RENAL AFFECTION IN URINARY SCHISTOSOMIASIS

THESIS PRESENTED

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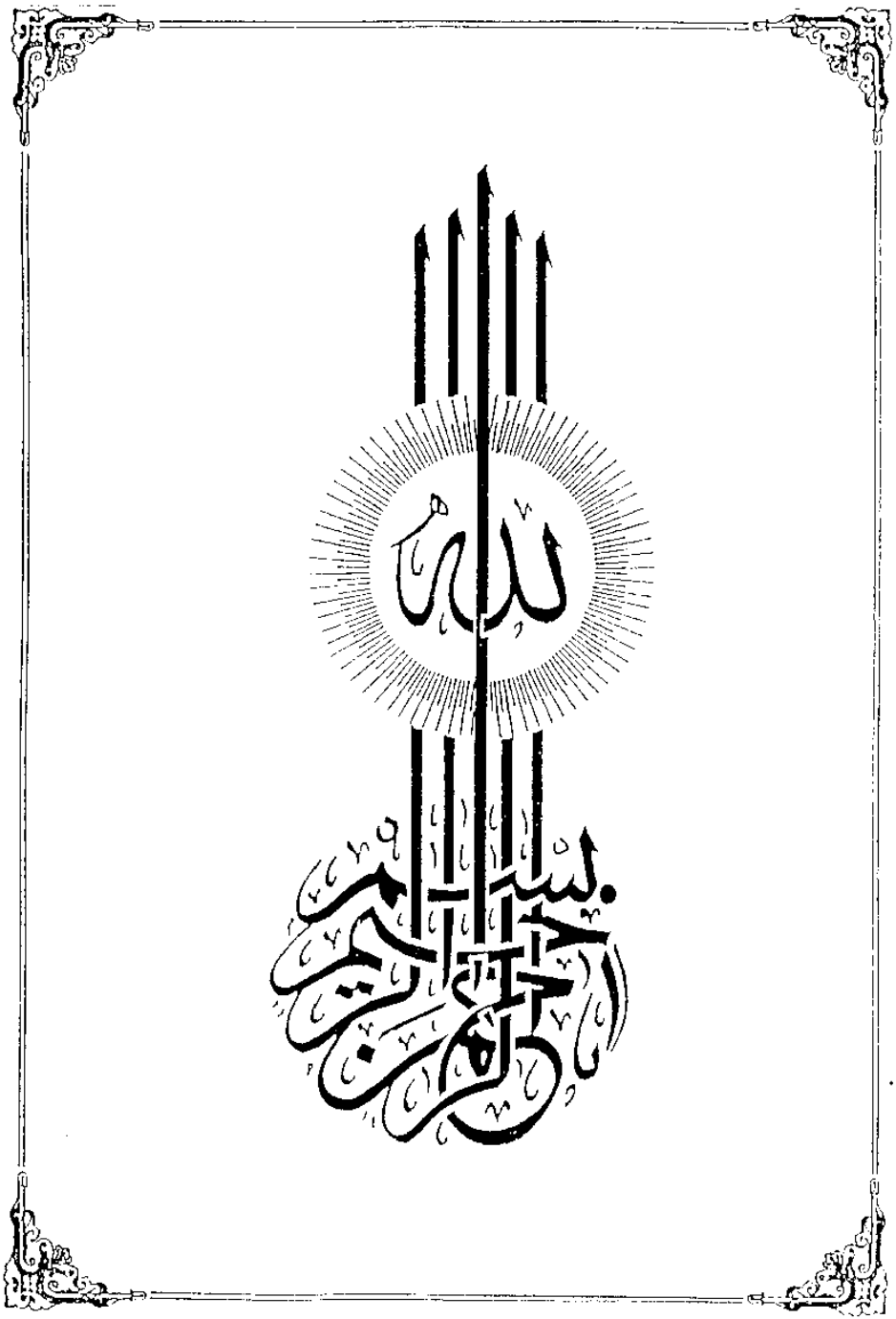

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INTRODUCTION AND AIM OF WORK

Schistosomiasis (Bilharziasis) has been found to exist in Egypt since the Pharaonic era (Wilcocks,1962). Schistosomiasis is the most prevalent disease in Egypt; and next to malaria, the most prevalent disease in the world (Kagan, 1962). Unfortunately the ill-effects of the disease are underestimated, as it is not directly a contageous or a killing disease.

Schistosomiasis is a national problem, not only because of its endemicity and high incidence, but also because it is intimately connected with our economic resources. The reduction in the total economic production has been estimated to be 30% as a result of incapacitation of a large segment of the population (Mousa, 1969). Farouq (1967) estimated the annual economic loss to be 560 million dollars per year.

Weir et al (1952) proved that all inhabitants in rural areas exposed to infection are usually infected. The number of infected individuals in Egypt was calculated to exceed 14 millions (Mc Mullen, 1963 and Farouq,1967).

It has been estimated that more than 16 millions amongst the population of Egypt suffer from this infection or from its complications (Mousa, 1969).

According to Scott (1936), Schistosoma haematobium infects 60% of the people in the Delta and those parts of the Nile Valley under perinneal irrigation but only 5% of the people in the districts under basin irrigation. After establishment of the Aswan Dam, most of the agricultural land will be under perinneal irrigation, and the aforementioned percentage is likely to be higher, despite the improvement in sanitation and hygienic equipments.

Urinary tract schistosomiasis, affecting children and young adults, is not generally considered as a serious infection so long as it is causing only haematuria and painful micturation. These symptoms cause little physical destress and have been frequently considered by children, their parents, and even some doctors as a phenomena of minor importance. The afore-mentioned impression is particularly held by Eldson-Dew (1967), Edington (1967), Macdonald and Foreayth (1968).

In sharp contrast to the above opinion, Girgis (1934), Honey and Gelfand (1960), Nabawy et al (1961) and Prates (1962), have reported that urinary bilharziasis in children and young adults is not the benign unimportant infection which many authorities consider it to be and that serious lesions of the bladder, ureters and kidneys occur at an early stage.

Renal involvement is considered to be one of the most important complications of urinary schistosomiasis. Chronic renal failure, the final inevitable end, supervenes, sometimes acutely, but more commonly insidiously over years.

While hepatosplenic and cardiopulmonary bilharziasis, especially the former, have been well studied, the renal affection in urinary tract schistosomiasis has not been adequately studied (Farid et al, 1967). Moreover, the great development achieved in nephrology discipline opened the field for exploring this problem.

However, the great bulk of the clinical literature is concerned more with the structural changes in the conduit system, than with the effects of obstruction

of 10 after antimonial therapy, while the blood urea nitrogen and the serum creatinine were within normal range in the 10 patients studied.

Wolf (1968) in Ghana, estimated the blood-urea nitrogen in 51 patients with urinary schistosomiasis and he found it abnormal only in one patient.

Since the kidney suffers secondarily to lower urinary obstruction by back pressure and/or secondary infection, the medulla is expected to carry the brunt of impairment early before the cortex; in spite of that, the medullary functions are still remaining without thorough study.

The AIM of this work is to study the functional and structural changes of the kidney at an early stage of urinary tract schistosomiasis, when the disease is still localized to the lower urinary tract and considered as a mild disorder, and to try to relate these changes to obstruction in the urinary passages and/or reflux associated with infection. It is of great

importance to find out whether these changes are reversible by antibilharzial treatment or not. Also one of the most important items of our study is to demonstrate the stage of the disease at which reversibility of these lesions could occur.

REVIEW OF LITERATURE

I - REVIEW OF THE LIFE CYCLE OF THE SCHISTOSOMA WORM

Bilharz (1852) in Kasr-El-Aini Hospital discovered the adult worm in the mesenteric veins of an Egyptian boy at postmortem. Manson (1893) realizing the presence of two types of ova suggested the existence of two different species of worms and Sambon (1908) discovered two species of worms. Ruffer (1910) demonstrated Schistosoma ova in the kidneys of Ancient Egyptian mummies dating back to 1220-1000 B.C. Lieper (1915-1918) discovered the intermediate host of the parasite in Egypt after it has been discovered by the Japanese workers in Schistosoma japonicum.

The life cycle of schistosomiasis involves man, or other warm-blooded vertebrates, and a suitable fresh water snail.

Schistosomal infection occurs early in life being 17% at 2-4 years and 45% at the age of 15 years, reaching

its maximum at 16–20 years, then it continues to fall gradually till it reaches only 20% at the age of 50 years (Azim, 1948). Mousa (1969) considers the maximum incidence to occur between 8–12 years.

In urinary schistosomiasis, ova are voided in the urine (mainly haematobium, and rarely mansoni). The number of ova varies from 500–3000 eggs per day for each worm in the body (Mousa, 1962). On reaching fresh water the living ova hatch their miracidia by a process of osmosis, the latter swim actively in water and are attracted to the appropriate snail intermediate host by chemotaxis and eventually reach the liver of the snail. In a month or so, cercariae begin to emerge from the snail in great numbers, each miracidium gives rise to about 250,000 cercariae all of one sex.

The cercaria is the infective agent to man and perishes in 48–72 hours if it does not reach the definitive host. At this stage of invasion the cercariae shed their tails, pierce the human skin, rarely the bucco-pharyngeal mucosa, and reach the general circulation either directly by going through the superficial