

CASE REPORT

DIABETIC FOOT DISEASE IN KING GEORGE HOSPITAL, VISHAKAPATNAM: A DESCRIPTIVE STUDY ON 100 PATIENTS

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ABSTRACT: Diabetic foot as the leading cause of lower limb amputation is one of the most important complications of diabetes mellitus. Effective treatment and formulation of prevention guidelines for diabetic foot require a thorough understanding of characteristics of diabetic foot patients and their ulcers, so we conducted this study to investigate these characteristics.

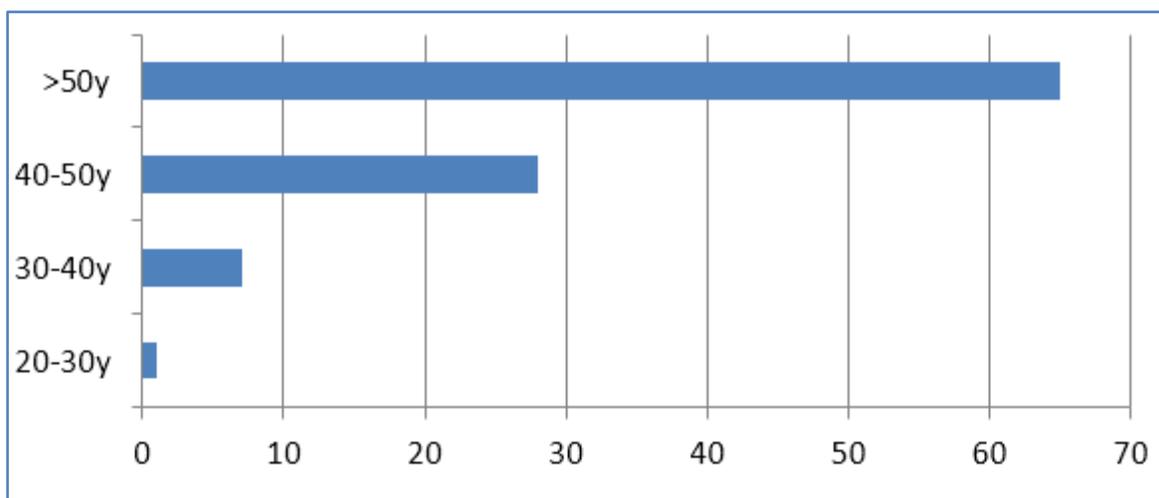
INTRODUCTION: Necessary data was collected from case sheets of 100 diabetic foot patients admitted in King Goerge Hospital, Vishakapatnam in the year 2014.

KEYWORDS: Diabetes mellitus Diabetic foot.

RESULTS: Case sheets of 100 patients with diabetic foot were included during the year 2014 in King Goerge Hospital, Vishakapatnam, India.

Mean age was 51.15. Most of the patiens developed diabetic foot between 5th and 6th decades of their life results including 7% of pts in age group 30-40 years 28% of pts in age group 40-50and 65% of pts above 50 years age group

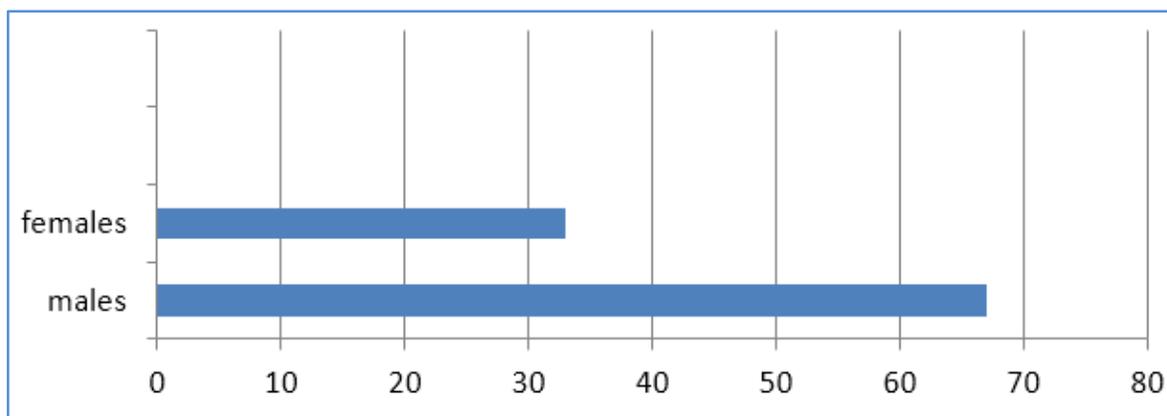
Age pattern of patients with diabetic foot:



Graph 1

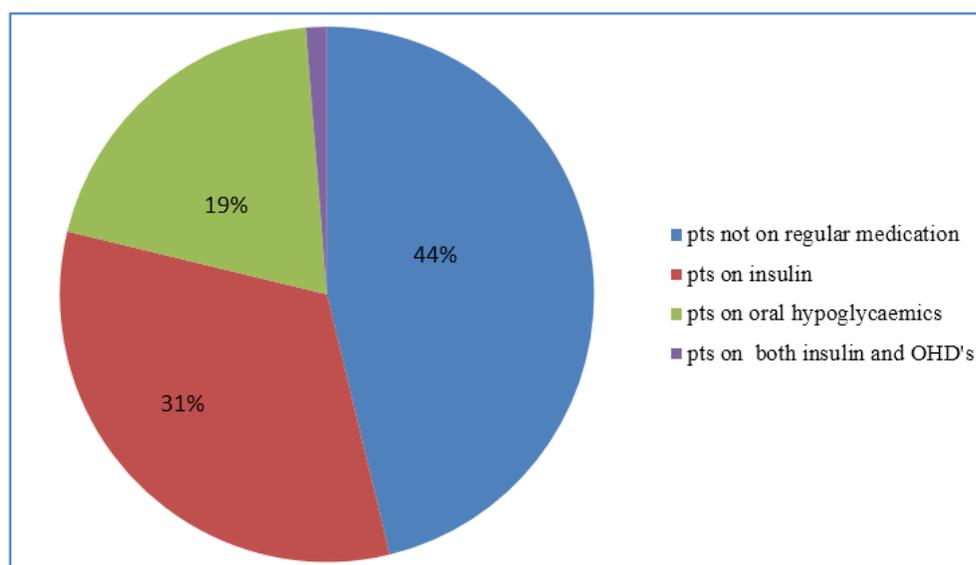
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Among these 67% were males and 33% of females:



Graph 2

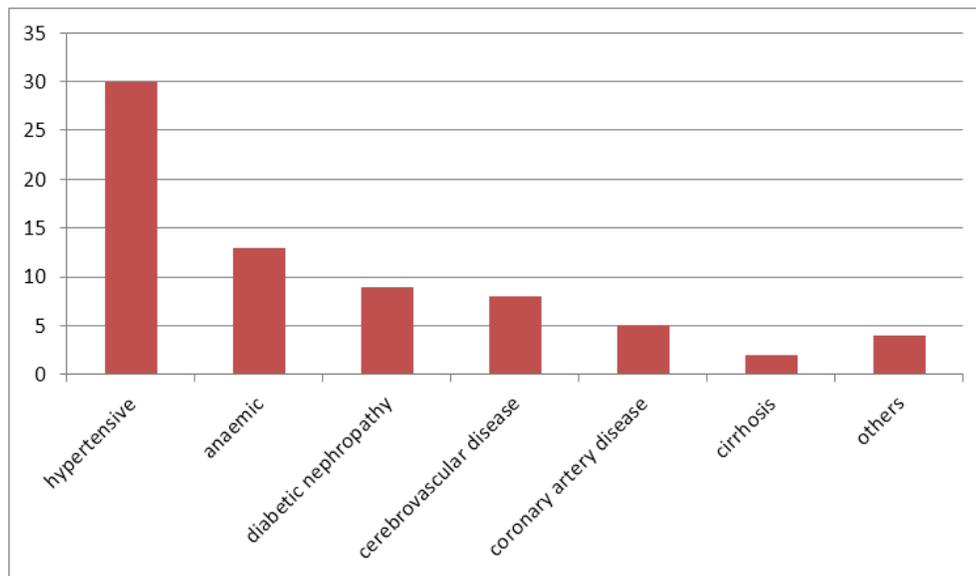
Only 56% of the patients with DM history were on regular medication among which 31% were on insulin 19% on oral hypoglycaemic drugs 6%on both OHD's and insulin.



Graph 3

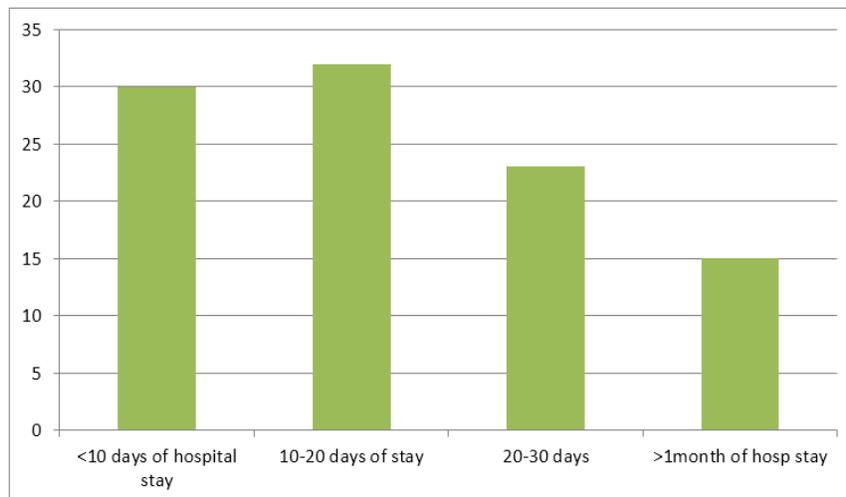
Patients were evaluated about presence of other comorbidities, most common concomitant comorbidity (30%) was hypertension followed by anaemia and diabetic nephropathy 8%with cerebrovascular disease 5% with coronary artery disease 2% with cirrhosis other comorbidities included infective endocarditis, leprosy, COPD.

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Graph 4

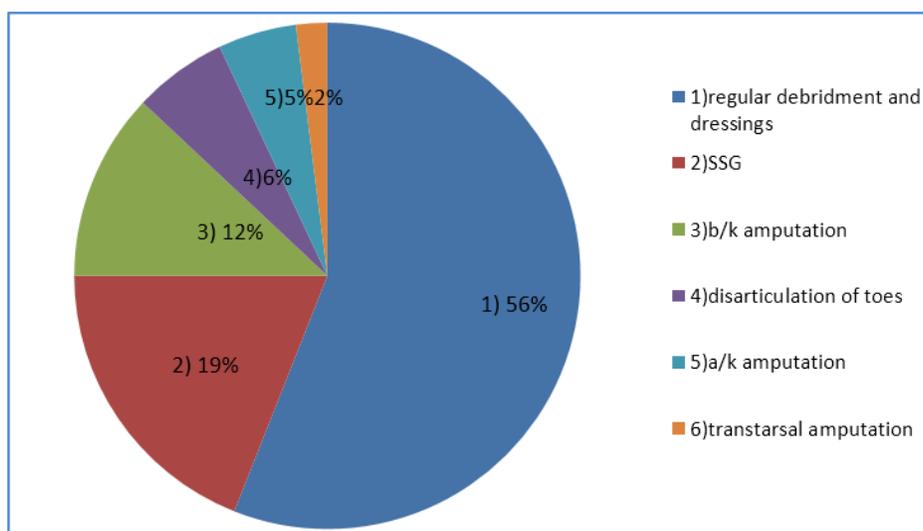
Among these 100 patients 30% of patients had less than 10 days of hospital stay, 32% of patients had 10-20 days of stay 23% patients had more than 20-30 days of hospital stay 15% stayed more than a month, Most of the diabetic foot patients thus needed an average stay of 20 days.



Graph 5

Among them majority(56%) of cases were managed with regular debridement and 19% underwent split thickness skin grafting following wound healing over healthy granulation tissue In patients who required amputations majority underwent below knee amputations (12%) and about 5% above knee amputation.

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Graph 6

Pus for culture and sensitivity was done for these cases out of which 47% cases showed positive for E.coli 38% cases positive for Klebsiella 15% cases showed positive for Proteus, Enterococcus, Staphylococcus, Streptococcus, Pseudomonas and treatment was given according to the sensitivity. Among 100 cases 18 cases expired during course of their treatment out of them 12 cases expired due to Acute on Chronic kidney disease 6 cases DKA with shock.

DISCUSSION: One of the most important and disabling complications of diabetes mellitus (DM) is the diabetic foot ulcers. Development of DF is believed to result from a combination of oxygen deficiency caused by peripheral vascular disease, peripheral neuropathy, minor foot traumas, foot deformities, and infection^[1,2] DF, with a lifetime development risk of 15% in all diabetic patients, accounts for more than half of the non-traumatic lower-extremity amputations in the world.^[3,4,5] DF is among the most prevalent causes of hospitalization and morbidity^[5] and is responsible for more days of hospital stay than any other chronic complication of DM.^[6]

DF lesions are significant health and socioeconomic problem as they exert adverse effects on patients' quality of life and impose heavy economic burden on the patient and the state due to rising the need for rehabilitative and home care services.^[7] Given the DF's high prevalence, heavy burden, and severe impact on patients' life quality, it is advisable that sufficient heed be paid to prevention of this particular complication of DM. Furthermore, while effective treatment and formulation of prevention guidelines requires a thorough understanding of characteristics of DF patients and their ulcers.

ETIOLOGY OF DIABETIC FOOT PROBLEMS: The important risk factors for foot ulcers in diabetic patients are peripheral neuropathy and ischemia.

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NEUROPATHY: The distal neuropathy of diabetes affects all components of the nervous system: sensory, motor, and autonomic, each of which contributes to foot ulcer development. Loss of nerve function correlates with chronic hyperglycemia, as reflected in the mean level of glycosylated hemoglobin over time. Ischemia of the endoneurial microvascular circulation induced by metabolic abnormalities from hyperglycemia is believed to be the underlying mechanism for nerve deterioration.^[8]

ISCHEMIA: The other major underlying cause of diabetic foot ulcers is peripheral vascular disease. Overall, atherosclerosis of the lower limbs in people with diabetes occurs at least two or three times more often than in people without diabetes and has a predilection for affecting the tibial and peroneal arteries of the calf with relative sparing of the arteries of the foot. This pattern differs from the general population, where more proximal atherosclerotic changes predominate.

Involvement of the peripheral autonomic nervous system has been proposed to explain the more distal distribution of lower limb atherosclerosis among diabetic patients. Autonomic dysfunction reduces the normal vasoconstriction that occurs in the lower leg arteries with standing and results in an increase in the intraluminal flow and pressure that is aggravated by gravitational forces. Reduced vasoconstrictive ability further reduces vessels' capacity to expand in response to systolic pressure. The combination of high flow and reduced wall motion encourages formation of plaque in calf arteries.^[9]

THERAPY FOR DIABETIC FOOT ULCERS: An understanding of the processes that precipitate and propagate foot ulcers in diabetic patients should dictate a rational approach to therapy.

BLOOD SUGAR CONTROL: Most foot ulcers have their origins in inadequate control of blood sugar, which results in development of lower limb neuropathy.

There is now excellent evidence that improved control of diabetes can markedly reduce the incidence of neuropathy.^[10] Host defenses against infection, particularly white blood cell function, are also adversely affected in the presence of elevated blood sugar levels but improve as blood sugar is reduced.

PRESSURE RELIEF AND AVOIDING FURTHER TRAUMA: Unrecognized injury from poorly fitting footwear is a frequent precipitating event for foot ulcers in patients with peripheral neuropathy and loss of protective sensation.^[11] Teaching patients to purchase appropriate shoes with sufficient width, depth, and arch support can prevent many foot ulcers from developing. Once an ulcer develops, on-going trauma from continuing to walk on the affected area prevents healing.

PERIPHERAL VASCULAR DISEASE: Improved peripheral blood flow after distal revascularization surgery has frequently salvaged limbs in diabetic patients with limb-threatening obstruction in the tibial or peroneal arteries who otherwise would have proceeded to amputation.^[12]

Unfortunately, comorbidity often dictates a nonsurgical approach to the problem of peripheral vascular insufficiency. Few medical options are available. Arterial vasodilators have largely been abandoned because they are ineffective. Pentoxifylline, officially defined as a

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hemorheologic agent that improves deformability of stiff red blood cells, has been used for peripheral vascular disease to improve distal flow, Antiplatelet agents like cilastazole can modify the natural course of peripheral arterial insufficiency, delaying progression of occlusive disease and decreasing the need for arterial reconstruction when used for primary prevention.^[13] Their role is essentially to maintain the flow that already exists and to reduce the potential for further thrombotic occlusion.

INFECTION: Infection arising from diabetic foot ulcers frequently leads to amputation. Deep infections require early, aggressive surgical débridement in addition to antibiotics. The choice of antibiotics ultimately, of course, depends on the results of bacterial culture. Initially, while cultures are being obtained, the spectrum of antibiotics should reflect the broad variety of bacteria isolated from these wounds in diabetic patients. Mixed bacterial flora involving Gram positive and Gram-negative aerobes and anaerobes are common. Culture technique is critical in obtaining reliable results from diabetic foot infections. Surface swabs from a foot ulcer site are notoriously inadequate for identifying the type of bacteria causing limb-threatening deep infection. The most accurate technique involves removing surface exudate from the ulcer, obtaining a small tissue biopsy from the base of the ulcer, and sending the sample of tissue to the laboratory in appropriate aerobic and anaerobic culture material.

TOPICAL ULCER THERAPY: Primary principles of good wound care incorporate regular débridement to remove necrotic material from the ulcer site and application of appropriate dressings. Encrusted, callused areas should be deroofed. Débridement is most efficient when forceps and scalpel are used for all wounds, preservation of a moist wound environment aids healing. Ideal topical therapy for ulcers should keep the ulcer bed moist, but not excessively wet so as to promote maceration. Papain containing preparations for chemical debridement are associated with better results.

Moistened gauze dressings are frequently used but have a tendency to dry out unless changed several times daily.

OTHER THERAPIES:

Hyperbaric oxygen: Hyperbaric oxygen has been used as adjunct therapy for diabetic foot ulcers, with a doubtful role.

Tissue engineering and growth factors: Despite adequate blood flow and good wound care, some neuropathic diabetic foot ulcers fail to heal. Impairment of the normal cellular functions involving growth factors and fibroblasts necessary for wound healing has been postulated to account for the failure to close these wounds.^[14]

Recent advances in technology have produced growth factors (such as platelet-derived growth factor) and tissue engineering of living skin replacements to replenish components of the wound care process. Because these newer technologies are expensive, however, they are generally reserved for patients with neuropathic foot ulcers who continue to exhibit poor healing despite

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optimal wound care with débridement, offloading of pressure surfaces, provision of a moist wound environment, and control of infection and ischemia.

CONCLUSION: Diabetic foot is more likely to develop in middle aged diabetic patients who are usually above 50 years with male preponderance with long duration of DM of >10 years with poor glycaemic control.

Most common comorbidities in these patients were hypertension or associated diabetic nephropathy. Gram negative species were the predominant species to be isolated on culture sensitivity of wounds of these patients.

Most of the patients with diabetic foot healed by regular debridement and few followed by split skin grafting, with about 20% patients going for amputation.

Diabetic foot infection are best treated empirically by 3rd generation cephalosporins which have good action on gram negative species and adding an antibiotic such as clindamycin or linezolid for gram positive and metronidazole for anaerobic coverage and later to be switched on to antibiotics according to the culture sensitivity profile. Mortality and morbidity in diabetic foot patients can be prevented by good glycaemic control, correction of malnutrition anaemia and hypoproteinaemia, appropriate and immediate treatment and adequate care of the foot

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