The Diabetic Foot

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Abstract

Management of foot problems in the patient with diabetes mellitus requires attention to each system affected by the disease. Appropriate treatment of common clinical problems affecting the foot in diabetic patients, such as ulcerations and fractures, depends on a thorough understanding of the pathophysiology of the disease. Treatment of neuropathy is directed at pressure relief and prevention of deformity. Infection is addressed with antibiotics, debridement, and improvement of the vascularity and oxygenation of the tissues. Amputation should be viewed, not as evidence of treatment failure, but as a reconstructive procedure, the goal of which is to regain energy-efficient ambulation. The orthopaedic surgeon can play a critical role in the team approach to the care of the diabetic patient with foot problems.

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Diabetes mellitus is a systemic disease affecting the nervous, vascular, skeletal, immune, and integumentary systems. Management therefore requires a multisystem approach that addresses each of these problem areas. The spectrum of foot involvement ranges from mild insensitivity to nonhealing ulcers, neuropathic arthropathy, osteomyelitis, and severe vascular insufficiency.

The orthopaedic surgeon has a critical role in the team approach to the diabetic patient with pathologic changes in the foot. With a background in extremity surgery, familiarity with the biomechanical function of the extremity in gait, and the ability to prescribe footwear, orthoses, and prosthetics, the orthopaedist is uniquely qualified to act as a leader of the diabetic foot team.[1] The team should take an interdisciplinary approach and involve the expertise of nurse educators, physical therapists, a prescription shoe specialist, and social services representatives, as well as input from the disciplines of vascular surgery, infectious diseases, endocrinology, and neurology. The basic principle of this team approach is to provide a setting wherein the patient with diabetes can receive complete foot care, ranging from education and prevention to aggressive treatment and continuity of follow-up, so that problems such as ulceration, infection, and amputation can be avoided.

Pathophysiology

Nervous System

Diabetic neuropathy is most common in patients with poor metabolic control. The sensory, motor, and autonomic divisions of the nervous system are involved. All diabetic neuropathies have in common the loss of myelinated and nonmyelinated nerve fibers. Poor regulation of microcirculation, oxygen delivery, and extraneural arteriovenous shunting contribute to ischemic and focal nerve-fiber loss. Hyperglycemia likely contributes to metabolic abnormalities of the peripheral nerves and is associated with elevation of the nerve sorbitol concentration; depletion of *myo*-inositol, Na^+, and K^+ stores; and a decrease in adenosine triphosphatase activity. All of these metabolic events lead to slowed nerve conduction and characteristic neural lesions. Aldose-reductase inhibitors decrease the excess conversion of glucose to sorbitol and have been shown to reverse some of the structural lesions associated with diabetic neuropathy.[2]

Sensory neuropathy predominates, appearing first distally and then progressing proximally in a stocking/glove pattern. Large-fiber involvement diminishes light-touch sensation and proprioception,

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resulting in ataxic gait and intrinsic muscle weakness of the hands and feet. Small-fiber involvement decreases pain and temperature perception, making the patient susceptible to repetitive injury.

The inability to respond to mechanical stress because of poor sensation is often responsible for initial tissue breakdown. Brand[3] has described three levels of mechanical stress that lead to the breakdown (ulceration) of tissue. High stresses concentrated on a small area may cause immediate injury by puncture or tearing. High-tension or shear stresses are generated by stepping on a small object, either on the ground or within the shoe. Moderate stresses are the most common cause of injury in the insensitive foot.

The ultimate tensile strength of the intact plantar skin approximates 1,200 lb / sq in. During normal walking, average pressures do not exceed 75 lb / sq in in a barefoot individual, 50 lb / sq in in a person walking in a leather-soled shoe, and 25 lb / sq in in a person walking in a shoe with a padded, molded insert. Pressures of at least 200 lb / sq in are necessary to produce pain in a sensitive foot.

The gap between the level of pressure that produces pain and the level of pressure that causes injury is abridged by the long-term repetition of moderate stresses. Using pressures as low as 20 lb / sq in for 10,000 repetitions per day, Brand showed that a progressive inflammation develops by the third day; a true ulceration, by the eighth day. People with normal sensation feel pain early in the inflammatory stage, relieve it, and allow the tissues to heal. When sensation is compromised, however, the pressure is not alleviated early enough to avoid tissue damage.

Stresses so low as to go unnoticed even by people with normal sensation (1 to 2 lb / sq in) are sufficient to block capillary blood flow, resulting in ischemia. This type and level of stress is seen in the patient with a tight shoe. The resultant discomfort of ischemia is noticed by the individual with normal sensation. If not addressed, even this amount of pressure over several hours can be enough to cause tissue damage.

In addition to a diminished pain response, diabetic neuropathy gives rise to neuropathic paresthesias, with accompanying symptoms of cutaneous contact hypersensitivity, superficial burning pain, and / or severe deep aching. These sensations may be caused by spontaneous depolarization of regenerating nerve fibers.

The autonomic nervous system regulates perspiration, skin temperature, and arteriovenous shunting. Loss of this function decreases the pliability of the plantar skin, causing it to form thick, stiff callus, usually in areas of pressure concentration. The callus becomes dry when there is loss of normal perspiration, leading to cracks and fissures through the dermis and creating a portal for bacteria and subsequent infection. Furthermore, as the hard callus thickens, it causes deep-tissue breakdown and necrosis.

Motor neuropathy is less common and involves conduction deficits secondary to demyelinization as well as involvement of the motor endplate. It may affect a single major peripheral nerve (e.g., the common peroneal nerve, resulting in a foot drop). More commonly, the motor neuropathy is distal and results in dysfunction of the intrinsic muscles of the foot. This leads to clawing of the toes, depression of the metatarsal heads, and subsequent abnormal pressure concentration on the plantar aspect of the forefoot. Dorsal ulceration of the toe results from extrinsic pressure by the shoe on the flexed proximal interphalangeal joint.[4]

Vascular System

The incidence of atherosclerotic peripheral vascular disease is increased in patients with diabetes mellitus, in whom the disease has an earlier onset and affects a higher percentage of women than in the nondiabetic population. Diabetic patients also have more diffuse involvement, which is more often bilateral and rapidly progressive. Although the pathologic changes in peripheral vascular disease may be the same in diabetics and nondiabetics, the rapid progression in diabetics is not accompanied by sufficient development of collateral circulation to prevent ischemia.

Peripheral vascular disease in the diabetic involves both the small and large vessels. A pathologic lesion at the subarteriolar level that correlates with decreased flow and ulceration has not been conclusively identified. Changes in basement membrane thickening and capillary permeability have been shown; however, no relationship between these histologic changes and the clinical appearance of ulceration has been documented.

Large vessels are the arteries of conduit, have few branches, and are found within fascial planes. Calcification of these arteries tends to be more circumferential and diffuse than in nondiabetics. Proximally, the most common sites of occlusion are the superficial femoral artery at the adductor canal, the aortic bifurcation, the common iliac bifurcation, and the common femoral artery bifurcation. In diabetics, the tibial and deep femoral arteries may be selectively obliterated. Between the knee and the foot, there is often diffuse involvement of the anterior tibial, posterior tibial, and peroneal arteries. These noncompressible calcified arteries are often visible on plain radiographs of the foot. Collateral circulation can often overcome a single level of occlusion, as is often seen in nondiabetics, but the

diffuse involvement of both large and small vessels complicates the treatment of vascular disease in diabetics.[4]

Immune System

Diabetics do not respond normally to established infection. Because of the systemic effects on the nervous and vascular systems, the skin of their feet has suboptimal durability. Small cracks or fissures in the skin allow entry of bacteria. Infection can then progress more easily due to the altered function of the white blood cells. Polymorphonuclear leukocytes have altered chemotaxis and respond less efficiently to the bacteria. Furthermore, the combination of malnutrition, hyperglycemia, and decreased oxygen tension and insulin concentration leads to inefficient anaerobic metabolism, increased acidity, hypertonicity, and edema, which in turn provide a favorable environment for bacterial growth and diminished function of polymorphonuclear leukocytes and antibiotics.[5] There is also decreased fibroblastic function and decreased collagen production and strength, which further compromises wound healing.

Evaluation

The patient who presents with a foot problem secondary to diabetes mellitus must undergo a thorough evaluation of each system affected by the disease.

The skin is visible and accessible and reflects the functional level of circulation and sensation. Evaluation of skin sensation includes qualitative and quantitative measures. Qualitative methods are light-touch and pin-prick sensation, two-point discrimination, and proprioception. Each of these is often decreased in the patient with sensory neuropathy and is usually distributed in a stocking-type pattern below the knee.

Quantitative methods are more objective. The most commonly used method entails use of the Semmes-Weinstein monofilaments, which are mounted nylon filaments of various sizes that are pressed into the skin perpendicularly until they bend. Thicker filaments require more force to bend and should be easier for the patient to feel. The size of the smallest filament that can be felt is considered the threshold of the patient's sensation. Although it has been reported that protective sensation is present if the patient can feel the 5.07 monofilament, approximately 10% of patients with sensation at this level develop ulcerations or neuropathic joints.

The biothesiometer has also been used to quantify sensation. It is an electrical device that delivers a measured, reproducible vibratory stimulus. The device has not gained widespread use.[1] Its limitations include age dependence, variability of applied pressure, and lack of natural history studies demonstrating predictability for diabetic ulcerations.

The vascular evaluation has a major impact on treatment choices. Qualitative measures include palpation of pulses, skin temperature, capillary refill, and hair and nail growth. Many quantitative methods have been proposed, the most recent trend being noninvasive assessment, including measurements of pressure, flow, and tissue oxygenation. Doppler examination yields pressure measurements at multiple levels (e.g., above the knee, below the knee, and in the ankle and toe).[1] Doppler measurement of flow depends on the compressibility of the vessels being studied. It is expressed as an absolute pressure with a flow wave or as part of the ankle-brachial index, which is a percentage produced by dividing the systolic pressure in the lower extremity by that in the brachial artery. An ankle-brachial index of 0.45 was initially considered to be a minimum requirement for healing of diabetic foot amputations. The reliability of this has been improved by examining absolute toe pressures and flow waveforms. The minimum absolute toe pressure for amputation healing is 45 mm Hg; that for ulcer healing, 60 mm Hg. Waveforms are depicted on pulse-volume recordings and are categorized as triphasic (normal), biphasic, or monophasic. The waveform characteristics reflect the compliance of the arterial wall being examined and allow a critical interpretation of the pressures given. The major limitation of pressure values is the falsely increased pressure necessary to compress calcified vessels. However, by locating discrete levels of occlusion in the large vessels, the findings from the lower-extremity Doppler evaluation and the physical examination should allow identification of patients who would benefit from vascular reconstruction.

Skin-perfusion pressure has also been used as a noninvasive assessment.[6] With this method one measures the minimum external pressure needed to prevent reddening of the skin after blanching. A photodetector is pressed against the skin by a manometer cuff inflated to a pressure above the systolic level. As the cuff is deflated, capillary inflow begins, and the skin perfusion pressure is read as the pressure of the cuff. Pressures of 31 to 40 mm Hg have been associated with a healing rate of 85% at the below-knee level.[6]

Determining transcutaneous oxygen diffusion ($TcPO_2$) is the most accurate method for assessing local skin vascularity and healing potential in the diabetic foot.[7] The $TcPO_2$ is measured at standardized points on the foot with use of a neonatal tissue

O_2 monitoring device, controlling for temperature. The skin should be heated to 45°C for 10 minutes. In the study by Pinzur et al,[7] healing of foot and ankle amputation wounds in nutritionally stable patients correlated with $TcPO_2$. The healing rate was 50% with a $TcPO_2$ of 1 to 19 mm Hg, 75% with a $TcPO_2$ of 20 to 29 mm Hg, and 92% at levels greater than 30 mm Hg.

The $TcPO_2$ measurement is also used in determining the need for hyperbaric oxygen treatments. Patients whose $TcPO_2$ levels improve after breathing 100% O_2 by nasal cannula or after a trial of hyperbaric oxygen may benefit from serial treatments to promote angiogenesis and tissue healing. The benefits of hyperbaric oxygen treatments for diabetic patients remain to be proved in randomized prospective trials. The $TcPO_2$ level is adversely affected by edema and cellulitis and is often improved if these conditions are treated.

The metabolic control of diabetes is usually managed by the primary-care physician or endocrinologist; however, the orthopaedist must be aware of the nutritional indices when assessing healing potential. A total serum protein concentration of 6.2 g/dL, a serum albumin level of 3.5 g/dL, and a total lymphocyte count of 1,500/mm^3 are minimal levels necessary to optimize tissue healing.[8]

Management of Common Problems

Ulceration

Ulceration in the diabetic foot results from a combination of neuropathy and extrinsic pressure from a shoe applied to a bony prominence. Ulceration is often the precursor of infection and subsequent osteomyelitis, necessitating ablative surgery. Many classifications have been proposed, two of which are shown in Table 1.[8] Approximately 70% to 90% of neuropathic ulcers occur in the forefoot; the heel is the next most commonly affected area, followed by the midfoot.

The treatment of the deformed foot without ulceration (grade 0) is directed at relief of pressure. This is done by using shoe modifications and molded inserts. In mildly involved feet, use of an oversized jogging or walking shoe with extra socks is an inexpensive initial treatment. An extra-depth shoe with a molded insole can be prescribed. Rigid insoles are not recommended for the insensate foot. The insole should be made of accommodative materials, usually the thermomoldable polyethylene foams that can be layered in several densities to give maximum pressure relief. Surgical correction should be considered for bony prominences that cannot be managed by nonoperative methods, provided the patient has sufficient vascular supply to allow incisional healing.

Table 1
Two Classifications of Ulceration

Wagner Classification[8]	
Grade 0:	No open lesions
Grade 1:	Superficial ulcer
Grade 2:	Deep ulcer
Grade 3:	Localized osteomyelitis or abscess
Grade 4:	Forefoot gangrene
Grade 5:	Gangrene of entire foot
Depth-Ischemia Classification[1]*	
Depth	
Grade 0:	At risk foot, no ulcer
Grade 1:	Superficial ulcer, not infected
Grade 2:	Deep ulceration
Grade 3:	Extensive ulceration with exposed bone and deep infection
Ischemia	
Grade A:	Not ischemic
Grade B:	Ischemia without gangrene
Grade C:	Partial forefoot gangrene
Grade D:	Complete foot gangrene

*As an example of how this classification system is used, a grade 2B lesion is a deep ulcer in a foot with vascular ischemia but without gangrene.

Once ulceration has occurred, it must be managed aggressively to halt progression. If pressure cannot be adequately relieved with molded insoles and shoe modification, the total-contact cast offers a good alternative.[9] The total-contact cast is a below-knee weight-bearing cast popularized by Brand[3] in the treatment of neuropathic ulcers caused by Hansen disease. There is very little padding, to allow exact conformation of the plaster to the contours of the leg and foot. This evenly distributes the pressure and shear stresses over the foot, effectively relieving the pressure over the ulcer and allowing it to heal. Myerson et al[10] used the total-contact cast for management of Wagner grade I and II lesions and reported 90% healing at a mean of 5.5 weeks. There was a 31% rate of recurrence over the 18-month follow-up period. Eighty-one percent of the recurrent ulcers healed in a second cast after 2 weeks of treatment. The recurrent ulcerations were usually associated with an underlying bony prominence.

Infection

Infection of the foot is a common reason for hospitalization of the diabetic patient. Infection often follows neuropathic ulceration or cracking of the skin. Prevention through the use of proper footwear and anticipation of problems is important. Routine examination of asymptomatic feet is essential because 49% of diabetic patients who present with foot infections will have an infection in the contralateral foot within 18 months.[11]

Staging the infection enables the physician to make the proper decisions for treatment. Infection should be staged with a system similar to that used for ulceration. Table 2 shows the University of Texas Medical Branch staging system.[5] This gives an anatomic description based on the depth of infection and the quality of the overlying tissue. Stages I and II can be treated nonoperatively with antibiotics, provided the source of increased pressure that caused the ulcer is addressed. If osteomyelitis or an abscess is present, necrotic tissue should be debrided and culture-specific antibiotics should be administered. After the final surgical debridement, soft-tissue infections are treated for 2 weeks and osteomyelitis is treated for 4 weeks.

The infection in a diabetic foot is often caused by multiple organisms, and their isolation should be attempted before starting antibiotic therapy. Surface swab cultures of an ulcer can be used to identify approximately 66% of the pathogenic bacteria. Enterococci and Gram-negative bacteria are often missed. Deep cultures obtained under surgical conditions have a higher accuracy. Aerobic and anaerobic cultures of both the soft tissues and the bone should be obtained. *Staphylococcus aureus* is the bacterium most commonly isolated, followed by *Streptococcus* species, *Enterococcus* organisms, and *Staphylococcus epidermidis.* Commonly isolated Gram-negative organisms include *Proteus mirabilis* and *Pseudomonas aeruginosa.* Anaerobes are cultured in about one third of patients. Table 3 lists the most common pathogenic organisms and their incidence.[12]

The vascular supply must be evaluated when treating an infection. An area of cellulitis with good blood flow allows delivery of antibiotics and nonoperative treatment. Areas that are poorly vascularized or necrotic cannot be adequately perfused with antibiotics (stage III and IV infections) and require surgical debridement to the level of viable tissue.

Once cultures have been obtained, an antibiotic regimen must be chosen. During the interval between the culture and the final determination of sensitivities, a broad-spectrum regimen is used to cover Gram-negative, Gram-positive, and anaerobic organisms. Antibiotic choice can be modified once the final sensitivities of the pathogenic bacteria have been obtained. Table 4 lists the common pathogens and the antibiotics commonly used to treat them.

Table 2
University of Texas Medical Branch Staging System for Musculoskeletal Sepsis

Stage*	Soft Tissue	Osteomyelitis	Septic Arthritis
I	Cellulitis	Medullary	Acute
II	Ulceration	Superficial	Chondrolysis
III	Abscess	Local	Chondrolysis and osteomyelitis
IV	Permeative or gangrene	Diffuse	Unstable joint

*System also involves classification according to physiologic class status: A, good immune system and vascular delivery; B, compromised locally or systemically; C, requires suppressive or no treatment due to minimal disability or treatment being worse than the disease. As an example of how this classification system is used, superficial osteomyelitis affecting only the surface of the bone in a compromised host would be designated stage IIB.

Neuropathic Joint Disease

Distal neuropathy can have a profound effect on the osseous architecture of the foot, turning a seemingly minor traumatic event into complete collapse. The destruction of neuropathic arthropathy has been attributed to cumulative microtrauma to joints that lack protective sensation. Because of the concomitance of erythema and hypervascularity, a disorder of the autonomic nervous system is considered a possible contributing factor. Documentation of progression in non-weight-bearing patients makes multifactorial causation likely.

Neuropathic arthropathy occurs in 0.15% to 2.5% of the diabetic population. Affected patients are more likely to be insulin-dependent. Thirty percent of these patients have bilateral involvement. The clinical presentation may be insidious, with a progressive flatfoot deformity, or its advance may be acute, accompanied by swelling and erythema. In patients with an acute onset, differentiation from acute infection is essential.

On physical examination, erythema, tenderness, crepitus, deformity, and even ulceration may all be present in either a Charcot foot or an infected foot. If the skin is intact, differentiation between these two conditions can be quite difficult. The erythema and edema of the neuropathic joint should dissipate if it is elevated above the level of the heart when the patient is in the supine position.[4] Laboratory studies should show a normal or only slightly elevated erythrocyte sedimentation rate and white blood cell count. Fortunately, osteomyelitis without the

Table 3
Pathogenic Bacteria in the Diabetic Foot

Organism	Incidence
Staphylococcus aureus	46%
Streptococcus species (nonenterococcal)	35%
Enterococcus species	29%
Proteus mirabilis	26%
Staphylococcus epidermidis	23%
Peptostreptococcus species	22%
Diphtheroids	19%
Pseudomonas aeruginosa	16%
Bacteroides species	16%

history or presence of ulceration is uncommon.

More sophisticated testing with nuclear scans and magnetic resonance imaging is available, but costly. Technetium and white blood cell scans have been used to establish the diagnosis of osteomyelitis. Scanning with indium-111–labeled leukocytes has better specificity for infection than technetium-99m scanning. When both studies are done together after a few days of antibiotic therapy and elevation of the affected foot, their accuracy is improved. The combination of a positive indium scan and a positive technetium scan in the same area is suggestive of osteomyelitis, whereas a negative indium scan is suggestive of a neuropathic joint. Magnetic resonance imaging has also been used, but it is difficult to distinguish the marrow edema of infection from that of neuropathic arthropathy.

Treatment of the patient with neuropathic arthropathy requires early recognition and long periods of immobilization with restricted weight-bearing. The Eichenholtz staging system describes the natural history of neuropathic changes in the feet of patients with tabes dorsalis but can be applied to diabetics with neuropathic arthropathy. Stage I is characterized by fragmentation and presents as a fracture, fracture dislocation, or pure ligamentous disruption. It should be treated with elevation for 1 to 2 days, followed by total-contact casting. The initial cast should be changed in 1 to 2 weeks; thereafter, the cast should be changed every 2 to 4 weeks. Weight-bearing on the affected foot should not be allowed until the inflammatory stage is over, which may take several weeks to several months. Progression of the process can be monitored on the basis of changes in skin temperature, volume, and the appearance on weight-bearing radiographs. The foot and ankle should be kept immobilized until the temperature has decreased to within 2°C of that of the uninvolved foot and should be protected thereafter. The goal of treatment is to prevent deformity. Stage I usually lasts 2 to 6 months.

Stage II disease is characterized by coalescence. The recognition of this stage is not made on the basis of a single radiograph, but by noting the gradual cessation of fragmentation on a series of radiographs. This stage marks the beginning of the reparative process. The temperature of the foot is decreased, along with the erythema and edema. The patient with midfoot or hindfoot involvement may then progress to weight-bearing in a total-contact ankle-foot orthosis. Rocker-bottom shoes with custom-molded inserts and steel shanks are preferable for patients with forefoot involvement. This is continued until bone consolidation and healing have occurred, which marks stage III. Stages II and III combined typically last 18 to 24 months.

The most common location for neuropathic destruction is the midfoot (60% to 70% of cases). The hindfoot accounts for 30% to 35% of cases. The changes generally occur in one area of the foot. Recurrence after bone consolidation is rare. Brodsky[4] has anatomically classified neuropathic joint disease on the basis of which joints are seen to be primarily affected on plain radiographs. Type 1 involves the midfoot, affecting the tarsometatarsal and naviculocuneiform joints, and presents problems of symptomatic plantar and medial bony prominences. Type 2 affects the hindfoot and causes problems with instability and slow bone consolidation. Type

Table 4
Antibiotic Selection

Organism	Antibiotic
Staphylococcus (methicillin-sensitive)	First-generation cephalosporin, β-lactamase-resistant penicillins (oxacillin, nafcillin), vancomycin, rifampin
Staphylococcus (methicillin-resistant)	Vancomycin
Enterococcus (group D *Streptococcus*)	Ampicillin, β-lactamase inhibitor (sulbactam or ticarcillin-clavulanate), vancomycin
Gram-negative (*Pseudomonas* species, *Proteus* species)	Ciprofloxacin, ofloxacin, ceftazidime
Anaerobes (*Bacteroides* species)	Clindamycin, metronidazole, ticarcillin-clavulanate

3, the least common, has two subcategories. Type 3A involves the tibiotalar joint and is problematic because of the instability it creates and the excessively long period required for bone healing. Type 3B is a pathologic fracture of the os calcis, which leads to pes planus and a widened heel, necessitating shoe modifications.

Surgical intervention in neuropathic joint disease should be approached with great caution. The overall treatment goal should be prevention of deformity and maintenance of energy-efficient ambulation. Papa et al[13] recently reported their experience with arthrodesis in diabetic patients with neuropathic arthropathy. Using selective open reduction and arthrodesis with rigid fixation for severe instability or a fixed deformity, they were able to salvage 93% of the affected feet. The fusion rate was 69%, but there was a 65% complication rate. A non-weight-bearing plaster cast was used for 2 months postoperatively, followed by a weight-bearing total-contact cast for an average of 5 months. A permanent polypropylene ankle-foot orthosis was then prescribed. Seventy-two percent of the patients underwent ankle fusion as an alternative to amputation. When embarking on such a treatment course, it should not be forgotten that 30% to 35% of patients with neuropathic joint problems will be affected bilaterally. Careful surveillance of the contralateral limb is necessary during the prolonged course of non-weight-bearing and immobilization of the operative limb.

Fractures

The treatment of fractures in diabetic patients must incorporate a respect for the possibility of underlying neuropathy. Thompson and Clohisy[14] recently reported a series of fractures in diabetic patients and identified juvenile-onset diabetes mellitus, neuropathy, nephropathy, and retinopathy as additional risk factors for fractures about the foot and ankle.

An acute fracture involving the ankle and tarsal bones should be treated with the nonoperative or operative protocol that would be appropriate for a nondiabetic patient. Non-weight-bearing immobilization until demonstration of healing is necessary to prevent deformity in both nonoperatively and operatively treated fractures. Once healing has been demonstrated, the patient can be progressed to a patellar-tendon weight-bearing orthosis or the Charcot restrained orthotic walker.

When a deformity cannot be managed with an orthosis, surgical reconstruction may be indicated. The goal of reconstruction is to obtain a plantigrade foot without ulceration that is capable of weight-bearing in an orthosis. Thompson and Clohisy[14] found that reconstruction failures occurred in 75% of the patients if ulcers were present at the time of surgery. Pseudarthrosis is not necessarily representative of treatment failure if the above-mentioned goal is attained, since it is usually not painful in this situation.

Amputation

Amputation should not be viewed as evidence of treatment failure, but rather as a reconstructive procedure, the goal of which is to regain energy-efficient ambulation. Limb salvage by means of a distal amputation has become an important issue because of the systemic nature of the disease. In 49% of patients with diabetic foot infections, a similar infection will develop in the contralateral foot within 18 months.[11] Diffuse involvement and excessive weight-bearing render the uninvolved limb particularly vulnerable to the complications of diabetes.

In a case-control study, Reiber et al[15] listed the following risk factors for amputation: insufficient mean below-knee and foot cutaneous circulation; ankle-arm blood pressure index less than 0.45; absence of lower-leg vibratory perception; a high-density lipoprotein subfraction 3 level less than 0.7 μmol/L; and no previous outpatient diabetes education. Many amputations are preventable through proper patient education.

Pecoraro et al[16] described causal pathways to amputation. A traumatic event causing tissue injury was identified in 73% of their cases. Shoe-related ulceration secondary to repetitive pressure was found in 36% of the cases. This underscores the importance of appropriate protective footwear, prescribed and monitored by a knowledgeable health-care provider. Furthermore, early and active involvement of a vascular surgeon may help maintain circulation to the distal extremity, so that if amputation is necessary, it can be done at the most distal functional level.

It is beyond the scope of this article to describe specific amputation procedures, but certain concepts should be applied to all amputation surgery in the foot. In selecting the amputation site, preoperative assessment is prognostic of success. Generally, noninvasive assessment using either $TcPO_2$ or Doppler pressure and pulse-volume recordings should allow accurate prediction of amputation healing with a less than 10% revision rate. A $TcPO_2$ level greater than 30 mm Hg and a great-toe pressure greater than 45 mm Hg have been associated with a healing rate of approximately 90%.

Surgical technique should keep trauma to the soft tissues and skin to a minimum. Resection back to bleeding, viable bone and soft tissue is the first step. Once all nonviable tissue has been removed, the surgeon can begin fashioning the flaps that will allow a tension-free closure. Digital, ray, transmetatarsal, and below-knee

amputations are the procedures most commonly performed. Midfoot and Syme amputations are desirable alternatives to below-knee amputations in some patients.

Although achieving a healed wound is the primary short-term goal, creation of a plantigrade, durable residual limb must be the long-term goal. Midfoot amputations are not contraindicated as long as a balanced foot can be maintained with selective tenotomy and/or tendon transfer. Skin grafts and flaps can be helpful; however, they are insensate and not very durable when placed directly over a weight-bearing bony prominence. When used in areas of minimal weight-bearing, such as the arch or dorsum of the foot, they can be a helpful adjunct.

Salvaging the foot can help preserve ambulation and may allow walking without upper-extremity aids that might be necessary if a higher amputation were done. If the salvaged foot is poorly functional and does not provide a durable weight-bearing platform, the patient may be better served by a below-knee amputation and prosthetic replacement. The amputation level is directly related to efficiency of ambulation in diabetic patients due to the often associated comorbidities of heart disease and peripheral vascular disease. When confronting amputation in the foot, the surgeon must not lose sight of the final goal of energy-efficient ambulation.

Summary

Diabetes mellitus is a systemic disease affecting more than 10 million persons in the United States. Increased longevity has accompanied the advances in the metabolic management of this disease. As patients survive longer, the musculoskeletal complications of diabetes will be increasingly encountered by the orthopaedist. Management of these problems requires a broad, multidisciplinary team approach that addresses the vascular, nervous, skeletal, immune, and integumentary systems. Evaluating the patient with a foot problem requires attention to each of these areas.

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