Diabetic hindfoot problems

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Abstract

Diabetes prevalence is reaching pandemic proportions in the 21st century. Since the discovery of insulin physicians and surgeons have been challenged by the long term complications of diabetes. Foot and ankle complications consume a great proportion of health-care resources. In recent times there have been great advances in understanding the basic science behind the aetiology of diabetic foot and ankle complications. The combination of abnormal biome-chanics, neuropathy and vascularity is integral to the pathogenesis of ulceration and Charcot arthropathy. The article discusses the unique biomechanical properties of a diabetic neuropathic lower limb.

Hindfoot and ankle ulcerations have a greater association with major lower limb amputation. Conservative treatment includes prescription of off-loading orthotics and local ulcer treatment. However heel ulcers can also be the result of overzealous surgical intervention.

Mirroring the increased prevalence of diabetes, Charcot arthropathy incidence is rising. We discuss the conservative and surgical manage-ment of this devastating complication of a neuropathic hindfoot and ankle. Conservative management is the mainstay of the initial phase of this disease though evidence is appearing of the benefits of early surgical intervention. Surgical interventions aim at preserving the lower limb and producing a stable plantigrade foot.

Keywords Biomechanics; Charcot; diabetes; neuropathy; ulcerations

Introduction

Hindfoot diabetic disease has a devastating influence on the functioning of the foot in general and has a significant morbidity associated with it. It requires protracted treatment and may lead to amputation.

Diabetes mellitus (DM) is a global disease with higher prev-alence amongst indigenous populations including Hispanics, Asians and Africans. Its prevalence is increasing exponentially in a pandemic pattern.[1] In patients with diabetes the incidence of foot ulcers is 2−6%,[2] the prevalence 3−8%[3] and the recurrence rate 70% after 5 years.[4] Lower limb morbidity, secondary to infection and ischaemia, continues to be a major problem as up to 60% of major amputations are performed in patients with diabetes.[5] The risk has been reported to rise from 1.7-fold once

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protective sensation is lost to 12-fold with both sensory loss and foot deformity.[6,7] The prevalence of diabetic peripheral neurop-athy (DPN) ranges from 13 to 68% in diabetic populations.[8] Charcot arthropathy is a debilitating complication of diabetes often associated with dense neuropathy. The prevalence of Charcot arthropathy is reported to vary from 0.08% to 7.5% amongst the diabetic population.[9] Foot and ankle complications of DM have a profound effect on the individual and exert enor-mous strain on the health care resources of the nation.

Biomechanics of the foot & ankle

The normal foot and ankle

The foot and ankle has two basic functions.

Shock absorption is an important function, which helps the foot to adapt to uneven terrain. During heel strike the foot is required to be unstable and flexible to allow adaptation to uneven surfaces. The subtalar joint plays a pivotal role in this aspect. It allows motion in three planes, which is described as pronation (a combination of eversion, abduction and dorsi-flexion) and supination (a combination of inversion, adduction and plantarflexion). Together the ankle and hindfoot act as a universal joint[10] allowing us to walk on different terrains.

The second function is to provide a stable platform to allow propulsion. The midfoot joint provides a stable platform with the subtalar joint locked during the propulsive stage. There is less movement of the midfoot joints as stability rather than flexibility is required of them.

The gait cycle consists of two parts: the stance and the swing phase. The stance phase is divided into three parts. The first is initial contact; also called heel strike as the heel usually makes initial contact with the ground. In a deformed 'rocker bottom' midfoot secondary to Charcot arthropathy the initial contact maybe with the midfoot. Thus 'initial contact' is the preferred term. The second part of stance phase is mid-stance. The ankle joint comes into play here as the second rocker. The tibia ad-vances forward in the sagittal plane with relative dorsiflexion of the ankle on a fixed planted foot. This propels the centre of gravity from the heel to the forefoot. In diabetic patients the stance phase is prolonged compared to controls[11].

The third and final part of the stance phase is the terminal stance or propulsion phase. This phase requires a stable rigid foot.

Changes in the diabetic foot

Ulceration in the diabetic foot occurs as a consequence of several contributory factors.

Gastrocnemius and/or Achilles contracture occurs in many diabetic patients. The aetiology is not well understood but may be due to inherent neuromuscular deficiency in the tibialis anterior and other ankle dorsiflexors and over activity of the gastrocsoleus complex. Gastrocnemius tightness is assessed by the Silfverskiöld test. It is positive if the ankle dorsiflexion im-proves with knee flexion. Equally reduced ankle dorsiflexion with knee in extension and flexion implies Achilles tightness. The diminished ability to dorsiflex the ankle leads to increased pressures across the arch and to the forefoot. This increases the risk of forefoot plantar ulceration. Gastrocnemius and/or Achil-les release has been shown to decrease the incidence of forefoot

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reulcerations[12] and aid in healing of pre-existing ulcers.[13] However Holstein et al. proposed that patients with sensory neuropathy involving the heel should not undergo this procedure as they found an unacceptable high rate of subsequent heel ulcerations after Achilles release. They also recommended that the surgeon should not be overzealous with Achilles lengthening as extreme dorsiflexion also puts the patients at risk of heel ulcerations.

Recurrent minor trauma in the neuropathic foot instigates ulceration. Involvement of the autonomic nerve fibres leads to anhidrosis or dry skin which encourages callus formation. Plantar pressure is higher at sites of callus formation.

High plantar foot pressures have been described as a risk factor for ulceration.[14] This is certainly an intuitive concept. However a recent study by Yuvuz in 2014 concluded that it was high plantar shear stress, rather than vertically oriented plantar foot pressures, that were closely related to callus and ulcer formation.[15] His study recommended orthotics to concentrate on reducing plantar shear stress rather than merely offloading the foot. The body reacts to repeated high pressures and microtrauma with callus formation to protect the skin from further damage. However, if callus formation becomes excessive it will contribute to higher pressure and should be removed regularly.

Diabetic patients suffer with reduced plantar tissue thickness. This has a strong association with increased plantar pressures and ulcerations.[16] Qualitative changes in plantar fat pad are thought to occur due to a non-specific fibrotic process. This fibrotic tissue affects the biomechanical properties of the plantar fat pad to act as a shock absorber and does not dissipate increased foot pressures associated with neuropathy.

Foot morphology can also predispose to plantar ulceration. Individuals with a valgus or varus hindfoot experience higher plantar pressures over a focussed area. A varus hindfoot has been associated with lateral heel or fibular ulcers whereas a valgus hindfoot is predisposed to medial heel or medial malleolar ulcers.[17]

These various biomechanical abnormalities complicated by peripheral neuropathy and diabetic micro and macro-vascular disease predispose patients to plantar foot ulcerations.

Diabetic hindfoot ulceration

It is estimated that diabetic patients have a 15% life-time risk of developing a foot ulcer. This often leads to further complications such as deep soft tissue infection, abscess formation and osteomyelitis. Diabetic foot ulcerations precede up-to 85% of all non-traumatic lower limb amputations. The triad of neuropathy, deformity and trauma commonly exist in these patients.[18]

Risk factors for ulceration
Fryberg et al. proposed the following list of risk factors for foot ulceration in diabetic patients.[19]
- Peripheral neuropathy
- Past history of ulceration or amputation
- Charcot neuroarthropathy
- Diabetic complications e.g. retinopathy, nephropathy
- Peripheral vascular disease
- Poor glycaemic control
- Increased duration of diabetes
- Trauma

- Impaired visual acuity
- Oedema
- Callus
- Limited joint mobility
- Structural foot deformity

Neuropathic ulcers are generally painless in contrast to ischaemic ulcers which are often painful. A long standing chronic non-healing ulcer may convert to a Marjolin's ulcer (squamous cell carcinoma).

Diabetic foot ulceration classification
One of the most commonly used classification systems is the Wagner-Meggitt (1979) and modified by Brodsky in 2003 (Table 1).

Biomechanical & anatomical considerations specifically pertaining to heel ulcers
Of all the multiple intrinsic risk factors for heel ulcers, which are imposed by diabetes, neuropathy and inadequate tissue perfusion are the most well-known. However, there may be additional risk factors that relate to the pathologically altered biomechanics of the diabetic foot, as diabetes might involve changes to the extents of loading in the soft tissues of the posterior heels, as well as to the soft tissue mechanical properties and sometimes to their geometry.

Diabetes is known to be associated with obesity, and hyperglycaemia gradually induces thickening and interlinking of collagen fibres in connective tissues, particularly in the soft tissues of the feet, which leads to progressive stiffening of these tissues that in turn, decreases their ability to spread and attenuate mechanical loads through tissue deformation. Soft tissue displacements (migration) and atrophy have also been documented in the diabetic foot, which means that the soft tissue layer overlying the posterior calcaneus might be not only pathologically stiffer, but also pathologically thinner.[20]

Abnormal heel strike position could also be an important factor in determining the site of ulceration. Hindfoot deformity in the sagittal plane is not uncommon in diabetic patients particularly in varus and this may well initiate ulceration in the lateral aspect of the heel. The shear forces generated by tangential weight bearing on the heel may produce significant trauma to the plantar skin and in the presence of neuropathy may accelerate the creation of ulcers in this area. Deformity also causes attrition on footwear, which may not be recognized in an insensate foot further compounding the problem.

Clinical examination
Particular attention should be paid to assessing peripheral sensation and blood flow, as well as examining the general appearance of the extremities. The examiner should inspect for differences in skin temperature, hair growth, pallor or colour, calluses, and dry or cracked skin. Additionally, the examiner should note any potential causes of elevated pressure overload such as bony prominences on the plantar aspect of the foot, toe flexor tendon contracture, or gastrocsoleus contracture. If ulceration is present, it is imperative to measure the size and depth of ulceration in addition to recording the quality of the ulcer base (e.g., granulation, fibrous, or necrotic tissue) to serve as a baseline while monitoring healing progress.

Peripheral sensory neuropathy is present in approximately 25% of the diabetic population, and accordingly it is imperative