Management of diabetic foot ulcers

Diabetic foot disorders are the rated as the number one cause of hospitalisation for diabetic mellitus patients in the United States and abroad. Among these disorders are, foot infection, ulcerations, cellulitis and gangrene. It is estimated that a 100 people per week lose a digit, foot or a lower limb due to diabetes mellitus8. Diabetic foot disorders and its long term complications account for direct medical budget of hundreds of millions of dollars annually, including lengthy hospital stay of patients and lengthened periods of disability. In the UK, diabetes cost the NHS over £5 million pounds per annum and the cost of diabetic foot complications including amputations was £252 million in 2003. Figures show that 1 in 10 foot ulcers result in the amputation of a foot or a leg. The most distinguishing foot complication of diabetes is the ulcer, which is of course a major risk factor for amputation. Mortality rates after amputation are as high as 50 percent in diabetic patients. Although the primary pathogenesis is neuropathy, immunopathy and vasculopathy (ischemia), diabetic foot ulcer is attributed to a number of other risk factors. Early detection of these risk factors as well as the foot ulcers themselves is crucial in the general management of diabetic foot ulcers and amputation prevention. Therefore, immediate and aggressive treatment of all diabetic foot ulcers can prevent worsening of the complication and the need for amputation. The objective of treatment as a result should be prompt intervention to allow good healing of the ulcer and once healed, to prevent its recurrence. However, the optimum care for foot ulcers rest upon the treatment teams' understanding of the pathophysiology associated with diabetic foot ulcers, familiarity with current methods of treatment and the concept that the multidisciplinary team

approach is the gold standard in preventing limb amputation. And above all, prevention of foot lesions should be ranked highest amongst all priorities.

EPIDERMIOLOGY There is no concrete data illustrating the true picture of the incidence and prevalence of diabetic foot ulcers. However, majority of the information gathered about its aetiology is based upon national hospital discharge survey (NHDS) data, which does not include a vast number of patients with ulcerations treated on the outpatient basis. Cross-sectional and population-based studies help to approximate the distribution and frequency of diabetic foot ulcers, while prospective cohort studies and retrospective case-control studies are instrumental in detecting associated risk factors for the foot lesions. According to Reiber et al’s epidemiological review, chronic ulcers represented 2. 7% of all diabetes related admissions and 46% of all admissions due to any ulcer condition. The 1983-1990 NHDS survey also revealed that the highest ulcer rates were found in individuals aged 45-64 years, with male preponderance over the female. The average length of stay (LOS) for diabetes patients discharged with ulcers was 59% more than in patients without ulcers, around 14 and 8 days, respectively. Numerous population-based studies record a yearly incidence of diabetic foot ulcers in the magnitude of 2% – 3% in both type 1 diabetes mellitus (IDDM) and type 2 diabetes mellitus (NIDDM) patients, while the prevalence ranges between 4% and 10%. These studies also suggest a widespread trend for higher prevalence of ulcerations with increasing age and duration of diabetes. Once ulcerations have occurred, recurrence rate can get to 50% in 2 years and 70% in 5 years. Despite the fact that mortality linked with diabetic foot ulcerations has not been recorded, 70% to 80% of amputations of the lower

extremity can be lead by foot ulcers. Amputation is one of the most common sequels in persons with diabetes mellitus now exceeds 100, 000 per year in the United States and amputations involving the leg, foot or toe(s) are not uncommon. Approximately 15% of patients with diabetic foot ulceration will require amputations due to uncontrolled infection, gangrene or failure of the ulcers to heal. Unfortunately, there is a 3-year survival rate of 50% after the amputation of a lower limb, while the 5-year survival rate is approximately 40%. Subsequently, less than half of the patients who end up with lower extremity amputation (LEA) of one limb go on to develop a severe contralateral ulcer within a space of 2 years, thereby putting the future of the other limb at risk as well.

AETIOLOGY/PATHOPHYSIOLOGY

Several factors have been implicated in association with diabetic foot ulcers. These factors include the intrinsic complications of diabetes mellitus in affiliation with some extrinsic factors and together predispose the diabetic patient to the risk of developing foot lesions. The trilogy of peripheral neuropathy, peripheral arterial disease (ischemia) and susceptibility to infection (immunopathy) are the main predisposing factors for lesions on the foot. The impact of peripheral neuropathy may not be easily detectable, with little or no signs and symptoms. Notwithstanding, its pathology advances fast and the end stage of tissue necrosis quickly reached. Distal sensory neuropathy can be seen in 20% to 50% of patients with type 2 diabetes. This decreases the protective sensation in the feet, leading to abnormal spreading of foot pressure and shear stresses with subsequent callus formation. This in turn diminishes the patient’s ability to perceive minor

trauma to the foot and this is demonstrable by vibration perception threshold (VPT) and insensitivity to a 10g monofilament, which conveys a 7-fold and 18-fold risk of foot ulceration respectively.

Distal motor neuropathy precedes atrophy of the intrinsic and extrinsic musculature of the foot, with accompanying deformities of the toes and metatarsals heads on the plantar aspects of both feet and consequent bunions on the 1st and 5th metatarsal-phalangeal joints. However, these irregularities of the feet cause an increase in the foot pressures, particularly around the bony prominences, thus resulting in more calluses forming. These calluses then go on to further increase the local subcutaneous pressure, ultimately resulting into haemorrhage beneath the callus, a lesion known as the “ pre-ulcer” is then formed. With progressive pressure mounting on the pre-ulcer, the overriding skin breaks down to produce an ulcer.

In addition, distal autonomic neuropathy could as well spark a plantar ulcer directly by reducing sweating in the feet with consequent drying and cracking of the skin28. Peripheral arterial disease and impaired cutaneous circulation are also important risk factors for both ulcerations and LEA. Peripheral arterial ischemia sometimes produces ischemic ulcers, but these are rare occurrences i. e. (1% – 2% incidence) than neuropathic ulcers (65%) or combined neuroischaemic ulcers (25%) 28. Transcutaneous oxygen tension (Tc pO2) levels less than 30mmHg and absence of peripheral pulses or past history of vascular surgery are strong separate predictors of ulceration.

Diabetic foot infections often set in and complicate already settled foot ulcers. Although, infections play an integral part in the pathway to lower limb amputation, there is inconclusive data with regards to the position of susceptibility to infection in causing ulceration. Even though, most ulcers are caused by minor foot trauma, and in some cases the patient takes no notice of because of the sensory neuropathy. These minor injuries (i. e. extrinsic factors which include; wearing ill-fitting/brand new shoes, hot soaks occupational hazards and to a lesser extent self-induced trauma by cutting toe nails or calluses) constitute the leading cause of acute precipitant of diabetic foot ulcers. In addition, there are also a number of intrinsic factors which could predispose diabetics to developing foot ulcers and they include; longstanding diabetes, past history of ulcers or amputation, age, weight, retinopathy, nephropathy and structural deformities of the foot (i. e. Charcot foot) have all been associated as risk factors for ulcerations. However, bad biomechanical function arising from the complications of diabetes generally leads to foot injuries in most diabetic patient.

ASSESSMENT OF DIABETIC FOOT ULCERS

A detailed and well organised evaluation of the lower extremities is crucial when commencing the treatment of a diabetic foot ulcer. Before carrying out the physical examination of the limbs, it is noteworthy to perform a quick inspection of the patient’s shoes for good fit, foreign objects and the wear and tear patterns. The clinical evaluation must include an appropriate assessment of the ulcer’s aetiology, its extent and depth, presence and severity of both local and systemic infection and peripheral vascular status.

A comprehensive assessment of the patient’s general health and glycaemic control, extent of peripheral neuropathy, a careful, yet detailed dermatologic and musculoskeletal examination should also be included in the evaluation. These assessments determine the ulcer’s healing rate, potential progression to LEA, and the likelihood of reoccurrence. Therefore, they should be accomplished urgently in the ambulatory or hospital setting and require a multidisciplinary team approach, with possible consults to the infectious disease specialist, podiatry, vascular and orthopaedic surgeons. Bilateral lower limb pulses must be examined. When pulses are diminished or not palpable, Doppler segmental pressures to the toes or TcpO2 measurement are indicated and the vascular experts should be brought on broad. The neurological evaluation should assess the patient’s sensorium and deep tendon reflexes. The ankle and knee reflexes are tested with the aid of a simple neurological hammer, while the important aspects in the evaluation of the sensorium are: reduced sensation to pain, light touch, hot/cold and vibratory sensation. Pain sensation is easily assessed with a disposable needle. A piece of cotton ball, lamb’s wool or 10-g monofilament can be used to evaluate the light touch and a 128-Hz or 512-Hz tuning fork or biothesiometer are approved for vibratory evaluation. Cold perception is also assessed by submerging the metal arm of a neurological hammer into cold water and then placing it against the patient’s skin.

Anatomical deformities such as hammertoes, previous foot amputation, or Charcot joints often produce high pressure areas which result in ulceration. The musculoskeletal evaluation cannot be done by visual inspection of structural findings alone, it must also include testing for muscle strength,

weakness, atrophy and contracture. Assessment of joint range of movement and gait evaluation with computerised plantar pressure analysis will also be of great value in appreciating the abnormal dysfunction contributing to ulceration.

Examination of the skin of both feet is also carried out with detailed attention to the quality and integrity of the skin around the interdigital areas. Changes in the colour of the skin often associated with spotted rashes and heel fissures are suggestive of a significant level of ischemia. Toenail changes and presence of subungual drainage are pointers to a proximal source of infection.

Clinical assessment of the ulcer should include a detailed description of its appearance as well as the measurement of the ulcer’s diameter with a wound measuring guide. Outlines of the ulcer on a translucent film or plastic sheet can also promote this process. This must be documented and retraced at subsequent visits to assess the treatment process. The depth and extent of the wound should be carefully explored with a blunt sterile probe. Special care must be taken to probe for hidden sinus tracts and subcutaneous abscesses or to identify tendon, bone or muscle or joint involvement. Ulcer depth is a significant predictor of healing rate, possibility of concurrent osteomyelitis and the chances for amputation.

The presence of infection is a huge cause of the need for hospitalization. Therefore, a general assessment with physical examination, laboratory investigations and radiographic studies is important in classifying infection as absent, mild, moderate or severe. This classification acts as a guide to

determine or select the initial antibiotic therapy and to decide when to hospitalise the patient. Clinical signs of infection such as purulent discharge, odour, cellulitis, fever and leucocytosis must be documented. However, Leucocytosis and fever might not always be noticeable even in the presence of acute osteomyelitis. Approximately 54% of patients with diabetic foot infections had normal white blood cell count and no fever44. Bacteria cultures of anaerobes and aerobes (both gram positive and gram negative) should be obtained from the base of the ulcer, bone or blood or from all three depending on the clinical setting. This helps in clarifying the true hidden pathogens and may facilitate the decision to adjust initial antibiotic therapy.

Physical examination of signs of infection in the patient centers on the presence or absence of systemic responses such as fever, tachycardia, sweats or hypotension and the appearance of the wound and adjacent tissues. Early signs of infection are evident by increased amount of exudates from the wound, base of the ulcer changes from pink granulation to yellowish- grey tissue, tenderness and induration around the ulcer. Infection should be considered severe when the patient present with systemic toxicity, signs of fascilitis or a rim of erythema around the ulcer greater than 2cm in diameter.

Laboratory investigation to confirm the presence of infection should include white blood cell count and differential which could show leucocytosis or a shift to the left or both, erythrocyte sedimentation rate (ESR) which when elevated above 40mm/hr is a strong indicator of osteomyelitis28. In addition, glucose, bicarbonates and creatinine levels are tested to rule out possible

hyperglycaemia, metabolic acidosis or azotemia from dehydration which strongly suggest the presence of a severe infection.

Radiological evaluation should be obtained promptly to ascertain the presence of fractures, foreign objects or signs of osteomyelitis. Plain x-rays have a low sensitivity, thus they should be interpreted with caution as changes in the foot caused by Charcot foot could mirror those osteomyelitis when seen on a plain x-ray. However, a normal plain x-ray of the foot does not rule out osteomyelitis, a repeat should be requested 2 weeks later to exclude occult osteomyelitis. As indicated, other imaging modalities can aid in the diagnosis of osteomyelitis such as CT scans, magnetic resonance scan (MRI) or leukocyte scans and each having their own strengths and limitations.

TREATMENT

Management of the foot ulcer is mostly determined by its severity, vascularity and the presence of infection1. Recognition of its root cause will serve as a guide during the course of treatment. However, a multidisciplinary team consisting of specialist from podiatry, orthopaedic surgery, vascular surgery, the infectious disease service and diabetic education service should be involved in the management. The multidisciplinary team approach is due to the complicated nature of the disease itself as well as managing the various comorbidities associated with foot ulcers. In addition, the approach has been demonstrated in clinical trials to produce significant outcomes in terms of improvement and reduce the incidence of major amputations. The wound should be immediately relieved of all pressures, elevated and rested at first presentation. Effective local wound care must be carried out and ill-

fitting footwear should be discarded and replaced with appropriate surgical or relief shoes for protection. And in cases where total nonweightbearing with crutches is impossible, a pressure felt padding or foam can be used in the surgical shoes. However, the total contact cast (TCC) is considered gold standard to protect neuropathic ulceration during ambulation due to its ability to eliminate high pressure areas under the foot. Adequate alternatives to the TCC are the “ Scotchcast Boot” or removable walking braces. Treatment of hyperglycaemia, ketoacidosis, renal insufficiency and other comorbidities that may coincide in the ulcerated patient should be treated simultaneously with the foot lesion. Consultations to internal medicine, endocrinology and cardiology are generally frequent when managing acutely infected patient who need to be hospitalised. Such consultations are usually sought early in course of treatment to ensure good metabolic control.

Diabetic foot infections are usually polymicrobial and as such initial antibiotic therapy should be broad-spectrum after obtaining good aerobics and anaerobic culture samples. Antibiotic therapy should be later modified according to the culture and sensitivity test and the patient’s clinical response to the initial therapy. Surgical debridement and drainage or local partial amputations are crucial adjuncts to antibiotic therapy. Underlying osteomyelitis usually present in moderate to severe infections and often requires aggressive bony resection of infected bone and joints accompanied by cultured -directed antibiotics for 4 – 6 weeks.

Foot ulcer patients with underlying ischemia should undergo revascularization with angioplasty or vascular bypass procedure if it’s anatomically possible. Even with severe distal arterial obstruction,

revascularization to return pulsation to the foot is a major part of the limb-salvage strategy and may be accomplished in such patients. However, where revascularization is not feasible or in cases of advanced infection or extensive necrosis, amputation at some level may be required.

Wound care is also necessary after surgical or sharp debridement of all callus and necrotic tissue58. Practically, a warm, moist environment conducive for wound healing should be maintained. This can be arranged using saline wet/dry dressings or special dressings such as semipermeable films, hydrogels, calcium alginates and hydrocolloids. Tissue-engineering dermis is a more recent class of biologic dressing and has been tested to be more effective than saline dressings. There is little evidence to support the role of topical enzymes and should be avoided. Although the role of topical growth factors in the healing rate of ulcers is beneficial, however, they are expensive and should be limited to patients whose ulcers cease to improve after 4-6 weeks of adequate therapy.

PREVENTION OF RECURRENCE AND AMPUTATION

Prevention is regarded as a major aspect in avoiding ulcer relapse and diabetic lower limb amputation. Recurrence rate with diabetic foot ulcers and LEA are as high as 50% -70% over three years. Comprehensive intervention programs tailored to individual patients can lower these rates and can be accomplished with a multidisciplinary team approach. Control of both macrovascular and microvascular risk factors is also of great importance. Patient education and re-education plays a primary, yet active role in this program and involves instruction in foot hygiene, the need for daily inspection, proper footwear and the necessity of prompt treatment of new

lesions. In addition, regular and frequent visit to a diabetic foot care program is crucial. The feet must be thoroughly inspected at every visit and should include debridement of calluses and ingrown toenails. This provides an excellent opportunity to back up self care behaviour as well as allowing early detection of new or imminent foot problems. Appropriate therapeutic footwear with pressure-relieving insoles and high toe box which protect the high risk foot are an essential element of the prevention program and have been associated with significant reductions in ulcer development. Subsequently, patients with major structural deformities may benefit from reconstructive surgery to prevent recurrent foot ulcers. Surgery may be especially suitable in patients who cannot be accommodated in therapeutic footwear. And because patients with healed ulcers are at risk for future ulceration, these preventive measures must be integrated into a long life strategy and treatment program.

CONCLUSION

Diabetic foot ulcerations, infections, gangrene and lower extremity amputations (LEA) are major causes of disability to patients with diabetes mellitus. And these often results in extensive periods of hospitalisation, substantial morbidity and mortality. Although not all such lesions can be prevented, it is certainly possible to reduce their incidence by proper management and prevention programs. A multidisciplinary team approach to diabetic foot disorders has been regularly proven to be the best method in achieving favorable rates of limb salvage in this high risk population. Foot care programs accentuating preventive management can reduce the incidence of foot ulceration through modification of self care practices,

appropriate evaluation of risk factors and formulation of treatment protocols directed at patient education/re-education, early intervention, limb preservation and prevention of new lesions. The joint team of medical, surgical, rehabilitative and footwear specialist should impart effective and coordinated services for acutely infected or ischemic inpatients as well as management for the outgoing patients. In general, the incidence and morbidity of diabetic limb amputations can be reduced if the above principles are embraced and integrated into everyday patient management protocol.