DIABETIC FOOT MANAGEMENT

BY

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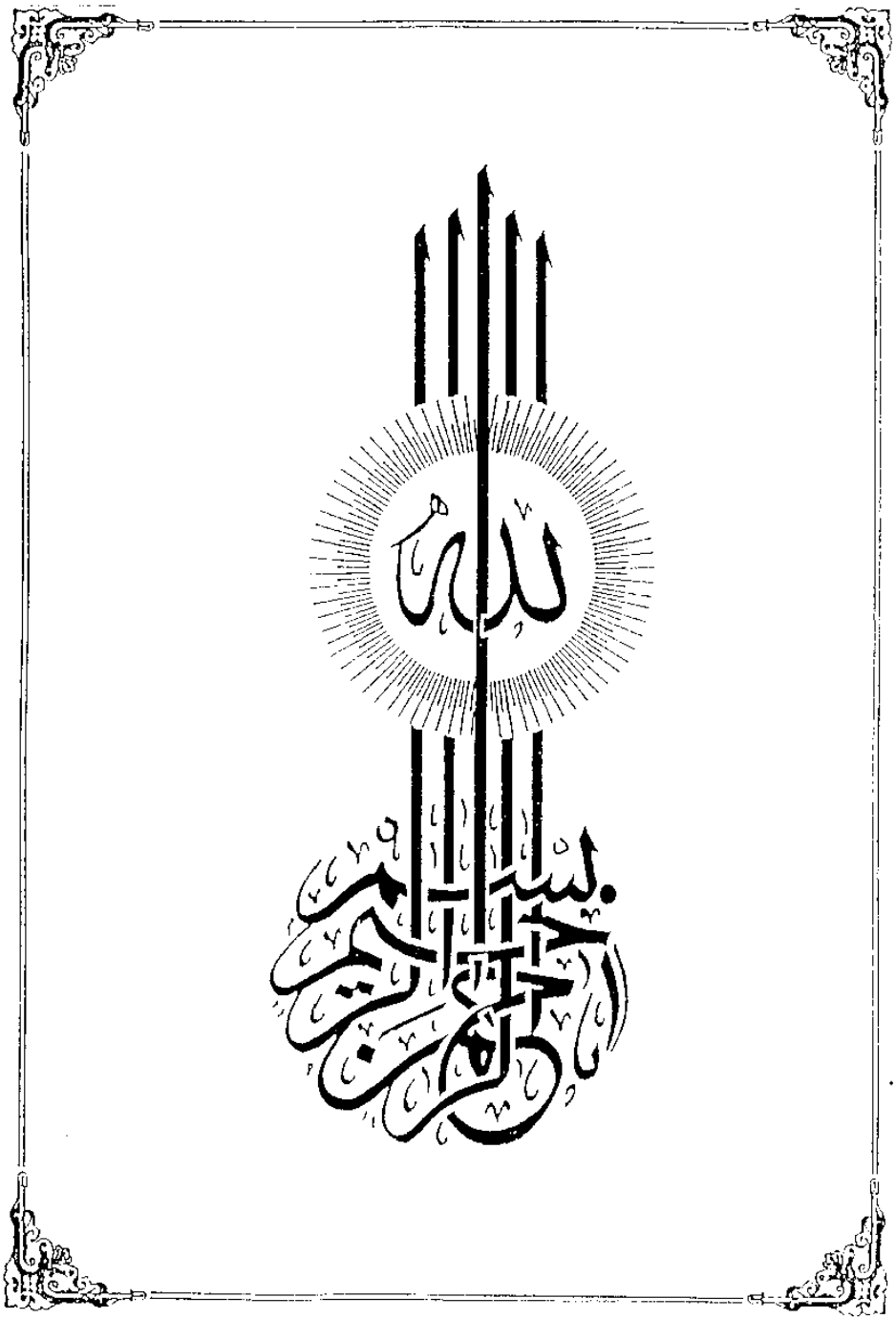

C O N T E N T S

I N T R O D U C T I O N

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Diabetic foot is a real challenge to the surgeon, to his skill proper judgement, his patience and his ability to make a good decision in the proper time.

The diabetic patient is a chronic one and so are his foot troubles.

If the surgeon is going to deal with and treat a diabetic foot he must be knowledgeable in the anatomy of the foot, aetiopathology and clinical presentation of a diabtic foot. together with proper understanding of the underlying angio-pathic and neuropathic precipitating lesions.

The management of a diabetic foot consists of good evaluation, good prophylaxis and treatment of established cases either conservatively or surgically.

Diabetic foot is also a social and economic problem.Its grave troubles bypass the patient to his family and community.

I. REVIEW OF LITERATURE

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Diabetic foot is the main problem that brings a diabetic patient to the surgeon. Such a poor patient faces the most devastating complication in the course of his illness.

Perhaps no where alse in the body do we see so clearly the ravages of these diabetic complications of vascular disease and neuropathy as in the foot of the diabetic, (Levin , M.E. 1977).

INCIDENCE

Survey's in the united states have shown 2 to 4% the population to have documented diabetes mellitus and a similar number to have abnormal glucose tolerance without having been previously diagnosed, (Cahill, Jr.,1979).

The incidence of foot lesions in the diabetic is so surprisingly high that it accounts for approximately 20% of diabetic patient admissions to hospitals, (Crofford , O. B. 1975)

It has been estimated that as many as 35 % of all diabetic patients , regardless of adequacy of treatment of the diabetic condition, develop some degree of neuropathy. These patients are prone to develop the complication of diabetic foot ulcer (Mal perforans).In Martin's series of 150 diabetics with neuropathy , 18 had mal perforans, (Martin, M.M. 1953)

The incidence of diabetic foot is the same for both sexes, (Malins, 1968). It increases with age and Obesity. Such changes usually start after 40,but most often between 60 and 70 years in old people, specially those who have a history of diabetes of 5 - 10 year's duration

There is no racial, residential or occupational difference in distribution , (Darling , et al, 1972).

In a postmortem study by Bell,1950,gangrene developed forty times more in diabetics than in nondiabetic subjects with atherosclerotic changes. Gangrene was a principal or contributing cause of death in about 65 % of diabetics with gangrene.

VASCULAR DISEASE

Complications of vascular disease are the most important causes of morbidity and mortality in the diabetic patient and have a lot to do in pathogenesis of diabetic foot.

Except for retinopathy and nephropathy ,angiopathic lesions observed in the diabetics are not pathognomonic of diabetes but differ from those of non diabeties in the following ;

(1) Occur at an earlier age , (Dry , Hines , Bell and Bencosome 1966 .)

(2) Progress more rapidly , (Warren , S. et al , 1966.)

(3) More Severe (Bell, 1957.) Bell has found that vascular changes leading to gangrene occur fifty-three times more frequently in diabetic men and seventy-one times more frequently in diabetic women when compared with the non-diabetic.

(4) Of more distal distribution affecting mainly the smaller more distal vesseles as the popliteal , anterior tibial and posterior tibial vesseles , (Haimovici and Oakley et al ,1968.) and (Marios et al , 1961.)

The profunda femoris artery , except for its origin was believed to escape angiopathic changes. A recent study by Terry A. King et al , 1984, proved that diabetics are more brone to involvement of profunda femoris artery with atherosclerotic changes than non-diabeties.

(5) Produces greater disability (Ferrier, T. M. 1965.)

Angiopathic changes in the diabetics can be divided into three categories ;-

(1) Diseases of Arteries mainly a theroslerosis and medial

(2) Diseases of arterioles mainly arteriolosclerosis.

(3) Diseases of capillaries including skin and muscle capillaries.

Atherosclerosis

Fibrous thickening of the intima of large vesseles apparently begins in infancy , (Haust , M.D. , 1971), if not in utero , and progesses throughout life ,(Wilens , S.L.,1951)

The cell recently implicated in the proliferative process leading to intimal thickening possesses the ultrastructural characteristics of smooth muscle cells,(Haust M.D.,et al , 1963) and (Ross , R.,et al ,1976.)

A second change commenly seen in the intima of large vesseles is the fatty streaks. These are belived to appear at an early stage but whether they are precursore of intimal thickening or not is still a point of controversy,(Williamson et al , 1977).

Medial Calcification

Here the media of muscular arteries, especially in the lower extremity , is calcified. The intima is not involved (at least not untill late in the course of the disease)

Arteriolosclerosis

It affects the smallest arteries ,that is those under 0.3 mm in diameter. It is characterised by hylinisation of arterioles.

It has been found to be much more pronounced in diabetics than in the general population.

Blumenthal et at, 1960, 1964, have called attention to proliferative changes in endothalium of arterioles and have suggested that such changes occur much more frequently in the diabetic than in the non-diabetic and that they have an immunogenic basis. More recently,Kilo and Williamson ,1977, suggested that these lesions are due to ischaemic injury possibly from occlusive disease of larger, more proximal vesseles.

Capillary disease.

It affects vesseles approximately 5-30 μm in diamter which lack a continuous layer of smooth muscle cells.

The single most characteristic ultra-structural feature of the various forms of diabetic micro-angiopathy, as evidenced by light and electron microscopic studies, is thickening of the capillary basement membrane (Kilo, C., Vogler, N., and Williamson, J.,1972)

Skin capillary changes.

Banson and Lacy, 1964, found that average skin capillary basement membrane width of the big toe was 13,300A^{o} in the diabetics. They found no correlation between basement membrane width and severity or duration of diabetes.

Muscle capillary changes.

Siperstein et al, 1968, reported that muscle capillary basement membrane thickening was demonstrable in over 90% of overt diabetics and was not influenced by age , sex , body weight or the duration or severity of diabetes.

Kilo et al, 1972 , and Pardo et al, 1972, however, have demonstrated highly significant thickening of capillary basement membrane as an aging phenomenon and revealed the presence of sex differences.

Williamson, J.R.,1971, found that muscle capillary basement membrane width increases greatly in the lower extremity in adults. Since the venous hydrostatic pressure increases as we proceed below the heart level in adults, strong evidence for an influence of venous hydrostatic pressure on capillary basement membrane width arised which theory was supported by the observation of Vracko , 1970.

Aetiology and role of insulin deficiency.

The pathogenic mechanisms involved in diabetic vascular disease are comparable to those of the non diabetics but are much accelerated.

Recently , there is some evidence that platelet release factors , which increase vascular permeability and stimulate smooth muscles to profiferate, are found, (Ross and Harker, 1976).These authors agree with Herman and Gorlin, 1967, Ostander et al , 1965, and Greenhalghet al,1971 that there is a close relationship between hyperlipidaemia, diabetes and premature atheroselerosis .

Smith , E. B., and Slater, R. , 1970 proved that the lipids of atherosclerotic plaques are derived from plasma lipoproteins. This comes in parallel with the work of Fredrickson et al, 1972 , and Greenhalgh et al, 1971, , that found that the prevalence of atherosclerotic vascular disease is increased in subjects with hyperlipidaemia and vice versa

Balodimos and Cahill, 1971, have pointed out that delayed or deficient insulin responses lead to increase in the level of circulating endogenous as well as exogenous plasma lipo - proteins. In diabetics glucose is metabolised to a polyol (Sorbitol). It has been suggested by Beaumont et al 1971, and Clements et al, 1969, that excessive metabolism of glucose to sorbitol may contribute to acceleration of large and small vessel disease.

According to Williamson and Kilo, 1977, capillary basement membrance thickening is greatly influenced by the duration of even mild insulin deficiency and the effect of a number of physiologic variables, for example, sex differences, aging , and venous hydrostatic pressure. Since the thickening is of segmentel nature , it is suggested that local or regional factors may play an important role in the pathogenesis of diabetic microangiopathy.

There is evidence that attempts to normalise blood glucose leveles may ameliorate the course of diabetic vascular disease as proved by the work of Jackson et al, 1975 , and Job et al , 1976.

Thickening of capillary basement membrane may play an important role in the increased susceptibility of diabetics to infection, (Banson et al , 1964,) Leucocytic emigration is affected specially in the lower extremity , where this thickening is the greatest. Lower extremity is therefore, the most susceptible part of the body to infection culminating in gangrene even in the absence of large vessel occlusion (Williamson and Kilo, 1977 .)

NEUROPATHY

Historical Aspects

Neuropathy in the diabetic patient was described by Rollo as early as 1790. Marchal following him in 1864 and in the following years detailed studies were done by many authors.

Incidence

Neuropathy is present in 50 % of diabetic patients, although clinically apparent in only about 5 % , (Bailey AA,1955) It may occur at any stage of the disease, even subclinical.

Clinical varieties.

Diabetic neuropathy is a common name for what probably constitutes several disorders of the peripheral system.Those conditions involving the lower extremities include ;-

(1) Progressive symmetrical distal polyneuropathy ,

(2) Ischaemic mononeuropathy multiplex,

(3) Neurogenic arthropathy ,

(4) Diabetic anhydrosis ,

(5) Diabetic amyotrophy ,

(6) Diabetic cold feet .

Aetiology.

Controversy still exists among different hypotheses for the causes of neurologic manifestations of diabetes.Field , 1966, and Eliasson , 1977, divided the pathogenic theories into two major groups; (1) Vascular insufficiency of neuvous system structures and (2) derangements of molecular metabolism in the cord and peripheral nerves, depending on the diabetic state. Fagerberg, 1965, published the results of sural

nerve biopsies from 151 patients, all of whom had diabetes , but not necessarily signs of neuropathy. The intra- neural vesseles were found to have thickened walls and narrow laminae. Two years Later, Bischoff demonstrated electron microscopic evidence of basement membrane thickening in endo - neural capillories.

Dolman CL , 1963, discoupled the nervous from the arteriolar alterations. Raff MC et al, 1968, Proposed the possibility of multiple small infarcts in the peripheral nerve trunks caused by ischaemic damage. The neuropathy is now thought to be due to a metabolic distrurbance and not ischaemia. The studies of Pirart (1978) proved that the incidence of neuropathy is related to the duration of diabetes and , to a lesser extent , to the degree of control of hyperglycaemia, but the mechanism by which prolonged elevation of the blood sugar damages nerves remains unknown, (Delbridge P J 1985)

Biochemical mechanisms that have been implicated include an accumlation of intraneural sorbitol (Gabbay K H,1975 and culebras A et al ,1981) ,a deficiency of nerve myoinositol(Greene DA et al 1975),glycosylation of nerve protein (Vlassara H et al 1983) and a reduction of axonal transport (Sidenius P et al 1982 .)

It seems more likly that neuropathy in most instances is produced combination of factors establised by the diabetic state and perhaps an inherent metabolic abnormality within the nerve itself corried by genes other than those of diabetes, (Levin M E 1977)