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Review Article
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Diabetic Foot Biomechanics and Ulceration

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ABSTRACT

Diabetes mellitus affects the structure and function of the foot leading to sensorimotor neuropathy, foot deformities, callus formation and also limits joint mobility. High plantar pressure and autonomic dysfunction interact and contribute to the development of foot ulceration. Trauma is also needed in addition to neuropathy and vascular disease to cause tissue breakdown. This trauma may be intrinsic, repetitive stress from high pressure or callus. The body responds to repetitive high pressures or microtrauma with callus formation. If callus formation becomes excessive it will contribute to high pressure. Ankle dorsiflexion and subtalar range of motion are reduced in diabetes, so also the motion at first metatarsophalangeal joints. These along with changes in tendon, muscles, and bone with thickening and hardening of skin and fascia also add to stiffness and limited joint mobility. These changes are secondary to enzymatic glycosylation. Offloading of the diabetic wound is a key factor in successful wound healing. Total contact cast is generally reviewed as a reference standard for offloading the diabetic wound however several useful alternatives also exist

Key words- *Biomechanics; General Surgery; Diabetic Foot; Derna.*

INTRODUCTION

The global prevalence of diabetes mellitus has been projected to nearly double from a baseline of 2.8% in 2000 to 4.4% by 2030 affecting over 350 million individuals.[1] The diabetic foot remains one of the most serious complications of diabetic Mellitus with plantar ulceration a common complication of diabetic neuropathy. Ulcers commonly occur under the first, second, and third metatarsal heads and most neuropathic ulcers result from excessive and repetitive pressure applied to the foot while walking.[2]

Normal walking requires sensory input to adapt and modify motor patterns and muscle output to carry out the desired task.[3] Fully functioning joints and bone combined with adequate muscle strength are also needed.[4]

Biomechanical anatomy of the foot.

*T*here are 26 bones, 33 joints, and about 100 muscles, tendons, and ligaments in the foot.The foot can be divided into 2 components. The first division is at the tarsometatarsal joint, it divides the foot into forefoot and rearfoot. The second component divides the foot at the third ray into medial and lateral arches. The medial arch is composed of the calcaneus, talus, navicular, the threecuneiforms, the first, second, and third metacarpals.[5] The lateral arch has the calcaneus, the cuboid, and the fourth and fifth metatarsals. These arches are connected transversely.The surface underneath these bony supports is called the vault of the foot.[6] This vault supported by healthy soft tissue provides the foundation for weight-bearing and functions. Besides bearing the weight of the body the foot function as a shock absorber.

The ankle joint is the major joint that controlssagittal plane movements of the leg that is essential for ambulation.[7]

The main motion of the first metatarsophalangeal joint and the lesser metatarsophalangeal joints is in the sagittal plane and that is dorsiflexion. One of the chief functions of the foot is its shock-absorbing property during heel strike and adaptation to the uneven surface of the ground while walking. The subtalar joint plays a major role in this. Subtalar joint moves in 3 planes described as pronation which is a combination of inversion, abduction, and dorsiflexion. Supination a combination of inversion adduction and plantar flexion.

The first metatarso phalangeal joint lies between the head of the 1st metatarsal and base of the proximal phalanx, lateral and medial sesamoid bones all enclosed in a single joint capsule. Dorsiflexion and plantar flexion occur at this joint.

Gait:

Gait is a series of steps and strides.A step is a single step, the stride is a one complete gait cycle.Step time is the time taken from one foot hitting the floor to the other foot hitting the floor. Mediolateral space between the two feet is called step width. To analyze the step cycle one foot is taken as a reference. The Gait cycle has a stance phase during which the limb is in contact with the ground and the swing phase

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when the foot is in the air for limb advancement. The stance or the weight-bearing phase is divided into three parts, the first is the contact phase with the heel being the first area of the foot in contact with the ground, the mid stance phase begins with opposite side toe-off and full forefoot loading and ends with the heel lift.The third phase has active propulsion and passive lift-off. Active propulsion begins with the heel lift of the support side and ends with the heel strike of the opposite side. Gradient horizontal and vertical forces are directed against the foot during this stage. The passive lift-off begins with opposite heel contact and terminates with supportive side toe-off. Normal joint mobility in the joints of the foot is needed to allow normal foot function.Subtalar pronation after heel strike is a major shock-absorbing mechanism. Limited joint mobility or structural abnormality could compromise flexibility and shock absorption. Thus, increasing stress on the plantar surface.

Limited joint mobility of the first metatarsophalangeal joint is the commonest cause of recurrent ulcers under the big toe.Limited ankle dorsiflexion results in increased pressure on the forefoot during the late stance phase of gait caused by an early heel rise or compensatory pronation. Planter shear stress results from forces exerted parallel to the skin and tend to cause a tear.Ground reaction forces (GRF) acts in all three dimensions under the foot.Vertical pressure is easily measured. When a person is standing the magnitude of the ground reaction force is equal to body weight, with each foot experiencing about 50% of the bodyweight distributed over the plantar surface.Which is predominantly a vertically directed force.

During walking the stress applied to the feet are much higher than when standing for several reasons.[9]

• The amount of time, both feet are simultaneously in contact with the floor is substantially reduced during walking.

• Rocker action allows different parts of the foot to make contact with the floor during different phases of the stance phase thus plantar surface area changes in size and location while the ground reacting forces progress from heel to the hallux.

• The ground reacting forces vary in magnitude with one peak during heel landing and a second peak during push off with the forefoot. Therefore, the heel and the forefoot experience much higher peak pressures than does the midfoot.

A minimum of 10000 steps per day is required to consider an individual active. If the steps per day are 5000 the individual is considered sedentary.

Measurement of pressure

A force applied to a small area can do more harm than the same pressure applied over a large area of the plantar surface. Increased plantar pressure is a strong risk factor for foot ulceration.[10,11] Pressures are expressed in kilograms per square centimeters or kilopascals $1kg/cm^2$ =98.1KPa. Normal-pressure values for the whole foot have been established for the optical pedobarograph[12,13] and the EMED platform.[14]

A threshold of 700 KPa has been suggested for predicting foot ulceration using an EMED platform.[15] A threshold of 1000 KPa has a high percentage sensitivity and specificity for the prediction of foot ulceration. Peak pressures over 875 KPa seem capable for prediction of the foot ulcer development.[16]

Diabetic patients with neuropathy and ulcers have higher peak forefoot pressures than diabetics without neuropathy. Charcot foot with a rocker bottom deformity is associated with increased plantar pressures.[17] Pressure that applies on tissues during walking under a callus or a scar from a healed ulcer can be almost 10-15 times higher compared to healthy people.[18] Almost all pressure measurement devices measure only vertical or normal pressure. Shear stress may be an important etiological factor in the development of plantar ulcers. The advantage of in-shoe measurement is that they can evaluate the effects of different types of footwear on pressure and pain relief. In-shoe measurements permit the actual assessment of the underlying effects of different types of insoles and footwear. Normal walking is not always able to predict the plantar pressures expressed during other activities.[19]

Changes in the foot caused by diabetes

Diabetes mellitus affects the structure and function of the foot. The factors contributing to the development of foot ulceration include sensorimotor neuropathy, foot deformity, callus formation, limited joint mobility, high plantar pressures, and autonomic dysfunction which interact. Diabetic neuropathy leads not only to reduced or loss of protective sensations but to changes in structure as well as the dryness of skin which can lead to excessive callus formation.[20-22]

Changes in foot structure that affect foot function can lead to high plantar pressure which is an important predictive risk factor for the development of diabetic foot ulceration.[23] Foot deformities including bony prominencesare predictive of increased plantar pressures and foot ulcerations.[22] Plantar tissue thickness is strongly associated with plantar pressures indicating a close relationship between the amount of cushioning soft tissue available and the pressure distribution of the forefoot. There are higher peak pressures at sites with reduced plantar tissue thickness.Qualitative changes of the fat pad have also been observed in the form of a nonspecific fibrotic process beneath the metatarsal head in patients with diabetic neuropathy. This fibrotic tissue affects the intrinsic biomechanics of the plantar fat to act as a shock absorber and dissipate increased plantar pressures associated with neuropathy.[25] Prominent metatarsal heads may be due to weakness of the intrinsic muscles of the foot leading to deformities in patients with diabetic neuropathy. Weak and atrophied muscles secondary to motor nerve involvement result in imbalance causing clawing of the toes, high arched foot, and high pressure of plantar surfaces.[26] Muscular atrophy is due to the replacement of muscles by fat because of denervation.[27] Weakness of intrinsic muscles causes prominent metatarsals.[28] There is the migration of fat pads distally with clawing

of the toes thereby making the metatarsals relatively unprotected.[29] Sub metatarsal fat pad is much reduced in neuropathy with toe deformity.[30] Changes in the plantar tissue in the form of increased stiffness occur in diabetic patients with a history of ulcers.[31] Increased hardness of the skin has been shown in diabetic patients with a history of foot ulceration.[32] Using MRI scan increased stiffness was shown for diabetic plantar tissue with higher shearer elastic moduli indicating vulnerable tissue for ulceration.[33] Charcot arthropathy usually causes gross deformation of the foot, peak plantar pressures in Charcot arthropathy are higher compared to patients with a neuropathic ulcer.[34] Abnormal pressure loading is seen in the case of patients with minor amputations either same or contralateral foot. Patients with partially amputated feet were also shown to exhibit abnormal foot function and pressure loading either in the same or in the contralateral foot.[35,36]

Amputation of the hallux greatly increases pressure under the metatarsal head.[37,38] Callus areasaffect loading and have been reported to be highly predictive of foot ulceration.[39]

Sudomotor dysfunction

It results from damaged postganglionic sympathetic C fibers innervating sweat glandscausingdryness of the skin. the vast majority of the patients with diabetes, the skin of the feet shows dryness compared to patients without neuropathy.In patients with autonomic neuropathy, there can be an abnormal opening of arteriovenous shunts reducing blood flow and impair tissue oxygenation and wound repair.[40]

Changes in muscles, tendons and bones

Plantar aponeurosis and flexor hallucis longus are thickened in neuropathic patients.[41] Thickening of the plantar fascia and tendon achilleas increase with bad diabetic control due to impaired glucose regulation and dependency on oxidative phosphorylation for energy production.[42] The mechanical and metabolic functions of the muscle are affected.In neuropathic diabetics ankle and knee muscle volume and maximal isokinetic muscle strength are reduced.[43] Muscle strength and sensation may be related to dorsiflexion at the ankle.Tibialis anterior which eccentrically controls flattening of the foot after the heel strike phase remains active for a longer time in neuropathic diabetics.[44]

Limited joint mobility

In neuropathic diabetics, dorsiflexion of the ankle joint may be limited due to anatomical, physiological or orthopedic factors.Joint motion in diabetes is altered due to non-enzymatic glycosylation.There is a linear relation between diabetes and deficits in foot morphology, especially with neuropathy. Exposure to hyperglycemia alters composition and functions.Nonenzymatic irreversible glycosylation of proteins and formation of advanced glycosylation end products (AGEs) typically reduce elasticity.[45,46]Skin in diabetics is thickened and less elastic.[47,48] Collagen bundles are thickened and disorganized due to irreversible glycosylation. The formation of AGEs reduces the remodeling of collagen fibers.[49] Keratin in the stratum corneum of diabetics is glycosylated.[50] Mobility of the subtalar joint will be reduced in ulcerated joint compared to the contralateral nonulcerated foot in diabetic neuropathic patients.[51] Ankle dorsiflexion and subtalar range of motion was reduced in diabetic patients with a history of plantar ulceration compared to patients without a history of ulcerations and non-diabetic controls.[52] Ulceration of the great toe has been associated with reduced range of motion at the first metatarsophalangeal joint.[53] Thickness of the plantar fascia of foot was increased in diabetic patients with and without neuropathy compared to non-diabetics.[54]

Biomechanical aspects of foot ulceration

Foot ulcers in diabetics are caused by peripheral neuropathy, peripheral vascular disease, foot deformity, increased foot pressures, and severity of diabetes.[55] Ulceration is caused by repetitive or excessive pressure on the surface of insensitive skin leading to tissue damage.A normal person would experience pain and avoidadding pressure.The body responds to repeated high pressures or microtrauma with callus formation to protect the skin from further damage but excessive production of callus will contribute to high pressures. Both nondiabetic and diabetic planus feet have been shown to experience greater peak pressures than non-diabetic rectus feet.An inverted heel position has been associated with lateral ulcers whereas an averted heel position has been associated with medial ulcers.[56]

It is not only the magnitude of the plantar pressure which is causing foot ulceration several other factors such as the rate of increased pressure, duration of high pressure, and the frequency of applied pressure to the skin matter. Diabetic patients with foot type characteristics different from the normal are likely to develop high foot pressures and ulcerations.

Biomechanical interventions to offload the foot

Preventive care in reducing foot pressure includes removal of callus, provision of pressure reducing insoles, and therapeutic footwear. Wearing microcellular rubber insoles footwear for 6 months was shown to reduce the hardness of plantar tissue and foot pressure distribution in diabetic neuropathic patients.[57]

Injection of collagen under callus in diabetic patients with previous neuropathic ulceration was shown to reduce the surface area of callus compared to the non-treated control group at 8 months -post-injection.[58] Liquid silicone injection has been used in the foot to improve cushioning at callus sites, corns, and painful areas.[59] Injected liquid silicone decreases peak plantar pressures and callus formation and increased plantar tissue thickness under silicone treated areas.[60]

Dorsiflexion metatarsal osteotomy elevates prominent metatarsal heads, balancing and redistributing weight-bearing forces more evenly across the forefoot.First metatarsophalangeal joint arthroplasty increases the range of motion at the articulation of the hallux to the first metatarsal.This technique commonly improves the healing of ulcers at hallux.Achilles tendon lengthening

(ATL) has been shown to increase dorsiflexion range and decrease forefoot plantar pressure and reduce the rate of ulcer recurrence.[61,62] The most important complication of achilleas tendon lengthening is the development of a transfer lesion to the heel.[63] There are risksofsurgery but the benefits of Achilles tendon lengthening outweigh the risk for patients with recurrent ulcers and limited dorsiflexion at the ankle joint. [64]

Offloading devices for Foot ulcers

Total contact cast (TCC) is a reference standard for offloading.Several other alternatives do exist. [65] TCC is very effective in off-loading but regular checking is difficult.The solution is a non-removable fiberglass cast with a treatment window at the ulcer site which allows daily inspection of the wound.[66,67] The use of a TCC is contraindicated in acutely infected or ischemic feet. However, ulcers with moderate ischemia or infection are effectively treated using a TCC.[68]

When peripheral arterial disease and infection are present or in the case of heel ulcers alternative off-loading methods are required as the outcome is poor.[68] Other devices such as half shoes, healing shoes, accommodative dressings are used which are not as effective as TCC but regular inspection of the wound is possible.TCC is a well-molded minimally padded cast that maintains contact with the entire plantar aspect of the foot and lower leg.A large proportion of pressure reduction over the forefoot is borne by the rear foot. Irremovable total contact cast (ITCC) has shown faster healing rates compared to the removable cast.[69] Ankle foot orthotics has been suggested to be a useful alternative to casting techniques to offload during wound healing and to prevent ulceration.[70] In addition to off-loading, wound healing needs debridement, treatment of infection, and in some cases revascularization.

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