HEPATITIS B – VIRUS MARKERS IN HEPATOSPLENOMEGALIC PATIENTS

THESIS

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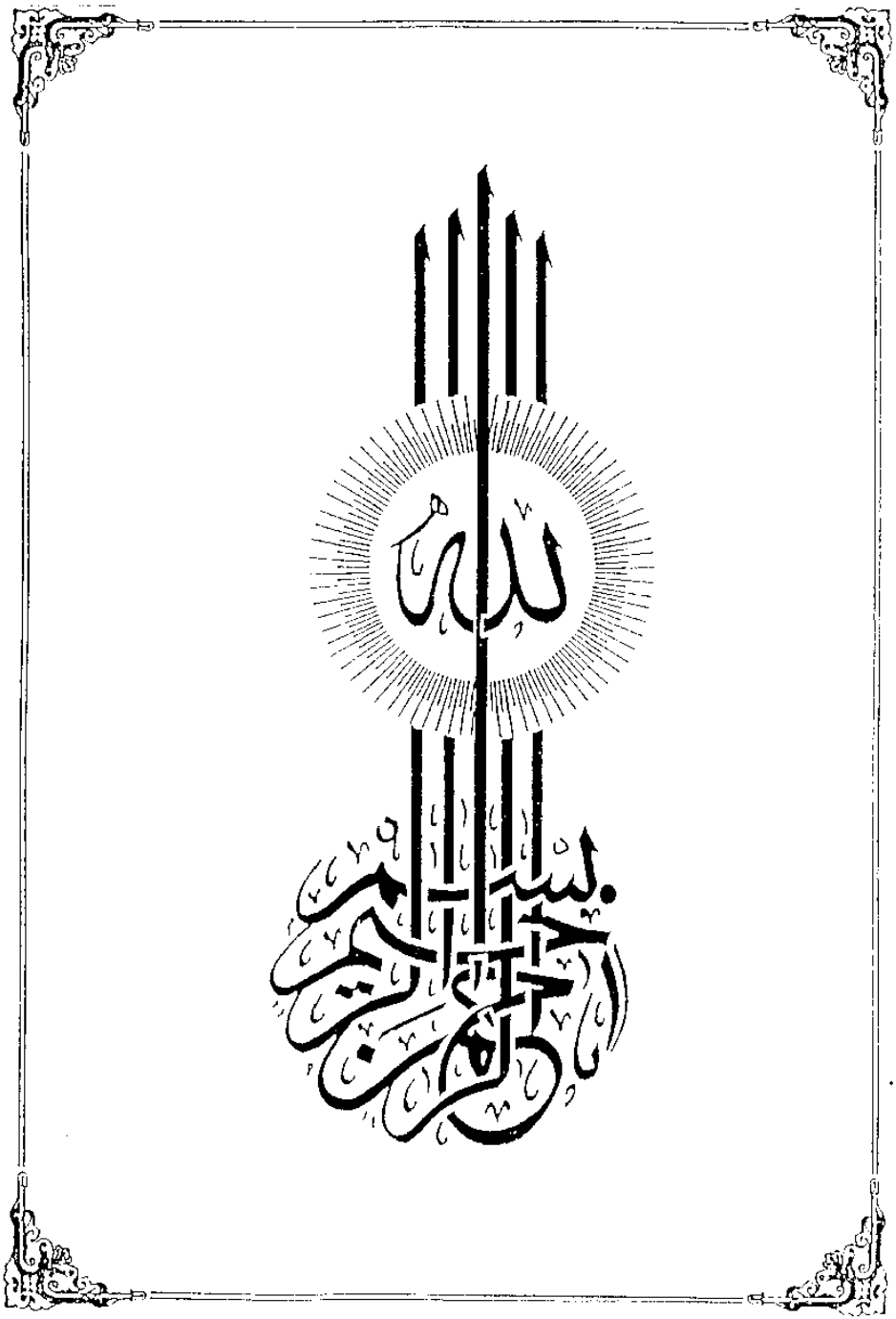
بسم الله الرحمن الرحيم

" قالوا سبحانك لا علم لنا إلا ما علمتنا
إنك أنت العليم الحكيم "

صدق الله العظيم

سورة البقرة - آية ٣٢



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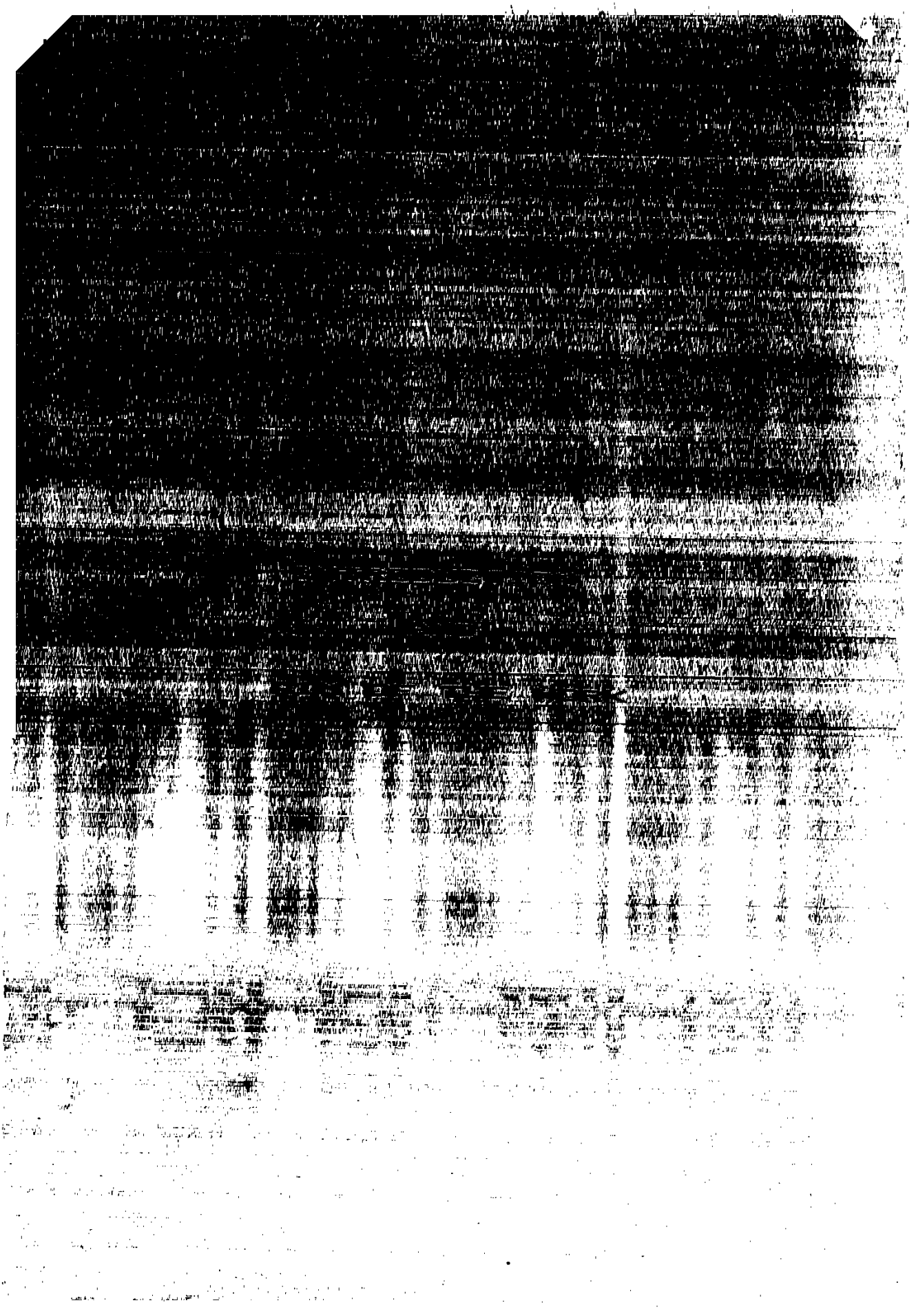

INTRODUCTION AND AIM OF WORK

Introduction :

When serological tests for hepatitis B virus [HBV] markers were developed hepatitis B verus was identified as the main cause of viral hepatitis in normal population. In Egypt, the frequency of hepatitis B surface antigenaemia among the normal population varies from [1.5%] to [5.7%] in urban and rural areas, [Noomon et al., 1973]. Among schistosomal patients this frequency was found to be 11.2% [El-Raziky et al., 1979]; 15.9% [Nooman et al., 1978]; 20.5% [Issa, 1979]; 22% [El-Zayadi 1985]. In fact scistosomal patients are more likely to retain the hepatitis B surface antigen [HBs Ag] for a longer period than the normal population if they are exposed to HBV. This was attributed to the immunological status of the host, [Nooman et al., 1973]. However, current evedence shows that cell-mediated immune mechanisms appear to play a key role in the resolution or persistence of HBV infection, [zucherman, 1981].

In Egypt, it's known that schistosomiasis plays an important role in hepatosplenomegaly. But, at the same time, there are many other causes of such a case. So, we think it's important to widen the scope in search of the frequency of HBV among hepatosplenomegalic patients in general.

Aim of work :

It was our aim to through light on:

The frequency of HBV markers among Egyptian patients suffering from hepatosplenomegaly.

REVIEW OF LITERATURE

VIRAL HEPATITIS

Knowledge of viral hepatitis has expanded greatly in the last ten years and several excellent monographs on the subject have been published, [Krugman and Groche, 1978; Vyas et al., 1978 and Sherlock, 1981].

Hepatitis is a severe viral disease of man that is actually increasing in incidence as a result of changing social conditions and lack of effective vaccine [Zuckerman, 1978]. In general, "Hepatitis" literally means inflammation of the liver, but practically it's an injury of hepatocytes that initiates inflammatory reations. Indeed, the injury is what is measured with the various hepatic tests and probably is responcible for many of the clinical symptoms. Inflammation may persist in and around the portal tracts after hepatocyte injury has subsided, and then it represents "self perpetuation" which may be related to immunologic ciculatory or virologic factors, [Popper and schaffner, 1970].

Many viruses are known to cause hepatitis. These include viral hepatitis type A [infectious hepatitis, epidemic catarrhal jaundice, short-ioncubation hepatitis] caused by hepatitis A virus [HAV]; viral hepatitis type B [Serum or transfusion hepatitis, homologous serum jaundice,

long-incubation hepatitis] caused by hepatitis B virus [HBV]; viral hepatitis type non-A, non-B caused by one or more non-A, non-B heptitis viruses, [Hoofnagle et al., 1985; Hollinger and Dreesman, 1986]. In addition, there is a new liver disease, viral hepatitis type D. It's caused by a recently discovered agent designated hepatitis D virus [HDV]. Generally speaking this is a defective virus which requires the presence of infectious HBV for its own replication [Rizzotto M, 1983]. Other viruses that may occasionally be implicated in hepatitis include human cytomegalovirus [CMV], Epstein-Barr virus, rubella virus, yellow fever virus, herpes simplex virus, and some enterovivuses [Hollinger, 1986; Aida A El Azim et al., 1987]. The great majority of clinically significant hepatitis infections are due to the first three hepatitis viruses mentioned above [shanson, 1982].

— ***Epidemiological and clinical features: viral hepatitis types A, B, and non-A, non-B*** :

It's evident that hepatitis B, so-called serum, and hepatitis A, previously called infectious hepatitis, are separate and distinct entities. The evidence is both clinical and immunologic. [Prince, 1968].

Feature	Type A	Type B	Non-A, Non-B
- *I.P.*	2-7 weeks [avg, 4-1]	4-20 weeks [avg, 11 – 4]	2-20 weeks [avg, 7 – 2]
- *Principle age distribution*	children, young young adults	adult	?
- *Seasonal incidence*	throughout the year but tends to fin autumn	throughout the year	throughout the year
- *Rout of Infection*	predominantly fecal-oral	parentral, sexual contact	predominanthy parentral
- *Occurance of virus in*			
*blood	-2 weeks before to <1 weeks after jaundice	Months to years	Months to years
*in Stool	-2 weeks before to 2 weeks after jaundice	Absent	Probably absent
*in urine	-Rare	Absent	probably absent
*in saliva	-Rare [saliva]	Frequently present	unknown
-Clinical & Laboratory Features			
Onset	usually abrupt	usually insidious	Insidious
fever >38 c	common early	less common	less common
Duration of transaminase elevation	2-6 weeks	2-6 + months	2-6 +months
Emmunogl-oulins [IgM levels]	Elevated	Normal to elevated	Normal to slightly elevated
Complications	Uncommon, no chronocity	Chronocity in 5-10%	Chronocity in 30-50%
Mortality rate [icteric cases]	< 0.5%	< 1-2 %	0.5-1%
HBs Ag	Absent	present	Absent

- *Immunity*			
Homologous	Yes	Yes	?
Heterologous	No	No	No
Duration	probably lifetime	probably life time	?
Gamma globulin prophylaxis	Regularly prevents jaundice	prevents jaundice only if gamma globulin is of sufficient potency against HBV	?

[Hollinger and Dienstag, 1985)

MODE OF TRANSMISSION OF HEPATITIS B VIRUS

In developed countries, the risk of exposure to hepatitis B appears to be greatest in certain groups of people [shanson, 1982]. These include :

1. Infants born to mothers with acute or chronic infection :

The virus is most likely to be acquired by neonatetes at parturition [Mc Donald et al., 1983].

2. Syringes, needles and other instruments :

Any instrument that breaks the skin of one person and then breaks the skin or mucosa of another without being sterilized has the potential for transmitting HBV [Koff and Galambos, 1982]

3. Transfusion of blood or blood derivatives

since long time viral hepatitis has been recognised to be the major hazard of blood transfusion and administration of blood derivatives. It has been demonstrated that paid blood donor presents a

significantly greater hepatitis B hazard to the recipient than does the volunteer replacement donor [Alter et al., 1975] .

4. Haemodialysis :

Hepatitis B is responsible for much morbidity in haemodialysis units among patients and staff and several staff members have died of it [Crosnier and Jungers, 1981].

5. Health service personnel :

Viral hepatitis appears to be an occupational hazard for health care workers and serologic evidence of HBV infection is found more commonly than in the general population. In contrast, members of health care personnel can be a source of HBV infection [Crob et al., 1981] .

6. Contact transmission :

This is suggested by several sets of epidemiologic observations as :

a. High proportion of infection among presons living in the same household as an HBs Ag carrier [Perrillo et al., 1979].