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THE EFFECT OF HEPATITIS B SURFACE ANTIGEN IN CARRIER MOTHER ON IMMUNOGLOBULIN E CONCENTRATION OF THE NEWLY BORN

Thesis

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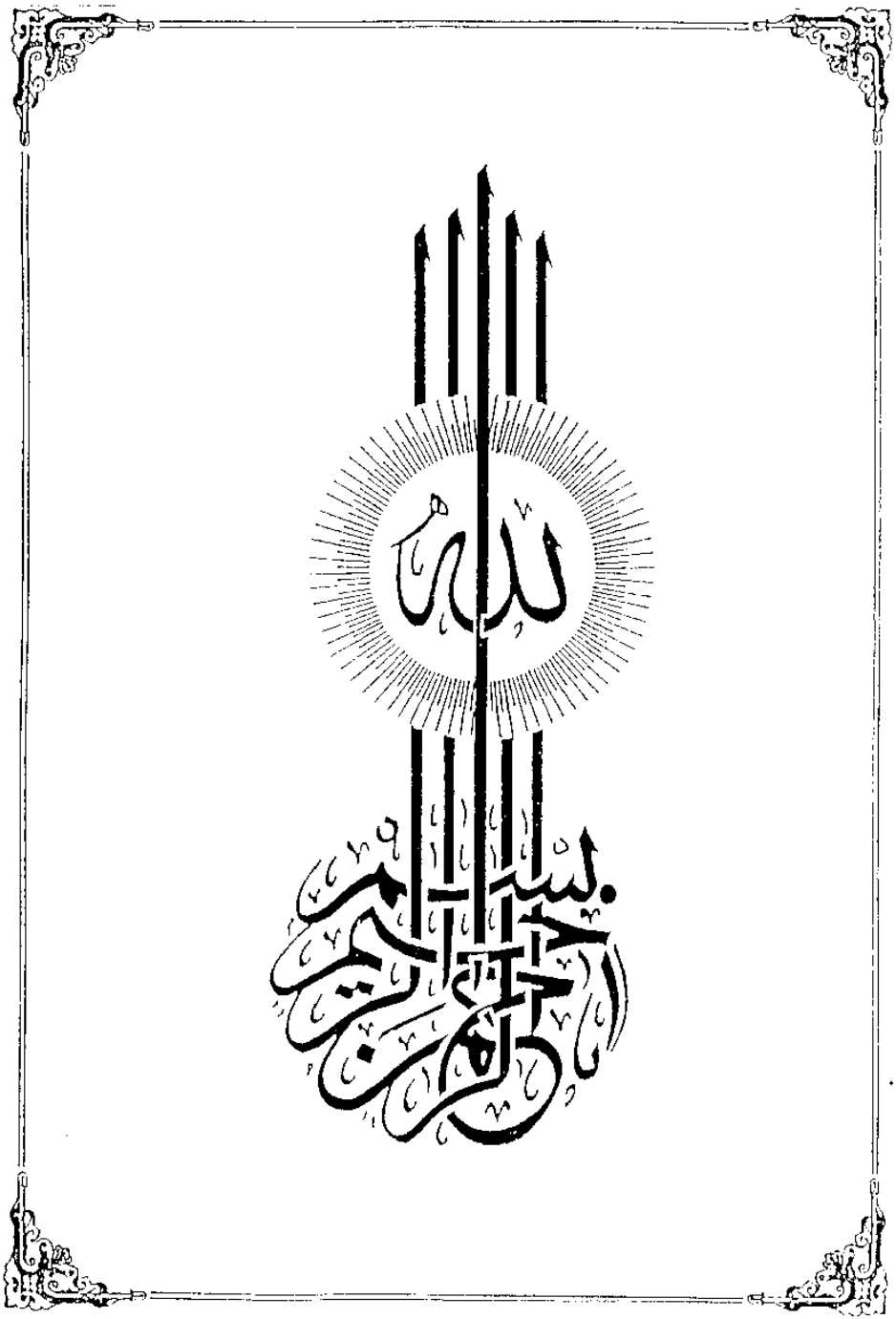

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INTRODUCTION

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Viral hepatitis is a worldwide major public health problem, with an estimated 200 million chronically infected individual **(Beasely et al., 1982)**. During the last 20 years, the notable progress in research of viral hepatitis has led to their classification into: hepatitis A, hepatitis B, hepatitis D (Delta agent) on serological basis. However, there is yet unidentified group designated as non A, non B hepatitis (NANB) which is only diagnosed by exclusion **(Zuckerman, 1985)**. Although the clinical illness produced by these agents may be similar, considerations regarding complications, transmission and risks are different **(Bernstein et al., 1980)**. Hepatitis B is considered the most serious, becaus of the development of the chronic clinical sequele, as chronic liver disease, chronic carrier symptomatic and asymptomatic states **(Olsak, 1984)**. The disease is endemic in certain areas, especially in Asia and Africa **(Woo et al., 1979; and Marnier et al., 1985)**.

Viral hepatitis is endemic in Egypt **(El Alamy et al., 1979)**. The chronic state was found to range from 2-10% **(Nooman et al., 1974; El-Alamy et al., 1979; Annual Report of Agouza Centre, 1980; Sherif et al., 1985)**.

Viral hepatitis is considered the most common cause of jaundice during pregnancy **(Wand, 1979 and Sherlock, 1981).** Acute infection with hepatitis B during pregnancy especially in the third trimester, carries on increased risk of prematurity, intrauterine growth retardation and foetal mortality **(Keys et al., 1972, Smithwick et al., 1972; Heiber et al., 1977 and Bernstein et al., 1980).**

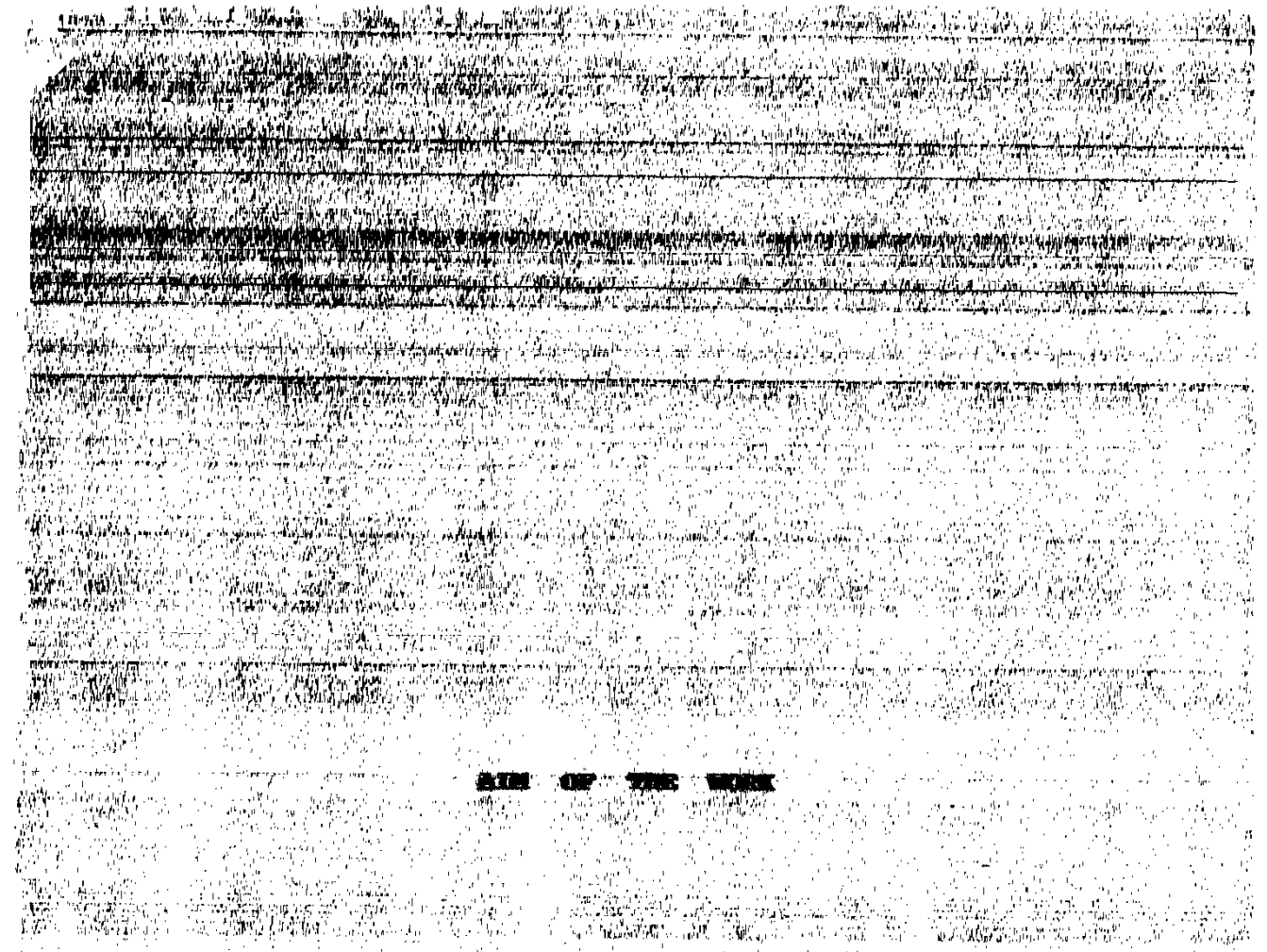
Transmission of hepatitis B virus (HBV) from the mother to the infant occurs when the mother is a chronic hepatitis B surface antigen (HBsAg) carrier or had acute hepatitis B during pregnancy, especially in the third trimester **(Isenberg, 1977; Chew, 1980, Olsak, 1984 and Sacher et al., 1985).**

The mode of transmission of HBV remains a point of much controversy, whether transplacental or through contact of the foetus with the infected maternal blood and vaginal discharge at the time of delivery **(Alessandro et al., 1983, Sacher et al., 1983 and Rull et al., 1984).**

Although most affected infants are asymptomatic, some develop variable neonatal liver disease ranging from asymptomatic carrier state, through mild elevation of liver enzymes with chronic changes seen in biopsy to

acute disease and fulminant hepatitis and do not survive **(Dupuy et al., 1975; Bernstein et al., 1980; Rull et al., 1984 and Brunell et al., 1985).**

The chronic carrier state of HBsAg in pregnancy has been studied in different populations all over the world it varied from 0.12% as in Denamark **(Skinhoj, 1972)** to 15.2% in Taiwan **(Stevens et al., 1975)**, according to the geographic distribution.



AIM OF THE WORK

We are aiming to elucidate the relation between maternal affection with hepatitis B-virus (HBs Ag positive) and IgE concentration in the fetus, in Ain-Shams University Maternity Hospital.

** **

REVIEW OF LITERATURE

VIRAL HEPATITIS

Acute viral hepatitis is a common and serious infectious disease caused by several viral agents and marked by necrosis and inflammation of the liver **(Hoofnagle, 1979).** During the last twenty years the notable progress achieved by research into viral hepatitis has led to their classification into hepatitis A, hepatitis B, hepatitis D (The Delta agent, a defective virus), epidemic non A hepatitis and by at least two non A, non B hepatitis **(Zukerman, 1985).**

Hepatitis A is caused by an entero virus type 72, and transmitted by the faeco-oral route, does not become chronic, and no chronic virus carriers exist **(Zukerman, 1978 and Deinhardt & Gust, 1982).**

Hepatitis B is caused by a hepanda virus type I, is transmitted parenterally or through close personal contact **(Deinhardt et al., 1983).** Hepatitis B becomes chronic in certain number of cases **(Nielsen et al., 1974; Klastskin, 1975; Pedeker, 1975; Velasco et al., 1978 and Zukerman, 1978),** and can lead to cirrhosis or primary hepatocellular carcinoma **(Arias & Shafritz, 1982).**

Non A, non B hepatitis was first recognized as a

disease associated with blood transfusion **(Prince et al., 1974)**. NANB hepatitis account for 60-90% of post-transfusion hepatitis in U.S.A **(Kim et al., 1980)**, and for 80-90% in Japan **(Furuta et al., 1980)**. A second form of NANB hepatitis mimic hepatitis A has been found responsible for water-borne epidemics of hepatitis in India **(Wong et al., 1980)**.

Hepatitis D is found only in patients who are infected with HBV. The Delta agent is a defective hepatitis virus-like transmissible agent that requires HBV synthesis for it's replication. Infection by Delta agent occurs either as a coinfection with hepatitis B or as a super infection of a hepatitis B carrier. Fulminant hepatitis and progressive chronic hepatitis are now well established sequelae of Delta agent infection **(Rizzetto et al., 1977, 1980 and 1983; Smedile et al., 1981; Deinhardt & Gust, 1982 and Redekar, 1983)**.

According to **WHO (1977), Deinhardt & Gust (1982) and Rizzeto et al., (1983)**, the nomenclature of the viral hepatitis viruses components and the corresponding antibodies is shown in Table (1) & (2)

Table (1) :

Nomenclature for viral hepatitis viruses, A, B, NANB (Components and corresponding antibodies).

Virus Component or Antibody	Definition
HA	Hepatitis A
HAV	Hepatitis A virus.
HAAg	Hepatitis A virus Antigen.
Anti-HAV	Antibody to hepatitis A virus without differentiation into immunoglobulin classes.
Anti-HAV IgG	Antibody to hepatitis A virus of IgG class.
Anti-HAV IgM	Antibody to hepatitis A virus of IgM class.
HB	Hepatitis B
HBV	Hepatitis B virus
HBsAg	Hepatitis B surface antigen.
HBcAg	Hepatitis B core antigen.
HBeAg	Hepatitis Be antigen.
Anti-HBs	Antibody to hepatitis B surface antigen.
Anti-HBc	Antibody to hepatitis B core antigen.
Anti-HBcIgG	Antibody to hepatitis B core antigen of IgG class.
Anti-HBcIgM	Antibody to hepatitis B core antigen of IgM class.
Anti-HBe	Antibody to hepatitis Be antigen.
NANB	Hepatitis non-A non-B.

After WHO, 1977 and Deinhardt & Gust, 1982).

Table (2) :

Nomenclature for Delta agent.

Current	Recommended
Delta hepatitis	type D hepatitis
Delta agent	Hepatitis D virus (HDV)
Delta antigen	Hepatitis D antigen (HDAg)
Antibody to ó Ag	Antibody to HDAg.
(Anti- ó)	(Anti -HD)

After Rizzetto et al., (1983 a).