture and Sensitivity Comparision.

<table>
<thead>
<tr>
<th></th>
<th>Gibbons et al[24]</th>
<th>Wheat et. al.,</th>
<th>Hughes et al.,</th>
<th>Present study</th>
</tr>
</thead>
<tbody>
<tr>
<td>Staph aureus</td>
<td>22</td>
<td>20</td>
<td>25</td>
<td>30</td>
</tr>
<tr>
<td>Streptococcus species</td>
<td>13</td>
<td>23</td>
<td>20</td>
<td>14</td>
</tr>
<tr>
<td>Pseudomonas species</td>
<td>03</td>
<td>4</td>
<td>0</td>
<td>18</td>
</tr>
<tr>
<td>E.Coli</td>
<td>07</td>
<td>5</td>
<td>03</td>
<td>10</td>
</tr>
<tr>
<td>Klebsiella</td>
<td>04</td>
<td>6</td>
<td>07</td>
<td>08</td>
</tr>
<tr>
<td>Proteus</td>
<td>11</td>
<td>9</td>
<td>11</td>
<td>06</td>
</tr>
</tbody>
</table>

In the present study the commonest organism cultured was staphylococcus aureus 15 (30%) which was similar to study conducted by Gibbons et al. and Hugues et al., studies.

Treatment
In the present series, 12 cases were treated by slough excision and regular dressing. 14 cases were treated by wound debridement 10 with SSG, 16 by disarticulation of single or multiple toes at the level of metatarsophalangeal joints. I & D and fasciotomy done in 6 and 10 cases respectively. Below knee amputation was M V H done in 4 cases and above knee amputation was done in 14 cases.

Proper control of diabetes is very important in diabetic foot management fasting and post prandial blood sugar estimations were well under control. Infection was treated with broad spectrum antibiotics. Patients were educated about care of the foot.

Table 09: Amputation.

<table>
<thead>
<tr>
<th></th>
<th>Collen’s Series (1962)</th>
<th>Osakakosainekin Hospital (2005)</th>
<th>Present Study</th>
</tr>
</thead>
<tbody>
<tr>
<td>Number of Cases</td>
<td>215</td>
<td>210</td>
<td>100</td>
</tr>
<tr>
<td>Number of Amputation</td>
<td>83</td>
<td>110</td>
<td>18</td>
</tr>
<tr>
<td>Percentage</td>
<td>38.6</td>
<td>52</td>
<td>18</td>
</tr>
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</table>

The amputation rate is much lower 18% compared to collen's series 38.6% in 1962. This could be due to, better education of the patient, better glycemic control, proper case of foot, proper use of antibiotics, extensive debridement and regular Dressing after amputation wound healed well. The patients were referred to rehabilitation center for properties.

CONCLUSION
This study consists of 100 cases of diabetic foot patients with emphasis on surgical management and its complications over a period of 24 months
- The youngest patient in present study series of 100 patients studied was 31 years, oldest 80 years. Most of the patients were of 61-70 years.
The male to female ratio was approximately 3.54:1. Male predominance due to their increased susceptibility to trauma, smoking, and alcoholism.

Commonest presenting lesion was ulcers, followed by gangrene and cellulitis.

Commonest site of lesion was dorsum of foot followed by fore foot and toes.

Trivial trauma is the initiating factor in more than half of the cases.

Duration of the diabetes varies from 4 months to 24 years.

More than 50% patients had infection in addition to ischemia or neuropathy. This study indicates that all these three factors can be present in a patient with diabetic foot lesions.

Minimum duration of stay in hospital was 10 days and maximum 150 days.

Most common microorganisms grown from culture taken from the lesion was staph. aureus followed by pseudomonas.

Conservative treatment consists of control of diabetes with human actrapid /mixtard /lente/ Glargine insulin along with appropriate oral or iv antibiotics was effective in most of the cases.

Wound debridement, slough excision, followed by dressing with povidine-iodine, metronidazole, collagenase, L-lysine, mupirocin, etc., dressings resulted in healing of ulcers. Split skin grafting, disarticulation, bellow knee amputation, and above knee amputation, were the other modes of treatment.

Mortality rate in the present study was 2%.

SUMMARY

- Diabetes is a lifelong problem, and the incidence of diabetic foot complications increases with age and duration of the disease.
- By identifying high-risk patient and tailoring a total foot care prevention program accordingly, the incidences of ulceration and lower extremity amputations can be reduced.
- Diabetic patients at risk for foot lesions must be educated about risk factors and the importance of foot care, including the need for self-inspection and surveillance, monitoring foot temperatures, appropriate daily foot hygiene, use of proper footwear, good diabetes control, and prompt recognition and professional treatment of newly discovered lesions.
- Ulceration, infection, gangrene, and lower extremity amputation are complications often encountered in patients with diabetes mellitus. These complications frequently result in extensive morbidity, repeated hospitalizations, and mortality. They take a tremendous toll on the patient's physical and mental well-being as well as impose a substantial economic burden, often removing the patient from the workforce and placing a financial drain on the health care system.

- Not all diabetic foot complications can be prevented, but it is possible to dramatically reduce their incidence through appropriate management and prevention programs. The multidisciplinary team approach to diabetic foot disorders has been demonstrated as the optimal method to achieve favorable rates of limb salvage in the high-risk diabetic patient. Good glycemic control will prevent acute complications such as DKA and hypoglycemia.

REFERENCES