Treatment Modalities in management of diabetic foot complications at a Tertiary Care Hospital

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Abstract

The feet are one of the most used parts of the body, are necessary for daily activities pertaining to locomotion, and are therefore subject to repetitive trauma. Trauma, in turn, may lead to open lesions that allow ingress of pathogenic microbes. In the diabetic person, the pathogenesis of foot infections is multifaceted. Out of 100 cases studied in 20 cases the wound healed well, and patients were discharged without any complications. While in 8 cases the resulting raw area needed split thickness skin grafting. In 27 cases the wound remained as chronic ulcer without any signs of healing. Out of 38 amputations (35 BKA & 3 AKA) 27 stumps healed well, while 9 of them ended in dehiscence and the remaining 4 cases of amputation died due to other associated comorbidities. Out of the 100 cases in 14 cases the patient died because of other associated comorbidities of diabetes.

Keywords: Diabetic Foot, Complications, Treatment Modalities

Introduction

It has been recognized that persons with diabetes are prone to foot problems. Recent advances in molecular biology have added substantial insight into the pathophysiology of the disease and opened new avenues for treatment. Enhanced non-enzymatic glycosylation of lipoprotein has been shown to impair the binding of glycosylated LDL to the LDL receptor. Glycosylated LDL enhances the formation of cholesteryl ester and accumulation human macrophages – formation of foam cells characteristic of the early atheromatous lesion [1]. It is also noted that, vascular smooth muscle cells exhibit increased growth on exposure to high glucose in vitro. Blue toe syndrome is sudden onset of pain in the toe with bluish discoloration associated with leg/thigh myalgia and a sharp demarcated gangrenous toe is seen in diabetic foot. This is due to cholesterol emboli that break off from an ulcerated atheromatous plaque in the proximal vessels. Warfarin is used in treatment [2]. Peripheral neuropathies are found in 55% of diabetics. The incidence of neuropathies increases with duration of disease and episodes of neuropathies increases with duration of disease and episodes of hyperglycemia. Peripheral neuropathy clearly renders the patient to unrecognized injury, which potentiates the risk of bacterial invasion and infection [3]. The feet are one of the most used parts of the body, are necessary for daily activities pertaining to locomotion, and are therefore subject to repetitive trauma. Trauma, in turn, may lead to open lesions that allow ingress of pathogenic microbes. In the diabetic person, the pathogenesis of foot infections is multifaceted. Vascular insufficiency, neuropathy, and decreased resistance to infection, possibly from metabolic imbalance, have all been implicated. In the individual patient, the relative contribution of each of these factors can vary. The presence of peripheral neuropathy can lead to an insensate foot, to impaired ability of the foot to sweat, in turn leading to drying, fissuring, and cracking, and to deformities arising from poorly perceived microfractures. These changes, in the presence of the diabetic state, with or without significant vascular impairment, can set the stage for the entry of bacterial pathogens and the progression of the infections process [4]. Diabetic patients also represent around 60 percent of nontraumatic foot or leg amputations, the majority of which are secondary to infectious complications.
Defects in immune function have been described as occurring in diabetics. White cell dysfunction in diabetes’s, adherence, and chemotaxis, phagocytosis, and killing ability has been described in diabetic patients. In general, these defects are aggravated by poor glucose control. Poor granuloma formation and poor healing have also been seen in diabetic mice [5].

Salvapanidian reviewed the different types of foot infections and their characteristics in 1982. These infections can occur in nondiabetic as well as diabetic persons, although the presence of the diabetic state can aggravate the risks and themorbidity associated with these infections. Foot infections can occur in the wake of acute or chronic trauma.

**Methodology**

**Source of Data**

This study was conducted comprising of 100 patients of Diabetic foot in the Department of Surgery at Medical College.

**Inclusion Criteria**

All the patients with Diabetes Mellitus presenting with foot ulcers, infection of foot and gangrene of foot.

**Exclusion Criteria**

1) Patients with foot infections without Diabetes.
2) Patients with ulcer and Gangrene of foot other than Diabetic

**Methods of Collection of Data**

- Detailed history taking.
- Clinical examination
- Investigations (Routine Laboratory investigation)
- Relevant special investigations.
- Conservative management with meticulous dressing and if needed major surgical interventions with its outcome.

**Results**

Majority of the septic lesions yielded Staphyllococcus aureus on culture of pus. Other organisms that were isolated are, Pseudomonas, Klebsiella, E. Coli, Proteus. Most of them were sensitive to Ampicillin, Gentamycin, and Amikacin. Some cultures yielded more than one type of bacteria.

| Bacterial                  | No of Cases | Percentage (%) |
|----------------------------|-------------|----------------|
| Staphylococcus aureus      | 44          | 44             |
| Pseudomonas                | 14          | 14             |
| Klebsiella                 | 20          | 20             |
| Coliform                   | 10          | 10             |
| Proteus                    | 8           | 8              |
| Non-Haemolytic Streptococi| 4           | 4              |

Out of 60 case of neuropathy 23(38.33%) needed and daily dressings, Fasciotomy and I & D was done in 13(16.67%) cases, disarticulation in 6(10%) of them, fore foot amputation in 2(3.33%), while 15 (25%) cases ended up having below knee amputation and 1 in above knee amputation.

In Vasculopathic form of diabetic foot (23 cases), majority ended up in having Below knee Amputation., i.e.,15 cases of 23 (65.22%), 3 case underwent disarticulation (13.04%), 4 cases ended in fore foot amputation (17.39% ) and 1 case in above knee amputation (4.35%).

Out of 17 cases of mixed neurovascular lesions 8 cases ended in disarticulation, 5 cases in below knee amputation (29.41%),while debrediment, Fasciotomy, fore foot amputation and above knee amputation was done in 1 case each (5.88%).
Out of 100 cases studied in 20 cases the wound healed well, and patients were discharged without any complications, while in 8 cases the resulting raw area needed split thickness skin grafting. In 27 cases the wound remained as chronic ulcer without any signs of healing. Out of 38 amputations (35 BKA & 3 AKA) 27 stumps healed well, while 9 of them ended in dehiscence and the remaining 4 cases of amputation died due to other associated comorbidities. Out of the 100 cases in 14 cases the patient died because of other associated comorbidities of diabetes.

Discussion
The commonest pathophysiological changes that lead to diabetic foot are neuropathy, ischaemia and infection Neurupathy changes seen in 52 cases. Ischemic complication was noted in 23 cases. And infective complication of foot noted in all cases. The incidence of gangrene in the present series is comparatively approximately equal to that of Bell series of 1960 and more than that of Diabetic Research center (2005) Chennai.

| Table 5: End Results of Diabetic foot |
|-------------------------------------|
| **End Result** | **No of Cases** | **Percentage** |
| Wound Healed Well | 20 | 20 |
| Split skin Graft | 8 | 8 |
| Chronic Ulcer | 27 | 27 |
| Amputation | 22 | 22 |
| Stump Dehiscence | 9 | 9 |
| Death | 14 | 14 |

In the present series, 24 cases were treated by slough excision, 10 with fasciomy, I & D in 4 cases, 17 by disarticulation and fore foot amputation was done in 7 cases. Below knee amputation was done in 35 cases and above knee amputation was done in 3 cases. Proper control of diabetes is very important in diabetic foot management. Fasting and postprandial blood sugar estimations were well under control. Urine sugar estimation was done twice daily. Infection was treated with broad spectrum antibiotics. Patients were educated about care of foot and Tab Trental (pentoxyphylline) was administrated to in-patients with ischemic lesions.

| Table 6: Gangrene – Comparison of incidence of gangrene in various series |
|-----------------|-----------------|-----------------|
|                  | No of cases     | No of cases with Gangrene | Percentage |
| Bell series (1960) | 964             | 236                  | 24.9       |
| Pennsylvania Hospital Series | 614             | 274                  | 44.78      |
| Diabetic Research center (2005) Chennai | 1319 | 64 | 5 |
| Present Study | 100             | 23                   | 23         |

The amputation rate is much 38% almost equal to that of Collen’s series 38.6% in 1962. And less than Osaka kosainek in Hospital (2005) study (52%). After amputation, wound healed well. The patients were referred to Rehabilitation center for prosthesis. In neuropathy 38.33% needed and daily dressings, while 25% cases ended up having below knee amputation and 1 in above knee amputation. In Vasculopathy, majority ended up in having Below knee Amputation 65.22%.

Conclusion:
- The prognosis in diabetic foot patients does not solely depend on local complications of diabetic foot but also on the other associated systemic comorbid conditions like cardiac failure, renal insufficiency, metabolic problems etc.
- Hence the treatment of diabetic foot complications is a team work headed by the surgeon with support from general physicians, cardiologists, nephrologists, physiotherapists, podiatrists, etc.

References
1. Lopes Verella MF, Klein RH, Lyon TJ. Glycosylation of low density lipoprotein enhance cholesteryl ester synthesis in human monocyte derived macrophage diabetes 1988;37:550-557.
2. Natarajan R, Gonzalez N, XVI, NEdler JL. Vascular smooth muscle cell exhibit increased growth in response to elevated glucose. Biochem Biophys Res Commun 1992;187:552-560.
3. Tooke JE. Microvascular hemodynamics in diabetes mellitus. Clin Sci 1986;70:119-125.
4. Mac Rury SM, Lowe GDO. Blood rheology in DM. Diabet Med 1990;7:285-291.
5. Harati Y. Frequently asked questions about diabetic peripheral neuropathies. Neurologic Clinic 1992;10(3):783-801.
6. Delbring L, Cetecelo G, Fowler C. The etiology of diabetic neuropathic ulceration. Br J Surgery 1985;72:1-6.
7. Bessman AN, Sapico FL, Tobatabai MF, Montgomerie JZ. Persistence of polymicrobial abscesses in poorly controlled diabetic host. Diabetes 1986;36:448.
8. Donovan DL, File TM. Team approach in the management of diabetic foot infections. Journals of Foot Surgery 26(1):S-12, 187.
9. Leslie CA, Sapico FL, Ginunas VJ, Adkins RH. Randomized clinical trial of tropical hyperbaric oxygen for treatment of diabetic foot ulcers. Diabetes Care 1988;11:111.