PERINATAL TRANSMISSION OF HEPATITIS B VIRUS

A THESIS

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By

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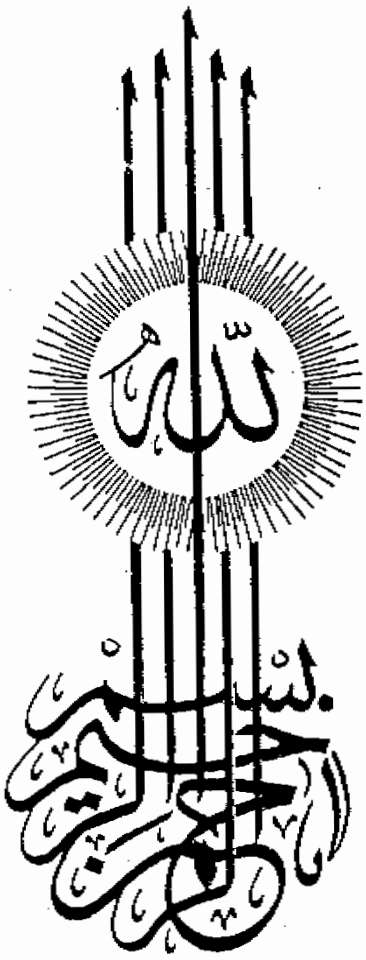
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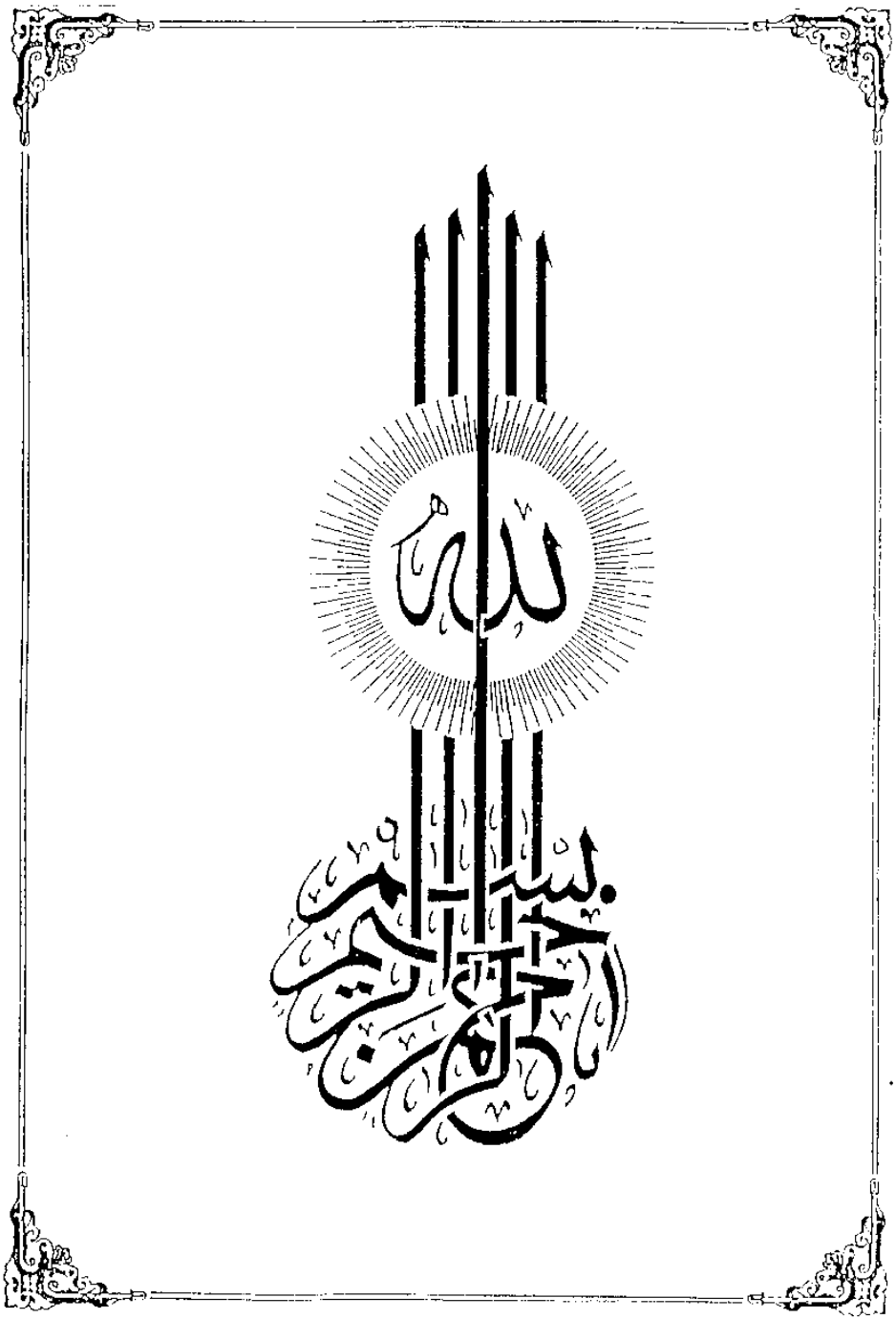

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Introduction

And Aim Of Work

INTRODUCTION AND AIM OF WORK

Hepatitis B virus (HBV) is a highly infectious pathogen that spreads along percutaneous, parenteral, sexual and perinatal routes ***(Gerety and Tabor, 1994).***

Of all the hepatotrophic viruses, HBV is associated with the greatest worldwide morbidity and mortality. This is because of the ease of transmission and the potential for progression to a chronic infective carrier state ***(Brown et al., 1990).***

Globally, about 5% to 10% of acutely infected patients will become chronic carriers of hepatitis B surface antigen (HBsAg) ***(Smego and Halsey, 1987).*** An estimated 40% of chronic HBV carriers will die of liver disease ***(Beasley, 1983).*** These patients run a high risk of developing liver cirrhosis and primary liver carcinoma ***(Prince, 1981).*** Furthermore, they serve as the infectious pool from which the virus is transmitted to uninfected individuals by blood or close contact ***(Michael et al., 1993).***

The reservoir of chronic HBsAg carriers in the world is estimated at more than 300 million person, with an incidence of around 20% in Africa and Asia ***(William, 1991).***

Approximately 40 percent of these carriers acquire the virus as a result of perinatal transmission, and another 35 to 40 percent become

infected during the preschool years. HBV is acquired during adulthood by only 10 to 15 percent of chronic hepatitis B carriers ***(Tong, 1989).***

This is particularly critical in populations with a high carrier rate where in some countries of the world more than 30% of women of childbearing age are HBV carriers, transmitting the infection to their offspring in a high percentage. In addition, the infected newborns become foci for further dissemination of HBV in the population because they develop so frequently a carrier state ***(Prozesky, 1983).***

Thus, immunoprophylaxis during the early years of life is important to prevent the hepatitis B carrier state and its potential consequences ***(Myron, 1989).***

Because of the effectiveness, safety and decreasing cost of HBV vaccine, worldwide eradication of the virus may be possible ***(Blumberg, 1989).***

As perinatal transmission of hepatitis B virus (HBV) represents one of the most efficient modes of HBV infection ***(Mancal et al., 1989),*** and the majority of HBV carriers worldwide becomes infected by transmission from asymptomatic carrier mothers to their infants.

This work is designed to :

1. Screen pregnant females for detection of asymptomatic carrier mothers (HBsAg positive ± HBeAg positive).
2. Estimate the incidence of perinatal transmission of HBV.
3. Show the correlation between the immunological status of the mothers and the rate of transmission.

4. Estimate the incidence of immune pregnant females (Anti-HBs positive).

Review Of Literature

REVIEW OF LITERATURE

HISTORICAL BACKGROUND

Early civilization regarded the liver as the seat of life ***(Lyon, 1978).*** Thus, it was not surprising that liver disease and jaundice were relatively well known to ancient people.

Although acute hepatitis following percutaneous exposure to human serum or blood was recognized in humans more than 100 years ago ***(Lurman, 1885),*** a causative infectious agent was not identified until 1960s, when hepatitis B surface antigen (HBsAg) was discovered ***(Blumberg et al., 1965).***

Many observations were made about the outbreak of epidemic jaundice. In 1885, the existence of a parenterally transmitted form of hepatitis was documented by ***Lurman (Lurman, 1885)***, who reported the development of jaundice in 191 of 1,289 shipyard workers in Bremen 2-8 months after they had received small pox vaccine prepared from human "lymph". Subsequent outbreaks were described later among patients treated in VD clinics receiving injections as a result of the use of improperly sterilized syringes and needles.

Similar outbreaks were recognized among patients with diabetes and rheumatoid arthritis treated in clinics and hospitals and among those receiving tattoos ***(Mosley, 1975).***

In 1937, it was reported from England that approximately 40% of 109 individuals receiving inoculations of a single batch of human measles convalescent serum developed jaundice after incubation periods up to 114 days. This is followed by an outbreak of similar nature in British troops given filtered human mumps convalescent plasma ***(Beeson, 1943).*** These clinical entities were grouped together as "homologous serum jaundice" ***(Memorandum, 1943).***

In 1942, epidemics of serum hepatitis occurred among USA army personnel immunized with yellow fever vaccine containing human serum to stabilize the product resulting in 28,000 cases with 62 deaths. Human serum was replaced by distilled water and no cases of jaundice were proved to be associated with the preparation ***(Morgan and Willianison, 1943).***

In ***1963, Blumberg and his colleagues*** identified an unusual antibody in the sera of two multiply transfused haemophilic patients. The antibody was found to react with an antigen from an Australian aborigine serum and was named Australia antigen (Au) (now designated the hepatitis B surface antigen or HBsAg) ***(Blumberg et al., 1965).***

By 1968, studies from two independent groups showed that (Au) antigen was found specifically in the serum of hepatitis B patients ***(Okochi and Murakami, 1968; and Prince, 1968).***

According to the epidemiological differences observed between

infectious hepatitis and homologous serum jaundice, the terms "hepatitis A" and "hepatitis B" were introduced in 1947 to categorize each of them respectively and these terms have been adopted by WHO committees on viral hepatitis.

In addition, a variety of terms were introduced renaming the antigen to reflect its association with hepatitis; as serum hepatitis antigen (SH antigen), Au/SH antigen, hepatitis antigen (HA) and hepatitis associated antigen (HAA).

In 1972, in order to avoid confusion, the Committee on Viral Diseases of the National Academy of Sciences (USA) recommended that the term hepatitis B antigen (HBsAg) be used to describe the antigen, and hepatitis B antibody (HBAb) or (anti-HBsAg) be employed for the antibody ***(WHO, 1977).***

In ***1970, Dane et al.,*** observed another larger virus-like particle in the serum of some patients with hepatitis B. This "Dane particle" is now known to represent the virion ***(Dane et al., 1970).***

The inner component or core of this virus is designated hepatitis B core antigen or Hbc. Antibody directed against Hbc is designated anti-HBc.

Subsequently, ***Maguis and Espmark*** described a third antigen-

antibody system related to infectivity, involving hepatitis Be antigen (HBeAg) and anti-HBe ***(Maguis and Espmark, 1972).***

The past years have witnessed a number of interesting facets of the hepatitis story and since 1975 studies on hepatitis B vaccine have been initiated.

HEPATITIS B VIRUS

Hepatitis B virus is a complex DNA virus belonging to the group of hepadna viruses ***(Deinhardt and Deinhardt, 1983).*** It has not been grown in tissue or cell cultures, and the disease has been transmitted only to man and the great apes ***(Edward, 1985).***

Characterization of HBV began with ***Prince's*** demonstration of hepatitis B surface antigen, the demonstration of the Australia antigen by ***Blumberg*** and Dane's recognition of complete virus particles in human serum. HBV has been fully characterized ***(Murray, 1983)*** and its properties are demonstrated in Table (1).

ULTRASTRUCTURE AND PHYSICAL PROPERTIES :

Three distinct structures have been observed in the serum of infected humans, and HBsAg resides on their surface :

1. The Dane particles (42 nm spherical particles).
2. Spherical particles (22 nm particles).
3. Filamentous forms (22 nm in diameter and 50-700 nm in length).