ASSOCIATED CLINICAL AND HISTOPATHOLOGICAL CHANGES IN BILHARZIASIS OF THE FEMALE GENITAL ORGANS

A Thesis

Submitted for the Partial Fullfilment of Master Degree

In Obstetrics and Gynaecology

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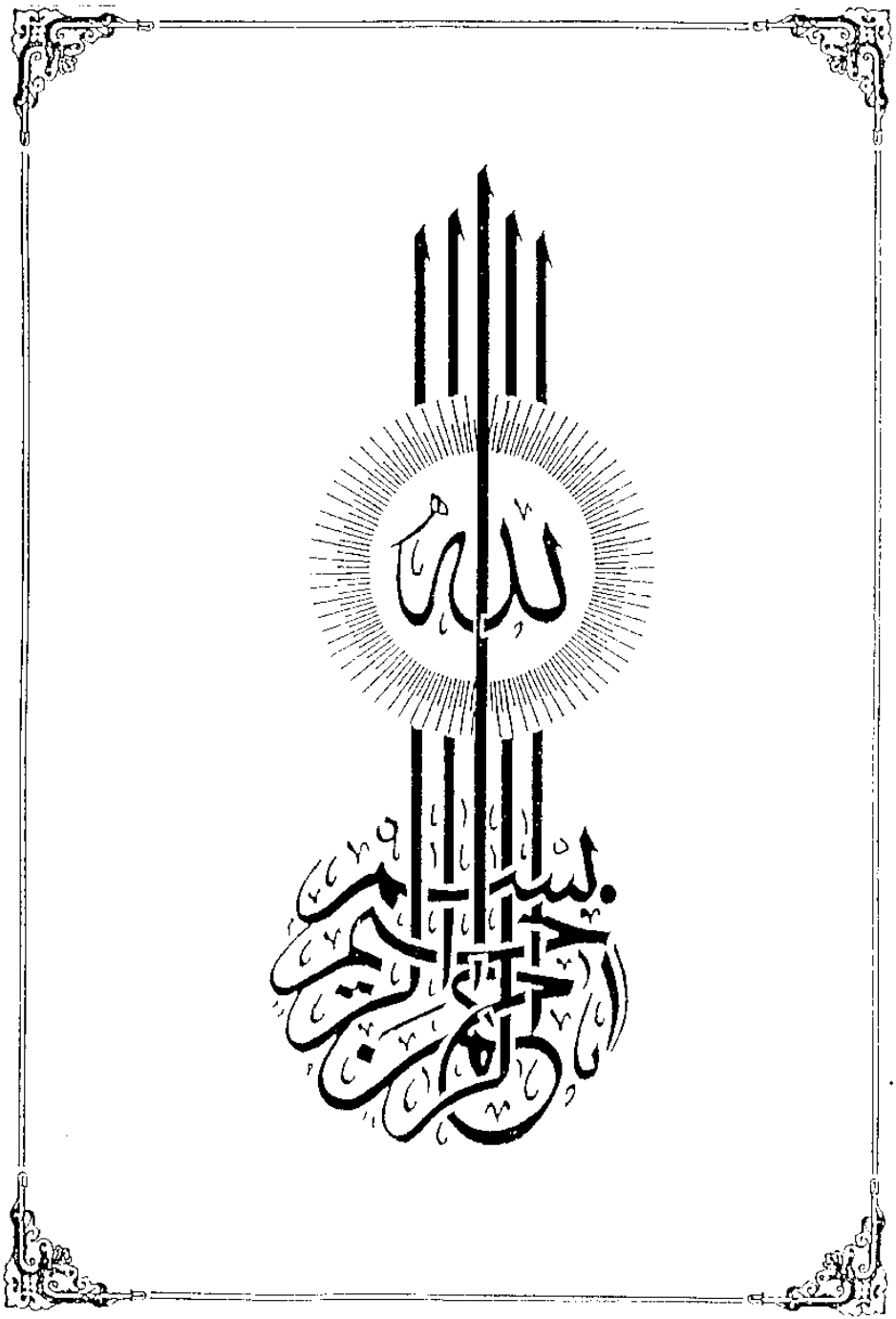
1990

Acknowledgement

I wish to express my deepest gratitude and most sincere thanks to ***Dr. Moneir Mohamed Fawzy El-Hao,*** *Assistant Professor of Obstetrics and Gynaecology Department, Ain Shams University, who inspired in me the spirit of research. I will allways remember his unforgettable sincere encouragment and kindness.*

I would like to express my deepest gratitude and cardinal thanks to ***Dr. Mohamed Adel El-Nazer,*** *Lecturer of Obstetrics and Gynaecology, Ain Shams Univeristy and* ***Dr. Ahmed Sa'ah Kamel,*** *lecturer of Pathology, Ain Shams Univeristy, for help and planning for this work, for their supervision and thier kind guidance throughout the course of this work.*

I wish to thank all the members of the Cytodiagnostic Unit, Obstetrics and Gynaecology Department, Ain Shams Univeristy Hospitals, for their excellent co-operation in the achievement of the parctical part of the work.



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INTRODUCTION

Schistosomiasis is a common and important parasitic disease affecting man in most of the tropics of Africa, Middle East, northern part of South America and South East Asia. It is caused by species of the genus schistosoma. According to WHO estimates, approximately 250 million people suffer from the disease and more than 600 million people are living in areas where schistosomiasis is endemic *(Danish, 1981)*. Schistsomiasis is endemic in Egypt and about eighteen million Egyptians are affected by the diseases *(Hadidy, 1964)*.

Schistosomiasis is essentially a disease of urinary or intestinal tract, but no other system in the body is immune. It may produce a variety of lesions in any part of the female genital organs from the vulva to the ovary.

Schistosomiasis of the female genital organs was first described in the Nineteenth Century. *Petridis (1899)* reported two cases of Schistosomal lesions of the cervix. In the same year *Madden (1899)* described three cases of the vulva, vagina and posterior lip of the cervix. Since then many others have reported on various aspects of the disease.

Magdi and Hefnawi (1951) reported that the incidence of sterility among patients with Bilharziasis of the different parts of the genital tract was 44%.

Attia (1962) referred to Bilharzial endometritis as a cause of abortion while *Magdi and Hefnawi (1951)* had considered it a rare lesion. Cervical carcinoma was recorded to be associated with Bilharziasis *(Shafeek, 1961)*.

AIM OF THE WORK

The wide prevalence of bilharziasis in Egypt has prompted us to look for the clinical presentation, complication or any other pathology that may be associated with bilharziasis of the female genital organs.

REVIEW OF LITERATURE

HISTORY OF DISCOVERY OF BILHARZIASIS

Our great ancestors recognized and described bilharziasis. They also advised it's prophylaxis and treatment. The papyri of Ebers gave several prescriptions for the treatment of haematuria. As the ancient Egyptians thought that the infection was introduced through the male urethra, they advised the use of a penile sheath for prophylaxis *(Ibrahim, 1929)*.

In the twelfth and thirteenth century, Bilharziasis was so prevalent in Egypt that haematuria was repeatedly mentioned by Arab physicians. Haematuria was described by their famous biographer Ibn Battouta who recorded that the males of Lower Egypt "menstruated" like their women; obviously mistook the haematuria of bilharzial infestation of the bladder for the monthly periods *(Khalil, 1928)*.

In 1851, the German Theodore Bilharz discovered the causative parasite at autopsy in the portal vein of a young man. Later on, he demonstrated the ova microscopically in the bladder lesions. He described ova with both terminal and lateral spines, but at that time he thought that they were produced by the same worm *(Hamilton, 1955)*.

In 1859 Cobbold suggested to call it genus Bilharzia immortalising the name of the man responsible for the

discovery. In 1870 Cobbold also conducted the first experiments to trace the life history of the parasite in an invertebrate intermediary host *(Magdi and Hefnawi 1951)*.

Sonsino (1893), an Italian helminthologist working in the public health laboratories with access to Kasr-el-Aini Hospital, published an important paper on Schistosoma Haematobium. For the next ten years he devoted himself to the task of solving the life history of the parasite. His work was mostly directed to fresh water snails which he thought to act as an intermediate host.

In 1888 Allan formulated the hypothesis that an unknown larval stage might enter the body through the skin. This interesting observation was based on the fact that the youths who bathed in the river water became infected while the girls who did not bathe remained free of the disease *(Christopherson, 1920)*.

In 1893 Looss came to Egypt from University of Liepzig as a special research professor at the Kasr-el-Aini Hospital. For over twenty years he made patient studies on the life history and the mode of transmission of the parasite. However his experiments showed that the infection most probably enter the human subject through the skin. This opinion was shared by others and later proved to be correct *(Looss, 1910)*.

Sambon in 1907 confirmed the presence of two species of Bilharzia as was stated by Manson in 1893, and established the presence of bilharzia mansoni as a separate species, producing lateral spined eggs and invading chiefly the bowel and bilharzia haematobium producing terminal spined ova, and invades chiefly the genito-urinary tract. Till this time, it was thought that bilharzial infection was directly transmitted from man to man *(W.H.O. 1957).*

In 1909 Miyairi and Suzuki of Japan worked out the life cycle of Bilharzia Japonicum. It became apparent that an intermediate stage in the life history of the parasite existed in the infected water, and was capable of invading the skin of victims. Following this key, *Miyairi and Suzuki (1913)* discovered that fresh water snails acted as the intermediate host *W.H.O. (1961).*

In Japan in 1914, Leiper and Atkinson saw the intermediate snail and noted its strange attraction to the miracidia and later found its liver packed with bifurcate cercariae. Later on, Leiper confirmed the same life cycle of Bilharzia haematobium and Bilharzia mansoni of Egypt *(Leiper 1918)*

In 1918 Christopherson of Khartoum showed that Bilharziasis can be definitely cured by tartar emetic administered by intravenous route *(Christopherson, 1920).*

In 1929 Khalil in association with Petro introduced a better tolerated antimony compound, Fouadin *(Khalil and Betache. 1930)*. Furthermore, from that time, modification of therapy of Bilharziasis have been achieved by different new drugs.

As the incidence of infection is much higher among males, bilharziasis of the female genital organs is relatively uncommon. In 1928 Shafeek began a detailed study of female genital bilharziasis at Kasr-el-Aini Hospital *(Shafeek 1949)*.

GEOGRAPHICAL DISTRIBUTION

Schistosoma haematobium is found chiefly in North and East Africa *(Rifaat and Morsy 1970)* and extends from Morocco through Algeria, Tunisia, Egypt and the Nile Valley to Sudan, East, Central, West-South Africa and the island of Madagascar and Mauritius. In Asia, the disease is restricted to the West, in Palestine, Persia, Iraq, Syria, Yemen and Bombay in India. The only European focus is in Southern Portugal and Spain, while the disease occurs in the island of Cyprus in the Mediterranean. In Egypt, it is found all-over the country although relatively less common in areas of basin irrigation in Upper Egypt (not used now).

Schistosoma mansoni is common in Africa chiefly the Nile Delta in Egypt, extending as far south to Cairo, East, Central and West Africa and Madagascar. The condition is fairly prevalent in Central and South America in the West Indes and Brazil.

Schistosoma japonicum is confined to the Far East, particularly, Japan, China, Formosa and Philippines.

Schistosoma intercalatum is confined to Congo, in areas where Schistosoma haematobium infection and cattle Schistosoma do not exist. It is a parasite of the veins of the large intestine of man and the adult resembles Schistosoma haematobium.

Schistosoma Matthei is met with in Southern Rhodesia and South Africa. It is a parasite of the veins of the large intestine.

LIFE CYCLE

The ova are deposited by the female worms in the smallest venules of the affected hollow viscus *(Khalil and Shaaban, 1962).*

The successful ova, assisted by the cytolytic action of the fluid secreted by the miracidium and the contraction of the hollow viscus move through the tissues towards the lumen of the affected organ and leave the body of the host with the urine faeces or menstural blood flow *(Shaaban, 1960).*

Coming into contact with water, the ova hatch and give rise to ciliated miracidiua which enter a fresh water snail which acts as an intermediate host. It is named either molluscum Bullinis (Schistosoma haematobium) or Planorbis (Schistosoma mansoni).

In the liver of the snail, the miracidium soon becomes a thin walled saccular sporocyst which produces several daughter sporocysts. In about six weeks, a tiny forked tailed cercariae escape from the snail into the surrounding water.

The cercariae must find a suitable definitive host to continue their life cycle. They enter the skin, cast off their tails and by direct motion and lysis gain entrance to the lumina of the superficial venules. They

are then carried by the blood to the right side of the heart then the lungs where some cercariae pass through the lung capillaries to the left side of the heart and are distributed all over the body. Only those which get to the mesenteric veins reach the hepato-portal system where they grow until sexual maturity. After maturity, they copulate and migrate against the venous blood system until they reach the small venous radicles of the portal vein and its tributaries. When the lumen of the venules allows no further progress, the female schistosoma leaves the male partner to deposit its ova.

Mansoni worms pass to the mesenteric and haemorrhoidal veins and settle in the venules of the small intestine, colon and rectum. Some enter the veins of the lower urinary tract.

Haematobium worms follow the same route and reach the haemorrhoidal veins and other pelvic and perineal veins that anastomose with the haemorrhoidal veins.