Minor amputations for diabetic foot salvage

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Abstract: Foot ulceration in diabetic patients is a frequent complication of diabetes mellitus (DM), necessitating hospitalization for control of infection, wound care and glycemic control. These patients are at risk for potential loss of the involved limb as well as for future loss of the contralateral limb. Diabetic foot is the consequence of peripheral neuropathy complicated by infrapopliteal peripheral vascular disease. Most of the patients present with chronic plantar ulceration and with cellulitis or an abscess. In a significant number of patients, it is observed that the frequency of life or limb threatening infection is less with an intact skin cover. Limb salvage employs the use of culture specific antibiotics, sharp debridement or a minor amputation, wound care and/or skin cover as the situation demands. 

Keywords: Minor Amputation, Diabetic Foot, Limb Salvage

Introduction

Diabetes mellitus is a chronic non-communicable disease, the prevalence of which is increasing significantly. In 2000 the estimated prevalence of diabetes worldwide was 171 million, with the most people being in India (17.7 million) and this number is projected to increase to 79.4 million by the year 2030 [1]. Diabetes mellitus is associated with various complications, amongst which foot ulceration is the most common, affecting approximately 15\% of all patients [2]. Foot related problems in diabetes lead to a marked impairment of the quality of life and this is more so true in the case of patients who have had a major amputation done [3]. Furthermore these patients have a mortality rate of upto 80\% following major amputation [4]. Minor amputations can be beneficial in these patients for reducing morbidity and mortality [5].

Pathophysiology of diabetic foot ulcers:

There is an interplay of various pathophysiologic mechanisms, which can ultimately lead to ulceration requiring surgical intervention. These can be broadly divided into three aspects:

i. Peripheral neuropathy
ii. Vascular disease
iii. Hemorheologic abnormalities

Peripheral Neuropathy: Fundamentally, peripheral neuropathy involving sensory, motor and autonomic pathways is the major cause of foot ulcerations. Autoimmune and microvascular mechanisms have been shown to cause segmental demyelination of the nerves [6]. The resultant neural oedema slows down axoplasmic flow significantly adding to neural dysfunction. It has been shown that peripheral nerve compression exists in these cases, which contributes to metabolic dysfunction. This double crush syndrome was first described by Upton and McComas [7]. Axonal swelling is further exacerbated by compression in a fixed space such as a bony foramen or a bony/ligamentous tunnel, examples of which are found in nerve entrapment syndromes.

Normal toe flexor extensor balance gets deranged due to intrinsic muscle weakness of the foot secondary to motor neuropathy. This results in midfoot collapse in longstanding cases. These Charcot deformities by virtue of their abnormal loading contribute to skin breakdown and ulceration [8]. Pressure above results in increased bacterial localization and multiplication. Denervation has been shown to lead to increased multiplication of bacteria [9], which is also a cause of ulcer development by itself. Trivial trauma often
results in disruption of protective epidermis which in presence of neuropathy and peripheral vascular disease along with infection results in an ulcer. Somatic neuropathy with reduced perception of pain, diminished proprioception and clawing of toes contribute to increased foot pressure. Autonomic neuropathy results in diminished or absent sweating. The skin becomes dry and gets fissured. Altered regulation of blood flow with a warm foot and Charcot neuropathy are responsible for callus formation [10].

Vascular Disease: It clinically presents with claudication, rest pain, cold extremities and reduced foot pulses ending up in foot ischemia and ultimately gangrene. Peripheral arterial disease is a major risk factor resulting in amputation and is more so true in patients with diabetes. Diabetes is associated with major changes in normal arterial function and these changes are proatherogenic [11]. About 20% of patients with peripheral arterial disease have diabetes [12].

Goldenberg had earlier concluded that microangiopathy with small vessel endothelial proliferation were thought to cause ulceration [13]. This concept was challenged by LoGerfo and Coffman in 1984 [14]. It has been found in amputated limbs that non diabetics and diabetics have same amount of intimal hyperplasia [15-16]. Transcutaneous oxygen tension (TcPO₂) studies done in diabetic and non diabetic patients with peripheral vascular disease show no significant differences when measured in the lower limb, hinting towards other factors contributing more to ulceration rather than lower cutaneous oxygen supply [17]. There is a thickening of the basement membrane in capillaries of muscles in diabetics, but not in capillaries of the skin [14].

Hemorheology: The increased levels of blood glucose in DM result in various changes which leads to decreased red blood cell deformability which in turn affects the viscosity of blood, especially in microvessels [18]. Platelet aggregation and increased fibrinogen levels also play a role in abnormalities of blood flow. This is explained by the lower bioavailability of nitric oxide (NO) in DM [19]. Viscosity is further increased by serum protein shifts from increased vessel permeability and leaking albumin and the elevation of globulin. All of these hemorheological changes impede wound healing and lead to progressive ulceration.

Management

All patients presenting with diabetic foot should be evaluated and managed via a multidisciplinary approach in order to reduce recurrent ulcerations which will also reduce the need for amputation [20-21]. Cases which require amputation include those with ischemic rest pain and life threatening infections which can't be managed by other modalities. Patients presenting with non-healing ulcers are also potential candidates. These patients should be thoroughly evaluated by vascular staff before being considered for amputation [22]. Minor amputations can in a majority of cases prevent the need of major surgical interventions, but increase the healing time [5].

Preoperative Management:

Preoperative management is very important as it influences the final post-operative outcome. It is directed towards the following [23]:

i. tight glycemic control
ii. infection control
iii. reducing oxidative stress
iv. assessment and correction of neuropathy and peripheral vascular disease
v. maintaining acid-base balance and electrolytes

Glycemic control is achieved with insulin sliding scale. Deep infection in the foot or leg must be ruled out. Plain radiographs may show evidence of clinical osteomyelitis at the base of an ulcer. Swab cultures and bone scans are of little value because each is non-specific. MRI does show better specificity compared to bone scan [24]. Broad spectrum antibiotics are used initially, modified as per culture sensitivity results. Oxidative stress can be reduced by reducing advanced lipoxidation end products (ALEs) and advanced glycation end products (AGEs). This could be achieved by tight glycemic control, dietary control and administration of anti oxidants [25]. Neuropathy is carefully assessed with a neurological examination. Bypass grafting or angioplasty may improve an ischemic limb.
Lack of heel sensation is a relative contraindication for Syme’s amputation. Ankle brachial indices above 0.7 are acceptable for reconstruction without obstruction of inflow. Arterial non-compressibility from medial calcification may elevate brachial index value. Angiography is the gold standard for assessing the vascularity of a limb.

Wound closure techniques: Single toe amputations are done for involvement of bone in the case of clinical ulceration of a single digit. If bone is not involved, toe sparing procedures such as local flaps or skin grafting are taken up. Possible predisposing factors such as tendon and joint abnormalities that produce ulceration are to be rectified to ensure prevention of recurrence. Toe ulceration is exacerbated by peripheral vascular disease and hence demands correction. Charcot arthropathy often results in metatarsal head ulcerations. Wounds greater than 2-3 cms can be closed with single or multiple local flaps from uninvolved adjacent toes in the form of fillet flaps [26].

This procedure offers better length and an intact transverse metatarsal arch. Ray amputations or metatarsal joint resection may be needed for osteomyelitis of metatarsals. Neurovascular toe island flap and plantar V-Y advancement flaps which comprise of skin, fat and fascia can be used to cover this defect [27-28]. Recurrent ulceration after transmetatarsal amputation needs more proximal mid foot amputations. Lisfranc tarsal metatarsal junction amputation and Chopart intertarsal junction amputation allow weight bearing without prostheses.

Charcot neuroarthropathy causes deformation of plantar arches due to denervation of intrinsic muscles. This disturbance in motor and sensory functions plays an important role in midfoot ulceration. Skin grafts, local flaps and free tissue transfer are the options available for wound closure in such cases. Severe wounds may warrant transmetatarsal amputation. Imbalance resulting from destruction of the insertion of tibialis anterior, tibialis posterior and peroneus in cases of mid foot amputation produces an equinovarus deformity. Tendo Achilles lengthening, tibialis anterior fixation or transfer may be helpful in correcting this deformity.

Glabrous skin with its fibrous septae over a thick heel fat pad resists shearing forces. Small to moderate sized defects of the heel can be covered with a suprafascial rotation of the heel pad [29]. Flexor digitorum brevis flap can be used for coverage of the heel. The plantar fascia is retained with the muscle to get good bulk [30]. Small wounds over the medial malleolus and heel can be covered using abductor hallucis whereas small wounds over the lateral mid and hindfoot areas could be covered using abductor digiti minimi. Loss of longitudinal and transverse arches in the neuropathic foot along with contracture of the tendo Achilles results in increased forefoot loading which can lead to metatarsal head ulceration ultimately leading to osteomyelitis. Lengthening of the tenuo Achilles is taken up routinely to overcome this.

Excision of medial and lateral sesamoid bones is usually indicated as these cause ulceration of forefoot including the metatarsal head region. Fifth metatarsal head ulceration is managed with excision of metatarsal head at the time of wound closure. Complete excision of at least one metatarsal head, often leads to reulceration called as transfer lesion in an adjacent metatarsal territory. This most often follows excision of the first metatarsal head and is least associated with fifth metatarsal head excision [31]. To counter this, resection is usually done with a plantar approach and osteotomies are performed with a linear dorsal approach. Resection of bony prominence is necessary while closing mid foot ulcerations.

Conclusion
In diabetic individuals peripheral neuropathy, angiopathy and hemorheological derangements significantly increase the lifetime risk of developing foot ulceration. The primary goal of managing diabetic foot is to identify patients at risk of developing foot complications and to institute preventive measures. If complications have already developed, the treatment should be directed towards preventing major surgeries and to salvage the limb. Early surgical management with minor amputation can reduce morbidity and mortality.
Reconstruction of neuropathic and ischaemic lower limbs is a challenging task for plastic, vascular and orthopedic surgeons. Better results are achieved with innovative multifaceted approaches. Timely executed minor amputation with an adequate preparation of the patient not only reduces the burden of infection and necrotic tissue but also promotes conservation of the limb by avoiding major limb amputation.

References


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