DIABETIC FOOT

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under the supervision

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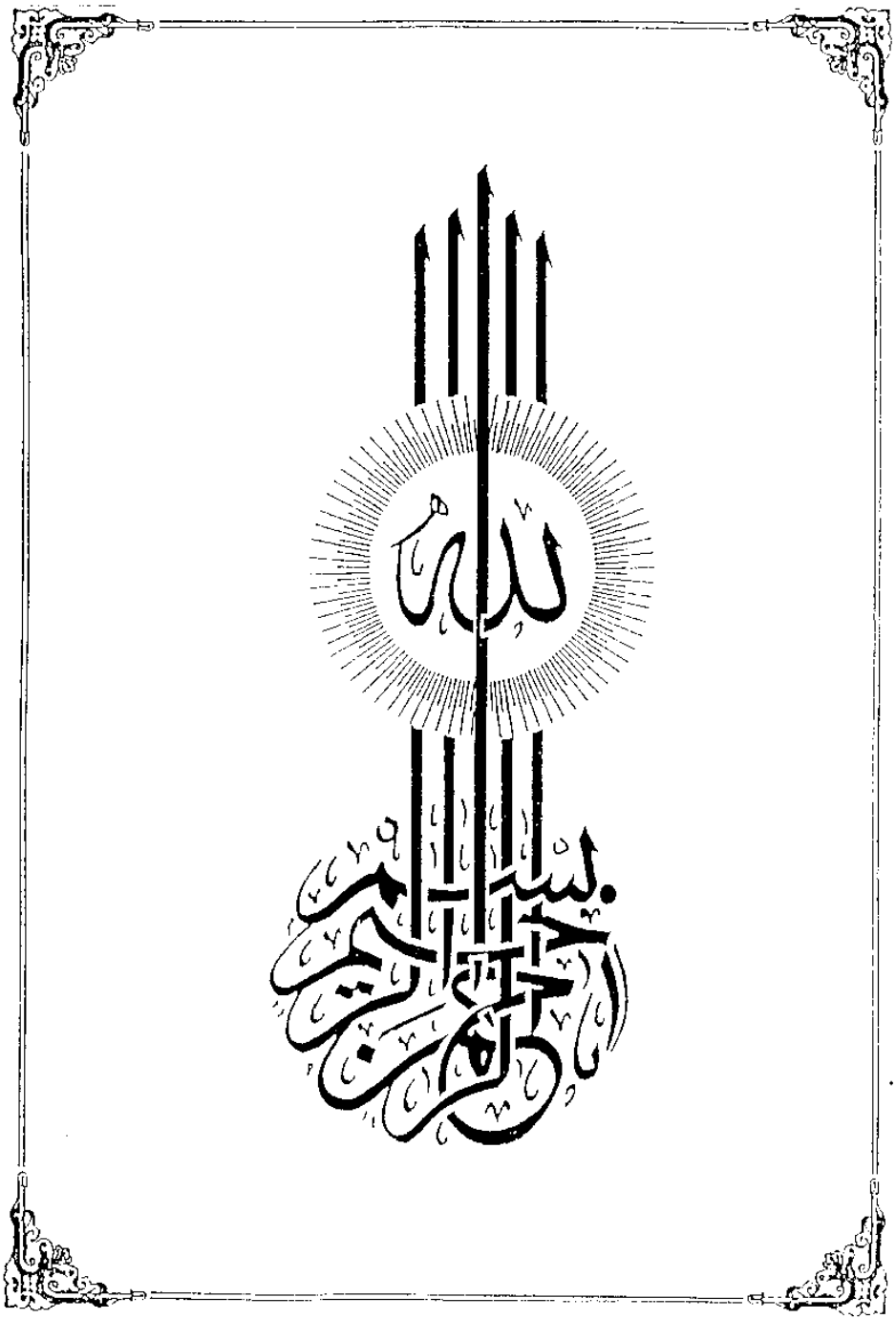

C o n t e n t s

INTRODUCTION

Diabetic foot constitutesa real challenge to the surgeon. The magnitude of the problem is evidenced not only by the number of cases but by the prolonged stay in hospitals, the repeated dressings , the medical and surgical treatment.

If the surgeon went to treat diabetic foot, he must be knowledgeable in the aetiopathology of diabetic foot, and be aware of signs and symptoms of angiopathies and neuropathies.

The general treatment include proper control of diabetes by crystalline insulin and proper antibiotic therapy.

The local surgical techniques are used to preserve as much of the foot as possible and to keep the patient ambulatory.

Prophylactic surgery is important line of treatment in a diabetic foot, to avoid problems need a long time and high cost to patient and community.

REVIEW OF LITERATURE

Diabetic foot is a challanging surgical problem complicated by the multificatorial pathogenesis and the fragility of the patient. Its management needs patience from both the surgeon and the patient.

Incidence

Surgical foot lesions are very common among diabetics. John Malins (1968) stated that in Britain, more hospital beds are occupied by patients with bad feet than all other complications of diabetes. Joslin (1973) attributes the cause of death in 20-32 % of the diabetic patients to complications of diabetic foot. Joslin clinic (1956 - 1964) received 440 cases (22%), out of 2000 total diabetic cases received, suffering from foot lesions.

There is no special predilection for diabetic foot to a special sex. The incidence of diabetic foot increases after the age of 40 to reach a maximum at about the age of 60, to decrease thereafter.

There are no significant effects of occupational, residental or racial factors on the incidence or severity of diabetic foot.

Pathology and Aetiopathology of Diabetic Foot

Mode of Onset :

Trauma is an almost constant precipitating factor in bringing about pathogenesis of diabetic foot to light, (Schwartz, 1974). Trauma is usually a trivial one,noticed or unnoticed by the patient due to the hyposthetic extremities. This trauma may take the form of a thorn or a sharp foreign body, badly fitting shoes or careless removal of a corn or a callosity. (Diabetes - Max and Harold Rifkin, 1973).

Aetiopathology :

The nain factors responsible for the initiation of diabetic foot in diabetic patients are the angiopathic changes, the neuropathic factors and infective element.

A- The Angiopathy:

It occurs at many levels; arterial, arteriolar and microcirculatory levels (Slovak 1975).

Types of Angiopathy

1. Atherosclerosis

It is a degenerative process involving primarily the large arteries with some endothelial proliferation and subintimal deposits of lipid material.

Calcification, ulceration and thrombosis,complicating atherosclerotic changes, aggravate the occlusive phenomenon (Strandness, 1964).

The atherosclerotic lesions occuring in diabetic foot differ from the non-diabetic lesions in the following

a. Occuring at a younger age (Dry, Hines, Bell and Bencosome, 1966).

b. Rapid progress.

c. Severer degree. Bell (1957) reported (25.8%) of diabetic mortality due to diabetic foot gangrene (198 case out of 786 total diabetic mortality).

d. More distal affection (Haimovici and Oakley et al, 1968) and Marios, Balodimos, (1961).

e. Produces a greater disability.

Atherosclerotic changes occuring in diabetic patients may be explained on the following grounds :

a - Arteriolar and microcirculatory angiopathy contribute to susceptibility of diabetics to atherosclerosis through involvement of vasa vasora (Pederson and Olsen 1962).

b - The capacity of the vessels to clear themselves from deposited cholesterol is diminished in diabetic patients due to impaired glucose uptake by arterial wall and consequently diminished phospholipid formation (Adams, 1963).

c - Diabetes causes or aggravates atherogenesis through hyper lipaemia and decreased fibrinolysis (Marvin E. Levin 1977).

II. Arteriolosclerosis :

It is a disease of arteries and arteriols more than 150 u. in diameter. Williams (1968), Waren and Lecompt, (1952); Semple (1953) and Lise (1942) agreed that there is a concentric laminated arrangement without much endothelial proliferation. The media is hypertrophied then hyalinized and the internal elastic lamina is reduplicated, then frayed.

Arteriolosclerotic lesions in diabetics differ from those occuring in non diabetic in the following:

a - The main brunt falls on the tibials and popliteal arteries and not on the femorals nor the iliacs (Schwartz, 1974) and (Haimovici, 1959).

b - The lesions in diabetics are multisegmental occlusions with diffuse mural changes proximally and distally (Allen Barker, 1955).

c - The condition is severer, occurs at a younger age and is much more common in diabetic than non-diabetics. Dry and Hines (1941) claimed that it is 11 times commoner in the diabetics.

d - The course is more rapid in diabetics with axial and centripetal spread.

e - Bell (1957), found that it is 53 times in men and 71 times in women in diabetics than in non-diabetics over 40 years and is often bilateral.

III. Microangiopathy :

This Microangiopathy affects blood vessels less than 150 u in diameter i.e. arterioles, capillaries and venules, (Williams, 1968),in the form of thickening of basement membrane followed by infolding of endothelial membrane and projections into endothelial cytoplasm with formation of masses of para-amino-salicylic acid + ve. material due to the presence of glycoprotein mucopolysaccharide residue interlaced in proliferating endothelium (Ellenberg, 1963). Such changes could be detected in the stage of prediabetes.

The thickened basement membrane of the micro-circulation might act as a barrier to the egress of intravascular nutriments into interstitial space.

This concept is given some support by Ismail et al., who observed a decrease in microvascular permeability to intravenously injected radioiodinated albumin in diabetic patients (Marvin E. Levin, 1977). There is however, no evidence to support this hypothesis, the information currently available indicates that thickened basement membranes of diabetic patients are at least as permeable as those of normal subjects, if not more so. Increased capillary permeability in subjects with diabetic microangiopathy has been demonstrated in the retina by fluorescein angiography and in skeletal muscle by isotopic techniques and is evidenced in the kidney by proteinuria (Skovborg, F. and Lauritzein, E.1965).

Recent information now suggest that abnormal platelets adhesiveness and agregation may be a critical factor in the genesis of microvascular lesions. These platelets adhesiveness and agregation has been demonstrated not only in patients with diabetic complications but also in patients with early diabetes and in prediabetic (Marvin E.Levin, 1977).

In general the angiopathy has a predominant role in the pathogenesis of diabetic foot through :

1. Microangiopathy which is present in all the cases and has a major deterimental effect on intravascular and transvascular perfusion. So even in presence of full bounding ankle pulses, there is always a sort of an occult ischaemia. This microangiopathy aggravates the deterioration in the defensive vascular, inflammatory reactions against traumaa and invasive organisms.

2. Microangiopathy aggravate the effect of atherosclerosis and arteriosclerosis .

3. Causation of neuropathy :
   Fagerberg, (1957), Waltman, (1959) and Williams (1968), documented that, ischaemia has a selective effect on the rapidly conducting large "A" nerve fibres, (Stein, 1964).

4. Microangiopathy hampers tissue perfusion with specific bactericidal plasma factors and interferes with granulocytic mobilization.

   In addition anoxic poorly perfused tissues give a better medium for anaerobic and microaerophilic organisms.

B. The Neuropathy :

Diabetic neuropathy can take many features and according to Williams (1968) it may affect :

I- Peripheral nervous system as :

a. Radiculopathy.

b. Mononeuropathy and mononeuropathy multiplex.

c. Peripheral polyneuropathy.

d. Autonomic neuropathy.

e. Cranial neuropathy.

II- Spinal Cord : as

a. Diabetic Myelopathy "Pseudo-tabes".

b. Diabetic Amyotrophy.

c. Myelomalacia : due to acute vascular lesion.

III- Cerebral :

Any form ranging from cerebral coma to acute or chronic cerebrovascular problems.

The type of neuropathy which is more related to diabetic foot is the polyneuropathy, but mononeuropathies and autonomic neuropathy may be related.

Rundles (1945) put the following explanations for the pathogenesis of diabetic peripheral neuropathy :

1. Angiopathy.

2. Disturbed carbohydrate metabolism and hyperglycaemia: More selective action on slow conducting small "C" fibres carrying pain and autonomic impulses

simulating the effects of toxic neuritis (Greenboum, 1964).

3. Dietary deficiency :

Of the vitamin "B", especially of vit. "B_1".

The common diabetic polyneuropathy has a distal bilateral symmetrical distribution. It is predominantly sensory. Williamson (1922) stressed on the element of vibratory sensory loss as an early sign. Gregresen (1968) , correlate vibratory sensory loss to the duration of diabetes with no sex difference.

Myelopathies presents with pseudotabes through affection of nerve roots near dorsal root ganglion with demyelination mononeuritis affects a major nerve trunk with sensory, motor and sympathetic denervation. Microangiopathy has a major role in its pathogenesis while metabolic disturbance has a major role in production of the polyneuropathy (Joslin, 1973).

In general , there are segmental demyelination, primary nerve and root changes with 2ry. muscle and cord changes. The primary changes is in the nerve cell body with secondary peripheral changes. Muscle biopsy shows patchy atrophy, nerve biopsy shows myelin breakdown, resulting collectively in loss of insulation which could be detected by E.M.G. changes.

The general effects begin by early loss of vibration sense due to the early affection of large fibres. Later on pain then touch perception is lost and lastely sense of position is impaired. Motor weakness is less prominent and atrophy is a late sign . Motor changes are early declared by fasciculations and denervation potentials on E.M.G. Sensory nerve conduction velocity is reduced quite early before any clinical manifestations.

Lost sensations of the foot deprives it from making automatic minor changes in weight bearing to be distributed uniformly and smoothly. The deprival of this protective mechanism causes maldistribution of pressure and initiated the formation of pressure sores.

This maldistribution, in absence of pain sensation and trophic impulses, begins the pathogenesis of diabetic foot. So deprivation of superficial and deep sensations in presence of unprotectable and unavoidable trauma to this helpless anaesthetic foot, is the major factor , in the pathogenesis of lesions of diabetic foot. This is also aggravated by the absence of the vital trophic impulses to help in repairing the damage. These elements in addition