Review Paper

Diabetic foot syndrome

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Abstract

The diabetic foot syndrome encompasses a spectrum of presentations ranging from an uninfected neuropathic ulcer to a swollen infected gangrenous foot with limb and life threat. The pathogenesis comprises a complex interplay of neuropathy, ischaemia and infection coupled with impaired host defence mechanisms in the diabetic. The misconception that ischaemia is due to untreatable, occlusive microvascular disease has generated a nilhistic attitude and bred the assumption that major amputation with resultant limb loss is always the eventual outcome. Arterial bypass procedures can restore pulsatile blood flow and together with meticulous foot care and aggresive surgical control of sepsis can salvage the limb that would otherwise have been lost.

Key Words: diabetic foot; microvascular disease

Introduction

The Diabetic Foot Syndrome is a term used to describe infection, ulceration and gangrene that occurs in the feet of people with diabetes. It is one of the most debilitating conditions of diabetes and occurs both in insulin and non-insulin dependent diabetes. Diabetic foot problems range from a punched out neuropathic ulcer under the metatarsal head with minimal infection to a swollen infected ischaemic foot with underlying osteomyelitis, limb threat, and possible life threat. The pathogenesis frequently is due to a combination of neuropathy, ischaemia, and infection. Fundamental to the management of the diabetic foot syndrome should be the understanding of the misconception that untreatable microvascular disease is responsible, and its sequela of generating a nilhistic attitude towards the condition. Patient and health professional education, painstaking foot care, aggressive management of foot sepsis, sophisticated arteriographic techniques and meticulous femorodistal arterial bypass revascularisation procedures have all led to a decreased amputation rate in diabetics. This paper attempts to review the multifactorial pathogenesis and multidisciplinary management of the condition.

Pathogenesis

The three pathogenetic mechanisms involved in the diabetic foot syndrome are neuropathy, infection and ischaemia. Seldom does each work in isolation. Rather, most foot problems result from a complex interplay among all three and possibly other factors such as altered foot pressures, limited joint mobility, glycaemic control, and ethnic background (Shaw & Boulton, 1997).

Peripheral Neuropathy

Changes of neuropathy may occur early. In an adolescent group with a mean duration of diabetes of 6 - 8 years, 29.5% and 28.4% were found to have at least one abnormal autonomic and peripheral nerve test respectively (Donaghue *et al.*, 1996). The causes of peripheral neuropathy are not completely understood but vascular and metabolic factors have been implicated. Sorbitol, produced by the increased metabolism of glucose as a result of chronic hyperglycaemia, accumulates in nerves and possibly results in increased endoneural tissue pressure with subsequent reduced blood flow in the vasanevorum and axonal ischaemia (Myers *et al.*, 1986). A defect in schwann cell metabolism resulting in delayed nerve conduction is also believed to be responsible (Chopra *et al.*, 1969) Other mechanisms implicated are decreased nerve myoinositol, reduced Na^+ / K^+ ATPase activity, microangiopathy, genetic and environmental factors (Shami & Scurr, 1994). Sensory, motor and autonomic nerves can be affected with their respective resultant sequel which at times act solely, but frequently act in synergy in the pathogenesis of the diabetic foot syndrome.

Sensory Neuropathy

Diminished sensation and lost of protective pain results in minor injuries that occur unnoticed, from ill fitting foot wear and penetrating or thermal injury. Diminished sensation may allow patients to tolerate ill fitting shoes, and diminished postural and joint position sense may interfere with normal gait and function of the foot thence exacerbating mechanical stresses and injury.

Motor Neuropathy

Motor neuropathy results in weakness of the intrinsic muscles of the foot and subsequent imbalance between long extensors and flexors of the toes. As a consequence deformities like high medical arch (pes cavus), hammer toes, subluxation of the metatarso-phalangeal joint and proximal migration of the metatarsal fat pads come about. The subsequent reduction in weight bearing area and loss of subcutaneous fat pads results in increased pressure, borne by areas overlying the metatarsal heads. This leads to hyperkerotosis and callus formation which itself is highly predictive of subsequent ulceration (Murray *et al.*, 1996). As a result the skin becomes less pliable, sheer stresses between it and the underlying bone results in cavitation and microhaemorrhage, which can be detected under the skin by MRI (Brash *et al.*, 1996). As the sheer forces continue the cavity becomes larger until it finally "button holes" out, displaying the full extent of the damage as an ulcer, when the callus and skin overlying the cavity is removed (Shami & Scurr, 1994).

Autonomic Neuropathy

Autonomic neuropathy from which 21.6% of diabetics suffer from results in loss of vasomotor control (Guy *et al.*, 1985), loss of sudomotor function and blunting of the inflammatory axon reflex. Loss of vasomotor control increases blood flow to the extremity, but much of this flow is channeled through skin and bone AV shunts such that other tissues may experience hypoperfusion (Watkins, 1992). Loss of the veno arterial reflex results in increased capillary pressure on standing, leading to chronic capillary hypertension (Watkins, 1992).

Lost of sudomotor function with resultant decreased sweating renders the skin less supple, predisposing to fissuring and breakdown of the skin envelope, possibly facilitating the entry of micro-organisms (Shami & Scurr, 1994). Lost of the axon teflex results in a diminished capacity to mount an inflammatory responsive thereby rendering a possible propensity to infection (LoGerfo,1995). Increased blood flow through adjacent bone may result in subsequent diabetic osteopathy. Autonomic neuropathy may also contribute to the development of medial calcific stenosis (Mueller *et al.*, 1994).

Infection

Although it is commonly believed that infections in general, are more common in diabetics, data supporting this remains controversial (Towne, 1995). Diabetics do however have a higher incidence of foot infections and impaired wound healing (Lipsky *et al.*, 1990). Autonomic neuropathy with subsequent impaired sudomotor function and blunted inflammatory reflex may be contributory.

Vascular occlusive disease with resultant decrease supply of phagocytes and diminished tissue oxygen tension has been implicated. Hyperglycaemia causing impairment of collagen synthesis and degradation, and impairment of leukocyte chemotaxis and phagocytosis is also thought to be contributory (Lipsky *et al.*, 1990).

Infection adversely affects diabetic control and uncontrolled diabetes adversely affects infection (Gibbons *et al.*, 1989). An infected ulcer can predispose to additional tissue loss because of increased metabolism which exceeds blood supply. Moreover inflammatory cells and some bacteria can secrete substances that act as procoangulants causing small vessel thrombosis with resultant worsening ischaemia and thence exacerbating infection (Marek & Krupski, 1995).

Ischaemia

Peripheral vascular disease is 20 times more common and 50% of diabetics develop this 10 years after the onset of diabetes (Gibbons *et al.*, 1995). Operative treatment of ischaemia in the diabetic was limited by the misconception that occlusive microvascular disease was responsible. This was due to light microscopy studies, where PAS positive material, thought to be due to endothelial proliferation, was found more prevalent in the arterioles of diabetics (Goldenberg *et al.*, 1959). Subsequent blind histology (Strandness *et al.*, 1964) and arterial casting (Conrad, 1967) studies have refuted the existence of this occlusive arteriosclerotic lesion, at one stage, thought, specific to diabetes (Towne, 1995).

Also physiologic evidence of increased and fixed peripheral vascular resistance in diabetic extremities, expected of this occlusive microvascular disease is lacking. No difference in vasomotor response between diabetic and non-diabetic controls have been found in the runoff bed after direct vasodilator (papaverine) injection at the completion of femoral-popliteal bypass (Barner *et al.*, 1971), and after experimentally induced reactive hyperaemia (Irwin *et al.*, 1988). Further, if an occlusive lesion existed in the microcirculation it would mitigate against the effects of arterial reconstruction (Mueller *et al.*, 1994).

Although now held to be non-occlusive, microvascular changes are present and may possibly be contributory in the pathogenesis of the diabetic foot. Electron microscopy studies have shown this PAS positive material identified in the arterioles on light microscopy to be due to basement membrane thickening of muscle capillaries (Siperstein *et al.*, 1968), and to be non-occlusive. It was approximately twice the thickeness that of non-diabetics and occurred in 98% of diabetics and in 50% of people with a diabetic genetic predisposition but whom had not yet had any manifestation of diabetes, suggesting that the thickening was an early lesion. The thickening was found to be increased in accordance with the duration of diabetes, suggesting a possible association with carbohydrate intolerance, but found not to be related to the severity of regulation (Siperstein *et al.*, 1968). In another study, it has been found in

88% of diabetics as compared to 13% in non-diabetics, patchy in distribution and to increase distally (Banson & Lacy, 1964).

Although non-occlusive, observed with an equivalent and sometimes increased capillary luminal diameter, and with unimpaired oxygen diffusion across it, this thickened basement membrane may impede the diffusion of nutrients and limit movement of leukocytes into areas of infection (Banson & Lacy, 1964). The flux of highly charged particles, most notably albumin out of the capillaries and into the interstitium is increased (Parving & Rasmussen, 1973). These changes could contribute to the susceptibility to impaired wound healing and infection.

The atherosclerotic lesions of diabetics are similar, histologically to that of non-diabetics. In diabetics the tibioperoneal arteries are more frequently involved with relative sparing of the more proximal, and the pedal vessels distally. This has been demonstrated by anatomical studies (Strandness *et al.*, 1964; Conrad, 1967) and detailed arteriographic evaluations (Menzoian *et al.*, 1989). This distribution possibly explains the early disappointing results of standard femoropopliteal reconstructions in diabetics and have prompted development of techniques for distal and infra-malleolar bypass grafting. Artherosclerosis occurs more commonly (Brand *et al*, 1989) and, is more rapidly progressive (Beach *et al.*, 1988) in the diabetic. Reasons for this are not entirely clear and several factors have been implicated.

Hyperglycaemia has been shown to be an independent risk factor in atherogenesis possibly due to abnormal glycosylation of the vessel wall leading to increase adherence of monocytes and macrophages, resulting in local accumulation of vasoactive substances, increased endothelial proliferation and vessel wall damage by free radicals (Brownlee *et al.*, 1987).

In diabetes, lipoprotein lipase (LPL), (which catabolises VLDL and chylomicrons into hepatically consumed remnants), activity falls. LPL substrates accumulate leading to a lipaemic picture of increased tryglycerides and decreased HDL. Tryglycerides in the peripheral circulation have a stronger association with artherogenesis than cholesterol, LDL or HDL (Seeger *et al.*, 1989). Lipid normalitisation may prevent or reverse early atherosclerotic changes (Olsson *et al.*, 1991).

Increased levels of free radicals (superoxides, hydroxyl radicals and peroxides), which are produced by macrophages and monocytes upon activation, and which can cause vascular damage (McCord, 1985) are found in diabetics (Jennings *et al.*, 1987). Diabetics are also less able to prevent oxidative damage by free radicals owing to reduced levels of ascorbic acid, a free radicle scavenger (Jennings *et al.*, 1987).

Elevated serum fibrinogen, a well established cardiovascular risk factor (Kannel *et al.* 1990) as well as clotting factors V, VII, X and VIII are found in diabetics. These increased clotting factors with, increased platelet aggregation and thromboxane synthesis and the increased serum viscosity found in diabetics may play a role in increased artherogenesis (Mueller *et al.*, 1994).

Hyperinsulinaemia, reduced physical activity, obesity and genetic factors have also been implicated as possible causes for the increased atherogenesis in diabetics (Shami & Scurr, 1994).

Medial calcific stenosis (MCS) also called Monckenbergs atherosclerosis which involves progressive degeneration and calcification of the tunica media of muscular arteries occurs more frequently in diabetics (Chantelau *et al.*, 1990). Ultimately, affected vessels may become long rigid tubes of calcium with an easily recognisable pipe stem appearance on X-ray (Kherty, 1984). Unlike the more common intimal artherosclerosis, medial calcific stenosis does not produce an anatomical stenosis (Niskanen *et al.*, 1990). It does however, decrease vessel compliance, which, may by preventing full systolic expansion, cause a functional stenosis (Neubauer *et al.*, 1984). Clinically, MCS has been demonstrated to decrease blood flow in calf arteries and has been associated with claudication (Niskanen *et al.*, 1990).

Clinical Presentation and Management

Prevention and Foot Care

After 20 years of disease, 45% of diabetics have sensory impairment and after 10 years, 50% will develop peripheral vascular disease (Marek & Krupski, 1995). Before foot problems develop, management should commence with education about foot care and disease natural history. Those with an increased risk of amputation, namely, male, those with end organ complications, neuropathy, vascular disease, foot deformity and previous ulceration (Mayfield *et al.*, 1996) should be placed on a meticulous foot survillance programme. Patient education with respect to detailed attention to diabetic foot care have reduced amputation rates by 33% (Reiber, 1992). Several large clinical centres have reduced the rate of amputation in diabetics by 44% to 85% after the implementation of educational and foot care programs (Marek & Krupski, 1995).

Therapeutic footwear protects the diabetic foot (Mueller *et al.*, 1997). New shoes preferably leather, as it more easily conforms to foot shape should not be worn for more than 2 hours at a time. During the day, shoes should be rotated after 2 hours to a different type in order to rotate the areas of high pressure concentration on the foot. Shoes should fit well to prevent movement within them, and should be equipped with appropriate insoles for added protection and to even out pressure distribution. Shoes and socks should be inspected for rough linings and foreign bodies that could cause injury. Socks should be checked for wrinkles and, mended socks should not be worn. Heavy cotton or woollen material provides better protection than nylon. The feet should be washed with gentle soap and

water and lanolizing lotions applied if dry.

Management Principles

Priorities in the management of diabetic foot are, the control of sepsis, the elimination of limb and life threat, the determination of the need for, and the feasibility of surgical revascularisation, and, psychosocial support towards illness acceptance and possible amputation. Factors which may possibly exacerbate the diabetic foot syndrome like congestive cardiac failure, oedema and nutritional deficiences should also be addressed (Gibbons *et al.*, 1995).

Clinically, diabetic ulcers can be classified as "simple" ulcer without limb threat or "complicated" with limb threat. The simple ulcer with no immediate limb threat, is superficial, less than 2 cm in diameter, does not communicate with tendon, joint or bone, is not associated with cellulitis and occurs in a reliable patient who has no systemic signs of sepsis and possesses good home and/or community support. This can be treated and investigated in the outpatients clinic with total contact casting and meticulous follow-up. Should there be no improvement after 24-48 hours they should be admitted for hospital inpatient treatment. (Gibbons *et al.*, 1995).

A complicated ulcer with limb threat, is larger than 2 cm in diameter, appears deep, is associated with accompanying cellulitis, communicates with tendon, joint or bone, occurs in an unreliable patient who does not have a good home and/or community support and exhibits systemic signs of sepsis. Frequently diabetic patients do not manifest symptoms and signs of sepsis because of the blunted inflammatory response and may only present with uncontrolled hyperglycaemia. These patients should be hospitalised for bed rest, diabetic control with insulin, broad spectrum intravenous antibiotics, superficial and deep bacteriological cultures, debridement to control sepsis, and evaluation with respect to the need for, and the feasibility of revascularisation. (Gibbons *et al.*, 1995).

Many ulcers can heal with local therapy. An aggressive clinical wound care protocol including the use of surgical debridement, arterial bypass, prosthetic orthotics and platelet derived growth factor has been shown to heal up to 75% of ulcers judged by referring physicians to call for amputation (Reiber, 1992).

Although presentation varies according to the predominant pathology and the extent of the disease process, frequently multiple factors are in operation. Pecacaro *et al.* (1990) in analysing the aetiology of diabetic amputations, found that 85% were attributed to ulceration, 81% to minor trauma, 81% to faulty wound healing, 61% to neuropathy, 59% to infection and 46% to ischaemia.

Neuropathic Ulcer

A purely neuropathic ulcer typically occurs on the plantar sur face of the foot overlying a prominent metatarsal head, is usually deeply punched out with surrounding heavy callus, and is usually painless. If infection can be excluded and blood supply found adequate for healing, conservative treatment and possibly conservative foot surgery can be tried.

Ischaemic Ulcer

An ischaemic ulcer is usually found on the digits secondary to, trauma incurred during clipping of the toe nails or from pressure of ill-fitting shoes. It can occur on the heel in a bed-ridded patient. The management includes arterial evaluation and revascularisation if feasible. Otherwise conservative debridement and amputation should be considered.

Peripheral Digital Gangrene

Diabetics frequently present with ischaemic gangrene rather than intermittent claudication and rest pain because of coexistent peripheral neuropathy (Menzoian *et al.*, 1989). Some of these will require revascularisation while those who have pedal pulses and warm feet (the minority), a conservative amputation or debridement procedure will heal without revascularisation.

Infected Ulcers

Because of the blunted inflammatory response, infected ulcers are sometimes difficult to diagnose. A trial of strict bed rest without antibiotics for 48 hours may settle an inflammatory arthropathy (LoGerfo *et al.*, 1995) but not an infected foot. Superficial wound cultures from the ulcer or sinus tracts are inaccurate and correlate with deep wound cultures in only 25% of the time because of wound colonisation (Wheat *et al.*, 1986). Specimens obtained with a needle or wound curette from deeper tissue are required to diagnose becterial infection in the absence of abscesses or pockets of subfascial pus (Towne, 1995).

The majority of diabetics with limb threatening foot sepsis have polymicrobial infections, with over 90% culturing gram positive bacteria, 50% gram negative bacteria, and 50% to 70% culturing anaerobes (Lipsky *et al,* 1990). The polymicrobial nature of these infections predisposes to the development of rapidly progressive and often synergistic gangrene (Williams & Hutchinson, 1974).

When infection is suspected, antibiotics should be commenced immediately and should be of broad spectrum and with cover against anaerobes. Should there be any abscesses, pockets of subfascial pus and nonviable infected tissue, surgical debridement must be performed expeditiously.

Osteomyelitis

Osteomyelitis is not uncommon and is particularly serious. It carries a poor prognosis and even with long term medical therapy patients eventually require am-

putations of affected bones (Marek & Krupski, 1995). Particular attention must be paid to the establishment of, or the exclusion of the condition.

The diagnosis or exclusion of osteomyelitis is difficult. A history of long standing ulceration with associated swelling and erythema arouses suspicion. Some have systemic manifestations of infection although this is uncommon.

Clinical features found to correlate with osteomyelitis are an ulcer size greater than 2 cm and an ulcer communicating with joint or bone on probing (Levin, 1992). Strict bed rest without antibiotics may result in improvement of Charcots Osteoarthropathy but not osteomyelitis. Plain X-ray may only depict radiological evidence only after 10 to 14 days of infection. X-rays must be interpreted carefully as, diabetic osteopathy, a bone resorptive process due to autonomic neuropathy closely resembles osteomyelitis on X-ray (Marek & Krupski, 1995). X-rays and bone scans may depict features of osteomyelitis but when negative, do not exclude the condition. Both have been found to correlate poorly with positive bone cultures (Shults *et al.*, 1989). Sinograms may detect the communication of an ulcer with bone or the joint space (Towne, 1995). Radionuclide bone scans may not differentiate osteomyelitis from other causes of increased bone metabolism, or increased vascularity such as cellulitis, neuropathic osteoarthropathy and a soft tissue abscess and its cavity. The accuracy of MRI has been encouraging but is by no means absolute (Cook *et al.*, 1996). It cannot differentiate acutely evolving diabetic osteoathropathy from osteomyelitis (Marcus *et al.*, 1996).

Septic Foot

The septic foot is the most dreaded component of the diabetic foot syndrome and results from a combination of vascular insufficiency with peripheral neuropathy which allows the infection to progress without any significant local symptoms. Patients often present with sepsis, uncontrolled diabetes and coexistent congestive cardiac failure. It must be recognised and treated as a surgical emergency with cardiovascular and blood glucose stabilisation. Surgical options include a guillotine amputation of the forefoot in the presence of the diffuse arterial disease and an ischaemic hindfoot, and foot salvage, if the hindfoot is warm and capillary filling prompt. After sepsis is controlled and the life threat "eliminated", healing potential and feasibility of revascularisation can be addressed.

Non-Invasive Investigations

Although tissue loss in the diabetic foot is frequently non-vascular in origin, because healing calls for the restoration of pulsatile blood flow (Gibbons *et al.*, 1995), revascularisation should be considered when possible. Noninvasive vascular assessment techniques are useful in estimating the extent of occlusive vascular disease and in the assessing of healing potential. However they have certain diabetic specific limitations due to the incompressibility of calcified vessels and difficulty in applying pressure cuffs around the ulcer bearing infected necrotic tissues. Thus no patient should be denied revascularisation or conservative foot amputation on the basis of noninvasive vascular investigations.

Substantial calcification in the small and medium size arteries with consequent vessel incompressibility results in spuriously high segmental ankle pressures, rendering ankle pressures unreliable in the assessment of healing potential. However segmental ankle pressures have been found reliable in predicting non-healing. Only 8% of extremity wounds healed when ankle pressures were less than 40mmHg and none healed were less than 30mmHg (Apelqvist *et al.*, 1989).

Less calcification in the pedal and digital vessels enable toe pressures to be measured more accurately and be more reliable in the assessment of healing potential (Bone & Pomajzl, 1981; Gibbons *et al.*, 1979). In general 85% to 100% of foot lesions will heal when toe pressures are > 40mmHg whereas fewer than 10% will heal if toe pressures are < 20 mmHg (Marek & Krupski, 1995).

Plethysmographic wave forms, when pulsatile, predict healing. All below knee amputations healed when pulsatile waveforms were obtained on pletyhsmographic tracings and 90% of foot lesions healed when there were pulsatile transmetatarsal tracings (Raines *et al.*, 1976). However, when there are no wave forms, more than 50% of lesions healed, thus rendering only a marginal negative predictive value of the procedure (Marek & Krupski, 1995).

Transcutaneous oxygen pressure (T_cO_2) values of less than 10mmHg correlate with nonhealing while when greater than 30mmHg predicted healing after major or minor amputation (Bunt *et al.*, 1996). However, measurement is time consuming, requires an experienced technician and may vary depending on measurement site.

Laser Doppler velocimetry, a measure of skin blood flow velocity and pulse wave amplitude have been found to be a reliable predictor of healing (Karanfilian *et al.*, 1986).

Arteriography

In the presence of tissue loss, intra-arterial digital subtraction angiography (IADSA) should always be performed in the investigation of whether arterial bypass revascularisation can be performed. Occassionally even when no runoff can be identified by IADSA surgical exploration may find the occlusion, treatable by reconstruction.

Vascular Bypass Reconstruction

With current arteriographic techniques more than 90% of diabetic patients with ischaemic foot lesions have been

found to have surgically correctable obstructive arterial lesions. A 10 year review of 330 threatened diabetic limbs found only 2.7% with distal ischaemia so severe as to necessitate primary amputation (Taylor & Porter, 1992). Although it is widely held that diabetic patients have increased surgical risk and relative diminished long term survival, some recent studies have not confirmed this (Gibbons *et al.*, 1995).

Results of peripheral bypass procedures in diabetics have been equivalent to and sometimes even better than that of non-diabetics (LoGerfo *et al.*, 1995). Complication and morbidity rates have been found comparable to diabetics undergoing amputation and non-diabetics undergoing revascularisation.

Patency rates in a series of in-situ femoral distal bypass were found to be of no significant difference between diabetics and nondiabetics (Levine *et al.*, 1985).

Five year limb salvage rates for femorodistal popliteal autologus vein bypass grafts of diabetics (44%) was found compared to that of non-diabetics (51.4%) (Reichle *et al*, 1979). However in the 10 yeat follow-up period, the diabetic patients did slightly less well.

Mortality and perioperative complication rates in a series of 321 patients undergoing infra-inguinal bypass reconstruction for limb salvage was 2.2% and 9.7% respectively. These rates were similar to those, of diabetic patients undergoing major amputation (Taylor & Porter, 1992).

Five year survival rates for diabetics undergoing a primary or revision bypass procedure for limb salvage was equivalent to that of in a concomittant non-diabetic group (Taylor & Porter, 1992).

Even in the presence of foot sepsis, a 92% graft patency rate at 36 months has been achieved (Tannenbaum *et al.*, 1992). Although until recently, not normal practice (Mueller *et al.*, 1994), Chang *et al.* (1996), have recently advocated urgent revascularisation with an autogenous conduit in the presence of foot infections within 48 hours of hospital admission and teported high rate of limb salvage.

Recognition of the diabetics propensity to tibioperoneal disease have led to the development and frequent utilisation of the dorsalis pedis bypass graft technique. In patients undergoing this procedure for limb salvage, Pomposelli *et al.* (1991), have reported a decrease of amputations at all levels, patency rates of 87% and limb salvage rates of 92% at 3 years.

Reasons for the equally good or better patency rates of paramalleolar bypass grafting in the diabetic are not entirely clear but could possibly be due to the earlier age when bypass is required, the quality of the venous conduit (Plecha *et al.*, 1996) and possibly relative lack of disease in the inflow and outflow arteries.

Percutaneous Balloon Transluminal Angioplasty (PTA)

PTA is a reasonably effective theraputic technique for treatment of isolated short stenosis in the aorto-iliac system and femoropopliteal system. It is feasible in a large percentage of diabetic subjects with occlusive disease and foot ulcers, and may be practicable in the popliteal artery and branches (Faglia *et al.*, 1996). It has been used to successfully, salvage diabetic limbs otherwise doomed to amputation (Hanna *et al.*, 1997). However, the effectiveness of PTA in diabetes, is not as good, possibly due to the predominant distal tibial disease and heavy calcification of the tibial vessels (Mueller *et al.*, 1994).

Lumbar Sympathectomy

Lumbar sympathectomy has limited success in the diabetic possibly due to autosympathetomy already having taken place and already rendered its effect. The cumulative success rate of lumbar sympathectomy in diabetics was found to be 19.2% compared with 51% success rate for a general atherosclerotic population (DaValle *et al.*, 1981).

Foot Sparing Surgery

When conservative measures do not result in improvement of foot ulcers, foot sparing surgery may be considered before cellulitis or deep spaced infection develop. These should be elective procedures in neuropathic patients who have normal arterial flow or patients who have undergone peripheral bypass revascularisation and have had sepsis controlled.

Advantages of foot preservation surgery are threefold. Firstly, forces that traverse the foot during walking are dissipated over a maximal area if as much distal tissue as possible is preserved. Secondly, ease of fitting and wearing shoes when the foot remains intact and custom made shoes may not be a necessity when some foot anatomy is preserved. Thirdly, it is better accepted by patients who are often terrified of having an amputation.

Procedures performed include ulcer excision, toe arthroplasty, metatarsal osteotomy, metatarsal phalangeal joint resection, metatarsal head resection and panmetatarsal head resection.

An excellent primary healing rate has been reported after metatarsal head resection (Martin *et al.*,1990). Although these procedures may be less ablative than amputations, ulceration may redevelop due to the resultant alteration of foot biomechanics (Gibbons, 1995). Currently these procedures are not widely performed.

Successful microvascular free tissue transfer has been reported as a reconstructive strategy after conservative excisional surgery of the diabetic foot (Armstrong *et al.*, 1997). The feasibility of this procedure as standard treatment is yet to be evaluated.

Summary

Diabetic foot problems utilise more hospital bed days than any other complication of diabetes. Fundamental to the management should be, the understanding that

the aetiology is multifactorial, patient education and meticulous prophylactic foot care have decreased amputation rate. Although microvascular changes are present they are non-occlusive. Surgical bypass revascularisation coupled with limb salvage foot sparing surgery have resulted in a decrease in major amputation rate.

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