HISTOCHEMICAL AND HISTOLOGICAL STUDIES OF THE EFFECT OF SCHISTOSOMA MANSONI INFECTION ON THE LIVER, SPLEEN AND PANCREAS OF HAMSTERS

Thesis
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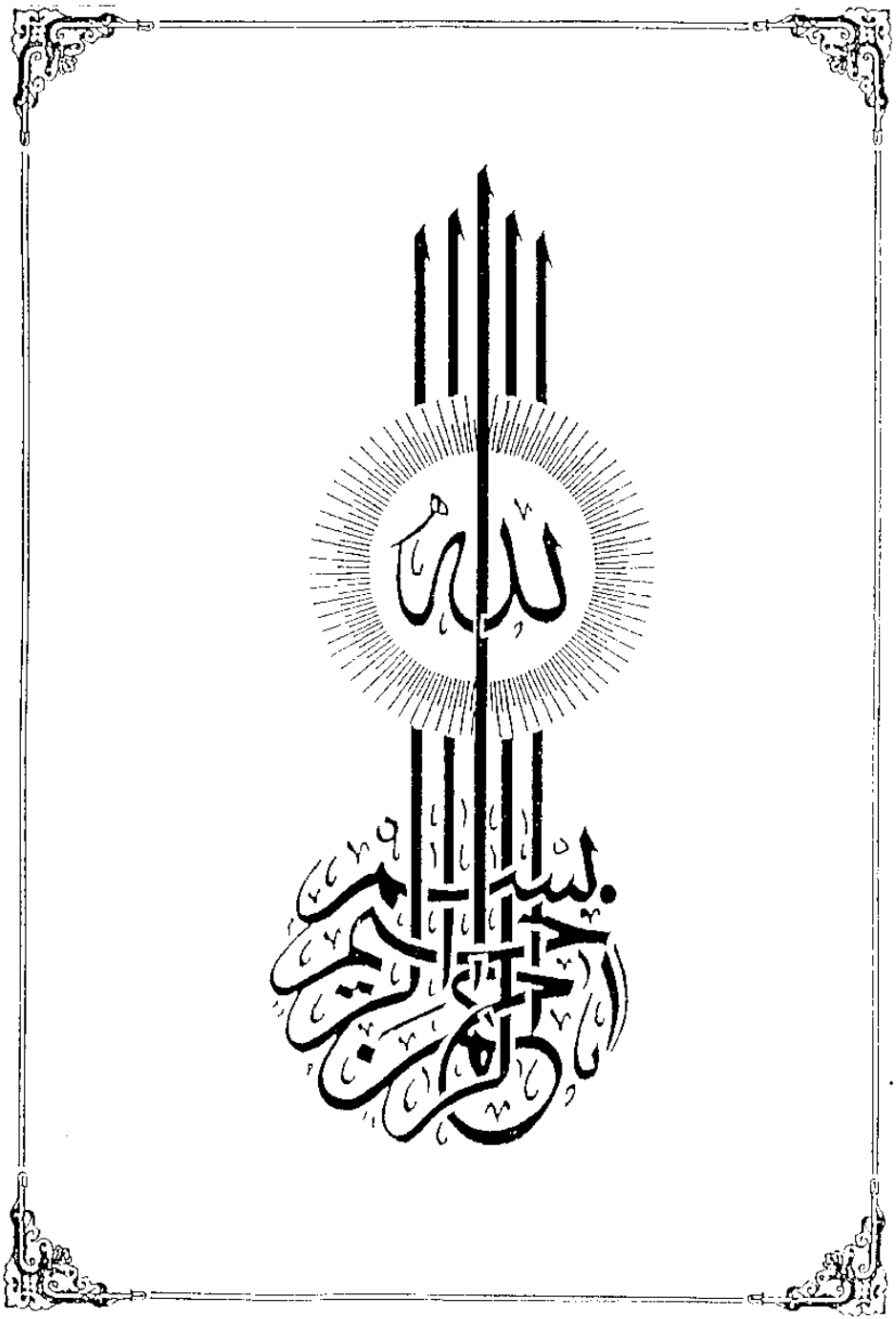

C O N T E N T S

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INTRODUCTION

The problem of Schistosomiasis has been existing since the dawn of history. The manifestations have been described in the papyrus scribed by Ebers and mentioned by Herodotus in his writing about Egypt, while the scientist Ruffer(1910) discovered the ova of the worm in the tissues of ancient Egyptian mummies. This disease continued to plague the people of the Nile valley with its secret safely hidden until the time of Theodor Bilharz; He was a German scientist, who discovered in 1851 the adult worms in the visceral veins of a child corpus in Kasr-El-Aini Hospital and this gave him the clue to prove the relation between these worms and the appearance of blood in urine.

Discoveries followed with Manson's recognition of another type of worms which was named after him (1902).

Lieper (1918), discovered the snails which transmit the disease and elucidated the life cycle of the parasite.

Christopherson (1918), described the effect of tartor emetic as a drug against bilharziasis.

Later, Khalil (1926), introduced the use of copper sulphate as a molluscicide against the snail vectors.

Schistosomiasis which is the Egypt major health problem, is also an international problem, not only affecting the health and the welfare of the individuals, but also interfering with the progress and the development of the community.

Accordingly, schistosomiasis has always attracted the attention of many workers in different fields, and several investigations have covered many aspects of the problem.

The application of the highly refined histochemical techniques opens a new field to study such relationship between the different cell components and their structure in normal and pathological conditions.

The present study is an attempt to achieve a new contribution by the application of cytochemical and histological techniques in studying some aspects of the problem of schistosomiasis on a cellular level.

HEPATIC SCHISTOSOMIASIS

Schistosomal hepatic fibrosis is the ultimate result of pathological processes provoked by the presence of ova and sometimes disintegrating worms usually of the mansoni type. The ova may be laid in the portal tributaries or may be carried to the liver from the intestine through the mesenteric portal system. They become impacted in the intrahepatic portal radicles where they excite bilharzial periportal granulomatous lesions. These lesions may be mild, of small number, scattered and later become fibrous producing no gross abnormality of the liver. They produce no significant clinical manifestations, except slight enlargement of the liver in the early infilterative stage. However, if the infection is extensive, heavy enough, repeated over long periods of time, it will produce widesperead pathological lesions which ultimately end in hepatic fibrosis associated with splenomegaly. The splenic enlargement is usually marked early in the course of the disease, as to mask the underlying pathological condition of the liver, hence the term Egyptian, or endemic splenomegaly is frequently applied to it.

Literatures dealing with schistosomal hepatic fibrosis classified it into two forms:

(1) The clay pipe stem cirrhosis Symmer (1904), later described as coarse periportal fibrosis by Hashem (1947),

(2) The nodular cirrhosis (Day, and Ferguson, 1909), defined as fine bilharzial hepatic fibrosis by Hashem (1947).

This is usually associated with various grades of enlargement of the spleen.

REVIEW OF LITERATURE

SCHISTOSOMIASIS OF THE LIVER

Liver involvement in schistosomiasis was first described in Egypt by Kartulis (1885), and since the original report of Symmer (1904), who first described the pathology of the disease, it had been accepted that *Schistosoma mansoni* produced the clay pipe stem cirrhosis of the liver.

Sambon (1908), by extensive study and comparison between the effect of *S. haematobium* and lateral spined ova, their pathological lesions and geographical distribution, came to conclusion that the lateral spined ova belonged to different species later defined as *Bilharzia mansoni* according to its first discoverer.

However, the matter was not completely settled except after the study of the life cycle of *Bilharzia mansoni* by Leiper (1918). This was confirmed in Egypt by Fairley and Manson (1920), in Brazil by Lutz (1919), and Later by Khalil in Egypt (1928).

The endemic form of Egyptian hepatosplenomegaly was described by Day and Ferguson (1909), who stated that it was an atrophic form of portal or multilobular cirrhosis, in which liver cells showed degeneration followed by regeneration and fibrosis as occured in Laennec's cirrhosis.

Day and Richards (1912), impressed by the great similarity of the Egyptian syndrome and Bantis disease, recommended the treatment by splenectomy, the results of which they considered satisfactory.

Kadi, (1923), drew attention to the great prevalence of hepatic and splenic enlargement amongst farmers complaining of chronic abdominal trouble. He regularly found schistosome ova in the faeces of such cases. On treatment with tartar emetic and thymol, great improvement was achieved. Accordingly, he suggested that this triple complex was the result of schistosomiasis.

Day (1924), accepted that the Egyptian splenomegaly was secondary to infestation of the liver by Schistosoma mansoni. He examined small portions of the liver histologically and found lateral spined ova in the tissues, in selected cases, He noted that slight infilteration or fibrosis really due to bilharziasis may be wrongly regarded as secondary to hepatic degeneration and that when complicating diseases were absent as in cases properly selected for splenectomy, the liver cells appeared healthy.

Hadson (1924), remarked the frequent association of schistosoma infection and hepatosplenomegaly in the northern most part of British Nyassaland, and concluded that

it was due to Bilharzia mansoni.

In the meanwhile Faust and Meleney (1924) described a similar syndrome in China and Japan, that was by Schistosoma japonicum.

Coleman (1926), studied the distribution of Schistosoma mansoni in Egypt and found that it was largely limited to districts of lower Egypt, where end canals prevailed, and remarked that in these districts the flow was sluggish and Planorbis boissyi (Biomphalaria-alexandrina) snails, the intermediate host of Schistosoma mansoni were plenty and hepatosplenomegaly was frequent.

Askanazy and Schiveizer (1927), studied five cases of Egyptian splenomegaly in which the spleens were extripated. There were lesions of the liver in the form of bilharzial pseudotubercles with ova of Schistosoma mansoni.

Hutchison (1928), found by microscopical examination of the liver, great increase in the fibrous tissue which contained large number of lateral spined ova. The liver cells were often atrophic due to compression by bands of fibrous tissue, and the endothelial cells contained a brown pigment.

Sorour (1928), used the term "periportal cirrhosis"

and considered that this diffuse bilharzial cirrhosis was caused by ova which were singly and diffusely deposited with no periportal distribution at all.

Petridis (1928), found that the liver contained schistosome ova. As the spleens were examined after removal by Askanazy, who drew attention to the presence of fibrosiderotic or sideromycotic nodules; he suggested that the syndrome might have dual etiology, one bilharzial and the other mycotic.

Many workers were not in favour of the bilharzial origin in the pathogenesis of bilharzial cirrhosis. Madden (1928), who commented that Day's views of the bilharzial origin of that syndrome had not yet been entirely accepted.

Likewise, Ibrahim (1928) stated that the bilharzial origin of the disease failed to explain the etiological relationship between the so called Egyptian splenomegaly with multilobular cirrhosis of the liver.

Khalil (1928), regarded bilharziasis of the intestine only a predisposing factor and so he considered the pathology of bilharzial cirrhosis in the liver, in line with that of laennec's cirrhosis.

Girges (1929), regarded the syndrome to be the result of deposition of ova over a long period, thus causing fibrosis of the liver and enlargement of the spleen.

El Daba (1930), suggested that as the cirrhotic condition of the liver was the dominating factor in this syndrome, while splenic enlargement was a purely secondary factor, and as the prognosis of any uncomplicated case depend entirely on the degree of hepatic efficiency, the name Egyptian splenomegaly should be changed to bilharzial cirrhosis.

Hashem (1931), reported eight cases of hepatosplenomegaly with diffuse bilharzial fibrosis of the liver and two cases of non bilharzial nature.

Girges (1932), attributed the hepatic lesion to the toxins released by the male worms.

Day (1933), described the pathological changes present in disease; from the portal venules the ova passed into the parenchyma and wandered for some distance, leaving a trail of destruction behind, marked by infilteration of small cells and eosinophils. Each ovum became surrounded