Definitive surgical treatment for diabetic forefoot ulcer

M. K. Rajendran*

Department of Plastic Surgery, Government Mohan Kumaramangalam Medical College Hospital, Salem, Tamil Nadu, India

Received: 08 October 2018
Accepted: 01 November 2018

*Correspondence:
Dr. M. K. Rajendran,
E-mail: drmkrajendran@yahoo.co.in

Copyright: © the author(s), publisher and licensee Medip Academy. This is an open-access article distributed under the terms of the Creative Commons Attribution Non-Commercial License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original work is properly cited.

ABSTRACT

Background: Diabetic foot ulcers are common among 15% of the diabetic population, at one stage of life that may progress to minor or major amputation, because of not healing with all modalities of the treatment. Since recurrent rate is >90%. But the surgical treatment of the forefoot ulcer so-called surgery offloading will heal the ulcer within a short time and prevent minor of major amputation. The purpose of this study was to compare different outcome measures in the audit of the management of diabetic foot ulcers.

Methods: Totally 20 diabetic patients were included in the study. The study was conducted in the department of plastic surgery, Government Mohan Kumaramangalam Medical College Hospital, from 2016-2018. Followed 20 patients with a surgical procedure called surgical offloading is by removing pressure element in head and neck of metatarsal bone involved.

Results: Among the 20 patients wound healed, in about 12 patients within 4 to 5 week and its remaining 5 patients wound healed in 5 to 6 weeks are in other 2 patients it takes. More than 6 weeks to heal. We followed the patients for the past 1 year, out of 20 patients, 2 patients have developed an ulcer in another site, not the same site. In all other patients wound healed completely without any recurrence.

Conclusions: In our study reducing the foot pressure by doing an excision of head and neck metatarsal surgically will completely remove the foot pressure over the ulcer site that helps to heal the ulcer without recurrence.

Keywords: Forefoot ulcer, Head and neck of metatarsal bone excision, Recurrence, Surgical management

INTRODUCTION

Diabetes is one of the most prevalent chronic diseases: in 2010, one study reported that 285 million adults worldwide had diabetes and this figure is projected to rise to 439 million by the year 2030. Such a profound demographic shift is likely to yield a corresponding increase in the prevalence of diabetes chronic complications, including those in the lower extremity, the diabetic foot. Foot ulcers are the common and devastating and feared complication of the diabetic population. Among foot ulcer forefoot ulcer is common. Among that 15% of the diabetic patients through one stage of life, suffer from forefoot ulcer. Fore foot ulcer is common among the patients with neuropathy which alter the biomechanics of the foot/curvature of the foot/loss of metatarsal pad of fat overheads of the metatarsals. Bone is prominent which produce increased, plantar pressure points over the forefoot that produce ulcer, once ulcer formed there remains always ulcer. Because of its recurrence rate is high with all modalities of the available treatment, like daily dressing with saline gauze, silver ionic gel, platelet growth factor, offloading shoes and TCC. It is estimated that 85% of these amputations are preceded by an infection. It is estimated that the rate of amputation can be diminished by 49-85% through implementation of an effective evidence-based prevention program, patient education, foot ulcer treatment by a multidisciplinary team, and periodic surveillance despite the efforts of conservative therapy there will always be a...
percentage of wounds that will require surgical intervention in order to heal the wound. Typically, this involves elimination of infection, surgical procedures designed to offload areas of increased pressure, improving diminished vascular flow or a combination of all of these. The peripheral arterial disease can lead to critical limb ischemia, either alone or when combined with an injury like a foot ulcer. The diabetic foot ulcer requires adequate circulation to heal. When this circulation is impaired and the oxygen demand exceeds supply from the arterial system, critical limb ischemia ensues, risking the loss of limb. There is often a common pathway that leads to amputation in those with diabetes. This pathway begins with the disease itself and usually progresses with a patient who develops peripheral neuropathy leading to foot ulceration.

METHODS

Totally 20 diabetic patients were included in the study. The study was conducted in the department of plastic surgery, Government Mohan Kumaramangalam medical college hospital, from 2016-2018. Followed 20 patients with a surgical procedure called surgical offloading are by removing pressure element in head and neck of metatarsal bone involved.

Inclusion criteria

Inclusion criteria were patient of all age, no sex differentiation, no fixed duration of the ulcer.

All these 20 patients underwent all types of conservative methods with results of not healing completely. Followed 20 patients with a surgical procedure called surgical offloading is by removing pressure element in head and neck of metatarsal bone involved.

Surgical procedure

- Under ankle block with a dorsal vertical incision over the corresponding shaft of the metatarsal bone.
- Extensor tendon identified and lateraled form the bone.
- Hand with the neck of corresponding metatarsal bone removed.
- Haemostasis obtained.
- Wound closed with 4-0 nylon.
- For the planter side of ulcer wound debrided thoroughly without suturing, thorough wash gave hemostasis obtained, non-adhesive dressing done. POP applied.
- A simple technique is done under local, without any morbidity, easily once can follow and understood.
- Results in me more satisfactory and rewarding.

Statistical analysis

The results are presented in tables and expressed by relative values and mean value, unpaired t-test) were applied. The level of statistical significance was p <0.05.

RESULTS

Certain predicted local changes were observed. Eight patients had gangrene, phlegmon seen in 4 patients, 3 had an ulcer with osteomyelitis, 3 had severe ulceration, 2 had phlegmon. Among the commonest causes of these pathways, it’s possible to consider some triggers such as peripheral neuropathy, foot deformity, abnormal foot pressures, abnormal joint mobility, trauma, peripheral artery disease. Both macro- and microvascular diseases are believed to contribute to the consequences of the peripheral vascular disease, resulting in the inability of the ischemic limb to heal itself properly.

Table 1: Local changes.

| Local changes          | No. of patients |
|------------------------|-----------------|
| Gangrene               | 8               |
| Phlegmon               | 4               |
| Ulcer with osteomyelitis| 3               |
| Severe ulcer           | 3               |
| Phlegmon               | 2               |

Table 2: Grade and duration of healing.

| Forefoot ulcer | No. of patients | Duration of healing |
|----------------|-----------------|---------------------|
| I              | 12 patients     | 4 weeks             |
| II             | 5 patients      | <6 weeks            |
| III            | 3 patients      | >6 weeks            |

Figure 1: View of common site involved in diabetic foot ulcer.

Among the 20 patients wound healed, in about 12 patients within 4 to 5 week and its remaining 5 patients wound healed in 5 to 6 weeks are in other 2 patients it takes. More than 6 weeks to heal. We followed the patients for the past 1 year, out of 20 patients, 2 patients have developed an ulcer in another site, not the same site. In all other patients wound healed completely without any recurrence. Following an injury to the skin, a set of complex biochemical events take place in a closely orchestrated cascade in order to repair the damage. The natural healing process can be divided into four stages: inflammation, granulation, epithelialization, and...
maturation. However, when a wound becomes delayed in healing, these four stages are interrupted and a large number of changes occur that result in the wound becoming chronic, lasting sometimes for years without healing. Unstable diabetes and colonization of bacteria are two of the primary causes of this chronicity.

Figure 2: Operative procedures for healing measures.

Figure 3: Healing process after 6 weeks of surgery.

**DISCUSSION**

Diabetes mellitus (DM) is a group of metabolic diseases in which a person has high blood sugar. The incidence of DM is 3.3% in a human population with equal rates in females and males. Diabetes mellitus increases the risk of long-term complications. Diabetic foot complications are the most common cause of nontraumatic lower extremity amputations in the industrialized world. The risk of lower extremity amputation is 15 to 46 times higher in diabetics than in persons who do not have diabetes mellitus. The vast majority of diabetic foot complications resulting in amputation begin with the formation of skin ulcers. Severe infections pose an immediate threat to the leg and, potentially, to the patient’s life. Thus, prompt surgical intervention is needed. Severe infections can occur when a mild to moderate infection is complicated by critical ischemia or when an adequately perfused foot shows a marked local involvement (e.g., necrotizing fasciitis or an infection with anaerobic gas-forming organisms) or systemic signs and symptoms (fever, hypotension, and vomiting, suggestive of bacteremia). Necrotizing fasciitis is a severe illness with a mortality rate of 24%-33%. It has classically been connected with β-hemolytic streptococci, but a recent review of 163 cases found that 71% of those with a positive result of tissue culture had polymicrobial infections. Several reports have documented an association of necrotizing fasciitis with diabetes mellitus. The presence of severe pain with a deep planar foot infection in a diabetic patient is often the first alarming symptom, especially in a patient with a previously insensate foot. Several factors may lead to quick deterioration and its attendant complications. Most ulcers occur on the planar surface of the foot, at the head of the metatarsal bones. With infection and subsequent cellulitis, edema can develop in the underlying compartment, resulting in a compartment syndrome. When the pressure in a compartment exceeds capillary hydrostatic pressure, micro vascular circulation is impaired. Patients with diabetic neuropathy may have higher compartment pressures than do no diabetic patients. This may result from the sorbitol pathway in the diabetic patient leading to an excess of split products; these, together with the hydrophilic sorbitol molecule, may lead to edema. Moreover, the oxygen concentration may be lowered by the greater oxygen affinity of glycosylated haemoglobin. The resulting increased capillary permeability leads to edema and high compartment pressure. This leads not only to tissue necrosis but also to thrombosis of the small arteries, resulting in a more extensive deep necrosis, even in a well-vascularized foot. In this research, the results of the treatment of diabetic foot were significantly different in relation to the approach. Prior to the adoption of a single multidisciplinary approach to the treatment of diabetic foot, there had been a much higher frequency of amputations compared to the second period, which is in relation with results of other authors.

**CONCLUSION**

Infection of the foot in diabetic patients is a serious complication that may lead to a major amputation. Early recognition and proper treatment are mandatory to avoid poor outcomes. Surgery must always be combined with antibiotics, and revascularization may also be necessary. The surgeon must have a thorough knowledge of the foot anatomy but also must be familiar with the defects in wound healing caused by diabetes. The outcome of surgery largely depends on the skill, care, and experience of the surgeon.

**ACKNOWLEDGEMENTS**

The author would like to thank the professors, Associate professors and Assistant professors, Department of Plastic Surgery, Government Mohan Kumaramangalam Medical College, Salem, Tamil Nadu, India for their valuable support in research work.
Funding: No funding sources
Conflict of interest: None declared
Ethical approval: The study was approved by the Institutional Ethics Committee

REFERENCES

1. Boulton AJM, Vileikyte L, Ragnarson Tenvall G, Apelqvist J. The global burden of diabetic foot disease. Lancet. 2005;366:1719-24.
2. Cavanagh PR, Lipsky BA, Bradbury A, Botek G. Treatment for diabetic foot ulcers. Lancet. 2005;389:1725-35.
3. Singh N, Armstrong DSG, Lipsky BA: Preventing foot ulcers in patients with diabetes. JAMA. 2008;293:217-28.
4. American Diabetes Association. Peripheral arterial disease in people with diabetes. Diabetes Care. 2003;26:3333-41.
5. Apelqvist J, Agardh CD, Castenfors J, Stenström A, Agardh CD. Wound classification is more important than the site of ulceration in the outcome of diabetic foot ulcers. Diabet Med. 1989;6:526-30.
6. Cassar K, Bachoo P, Ford I, Greaves M, Brittenden J. Markers of coagulation activation, endothelial stimulation, and inflammation in patients with the peripheral arterial disease. Eur J Vasc Endovasc Surg. 2005;29:171-6.
7. Fejfarová V, Jirkovská A, Petkov V, Boucek P, Skibová J. Comparison of microbial findings and resistance to antibiotics between transplant patients, patients on hemodialysis, and other patients with the diabetic foot. J Diabetes Complications. 2004;18:108-12.
8. Fowler MJ. Microvascular and macrovascular complications of diabetes. Clin Diabetes. 2011;29:116-22.
9. Gensler SW, Haimovici H, Hoffert P, Steinman C, Beneventano TC. Study of vascular lesions in diabetic, nondiabetic patients: Clinical, arteriographic, and surgical considerations. Arch Surg. 1965;91:617-22.
10. Hope SA, Meredith IT. Cellular adhesion molecules and cardiovascular disease. I. Their expression and role in atherogenesis. Intern Med J. 2003;33:380-6.
11. Leskinen Y, Salenius JP, Lehtimäki T, Huhtala H, Saha H. The prevalence of peripheral arterial disease and medial arterial calcification in patients with chronic renal failure: Requirements for diagnostics. Am J Kidney Dis. 2002;40:472-9.
12. Nielsen LB. Atherogenicity of lipoprotein(a) and oxidized low-density lipoprotein: Insight from in vivo studies of arterial wall influx, degradation, and efflux. Atherosclerosis. 1999;143:229-43.
13. Pradhan AD, Rifai N, Ridker PM. Soluble intercellular adhesion molecule-1, soluble vascular adhesion molecule-1, and the development of symptomatic peripheral arterial disease in men. Circulation. 2002;106:820-5.
14. Raymakers JT, Houben AJ, Vd Heyden JJ, Tordoir JH, Kitslaar PJ, Schaper NC. The effect of diabetes and severe ischemia on the penetration of ceftazidime into tissues of the limb. Diabet Med. 2001;18:229-34.
15. Ridker PM, Cushman M, Stampfer MJ, Tracy RP, Hennekens CH. Plasma concentration of C-reactive protein and risk of developing the peripheral vascular disease. Circulation. 1998;48:192-6.
16. Ridker PM, Stampfer MJ, Rifai N. Novel risk factors for systemic atherosclerosis: A comparison of C-reactive protein, fibrinogen, homocysteine, lipoprotein(a), and standard cholesterol screening as predictors of peripheral arterial disease. JAMA. 2001;285:2481-5.
17. Roldan V, Marin F, Lip GY, Blann AD. Soluble E-selectin in cardiovascular disease and its risk factors: A review of the literature. Thromb Haemost. 2003;90:1007-20.
18. Tzoulaki I, Murray GD, Lee AJ, Rumley A, Lowe GD, Fowkes FG. Inflammatory, hemostatic, and rheological markers for incident peripheral arterial disease: Edinburgh Artery Study. Eur Heart J 2007;28:354-62.

Cite this article as: Rajendran MK. Definitive surgical treatment for diabetic forefoot ulcer. Int Surg J 2018;5:3982-5.