Al Ameen J Med Sci 2015; 8(4):245-248 ● US National Library of Medicine enlisted journal ● ISSN 0974-1143

MINI REVIEW ARTICLE CODEN: AAJMBG

Minor amputations for diabetic foot salvage

Salah Y. Habel[1*] and Mohammed Kashif Habeeb[2]

[1]Department of General Surgery, El-Wahda Hospital, Derna /Omar El-Mukhtar University, Libya and [2]Department of General Surgery, Dr. Munir Bangi Hospital, Railway Station Road, Bijapur, Karnataka, India

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 amputed limbs that non diabetics and diabetics have same amount of intimal hyperplasia [15-16]. Transcutaneous oxygen tension (T_CpO_2) 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 evaluted 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 neuroarthopathy 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 tendo Achilles is taken up routinely to overcome this.

Excision of medial and lateral sesamoid bones is usually indicated as these cause ulceration of fore foot 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 excison 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

1. Wild S, Roglic G, Green A, Sicree R, King H. Global prevalence of diabetes-estimates for the year 2000 and projections for 2030. *Diabetes Care.* 2004; 27(3):1047-53.
2. Shankhdhar KLK, Shankhdhar U, Shankhdhar S. Diabetic foot problems in India: An overview and potential simple approaches in a developing country. *Current Diabetes Reports* 2008; 8:452-457.
3. Carrington AL, Mawdsley SK, Morley M, Kincey J, Boulton AJ: Psychological status of diabetic people with or without lower limb disability. *Diabetes Res Clin Pract.* 1996; 32:19-25.
4. Reiber GE. Epidemiology of foot ulcers and amputation in the diabetic foot. Levin and O'Neal's The Diabetic Foot. Bowker JH, Pfeifer MA (eds). *St. Louis, CV Mosby,* 6th Ed, 2001; 13-32.
5. Svensson H, Apelqvist J, Larsson J, Lindholm E, Eneroth M. Minor amputation in patients with diabetes mellitus and severe foot ulcers achieves good outcomes. *J Wound Care.* 2011; 20:261-2, 264, 266.
6. Said G, Slama G, Selva J. Progressive centripetal degeneration of axons in small fibre diabetic polyneuropathy. *Brain.* 1983; 106(4):791-807.
7. Upton ARM, McComas AJ. The double crush in nerve entrapment syndromes. *Lancet.* 1973; 2:359-362.
8. Rogers LC, Frykberg RG, Armstrong DG, et al. The Charcot foot in diabetes. *Diabetes Care.* 2011; 34(9):2123-2129.
9. Richards AM, Floyd DC, Terenghi G, McGrouther DA. Cellular changes in denervated tissue during wound healing in a rat model. *Br J Dermatol.* 1999; 140: 1093-9.
10. Bowering CK. Diabetic foot ulcers: pathophysiology, assessment, and therapy. *Can Fam Phys.* 2001; 47:1007-1016.
11. American Diabetes Association. Peripheral arterial disease in people with diabetes. *Diabetes Care.* 2003; 26(12):3333-3341.
12. Murabito JM, D'Agostino RB, Silbershatz H, Wilson WF. Intermittent claudication: a risk profile from the Framingham Heart Study. *Circulation.* 1997; 96:44-49.
13. Goldenberg SG, Alex M, Joshi RA et al. Nonatheromatous peripheral vascular disease of the lower extremity in diabetes mellitus. *Diabetes.* 1959; 8:261-273.
14. LoGerfo FW, Coffman JD. Current concepts: vascular and microvascular disease of the foot in diabetes. *New Eng J Med*. 1984; 311(25):1615-1619.
15. Conrad MC. Large and small artery occlusion in diabetics and nondiabetics with severe vascular disease. *Circulation* 1967; 36:83-91.
16. Strandness DE, Priest RE, Gibbons GE. Combined clinical and pathologic study of diabetic and nondiabetic peripheral arterial disease. *Diabetes.* 1964; 13:366-372.
17. Wyss CR, Matsen FA, 3rd, Simmons CW, Burgess EM. Transcutaneous oxygen tension measurements on limbs of diabetic and nondiabetic patients with peripheral vascular disease. *Surgery.* 1984; 95(3):339-346.
18. Cho YI, Mooney MP, Cho DJ. Hemorheological Disorders in Diabetes Mellitus. *Journal of diabetes science and technology (Online).* 2008; 2(6):1130-1138.
19. Grant PJ. Diabetes mellitus as a prothrombotic condition. *J. Intern.Med.* 2007; 262:157-172.
20. Dargis V, Pantelejeva O, Jonushaite A, et al. Benefits of a multidisciplinary approach in the management of recurrent diabetic foot ulceration in Lithuania: a prospective study. *Diabetes Care.* 1999; 22:1428-31.
21. Korzon-Burakowska A, Dziemidok P. Diabetic foot - the need for comprehensive multidisciplinary approach. *Ann Agric Environ Med.* 2011; 18:314-317.
22. International Diabetes Federation Clinical Guidelines Taskforce. Global guideline for type 2 diabetes. *Brussels: IDF,* 2012; 14:93.
23. Soghomonyan N. Perioperative management of diabetic foot. *Frontiers in Pharmacology.* 2014; 5:91.
24. Kattapuram TM, Treat ME, Kattapuram SV. Magnetic resonance imaging of bone and soft tissue infections. *Curr Clin Top Infect Dis.* 2001; 21:190-226.
25. Meerwaldt R, Links T, Zeebregts C, Tio R, Hillebrands JL, Smit A. The clinical relevance of assessing advanced glycation endproducts accumulation in diabetes. *Cardiovasc Diabetol.* 2008; 7:29.
26. Emmett AJJ. Filletted Toe Flap. In: Strauch B, Vasconez LO, and Hall-Findlay EJ. Editors. Grabbs encyclopedia of flaps Vol III. 2nd edn. Philadelphia. *Lippincott-Raven.* 1998; 1833-5.
27. Buncke HJ, Colen LB. An island flap from the first web space of the foot to cover plantar ulcers. *Br J Plast Surg.* 1980; 33:242-4.
28. Colen LB, Replogle SL, Mathes SJ. The V-Y plantar flap for reconstruction of the forefoot. *Plast Reconstr Surg.* 1988; 81:220-228.
29. Shanahan RE, Gingrass RP. Medial plantar sensory flap for coverage of heel defects. *Plast Reconstr Surg,* 1979; 64(3):295-8.
30. Ikuta Y, Murakami T, Yoshioka K, et al. Reconstruction of the heel pad by flexor digitorum brevis musculocutaneous flap transfer. *Plast Reconstr Surg.* 1984; 74:86-96.
31. Molines-Barroso RJ, Lazaro-Martinez JL, Aragon-Sanchez J, Garcia-Morales E, Beneit-Montesinos JV, Alvaro-Afonso FJ. Analysis of transfer lesions in patients who underwent surgery for diabetic foot ulcers located on the plantar aspect of the metatarsal heads. *Diabetic Med.* 2013; 30:973-6.

*All correspondences to: Dr. Salah Y. Habel, Professor & HOD, Department of General Surgery, El-Wahda Hospital, Derna /Omar El-Mukhtar University, Libya. E-mail: salah.ha20@yahoo.com