INCIDENCE AND TYPES OF INFECTIONS AMONG EGYPTIAN DIABETICS

THESIS

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By

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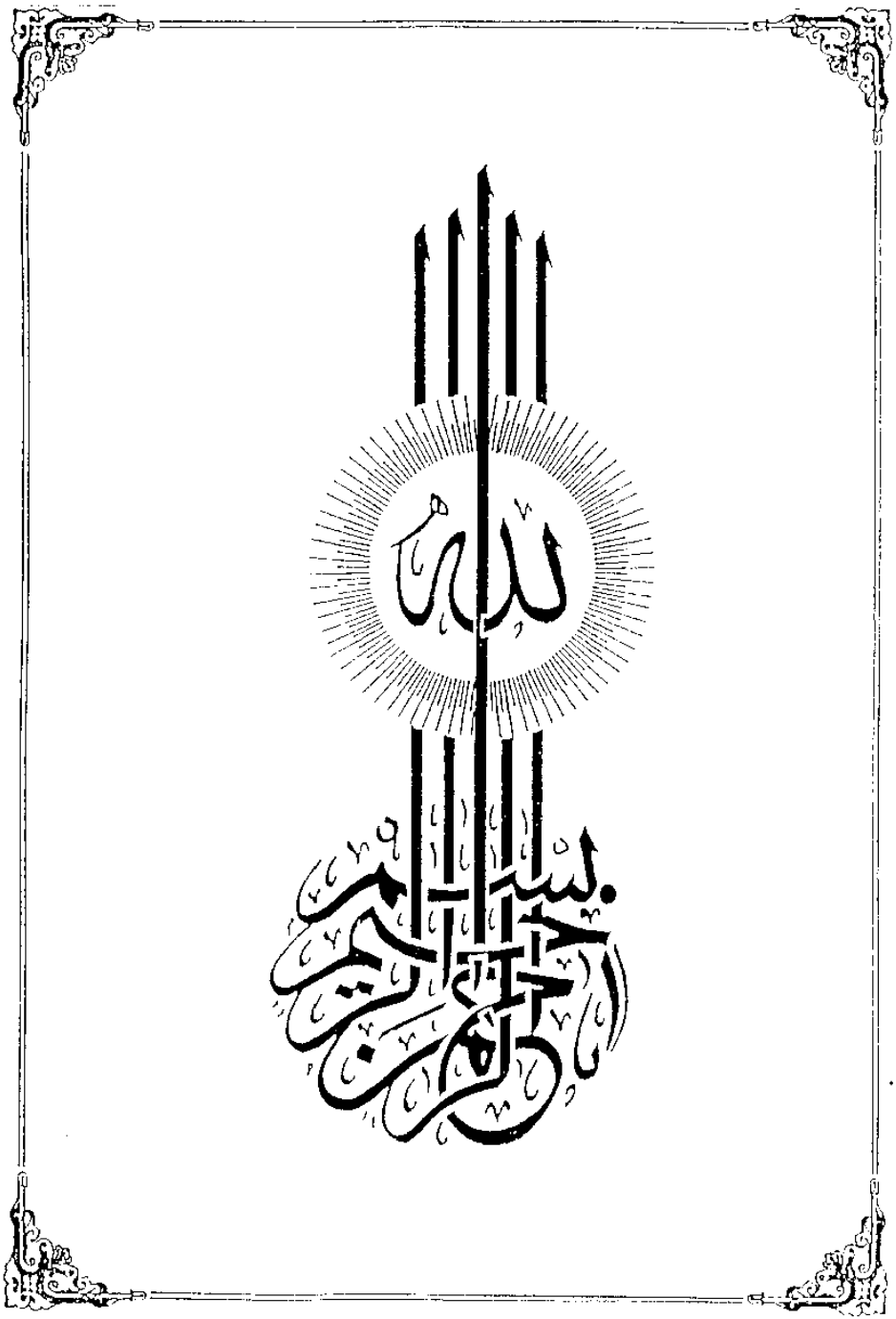
TO MY WIFE



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INTRODUCTION

The relationship between diabetes and infection has been, for many years, a topic generating intensive interest, extensive argumentation and research within the medical profession.

Clinicians have long suspected that diabetic patients are inherently more susceptible to infection than nondiabetics (Bagdade, 1976), and that infections that do occur are more severe (Axline, 1982). The patient with uncontrolled diabetes has less than normal resistance and infection has often been the cause of death in diabetic, particularly prior to the introduction of insulin and modern chemotherapeutic agents and antibiotics (Joslin et al, 1959).

In the preinsulin era the average life span for a diabetic was 1.5 years after the diagnosis was made. In the postinsulin era before the availability of antimicrobial agents the average life span was seven years after the diagnosis. In the current postantimicrobial therapy era the life span of diabetics is nearly two-thirds that of the nondiabetic patient (Axline, 1982).

It is doubtful, however, that diabetes can be caused by an infection or made permanently more severe except by certain rare infections of the pancreas itself or except in those individuals in which latent or unrecognized diabetes is activated by the stress of an infection (Joslin et al, 1959).

Early studies failed to show the exact nature of the lowered resistance to infection which is common in uncontrolled diabetes (Joslin et al, 1959) and found no consistent abnormality in the host defense mechanism of diabetics with well-controlled disease. These findings were consonant with clinical experience that diabetics resist infection normally when adequately treated (Bagdade, 1976).

More recently, an increasing body of evidence has suggested that insulin deficiency alters metabolism in a number of different tissues, causing disturbances that may complicate management of diabetic patients.

Studies of host defense mechanisms in insulin deficient patients and in experimental animal models, with and without ketoacidosis, suggest that moderate to severe insulin deficiency may impair the ability of granulocytes to perform a number of vital defense functions (Bagdade, 1976).

Rayfield et al (1978) suggested that the increased susceptibility to infection in diabetics may relate to local factors in specific target organs as well as to generalized defects in bactericidal activity and opsonic activity.

OBJECTIVE OF THE THESIS

The objective of this thesis is to study the following :

1) The incidence of infections in patients with diabetes mellitus.

2) The types of infection.

3) The localization of the sites of these infections.

REVIEW OF THE LITERATURE

Common infections in diabetes comprise skin and urinary tract infections. Less common infections include tuberculosis, gas gangrene and mucormycosis (Bagdade, 1976).

Skin Infections :

The skin is a unique organ, but its uniqueness causes complications in patients with diabetes mellitus. A brief review of basic anatomy and recent physiology research aids in understanding these complications.

The skin has three major compartments :

1) The intracellular compartment (primarily the epidermis).
2) The extracellular extravascular area (the connective tissue with its ground substance "sea".
3) The extracellular vascular tree.

The intracellular compartment, the epidermis, is concentrated on the outside and provides the main barrier that separates the internal milieu from the hostile environment. The epidermis along with its pilosebaceous apparatus constantly replaces itself every month to maintain its continuous encompassing integrity.

Below the epidermis is the dermis or the main body of the skin (approximately 95 percent of the skin by weight). This large extravascular compartment normally holds about 5 percent of the body's water. The solgel character of the dermis allows diffusion in and out of this compartment at a rate lower than that in any other significant extracellular extravascular compartments of the body. Its glucose content is a transudate from the blood (Fusaro and Goetz, 1971). Fusaro (1965) has shown that during a rapid rise in glucose concentration in the blood (i.e., after rapid intravenous injection of dextrose) approximately 15 percent of the blood glucose load appears in this dermal compartment and reaches peak concentration 30 minutes after the injection. The blood glucose levels return to prefasting levels in one hour; whereas, the dermal glucose concentration does not return to prefasting levels for two to three hours. This excess dermal glucose (amount above fasting levels) constitutes a large passive glucose reservoir that helps to prevent hypoglycemia because the skin slowly leaks glucose back to the blood. In fact, as a storage organ the skin is second to the liver in maintaining blood glucose levels.

The skeletal muscle (intracellular) absorbs the vast majority of blood glucose (nonportal), but cannot return glucose directly to the blood; therefore, the muscle cannot act as a direct balancing reservoir as does the skin. In addition, the extracellular compartment of the muscle has physicochemical characteristics similar to those of the

vascular compartment and is an extension of that compartment; therefore, the extracellular muscular component is in rapid equilibrium with the vascular tree and does not contribute to sequential buffering capacity (Fusaro and Goetz, 1971).

Although there are no diseases of the skin which are absolutely unique to diabetes, certain affections are more common in diabetics than nondiabetics. Among these are infections with furuncles, boils and carbuncles, formerly more common than now. A diabetic is particularly prone to infections if he has dry or cracking skin (Joslin et al, 1959).

The slow glucose diffusion in dermis becomes even more prominent in the diabetic (Fusaro and Goetz, 1971). Fusaro (1965) has shown that in diabetics dermal glucose disappearance rates are very low 0.3 percent per minute versus the normal of 2 percent per minute. Thus, directly under the epidermis there is a large pool of glucose that provides an ideal medium for cutaneous infection, a common skin problem of diabetics (Fusaro and Goetz, 1971).

The cutaneous surface of the skin has a bacterial population of both pathogenic and nonpathogenic organisms, (Marples, 1965), both capable of producing infections. The potential for cutaneous infection is always present; however, the integrity of the epidermal barrier must be broken before bacteria has access to the internal milieu.

Once the organisms have gained access to the internal compartment (extracellular extravascular ground substance "sea"), the "rich" glucose concentration of the diabetic's dermal compartment is fertile soil for bacterial growth. Bacteria and Candida grow rapidly in the presence of glucose, which they prefer as an energy source (Fusaro and Goetz, 1971).

Staphylococcal and candidal mucocutaneous infections are common in the general population, and their frequency is not increased in well-controlled diabetic patients (Bagdade, 1976). However, candidal vaginitis and Candida infections especially in the intertriginous areas (groin, axillae and inframammary) are extremely common in the presence of hyperglycemia and may be refractory to treatment if improvement in diabetic control and lowering of blood glucose levels are not achieved simultaneously (Fusaro and Goetz, 1971 ; Bagdade, 1976). Candida originates in the gastrointestinal tract and thrive in the moist climate of the intertriginous areas of the body, where they find the moisture they require to carry on metabolic processes and acquire food (e.g., glucose) from the environment. The anus acts as a source of Candida, which spreads from the anogenital area by direct contact. In addition, the hands, especially during sleep, transport the organism to the rest of the body. In the diabetic, especially the obese female, the high moisture and glucose content of the intertriginous regions invite proliferation of Candida (Fusaro and Goetz,1971).

The characteristic clinical appearance of candidiasis is a fiery red, exudative dermatitis with lesions that are sharply demarcated but shows small pustular satellite lesions beyond the periphery (Fusaro and Goetz, 1971).

Superficial candidal intertrigo is less important in itself but is a possible entry point for secondary bacterial invasion (Bagdade, 1976).

Infection of the Diabetic Foot

Infections of the diabetic foot dictates the consideration of two important pathogenic features :

1) The underlying metabolic disorder with its common tissue degenerative complications.

2) The bacteriologic flora of the lower limb.

Considering the bacteriology of the skin, on the drier portions of the lower leg of the flora is quite restricted (about 1000 bacteria per square centimeter) due to the physical and chemical conditions on the surface. The critical importance of moisture in bacterial and fungal growth is indicated by the density and species variety of organisms in the toe webs, which are even greater than in the axilla, where more than 1 million bacteria per square centimeter can be recovered (Beaumont et al, 1971).