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Review

The diabetic foot management – Recent advance

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HIGHLIGHTS

- Diabetic neuropathy and peripheral vascular disease are the main etiological factors in foot ulceration.
- Wagner's classification is one of the most widely used and universally accepted grading systems for DFU.
- Assessment of peripheral neuropathy and evaluation of peripheral arterial status are the two important investigations in a diabetic foot.
- Management of diabetic neuropathic ulcer by appropriate and timely removal of callus, control of infection and reduction of weight bearing forces.
- Management of diabetic ischaemic foot is medical management, surgical management and percutaneous transluminal angioplasty.

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ABSTRACT

Diabetic ulceration of the foot represents a major global medical, social and economic problem. It is the commonest major end-point of diabetic complications. Diabetic neuropathy and peripheral vascular disease are the main etiological factors in foot ulceration and may act alone, together, or in combination with other factors such as microvascular disease, biomechanical abnormalities, limited joint mobility and increased susceptibility to infection. In the diabetic foot, distal sensory polyneuropathy is seen most commonly. The advent of insulin overcame the acute problems of ketoacidosis and infection, but could not prevent the vascular and neurological complications. Management of diabetic neuropathic ulcer by appropriate and timely removal of callus, control of infection and reduction of weight bearing forces. Management of diabetic ischaemic foot are medical management, surgical management and percutaneous transluminal angioplasty of stenosed and occluded lower extremity arteries. Foot ulceration in persons with diabetes is the most frequent precursor to amputation.

1. Introduction

Diabetic ulceration of the foot represents a major global medical, social and economic problem. It is the commonest major end-point of diabetic complications. Diabetic neuropathy and peripheral vascular disease are the main etiological factors in foot ulceration and may act alone, together, or in combination with other factors such as microvascular disease, biomechanical abnormalities, limited joint mobility and increased susceptibility to infection.

Ulceration rarely results from a single pathology. It is the interaction of contributory causes which leads to the breakdown of the foot at risk [1]. The neuropathic foot, for example, does not spontaneously ulcerate. It is the combination of insensitivity and either extrinsic factors e.g. walking barefoot and stepping on a sharp object, or simply wearing ill-fitted shoes, or intrinsic factors such as diminished sensation and the development of a callosity which progresses to an ulcer on walking. Neuropathy is the most significant pathology in the pathway to ulceration [2].

Diabetic foot disease is an important problem confronting the diabetologists, internists and surgeons [3]. The advent of insulin overcame the acute problems of ketoacidosis and infection, but could not prevent the vascular and neurological complications. Foot is the most vulnerable part in a diabetic. It is exposed to frequent trauma and requires a sensitive sensory protection, which is often lacking in a diabetic. The foot, being farthest away from the central nervous system and hemodynamically disadvantageously placed, becomes the common site of complicated lesions. Foot ulceration in persons with diabetes is the most frequent precursor to amputation [4,5]. Overall, patients with diabetes are more likely to have an amputation than patients without diabetes [6].

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2. Etiopathogenesis

In a large prospective study by Lavery et al. [7], significant independent risk factors for DFI included wounds that penetrated to bone, wounds with a long duration, recurrent wounds, wounds with a traumatic aetiology and the presence of PAD.

Although the etiopathogenesis of diabetic foot disease is multifactorial, three main factors, namely neuropathy, ischaemia and infection lead to tissue necrosis and ulcer formation [8]. Other factors are foot biomechanics and weight bearing, peripheral vessel calcifications, trauma, (possibly) diabetic autonomic neuropathy and microangiopathy and diabetic skeletal disease.

2.1. Neuropathy in diabetic foot

In the diabetic foot, distal sensory polyneuropathy is seen most commonly. However, motor and autonomic fibres may also be involved and combined neuropathies frequently occur. The development of a neuropathy is linked to poor glycaemic control over many years and it increases in frequency with both age and the duration of diabetes.

Multiple factors such as blood glucose concentration, blood lipids, structure of myelin sheath and its permeability, axonal flow and micro and macroangiopathy of the peripheral nerves contribute to the production of diabetic neuropathy [9]. Longitudinal data from the Rochester Study [10] supported the contention that the duration and severity of exposure to hyperglycemia influenced the severity of the neuropathy. Current research on diabetic neuropathy is focused on oxidative stress, advanced glycation-end products, protein kinase C and the polyol pathway [11].

2.2. Blood flow in diabetic neuropathic foot

Recent studies have shown that the blood flow is increased in diabetic foot. The latter is due to the arterio-venous shunting and dilated and stiff peripheral arteries [12]. The pulsatility index which is inversely proportional to the quantity of the blood flow is markedly reduced in diabetic foot. The normal Doppler flow pattern is triphasic: a forward flow in systole followed by a reverse flow and a further short forward flow in diastole. In diabetics there is an increased forward flow with the absence of reverse flow.

2.3. Skeletal changes in diabetic foot

Due to increased blood flow to the lower limb there in enhanced blood supply to the bones of the diabetic foot.

2.4. Stiffening of arterial wall

The medial wall calcification in the peripheral vessels in the lower limb raises ankle brachial systolic pressure and shortens transit times of pulse wave. In Charcot's diabetic neuroarthropathy vascular calcification is found in about 90% [13].

Ward et al. have shown that rapid increase in flow of blood bypasses small vessels and the capillary nutrient circulation and results in a relative distal ischaemia [14].

The enhanced blood flow, vasodilatation and arteriovenous shunting, all arising out of sympathetic denervation leads to abnormal venous pooling and oedema. Atherosclerosis of the large vessels of the leg in a diabetic is often multisegmental, distal and bilateral.

Diabetic angiopathy is reported to be the most frequent cause of morbidity and mortality in diabetic patients [15]. Macroangiopathy manifests as a diffuse multisegmental involvement typically involving the infrapopliteal vessels, and is also associated with compromised collateral circulation.

Parving and Resmusen have demonstrated functional abnormality in the form of leakage of albumin from the capillaries to the interstitium; however an occlusive microvascular disease in the diabetic foot has not been clearly demonstrated [16].

The presence of neuropathy makes the feet insensitive and the diabetic patient is often not aware of even a severe mechanical trauma and gross infection.

3. Presentation

Patients present with a variety of complaints ranging from local to systemic signs of infections. Local signs of infection may include pain/tenderness, erythema, oedema, purulent drainage and new-onset malodor. Systemic signs of infection include anorexia, nausea, vomiting, fever, chills, night sweats, change in mental status and a recent worsening of glycaemic control.

Wagner's classification is one of the most widely used and universally accepted grading systems for DFU, consisting of six simplistic wound grades used to assess ulcer depth (grades 0–5) [17]. This classification is limited by the inability to recognize ischaemia and infection as independent risk factors in all classification grades [18] (Table 1).

A more recently proposed and popularized DFU classification is the University of Texas Health Science Center San Antonio (UT) classification system [18]. This system incorporates a matrix structure of four grades of wound depth with subgroups to denote the presence of infection, ischaemia or both (Table 2).

Wounds with frank purulence and/or two or more local signs of inflammation such as warmth, erythema, lymphangitis, lymphadenopathy, oedema, pain and loss of function may be classified as 'infected.'

Lower extremity vascular insufficiency is made by a combination of one or more clinical signs or symptoms of claudication, rest-pain, absent pulses, dependent rubor, atrophic integument, absence of pedal hair or pallor on elevation. From the practical point of view the diabetic foot can be divided into two major types: (1) Ischaemic diabetic foot and (2) Non Ischaemic neuropathic diabetic foot [8].

4. Investigations

Assessment of peripheral neuropathy and evaluation of peripheral arterial status are the two important investigations in a diabetic foot.

Accurate sensory testing in diabetic neuropathy is of paramount importance in the diagnosis, objective quantification and monitoring natural evolution or effects of therapy. These involve testing for (1) Vibration perception Threshold (VPT) {assessed by a Biothesiometer}, (2) Thermal Discrimination Threshold (TDT) {assessed by using 1. Marstock stimulator 2. Automated thermal threshold tester [19–21]}.

Although angiography and visualisation of the vascular tree is

Table 1
Wagner classification system.

Grade	Wound depth
0	Pre-ulcerative area without open lesion
1	Superficial ulcer (partial/full thickness)
2	Ulcer deep to tendon, capsule, bone
3	Stage 2 with abscess, osteomyelitis or joint sepsis
4	Localized gangrene
5	Global foot gangrene