DIABETIC FOOT

ESSAY

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BY

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A C K N O W L E D G E M E N T

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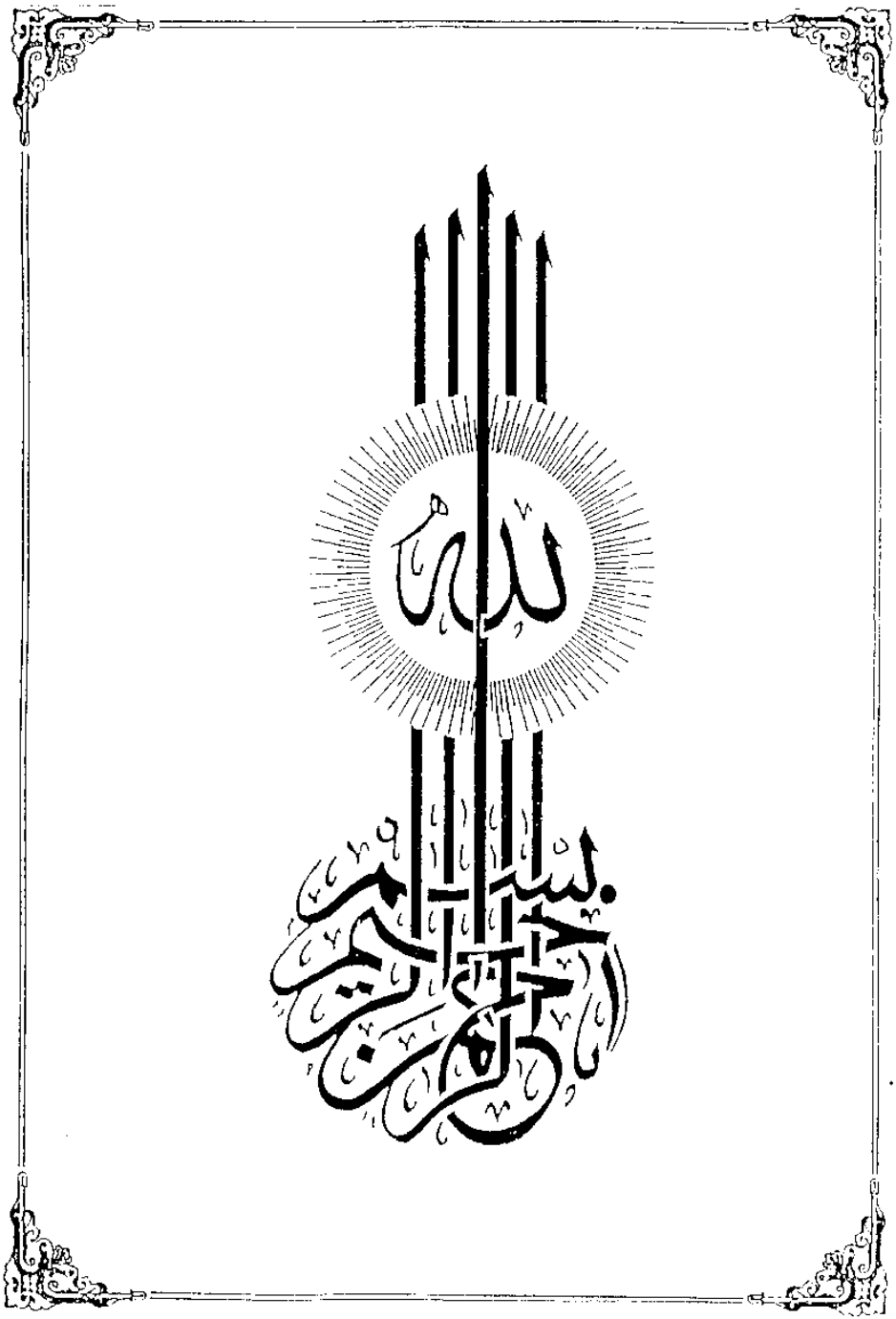

1- Introduction

The anatomical pecularities of the foot with its tense fascial plantar envelope which entraps inflammatory cellular exudate under tension, with its muscle layers and potential fascial spaces and with the long tendons constituting a high way for spread of infection from the sole to musculus-facial planes of leg, will make a trivial infection, if not properly and promptly managed, a virulent necrotising and dissecting one that can threaten limb and life.

The presence of vascular affection in various degrees is an important cause adding to the morbidity and mortality of diabetic foot patients, and the much greater incidence and severity of pathologic changes in the lower extremity vessels of diabetics compared with non diabetics attests to the importance of vascular changes in the genesis and prognosis of diabetic foot.

As regards the neurological changes, no part of the body of the diabetic patient is as vulnerable as his foot, as his foot, as it on top of the severe peripheral neuritis, it requires on an accurate monitoring of blood flow by the proper balance between the sympathetic and parasym pathetic nervous systems in the presence of a normally

2. Anatomical Considerations

the extent that the plantar skin is so fixed to deeper plantar fascia and aponeurosis to be considered as an unyielding strata that can keep any exudation or collection under very high tension leading to not only to very destructive undesirable effects, but also its oedema presents early and easily on the dorsum of the foot where the tissues are supple and yieldable.

The nail folds constituting two paronechial potential spaces connected with each other posteriorly, those spaces are frequently infected.

The sciatic nerve furnishes the main motor and sensory innervation of the foot, derived from the 4th and 5th Lumbar and the first and second sacral segments.
Other contributions from the femoral nerve through the saphenous are given.

The autonomic nerve supply is in its most part is furnished through the arterial branches by the plexuses around the arteries.
The arterial supply of the foot is derived from the popliteal artery through the anterior and posterior tibial arteries.

The sole of the foot is largely dependent on the posterior tibial artery through its two divisions: the lateral & medial plantars, so much so that in blockage of the posterior tibial endangers to a great extent the viability of the foot as the collaterals with the dorsalis pedis of the anterior tibial become insufficient to cope with the needs of the foot especially when these arteries become the site of diabetic presenile angiopathy.

The muscles, tendons of the sole of foot are arranged into four strata enclosed between an osteo-fascial tense compartment formed by the bones of foot and the thick, dense plantar aponeurosis - This space communicates freely with the posterior leg compartment through a tunnel undercover the abductor hallucis muscle on the medial surface of the oscalcis transmitting the tendons of tibialis posterior, flexor hallucis, and the flexor digitorum longus and the posterior tibial artery and nerve with their divisions lateral and medial plantars.

This communication is of very important surgical bearing on management of diabetic foot infections, when infection

if neglected can eventually reach the leg making the prognosis more worse and surgical infection more drastic. (Lawrence E. O'neal, 1977)

PATHALOGICAL CHANGES IN DIABETIC FOOT

Diabetic foot, which should be considered as a major surgical problem, is a real challenge to the medical attendants specially the surgeons, being contributed by multifactorial items aggravated by the fragility of the diabetic patient, and the delay in starting the prompt and the drastic measures, that it deserves.

Incidence: John Molins (1968) stated that in Britain at least more hospital beds are occupied by patients with bad feet than all other complications of diabetes put together. Joslin (1973) attributes the cause of death in 20-32% of diabetic population to complications of diabetic foot.

In Egypt, inspite of the absence of official national dependable statistics, yet among the approximate of three millions diabetics, diabetic foot is recording a real increase in incidence amounting to 15-20% in many sporadic centers.

Age and Sex: Though prevalent in both sexes in such a widely spread familial and hereditary disease as diabetes, yet there is a fairly marked preponderance

in males than females in the ratio of 5:3, most probably related to the predominence of the multi-factorial items underlying the pathogenesis of the case and to the nature of life and function in males.

The highest incidence occured at fairly younger age group in the fifth decade as compared to the reported seventh decade in abroad cases. (Haimovies; 1970, Smith & Casingol, 1969).
Though, there is no apparent reason for one foot to dominate the other, except for local causes of trauma or infection acting as an exciting factor. (Abdelrazek F. Masoud et al., 1980.) yet the right foot is materially more affected than the left, most probably due to functional physiological attitude. Moreover, it has been observed that the diabetic foot is not parallely related to the severity of the case of diabetes as shown by the high blood sugar level, but it is more dependant on the duration of the disease even of low blood sugar level, and also on negligence in the treatment of the diabetic phenomena.

Among others, three important factors influence the picture and course of the diabetic foot and decide to a great extent the management and the prognosis of the case.

A. Angiopathy: The most important of these factors, expresses itself in the following patterns:

1. Arteriosclerotic changes that start early in age in diabetic patients due to the availability of the predisposing factors in the diabetes, is a degenerative process that affects the medium and small sized arteries. That is why we refers to these senile changes that usually occur in non-diabetic patients around the sixties, to "presenile" arteriosclerosis.
They start by patches of fibrous thickening of the intima (Haust 1971, Wilens 1951) stimulated by mechanical and haemodynamic forces acting on the wall of the vessel. Later on, fatty streaks show themselves composed of accumulation of the lipids derived from the circulation, either deposited in a free form, or within macrophages as foam cells in the intima.

Ultimately, they progress to atheromatous plagues augmented by dietry habits, plasma lipoproteins, sex hormones and the diabetic disease, leading

to narrowing the lumen of the vessel and complicated by deposition of a thrombus on its roughened surface, resulting in all the sequalae of obliterated vascular territory. This interaction between plasma lipoproteins specially low density ones, and the arterial smooth muscle cells is of particular importance in the genesis of asterioselerosis. (constantinides, P. 1965, Geer, J.C., Mc gill, H.G., Jr, and strong, J.P., 1961, Getz et al. 1969). The vessel most frequently involved in diabetics is the tibials and popliteals.

At a very late stage the media becomes involved by collagen tissue, firosis, calcium deposition. The articles under 0.3 mm in diameter, modulating the peripheral resistance and regulating blood flow through the capillary bed, plays the most important role in contribution to tissue ischemia in diabetic patients either by functional vasoconstriction or by these organic arterioselerotic changes that occur at an earlier age as mentioned before, progress rapidly and occur massively in a generalised form in diabates,

hence the diabetic foot.

Although in the opinion of Josef R. Williamson et al. in 1977, more study is needed before the significance of this asterioles disease can be reliably assessed in the pathogenesis of peripheral vascular disease complications in diabetes.

2. Microangiopathy; which is the single most characteristic ultrastructural feature of the various forms of diabetic angiopathy, presenting as thickening of capillary basement membrane, which normally not only would appear to serve as a type of molecular sieve, but may also provide some structural stability to the cabillary wall. Banson and Lacy 1964 found that average skin capillary B.M. width of the big toe was 13.300 A in diabetics compared with 5900 A in non diabetics with no correlation at the same time between B.M. width and severity or duration of diabetes.

While siperstein et al. in 1968, considered that diabetic microangiopathy, an initial lesion in diabetes, is not the consequence of insulin deficiency but an independent manifestation of

diabetes unrelated to carbohydrate intolerance which in itself, may be the consequence of capillary B.M. thickening, yet Gunderson in 1978 stated that it is secondary to metabolic disturbance and is preventable by optimal treatment. On the contrary, Kilo, Vogler and Williamson in 1972 and pardo et al. in the same year have demonstrated highly significant thickening of caillary B.M. as an aging phenomenon in non diabetic subjects.

Varcko in 1970 and Williamson et al. in 1971, observed that B.M. thickening in the diabetic is manifested more in the most dependant part of the body, (i.e) the lower extremity, attributing it to the increased hydrostatic pressure. But as Szirtes in 1970 has shown increased platelets adhesiveness and aggregation in diabetics, Sagel et al in 1975 suggested that it may be the critical factor in the genesis of this micro vascular lesion.
The etiology of this tendency towards increased platelet aggregation in the diabetic has been attributed to the presence of a plasma factor