SURGICAL MANAGEMENT OF DIABETIC FOOT AND ITS COMPLICATIONS

Essay

Submitted for Partial Fulfillment of Master Degree in General Surgery

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List of Abbreviations

ABI Ankle-brachial index

B cell B. Lymphocyte cell

BUN Blood urea nitrogen

C' Complement &

CT Computed tomography

DCCT Diabetes control and complication trial

DM Diabetes mellitus

ESR Erythrocyte sedimentation rate

FBI Femoral-brachial index

GDM Gestational diabetes mellitus

IDDM Insulin-dependent diabetes mellitus

IGT Impaired glucose tolerance

INR International normalized ratio

LEA Lower extremity amputation

MHC Major histocompatibility complex

MRI Magnetic resonance imaging

MRSA Methicillin resistant staphylococcus aureus

NIDDM Non insulin-dependent diabetes mellitus

PAD Peripheral arterial disease

PN Peripheral neuropathy

PT Prothrombin time

PVD Peripheral vascular disease

PVR Pulse volume recordings

S.aureus Staphylococcus aureus

SDS Special dressing service

T cell T. Lymphocyte cell

Tc T. lymphocyte cytotoxic

Th ¹ T. lymphocyte Helper ¹

Th † T. lymphocyte Helper †

WBC White blood cells

WHO World Health Organization

NTRODUCTION

Diabetes mellitus is a chronic metabolic disease, with pathologic changes seen frequently in the peripheral vascular, nervous, skeletal, and integumentary systems. The foot is often the first part of the body to show the adverse effects of diabetes, being the farthest from the heart, the most abused and often the unexamined and neglected appendage on the body, often times demonstrates the most devastating complications which if neglected, can lead to loss of limb and sometimes death (Chauchard et al., $\gamma \cdot \cdot \cdot 1$).

70% of all diabetics are expected to develop severe foot problems at some point in their lifetimes (Cunha et al., *...), the risk of amputation is \o-fold higher in diabetic patients and o out of 7 amputees are diabetics (Vayssairat et al., * · · 1).

The pathologic mechanisms by which diabetes creates alterations in the neural, vascular, and immunologic systems are complex (Armstrong et al., 1997).

Diabetic foot infections range from cellulitis to chronic osteomyelitis, and globally, they are the most common skeletal and soft tissue infections in patients with diabetes. Mortality is not common, except in unusual circumstances. The mortality risk is highest in patients with chronic osteomyelitis and in

those with acute ne

those with acute necrotizing soft tissue infections (Perencevich et al., ** • • \$).

There are three main factors involved in the pathology of the diabetic foot; Neurophathy, Ischemia and Infection. The relative role played by each element can vary, but community studies have shown that of these patients ''w were associated with neuropathy alone, ''o'w with arterial disease predominantly, whilst ''o'w had both neuropathy and vascular disorders (*Inlow et al.*, ''···).

Metabolic changes compromise the host defense mechanism, white blood cells utilize glucose to produce lactic acid at a decreased rate in diabetics. This is crucial because lactic acid has a bactericidal effect in a low oxygen environment (*Kreyden et al.*, **••**).

Risk factors and pathogenesis for ulceration and amputation are quite similar because $^{\wedge \circ}$ % of diabetes lower extremity amputations have an ulceration in their causal sequence (*Pecoraro et al.*, 199).

The high risk patient for lower extremity amputation has one or more of the following: Loss of protective sensation, absent pedal pulses, severe foot deformity, history of foot ulcer and prior amputation (*Chauchard et al.*, **.**).

Early, surgical debridement, antibiotics, dressings, rest of injured area, correction of ischemia and control of diabetes are essential to prevent amputation (*Green et al.*, *··*).

In all diabetic foot infections a primary consideration is whether or not surgical intervention is required, e.g. for undrained pus, wound debridement or revascularization. Debridement has been shown to enhance and shorten the healing process. When necrotic tissue, crusted exudates, and fibrinogen are removed, growth factors are released and fibroblasts and keratinocytes migrate more easily into the wound (Steed et al., 1997).

Adjunctive treatment, besides the established treatments mentioned above, other modalities have been advocated, growth factors (becaplermin and platelet - derived wound growth factor). Hyperbaric oxygen therapy in the treatment of serious wounds that have not responded to established treatments, especially when complicated by ischemia (Stuck et al., 1990).

AIM OF THE WORK

he aim of this work is to review the anatomy and pathology of diabetic foot and the focus on investigation, presentation of diabetic complications and to present medical and surgical management of diabetic foot.

ANATOMY OF THE FOOT

The Bones of the Foot: (Fig. 1)

ones of the foot include tarsal bones, metatarsals and phalanges. Tarsal bones are calcaneus, talus, navicular, cuboid and the three cuneiform bones. Only the talus articulates with the tibia and fibula at the ankle joint (*Levin*, 1997).

The calcaneus is the largest bone of the foot and forms the prominence of the heel, it articulates with the talus through its anterior surface.

The talus articulates above at the ankle joint with the tibia and fibula, below with the calcaneus and infront with the navicular bone, it possesses a head, neck and body. The navicular bone has a tuberosity on the medial border of the foot one inch infront of and below the medial malleolus which gives attachment to the main part of tibialis posterior tendon. The cuboid bone has a deep groove on its inferior aspect which lodges the tendon of the peroneus longus muscle.

The three cuniform bones are small wedge shaped articulating proximally with the navicular bone and distally with the first three metatarsal bones. Their wedge shape contributes to the formation of the transverse arch of the foot (Sinnatamby, 1999).

The sesamoid bones:

Sesamoid bones, like the seeds after which they are named, are usually more or less ovoid nodules a few millimeters in diameter, although their shape and size vary, some being quite large. These are not always completely ossified, and may consist of dense fibrous tissue cartilage and bone, in varying proportions, but the majority is, to some extent at least, ossified. They are almost always embedded in tendons either in close relation to articular surfaces or in situations where tendons are sharply angled round a bone surface. In both cases, the surface of the sesamoid related to another bone is covered with articular cartilage and actually slides over the opposed bone, which is itself usually an extension of an articular surface. This arrangement entails that the tendons concerned are to some extent fused with the joint's articular capsule.

In the foot, two both of which may be duplicated (sometimes leading to fallacious diagnosis of fracture), are always present in the tendons of flexor hallucis brevis, on the plantar aspect of the metatarsophalangeal joint of the hallux. They are firmly tied in with the ligamentous strucutres around them, including the most medial part of the plantar aponeurosis. Single sesamoid occurs not infrequently in the plantar aspect of the capsule of the same joint in all the other toes, and also in the interphalangeal joint of the great toe.

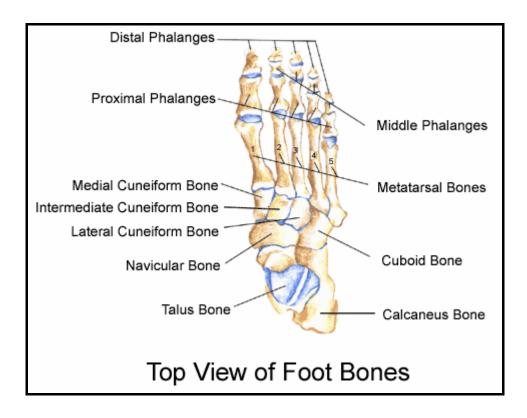


Fig. (1): Bones of the foot (Anne, 1997).

Sesamoid bones, or cartilages, which are not associated with a synovial joint, occur more frequently in the lower limb. As that appearing late in life and therefore perphaps adventitious in character, occurs in the tendon of tibialis anterior where is in contact with the distal part of the medial surface of the medial cuneiform bone. Similarly, the tendon of tibialis posterior may contain as sesamoid nodule where it glides over the medial side of the head of the talus (*Warwick*, 1977).