NEW LIGHT ON VIRUS B HEPATITIS

THESIS

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Master Degree in Clinical Pathology

By

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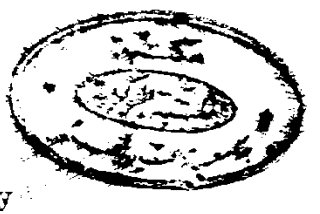

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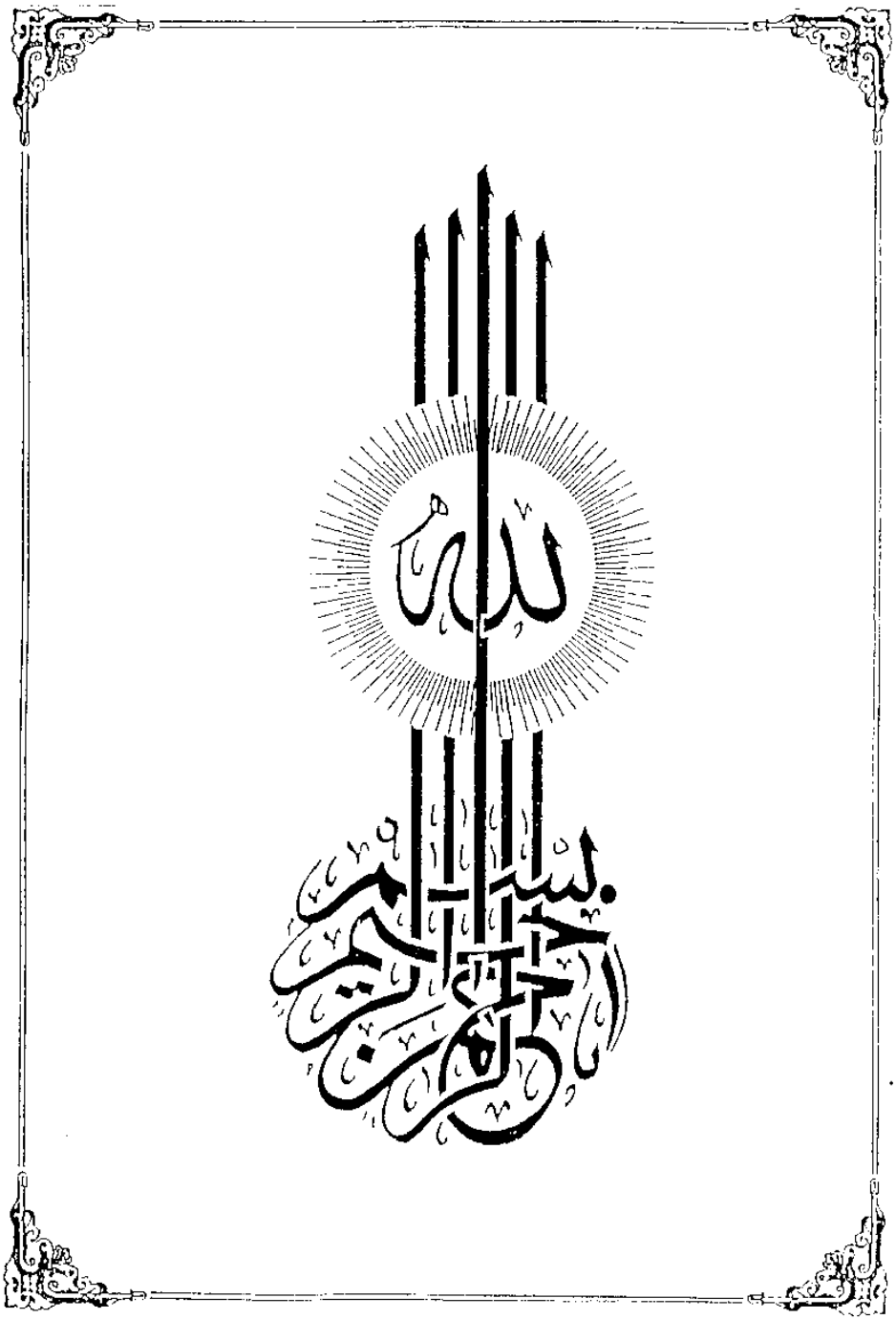

Contents

" INTRODUCTION "

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Infection by hepatitis B virus produces a spectrum of clinical and laboratory observations, these range in severity from asymptomatic infection to fulminant disease leading to death in a few days.

Blumberg (1964) discovered the australia antigen which is present on the surface of large dane particle (Dane et al 1970). By the use of electron microscopy australia antigen or hepatitis B surface antigen (HB_s Ag) has been identified in the nuclei of liver cells in patient infected with virus B (Blumberg et al 1968).

The hepatitis B core antigen (HB_c Ag) was discovered later by (Almeida et al, 1969) and the e antigen by (Magnues et al.1977).

The presence of a parenterally transmitted form of hepatitis was first reported by Duncan in 1885, but later studies done by Prince and Burke in 1970 suggested the transmission of hepatitis B virus by non parenteral route.

" AIM OF THE WORK "

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Is to review the recent studies on the aetiologic agents of the virus B hepatitis, the pathogenesis of infection, the immunological aspects, the various laboratory methods of diagnosis and also the concepts of prophylaxis.

" REVIEW OF LITERATURE "

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Epidemiology of virus B hepatitis :

The prevalence of viral hepatitis including hepatitis A and B is markedly different from country to country (Ling et al, 1972).

Mass surveys for study of epidemiology of virus B hepatitis have already been performed all over the world, positive rates of hepatitis B surface antigen in general population are markedly different in various areas. The highest positive rate, as high as 15%, was reported from Southeast Asia and Africa. In Japan the rate is 2 -3 % and 0.1- 0.2% in United States (Toshio Shikata, 1978).

Hepatitis B surface antigen carrier state in the world has been estimated to be over two hundred million (Szmuness 1978).

The incidence of hepatitis A is most predominant and appear to be endemic in tropical countries, in South Asia, Africa, and in mediterrnean areas.

The agent of viral hepatitis is more resistant to heat and chemicals than most human viral and bacterial pathogens.

Boiling for a few minutes is not sufficient to kill the virus but hot air oven at 180 degrees centigrade for one hour or boiling for a minimum of ten minutes have been recommended by WHO for sterilization of instrument (1964)

Viral hepatitis is endemic in Egypt, because of the very frequent, usually non indicated, parenteral administration of vitamins and calcium as tonics, also the habit of nurses moving from one house to the other to give injections with the same syringe, (Mohammad Sabbour and Zoheir, I978) .

On the other hand, in Japan there have been no large epidemics for over twenty years, at present only few sporadic cases, and a few small out-breaks in specialcondition have been reported (Toshio Shikata, I978).

The incidence of hepatitis in Asia and Africa is very high compared to that in European countries and United States (Zukerman and Howard, I975) .

Mode of transmission of virus B hepatitis :

This form of viral hepatitis has been shown to occur in a small proportion of patients after transfusion of blood or blood products that contain the infective virus (Havens 1965), and those receiving measles, mumps or yellow fever vaccines (Freeman, 1946).
It has been noted also after intradermal, subcutaneous, intramuscular or intravenous injections or following venepuncture for withdrawal of blood., under all these circumstances contamination of the syringe with the B virus is the cause (Blumberg, et al 1968).

A nonparenteral route of spread of hepatitis type B is indicated by serologic surveys that have shown a high prevalence of hepatitis B surface antigen among patients who deny parenteral exposure (Prince et al, 1970) , in family contacts of hepatitis B surface antigen positive members (Blumberg et al, 1970), in countries where parenteral exposure is at a minimum (Blumberg et al, 1968) and in large crowded institutions.
Recent detection of the antigen in semen and saliva of hepatitis B surface antigen carriers (Heathcote, et al,1973)

acquire the surface antigen within one to six months (Tong et al 1981).

On the other hand, vertical transmission is much less frequent if the mother is an asymptomatic carrier or if infection occurs in the first two months of gestation (Stevens et al, 1975).

The affected newborn may develop mild hepatitis, remaining hepatitis B surface antigen positive and becoming an adult chronic carrier.

Pathogenesis of virus B hepatitis :

It has been persumed that direct cytopathic effects of hepatitis B virus itself on liver cells are less likely responsible for the pathogenesis; because of the presence of asymptomatic chronic hepatitis B surface antigen carriers with abundant hepatitis B surface antigen in the cytoplasm and hepatitis B core antigen in the nucleus of hepatocytes and yet no enzymatic and histological evidence of hepatocyte necrosis.

On the other hand in acute hepatitis as well as chronic active hepatitis, hepatitis B virus antigenic materials in the liver tissue are rather scanty.

In fulminant hepatitis, demonstrable hepatitis B surface antigen in the liver tissue by immunofluorecent method is very scanty (Lee et al 1975).

These seem now to be a general agreement that host immune responses to hepatitis B virus or its associated antigens are responsible for the pathogensis of liver cell necrosis in virus B hepatitis (Toshio shikata, 1978).

The immune humoral response against hepatitis B surface antigen could be incriminated as one of the important factors in the pathogenesis of acute hepatitis (Lee et al,

1975).

In fulminant hepatitis marked liver cell destruction is seen, whereas hepatitis B virus associated antigens in the liver tissue are very scanty(Toshio Shikata, 1978). On the other hand, immunoglobulin especially IgG and complement are abundantly demonstrated in the necrotic areas, also numerous antibodies producing cells for hepatitis B surface antigen were observed in the lymphoid tissue of the cases with fulminant hepatitis (Toshio Shikata 1978). Cellular immune reaction also appear to play an important role in the hepatitis B virus infection and the liver cell injury.

We can observe histopathological findings suggesting the morphological expression of killer lymphocytes or immunologically activated T lymphocytes in action against the liver cell with hepatitis B surface antigen in the cytoplasm. In those cases, small lymphocytes and macrophages in close contact with hepatitis B surface antigen containing hepatocytes were observed.
Variable degenerative changes such as shrinkage, partial lysis, lytic necrosis were observed in these hepatitis B surface antigen containing hepatocytes (Toshio Shikata 1978).