DIABETIC FOOT

Essay

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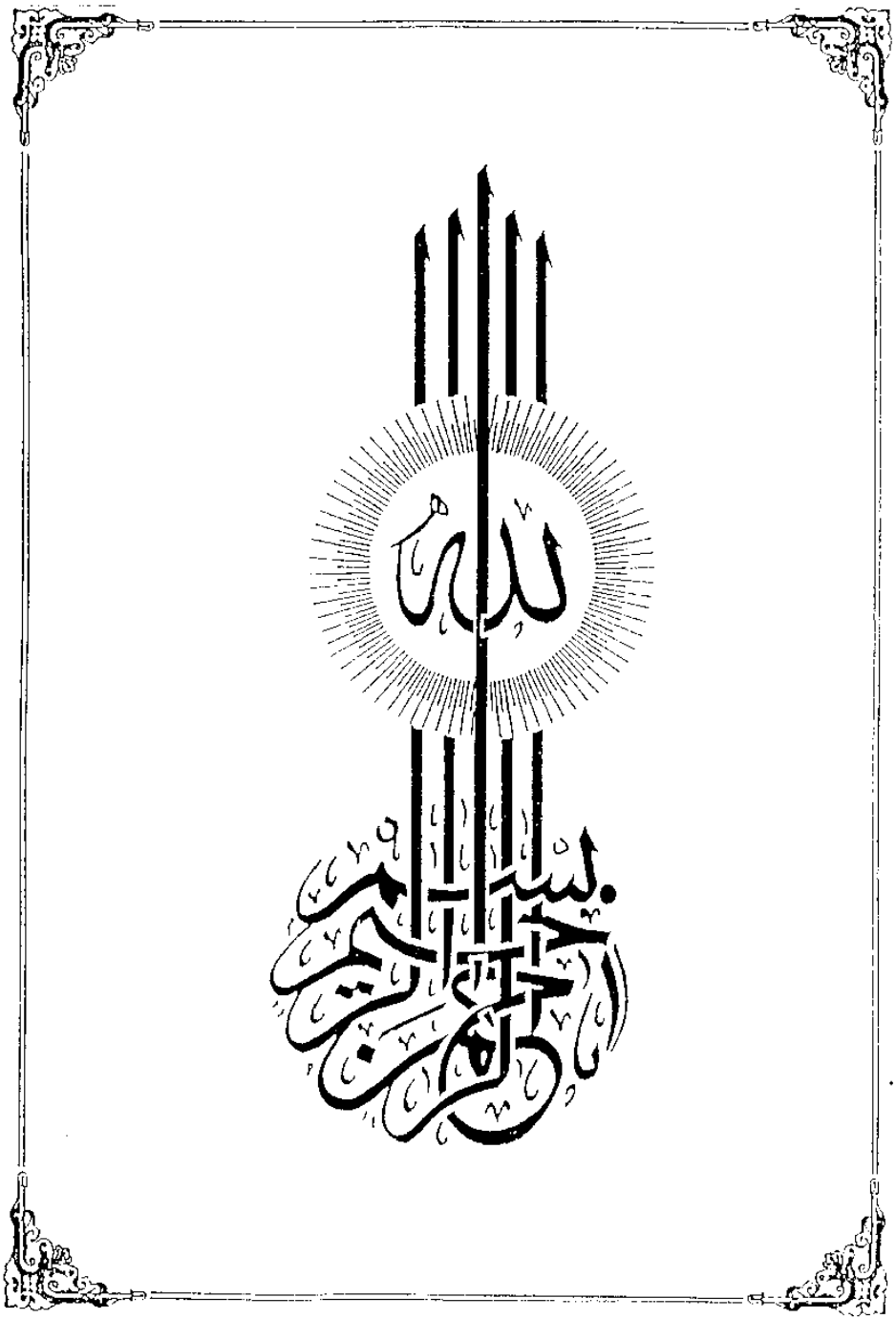

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INTRODUCTION

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The foot of the diabetic is especially susceptible to the diabetic complications of vascular disease and neuropathy. The interaction of these complications and perhaps hyperglycemia per se produces a wide panorama of clinical findings ranging from disorders of the nails, callus formation, and skin lesions to the involvement of the muscles and bones.

In such a vulnerable foot, trivial trauma may quickly lead to ulceration, infection and gangrene and to the final cataclysmic event, amputation.

Even the term (diabetic foot) is referred to now to describe a characteristic unique clinical entity in diabetic, peripheral arterial disease, neuropathy and infection contribute in variable degrees to the problem.

The relative incidence of foot, foot-leg and hand lesions in diabetics is 90%, 7.6% and 3.4% respectively. (Armed Forces Medical Journal,vol. XXII no. 2, 1980).

The anatomical peculiarities of the foot with its tense. fascial planter envelope which entraps inflammatory cellular exudate under tension, with its muscles layers and potential fascial spaces and with the long tendons constituting a high way for spread of infection from the sole to the leg in the neglected cases.

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 to nondiabetic, indicates 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, it requires an accurate monitoring of blood flow by the proper balance between the sympathetic and parasympathetic nervous systems in the presence of normally functioning muscles to assist and insure the flow of the blood. Finally, the diabetic foot is very liable to infection, this is because of two important pathogenic features which are the underlying metabolic disorders in diabetics and the extensive and widely variable bacteriologic and mycologic flora that contaminate the foot, as a normal constant flora.

The rationale of conservative management with control of the diabetics will provide a satisfactory function of locomotion and support. Diabetic foot is a highly delicate and complicated problem which should be handled from the start by a specialist and should not be looked at lightly whatever the initial presenting lesion seems trifle.

ANATOMY

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The skin of the dorsum of the foot contains hair follicles, sweat glands and some sebaceous glands. Its thickness is about 5 mm with the thickest areas covering the heel and distal metatarsals to sustain weight bearing effort. It has neither hair follicles nor sebaceous glands, but have some sweat glands.

The collagenous fibres of the dermis are connected to the deep fascia by dense fibrous septa.

The nails are cutaneous appendages. The nail folds constituting two paronychial potential spaces connected with each other posteriorly.

Those spaces are frequently infected (Lawrence, E.O'Neal, 1977).

The deep fascia on the dorsum of the foot forms a "Y" shaped thickening (inferior extensor retinaculum) which prevents the bow-stringing of the extensor tendons as they pass across the front of the ankle joint acting like an ankle strap.

The subcutaneous tissues in the sole differs from that of the rest of the body in being more fibrous. Fibrous septa divide the tissue into small loculi which are filled with a rather fluid fat under tension, so that the tissue bulges. This makes a shock absorbing pad especially over the heel.

The septa anchor the skin to the underlying plantar aponeurosis to improve the grip of the sole (Last, 1977).

The plantar aponeurosis is composed of dense white fibres running longitudinally from the heel to the toes. The foot is divided by the planter aponeurosis into superficial and deep plantar spaces.

The superficial plantar space is further divided into web spaces, corresponding to the position of the webs of the toes, interdigital spaces and the heel space, corresponding to the calcaneous. Web spaces are four triangular regions between the dorsal and plantar skin filled with loose fat that bulges between the divisions of the plantar fascia. Interdigital spaces are subcutaneous areas that lie between the five digital slips of the plantar aponeurosis. The shafts of the metatarsals border the spaces on the sides.

The deep plantar space is divided into central, medial and lateral spaces by vertical fibrous intramuscular septa arising from the borders of the central part of the plantar aponeurosis. Fig. 1

The space between the medial and lateral intramuscular septa is the central deep plantar space. It is further divided by the four layers of plantar muscles. The space medial to the intramuscular septum is the medial deep plantar space and those lateral to the lateral intramuscular septum are the lateral deep plantar spaces.

Fibrous septa extend from the phalanges to the medial and lateral aspects of each toe.

Extensor digitorum brevis is the only muscle of the dorsum of the foot while the plantar aspect has four layers of muscles.

The lumbricals are in the second layer and attached to the flexor digitorum longus tendon transmit the infection from the web to the deep layers. Fig. 2

There is also a communication between the sole and posterior leg compartment through a tunnel under cover the abductor hallucis on the medial surface of the sole transmitting the tendons of tibialis posterior, flexor digitorum longus and posterior tibial artery and nerve. Through this communication, the infection can spread to the leg in neglected cases.

The sciatic nerve is the main motor and sensory innervation of the foot, derived from L_4, L_5, S_1 and S_2.

Other supply from the femoral nerve through the saphenous nerve are given.

The autonomic nerve supply is furnished through the arterial branches by plexuses around the arteries.

The arterial supply of the foot is derived from the popliteal artery by its two divisions, the anterior and posterior tibial arteries.

The venous drainage of the foot follows the arterial supply except in the medial superficial marginal veins from which originate the long saphenous and the lateral marginal vein from which originates the small saphenous (Lawrence E. O'Neal, 1977).

PATHOLOGICAL CHANGES

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A further causes of illness and disability in diabetics are the complications affecting the lower limbs; more hospital beds are occupied by diabetic patients with such complications than for all other causes associated with the disease (Dormandy, 1979).

Oakelt et al. (1956) drew attention to the importance of peripheral neuropathy as a cause of plantar ulceration, and described three factors: (arterial disease, neuropathy and sepsis) as being the essential causes of foot lesion in diabetics. This concept was extended by Du Plessis, 1970, who emphasized the importance of inter-relationship between three principal etiological factors: vascular disease, haematological changes and neuropathy and the development of lesions in diabetic foot.

I) Neuropathy

Diabetic neuropathy can affect both the peripheral nerves and central nervous system. At least 35% of all diabetics will develop a degree of neuropathy that can be detected on gross clinical examination.

It was long assumed that these neurologic changes were secondary to changes in the vasa nervorum, until Thomas and Lascelles (1966) found that there are rarely changes in the local vascular supply of nerves. Ischemic infarction is sometimes seen, but this generally presents a rapid-onset

mononeuropathy, not as more diffuse pattern usually seen in diabetics (Ellengberg et al., 1959).

Charpra et al. (1969) demonstrated that the peripheral neuropathy of diabetics is more likely the result of an intrinsic metabolic abnormality of the Schwann cell. This almost involves more than one enzyme system, but which, if any of these defects is primary or purely speculative at the present time.

Pathological examination of affected nerves shows segmental demyelinations and axon loss, most severe distally (Thomas & Lascalles, 1966). This was originally thought to indicate that both the Schwann cell and the axon were primarily affected. However, it is currently thought that the demyelination could be secondary to axonopathy (Dyck et al., 1980). The neuropathy is now thought to be due to a metabolic disturbance, and not ischaemia. The studies of Pirart (1978) gave scientific evidence to the clinical impression 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 remain unknown.

Biochemical mechanisms that have been implicated include an accumulation of intraneural sorbitol (Gabbay, 1975 and Culebras et al., 1981), a deficiency of nerve protein (Vlassara et al., 1983), and reduction of axonal transport (Sidenius & Jakobsen, 1982).