Excerpta Medica

The American Journal of Surgery

The American Journal of Surgery 190 (2005) 295–299

Principles of management: the diabetic foot

T. Jeffery Wieman, M.D.[a,b,*]

[a]The Cancer Institute, 4323 Wornall Road, Kansas City, MO, USA
[b]Department of Surgery, University of Missouri–Kansas City, Kansas City, MO, USA

Manuscript received April 13, 2005; accepted manuscript April 15, 2005

Abstract

Diabetes mellitus affects 5–10% of the US population at some point in their lives. Hyperglycemia produces serious chronic complications. Peripheral neuropathy is one of the most serious of these. Peripheral neuropathy, in the lower extremities, leads to plantar foot ulceration. Secondary infection of these ulcers is by far the leading cause of major amputations of feet and legs. Proper preventative care will dramatically reduce ulcer formation and costs related to this complication.

Keywords: Diabetes; Neuropathy; Ulcer; Prevention

Diabetes mellitus is a serious metabolic disease affecting approximately 5% to 10% of the United States population. Similar demographics exist around the world [1]. Variation in prevalence depends on the genetics and cultural features of a given population. The disease is a significant health risk in virtually every country. Diabetes has historically been divided into 2 categories: type I and type II. Type I diabetes is defined as a disease of insulin deficiency and develops most frequently in childhood or juvenile years. Type II diabetes is frequently called adult-onset, non–insulin-dependent, or insulin-resistant diabetes. Although these categories are useful for understanding the disease, strictly speaking, there is an overlap in the pathophysiology of type I and type II diabetes. For the purposes of the current discussion, a detailed review of the differences is not necessary. Foot complications occur in both forms of diabetes and are related more to the period of time that the illness has been present than to the age of onset [2].

Protracted hyperglycemia has a number of serious deleterious effects on the body. Major complications include accelerated peripheral atherosclerosis, complicated coronary artery disease, obesity, renal insufficiency, visual deterioration, and peripheral neurologic degeneration. The cumulative effects of diabetes detract from quality of life and shorten life span. The disease increases health care needs and expenses enormously [3].

Recently it has been shown that careful glucose control can significantly decrease the complications of diabetes. Much effort has been expended to help diabetics maintain near-normal glucose levels. Those patients who have been successful have much better outcomes. For a variety of reasons, however, good blood glucose control is not easily obtained in a many patients. Therefore, the management of the complications is still a major focus of medical care.

The current discussion will focus on lower-extremity complications including neuropathy, skin ulcerations, peripheral vascular disease and serious infections of the skin, soft tissues, and skeleton. Issues related to cause, management, and prevention will be addressed, and a brief review of the economic features of the problem will be provided [4].

Cause of Lower-Extremity Morbidity

The fundamental cause of lower-extremity complications in diabetes is chronic hyperglycemia leading to hyperglycosylation of various body tissues. Hemoglobin A1c is a measure of the amount of glucose bound to the hemoglobin A molecule. It is probably the most readily recognized example of hyperglycosylation in diabetes. Hemoglobin A1c is used to measure blood glucose control during an extended period (eg, several weeks). The higher the blood glucose level, the higher the bound glucose and the A1c value. Similar measurements can be made in other cells and membranes. The higher the serum glucose during a long

* Corresponding author. Tel.: +1-816-932-2823; fax: +1-816-932-2823.

0002-9610/05/$ – see front matter
doi:10.1016/j.amjsurg.2005.05.030

period of time, the higher the amount of glucose bound to cell membranes and other structures. High levels of bound glucose exert a number of deleterious affects on the body mechanically and physiologically [5].

High levels of glucose cause cell membranes to lose pliability and function abnormally. In the case of red or white blood cells, the mechanics of the "stiff" cells lead to impaired movement, attenuating the normal physiologic behavior of these critical blood components. Excess glycosylation of cell membranes of blood vessels, tendons, and connective tissues causes them to be rigid and more subject to mechanical injury on both a macroscopic and microscopic level.

Physiologic changes induced by "tissue hyperglycemia" of the lower extremities include potential decrease in oxygen exchange by limiting the actual exchange process or through induction of damage to the autonomic nervous system causing shunting of oxygenated blood away from the skin surface. There is a link between chronic hyperglycemia and the accelerated progression of atherosclerosis. This particular issue is quite important but complex and will not be the subject of further discussion in this review.

Nerves are damaged by hyperglycemia in number of ways. They are affected differently depending on their size and degree of myelination. As a general rule, the smaller the nerve diameter and the less myelinated the nerve, the more readily it is injured. Hence, the small, unmyelinated nerves involved in sensing light touch and temperature are most readily affected early in the disease [6]. As with the vascular effect, there is a great deal of variation from patient to patient. Individual genetic characteristics play an important role in of expression of morbid complications.

At least three mechanisms of nerve injury result from chronic hyperglycemia: a metabolic effect, a mechanical conduction defect, and a compartment compression effect. The first appears to result from accumulation of deleterious metabolites intracellularly. The second is a membrane conduction defect, and the third is the result of nerve compression secondary to swelling associated with water accumulation in the nerve. Water content is tied to the inherent water bound to glucose in membranes. As the amount of glucose in a membrane increases, it binds more water and leads to nerve swelling. Consequently, a nerve may become compressed at certain confining anatomic locations such as the posterior compartment behind the medial malleolus.

Decreases in tissue oxygen, combined with impaired sensory and motor nerve function during a prolong period of time, create an environment in which traumatic events occur and can produce what is known commonly as a diabetic foot ulcer. The acute events are the result of a pressure-induced tissue breakdown [7]. The pressure event is often not noted, and pathologic changes in plantar pressure distribution go unrecognized until tissue death has occurred. Very often the event is something as trivial wearing a wrinkled sock, a change of footwear, or an altered gait cycle [8]. The hypesthesia that results from the nerve dysfunction eliminates the normal pain-and-trauma recognition sequence. The result of the trauma is tissue breakdown in a hypoxemic, neurologically impaired location such as the planter surface of the foot.

Tissue death begins in the subcutaneous area beneath a skeletal prominence, notably a distal metatarsal head. Repeated trauma critically injures soft tissues by crushing the connective tissues between the bone and the skin. Cellular hypoxemia prevents repair of the tissue injury. The end result is tissue destruction that eventually penetrates to the plantar surface of the foot where it is recognized as an ulcer [9].

Management

There have been as many descriptions of the management of diabetic foot ulcers as there are descriptions of wound care management in general [10]. An in-depth review of these widely varied techniques is not possible within the limitations of the current manuscript. A brief discussion of currently accepted therapy based on causation will be presented, and the focus of the comments will be the key factors of tissue hypoxia, nerve injury, and pressure trauma. Consideration will also be given to the major morbid consequence of foot ulcers, ie, infection.

It should be noted at the outset that the treatment of diabetic foot ulcers has been the subject of some of the most inflexible, dogmatic teaching in all of medical and surgical education. Unfortunately, much of this dogmatic approach remains in place, not yet contested by reasoned, scientifically based concepts.

It is still widely believed worldwide, and to some degree in the United States, that it is not good practice to operate on the diabetic foot and that if an operation is performed, it will nearly always lead to a major amputation. Therefore, the "best" means of managing diabetic foot disease is to amputate the affected extremity at an early point in the natural history of an ulcer. Although this management practice may be "best" in rare instances, it incontestably is not "best" for the majority of patients. As will be described later, prevention is the best treatment. Unfortunately, despite increasing efforts to prevent ulcers, many still develop. Therefore, a discussion of basic ulcer care follows.

Nonsurgical management

Establishment of the correct causal diagnosis if the first step to preventing ulcers. Oxygen deficiency caused by macrovascular and microvascular pathology is of primary importance [11]. Perfusion of the injured area is crucial to healing. If adequate tissue perfusion cannot be maintained or restored, no other management process can be effective. Therefore, accurate physical and physiologic examinations to assure adequate perfusion should be completed, and remediation of oxygen deficits must be accomplished at the outset of care. Many schema and algorhythms have been described to accomplish this end. Specific techniques for revascularization change constantly and will not be detailed here.

Local wound care must be addressed once a plan for achieving adequate tissue perfusion has been established. Cleansing the ulcer and maintaining regular inspection of the contaminated site provides a healthy environment for wound healing. Frequent observations are required because these wounds have a propensity o become infected and progress rapidly to a life-endangering, systemic illness.

The use of topical agents to control wound colonization remains unproven but may have efficacy after surgical debridement. Use of specific topical antibiotic agents is not recommended. Broad-spectrum antimicrobial agents such as silver sulfadiazine may have a role in suppression of colonization, but their value has never been unequivocally proven.

Minimization of weight bearing is important because excessive pressure and shear force are major contributors to continued tissue injury. Even short periods of high pressure will cause further wound breakdown and must be avoided. Bed rest, crutches, casting, and special orthotics can play important roles in achieving pressure relief. Implementation of a particular method is patient specific. Each measure has its own advantages and drawbacks. A working knowledge of the benefits and limitations of each is necessary to optimize individual patient outcomes.

Systemic antibiotics are essential in infected wounds. Protracted use may be justified in some cases, but short, defined, and directed courses are the norm. Choices of agents depend on identification of systemic pathogens. Local tissue swabs are misleading, but quantitative tissue cultures have been used with success [12]. Consideration of the possibility of osteomyelitis is mandatory. If osteomyelitis is present, surgical removal of the bone is nearly always required.

Use of adjunctive nonoperative therapies can be considered, but inconsistent results should be expected. There is some scientific evidence to support the use of topical platelet-derived growth factor and vacuum-assisted wound closure [13,14]. The application of other surface agents and the treatment with hyperbaric oxygen have very little, if any, good data to support their routine use in management.

Surgical management

Surgical management is the mainstay of treatment. Abundant data shows that extensive debridement of wounds and removal of devitalized tissue is the single most important therapeutic step leading to wound closure and limb salvage [15]. Repeated removal of infected and traumatized tissues must be carried out for most patients. This, associated with good wound care as described previously, will lead to closure of the majority of ulcers. Remembering that these are biologically as well as chronologically chronic wounds is very important. For that reason, good outcomes are measured in weeks and months rather than in hours and days. Six to ten weeks is the expected time for healing of these complicated lesions [16].

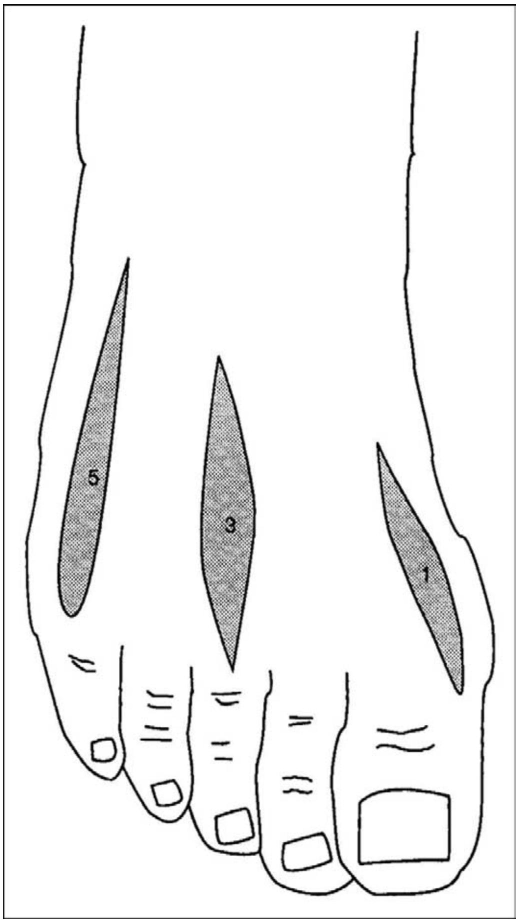

Fig. 1. Skin incisions for excision of three metatarsal heads. Note the extension of the incisions onto the forefoot and the lateral placements for the first and fifth metatarsals. Reprinted with permission from Griffiths and Wieman [24].

Chronic injury from pressure trauma is the direct cause of foot ulceration and the most frequent reason for recurrences. One must address pressure redistribution issues to obtain the best long-term outcomes for patients with ulcers and for those at high risk. Proper orthotics and shoes are indispensable in patient management. A host of other potentially beneficial equipment and devices are available to help accomplish pressure decrease. Ultimately, however, permanent anatomically based pressure decrease must be accomplished. Operative treatment is usually required to achieve this.

A number of operative procedures have been devised to decrease the pressure on the planter surface of the foot and allow wound healing to occur. Foremost among those is the metatarsal head resection (Figs. 1 and 2), in which the prominent bone beneath the plantar forefoot ulcer is removed [17]. Several articles from the Department of Surgery at the University of Louisville have demonstrated the

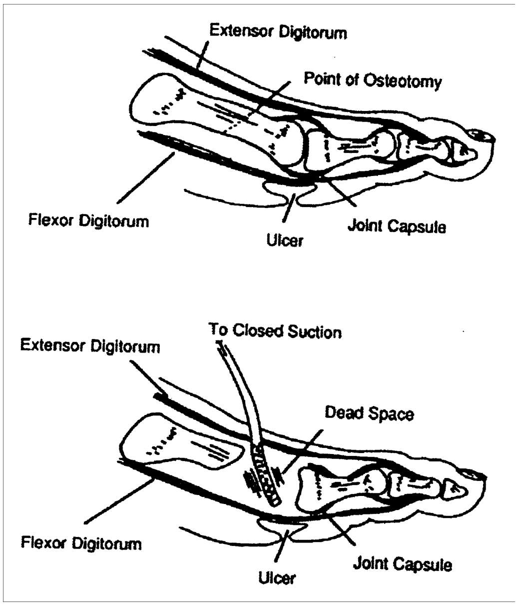

Fig. 2. Line of division of the shaft and appearance of beveled end at the end of the procedure. Reprinted with permission from Griffiths and Wieman [24].

safety and efficacy of this operation, which as a result has become standard therapy in many specialized institutions. Variants of the bone removal procedure to achieve pressure decrease have been developed to manage other sites and have proven similarly valuable [18,19].

After surgery, good wound care, education, foot and skin hygiene, frequent examinations, and adjustments in footwear are required along with appropriate diabetes management. If these steps are taken appropriately, a many-fold decrease in ulcer recurrence will result and an even greater decrease in the prevalence of major amputation should be expected.

Prevention

During the course of the past 2 decades, physicians have learned a great deal about the cause of diabetic complications, specifically the cause of diabetic foot ulcers. Maintenance of near-normal blood sugar has been shown to decrease the frequency of serious complications including most notably, peripheral neuropathy. Because euglycemia is difficult to maintain across decades, complications still represent major personal and public health dilemmas. Studies done at the University of Louisville Department of Surgery have demonstrated that serious complications in the lower extremities can be prevented or minimized in the majority of patients. Regular foot examination, patient education, simple hygienic practices, and provision of appropriate footwear combined with prompt treatment of minor injuries can decrease ulcer occurrence by 50% and can decrease the need for major amputation in nonischemic limbs to near zero [20,21].

Economics

Diabetes has a huge impact on personal and nationwide economics. The individual cost to receive adequate general care for diabetes has increased dramatically in that past 2 decades. The cost of diabetic medication and the intensity of treatment for heart disease, renal failure, and the like have reached shocking proportions. It is worth noting here that as frequent and expensive as treatment of diabetic renal disease is, the cost for the management of diabetic foot complications exceeds all of the costs for renal dialysis for all diabetic patients in the United States. The cost of treating foot complications is largely preventable using the simple steps listed previously [22].

A study of expenses performed in the department in 1998 indicating the following: The cost to treat one simple ulcer was $5000 to $8000. An admission for an infected ulcer cost approximately $15,000. Treatment of an episode of osteomyelitis cost $25,000 to $30,000, and an amputation cost between $50,000 to $150,000 in direct expenses. Indirect costs, ie, those related to family support, disability, and loss of independence, are many times the cost of the direct expenses. The numbers are staggering compared with the cost of a year of preventive care, which is roughly $1000 including a pair of shoes [22,23].

Comments

Diabetic foot ulcers are common and serious complications of chronic diabetes mellitus. Ulcers and accompanying infections lead to major health dilemmas and economic morbidity. Basic principles of wound care, including surgical and nonsurgical components, are essential to effective healing of these wounds. The therapy should be directed toward the causes: chronic tissue hypoxia, nerve injury, and pressure trauma. The majority of ulcers can be healed with proper local care if tissue hypoxia can be controlled. Prevention of foot ulceration is possible, desirable, and the most cost-effective means of managing this morbid complication of diabetes.

References

[1] Boulton AJ. The diabetic foot: a global view. Diabetes Metab Res Rev 2000;16(suppl 1):S2–S5.
[2] Reiber GE, Boyko EJ, Smith DG. Lower extremity foot ulcers and amputations in diabetes. In: National Diabetes Data Group (U.S.). Diabetes in America (NIH publication no. 95-1486). 2nd ed. Bethesda, MD: National Institutes of Health, National Institutes of Diabetes and Digestive and Kidney Diseases; 1995.
[3] Obrien IA, Corrall RJ. Epidemiology of adult diabetes and its complications. N Engl J Med 1998;318:1619–21.
[4] Reiber GE. The epidemiology of diabetic foot problems. Diabet Med 1996;13:S6–S11.
[5] Feig DS, Palda VA, Lipsome L. From the Canadian Task Force on Preventative Health Care. Screening for type 2 diabetes. UKPDS. Update 2005.

[6] Boulton AJ. The diabetic foot: neuropathic in etiology. Diabet Med 1990;7:852–8.
[7] Cavanaugh PR, Ulbrecht JS, Caputo GM. New dynamics in the biomechanics of the diabetic foot. Metab Res Rev 2000;16(suppl 1):S6–S10.
[8] Patel VG, Wieman TJ. Effects of metatarsal head resection for diabetic foot ulcers on the dynamic plantar pressure distribution. Am J Surg 1994;167:297–301.
[9] Delbridge L, Ctertcteko GH, Fowler C, et al. Etiology of diabetic neuropathic ulceration of the foot. Br J Surg 1985;72:1–6.
[10] Levin ME. Diabetic foot ulcers: pathogenesis and management. J ET Nurs 1993;20:191–8.
[11] Hunt TK, Hussain Z. Wound microenvironment. In: Cohen IK, Dinglemean RF, Lindblad WJ, eds. *Wound Healing: Biochemical and Clinical Aspects*. Philadelphia, PA: Saunders; 1992:274–81.
[12] Robson MC. Wound infection: a failure of wound healing caused by an imbalance of bacteria. Surg Clin North Am 1997;77:637–50.
[13] Wieman TJ, Smiell JM, Su Y. Efficacy and safety of a topical gel formulation of recombinant platelet derived growth factor BB (becaplermin). Diabetes Care 1998;21:822–7.
[14] Carson SN, Overall K, Lee-Jahshan S, et al. Vacuum-assisted closure used for healing chronic wounds and skin grafts in the lower extremities. Ostomy Wound Manage 2004;50:52–8.
[15] Steed DL. Foundations of good ulcer care. Am J Surg 1998;176(suppl 2A):20S–5S.
[16] Griffiths GD, Wieman TJ. Meticulous attention to foot care improves the prognosis in diabetic ulceration of the foot. Surg Gynecol Obstet 1992;174:49–51.
[17] Griffiths GD, Wieman TJ. Metatarsal head resection for diabetic foot ulcers. Arch Surg 1990;125:832–5.
[18] Wieman TJ, Griffiths GD, Polk HC Jr. Management of diabetic midfoot ulcers. Ann Surg 1992;215:627–32.
[19] Wieman TJ, Patel VJ. Treatment of hyperesthetic neuropathic pain in diabetics. Decompression of the tarsal tunnel. Ann Surg 1992:221: 660–5.
[20] Griffiths GD, Wieman TJ. Meticulous attention to foot care improves the prognosis in diabetic ulceration of the foot. Surg Gynecol Obstet 1992;174:49–51.
[21] Wieman TJ. Clinical efficacy of becaplermin (rhPDGF-BB) gel. Am J Surg 1998;176(suppl 2A):74S–9S.
[22] Wieman TJ, Mercke YK, Cerrito PB, et al. Resection of the metatarsal head for diabetic foot ulcers. Am J Surg 1998;176:436–41.
[23] Ramsey SD, Sandhu S. Incidence, outcomes, and cost of foot ulcers in patients with diabetes. Diabet Care 1990;22:382–7.
[24] Griffiths GD, Wieman TJ. Metatarsal head resection for diabetic foot ulcers. Arch Surg 1990;125:832–5.