INCIDENCE OF VIRUS HEPATITIS AFFECTION IN PATIENTS WITH SCHISTOSOMAL HEPATOSPLENOMEGALY WITH POSSIBLE EFFECT ON DEVELOPMENT OF OESOPHAGEAL VARICES

THESIS

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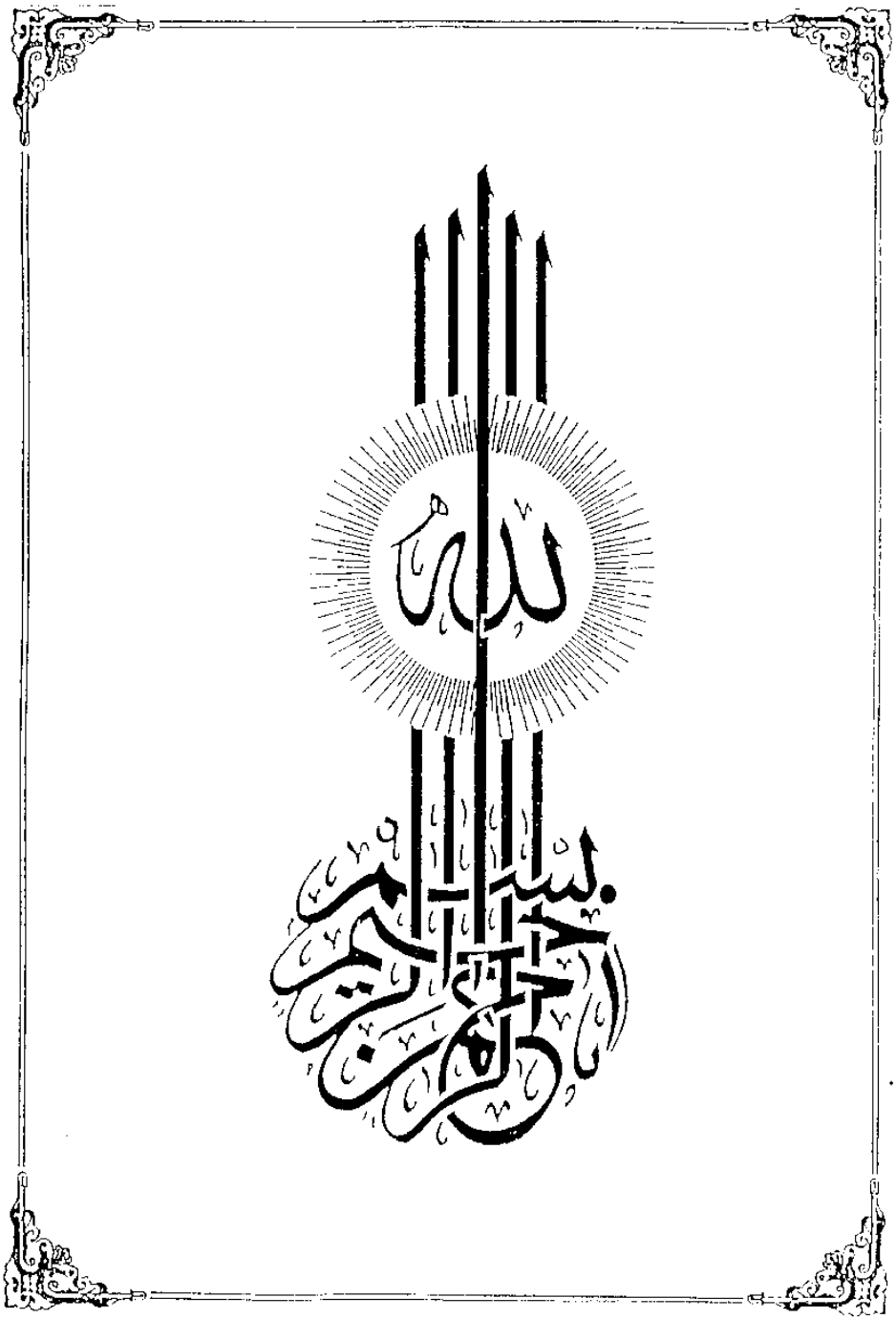

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LIST OF ABREVIATIONS

Anti-HBc	antibody to hepatitis B core antigen
Anti-HBs	antibody to hepatitis B surface antigen
Ac VH	acute virus hepatitis
Ac VHA	acute virus hepatitis A
Ac VHB	acute virus hepatitis B
A VHB	active virus hepatitis B
AVH non-A,non-B	active virus hepatitis non-A, non-B
BHN	bridging hepatic necrosis
CAHB	chronic active hepatitis type B
CAH non-A,non-B	chronic active hepatitis non-A, non-B
CLH	chronic lobular hepatitis
CPH-B	chronic persistant hepatitis type B
CPH non-A,non-B	chronic persistant hepatitis non-A,non-B
ELISA	enzyme linked immuno-sorbant assay
ENANB	endemic & epidemic hepatitis NANB
ETNANB	enterically transmitted hepatitis NANB
GGH	ground glass hepatocytes
HAV	hepatitis A virus
HBV	hepatitis B virus
H.C.V	hepatitis C virus

LIST OF ABREVIATIONS

H.D.V	hepatitis D virus
H.E.V	hepatitis E virus
H.NANB.V	hepatitis non-A, non-B virus
HBc Ag	hepatitis B core antigen
HBs Ag	hepatitis B surface antigen
RIA	radio-immunoassay
VHA	virus hepatitis A
VHb	virus hepatitis b

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INTRODUCTION

** *INTRODUCTION* **

The clinical course of hepatic schistosomiasis is variable. Some patients develop portal hypertension with oesophageal varices, but may or may not bleed. In other patients, the clinical presentation takes the form of progressive hepatic dysfunction ending in liver cell failure. So, it is obvious that additional factors might be responsible for this variability in the course of the disease. It is not true that these are different stages in the course of the same condition, as some patients die of haematemesis and others live enough to die of liver cell failure without the slightest evidence of presence of varices.

It has been postulated that some factors may be added to the bilharzial hepatic lesions that would direct the course of the disease here or there. Among those factors is superadded or concomitent viral hepatic affection.

Reports from Egypt *(EL-ROZIKY etal., 1979 and EL-ROOBY, 1985)*, Kuwait *(AL-NAKIB etal., 1982)*, Malawi *(MOLYNEUX and JENSEN, 1978)* and Sudan *(DANESHMEND etal., 1984)*, have indicated that rates of hepatitis B. virus antigenemia were

significantly higher in schistosoma mansoni infected persons, especially in those with hepato-splenic schistosomiasis (*LYRA etal., 1976*) than in the uninfected control group. A higher prevalence of H.B.V. antigenemia has also been found in patients with hepato-splenic schistosomiasis compared to those with intestinal form of the disease (*LYRA etal., 1976 and PRATA, 1982*).

EL-ROZIKY etal. in 1979 found that patients with Schistosoma mansoni infection showed a significantly higher frequency of HB s Ag and anti- HB s than those with schistosoma Haematobium infection.

The theories explaining why schistosoma mansoni infected patients are more vulnerable to H.B.V. infection or have a higher H.B.V. prevalence rate include :

a) Impaired cell mediated immunity which reduces the host resistance (*BASSILY etal., 1979 and EL-ROOBY, 1985*).

b) Low socio-economic condition which increases the risk of exposure (*EL-ROZIKY etal., 1979*).

c) Repeated parentral treatment in the past with intra-venous or intra-muscular anti-schistosomial drugs and frequent blood transfusions (*ZAKARIA etal., 1979*).

d) Diffuse liver fibrosis which has been cited as an optimal

milieu for viral replication in hepatocytes (*ZAKARIA etal., 1979 and BASSILY etal., 1979*).

However, the mechanisms underlying the persistance of H.B.V. antigenemia in schistosomal patients remain obscure and speculative (*NOOMAN, 1977*). Whereas the relationship between H.B.V. and schistosoma mansoni infection is controversial, there is general agreement that H.B.V. infection is always high in hepatic schistosomiasis due to schistosoma mansoni (*AL-NAKIB etal., 1982 ; ANDRADE etal., 1977 ; ANDRADE etal., 1978 ; DANESHMEND etal., 1984 and TOSSWILL and RIDLEY, 1986*).

No general consensus has been reached about the mechanism of interaction between H.B.V. infection and hepatic schistosomiasis. Some authors believe that H.B.V. infection causes early stage schistosomiasis (intestinal disease) to develop to an advanced stage (hepatic or even decompansated hepatic disease). Other authors believe that hepatic schistosomiasis leads to greater susceptibility to H.B.V. infection (*BASSILY etal., 1983 ; LYRA etal., 1976 and ZAKARIA etal., 1979*). However, when H.B.V. infection and hepatic schistosomiasis are associated, the prognosis is jeopardized.

H.B.V. infection modifies the liver pathology in patients with hepatic schistosomiasis and frequently leads to chronic active hepatitis. Moreover, liver function deteriorates and compansated hepatic disease tends to be decompansated *(ANDRADE etal., 1977 ; ZAKARIA etal., 1979 and BASSILY etal., 1983)*. Hepatic failure is not infrequently seen in those cases.

Although bilharzial infection is contracted by Egyptian farmers at a very young age, usually in childhood, yet the rush of cases presenting at hospitals for complications as bleeding or liver cell failure usually are at ages of 20 to 40 years *(KAMEL etal., 1968)*. This may be taken as an indirect proof that bilharzial hepatic disease follows a phasic course. A lag of many years elapse before the disease declares itself around the age of 20 to 40 years, then complications as haematemesis or liver cell failure or both start to appear and end the life of a good number of patients, so that they rarely live beyond 60 years. Or may be a missing factor which is contracted by some bilharzial patients later in their lifes which causes more deterioration in the hepatic reserve and accentuates the degree of portal hypertension.

Oesophageal varices demonstrated by barium meal and oesophagoscopy are found in 80% of patients with hepatic schistosomiasis *(PRATA, 1982)*.

The reported incidence of haematemesis due to oesophageal varices in patients with pure bilharzial hepato-splenomegaly ranges between 8% *(POPE etal., 1980)*, 11% *(BIBAWI, 1954 and MOUSA, 1960)* - , and 20% *(GIGASE, 1982 and NASH etal., 1982)*.

These data show that not all patients with schistosomal infection develop hepatic course of the disease. Also, some of the patients with hepatic schistosomiasis develop oesophageal varices as sequele of portal hypertension. But minority of these patients with varices due to pure bilharzial affection will develop haematemesis. Also, there is a lag period of many years between contraction of the disease and the development of complications.

This study aims at identification of the possible relationship between superadded viral hepatic affection and hepatic schistosomal affection as regards the production of complications such as portal hypertension and liver cell failure.

REVIEW OF LITERATURE

** *PATHOLOGY OF HEPATIC SCHISTOSOMIASIS* **

Although *KARTULIS* (1885) described the liver involvement in schistosomiasis, *SYMMERS* (1904) first described the pathology of the disease. He noticed that extensive scarring and thickening of the portal tracts occured in response to the deposition of ova of schistosomes at these sites. He referred to this type lesion as clay pipe-stem cirrhosis.

SOROUR (1928) used the term peri-portal cirrhosis instead. *HASHEM* (1947) on the basis that the bilharzial lesion does not cause necrosis of the liver tissue followed by regeneration nodules suggested the term "coarse peri-portal bilharzial fibrosis" rather than cirrhosis.

DAY (1924) noticed that several fatal cases of hepato-splenomegaly showed at autopsies a diffuse infilteration of liver but no typical pipe-stem cirrhosis as that described by Symmer. He considered that the type of cirrhosis produced by bilharzial infection depends on the number and rate of deposition of ova. When the ova are deposited in small numbers over a long period the usual form of diffuse