CERTAIN BIOCHEMICAL AND HEMATOLOGICAL PARAMETERS IN SOME CASES OF INFECTIOUS HEPATITIS

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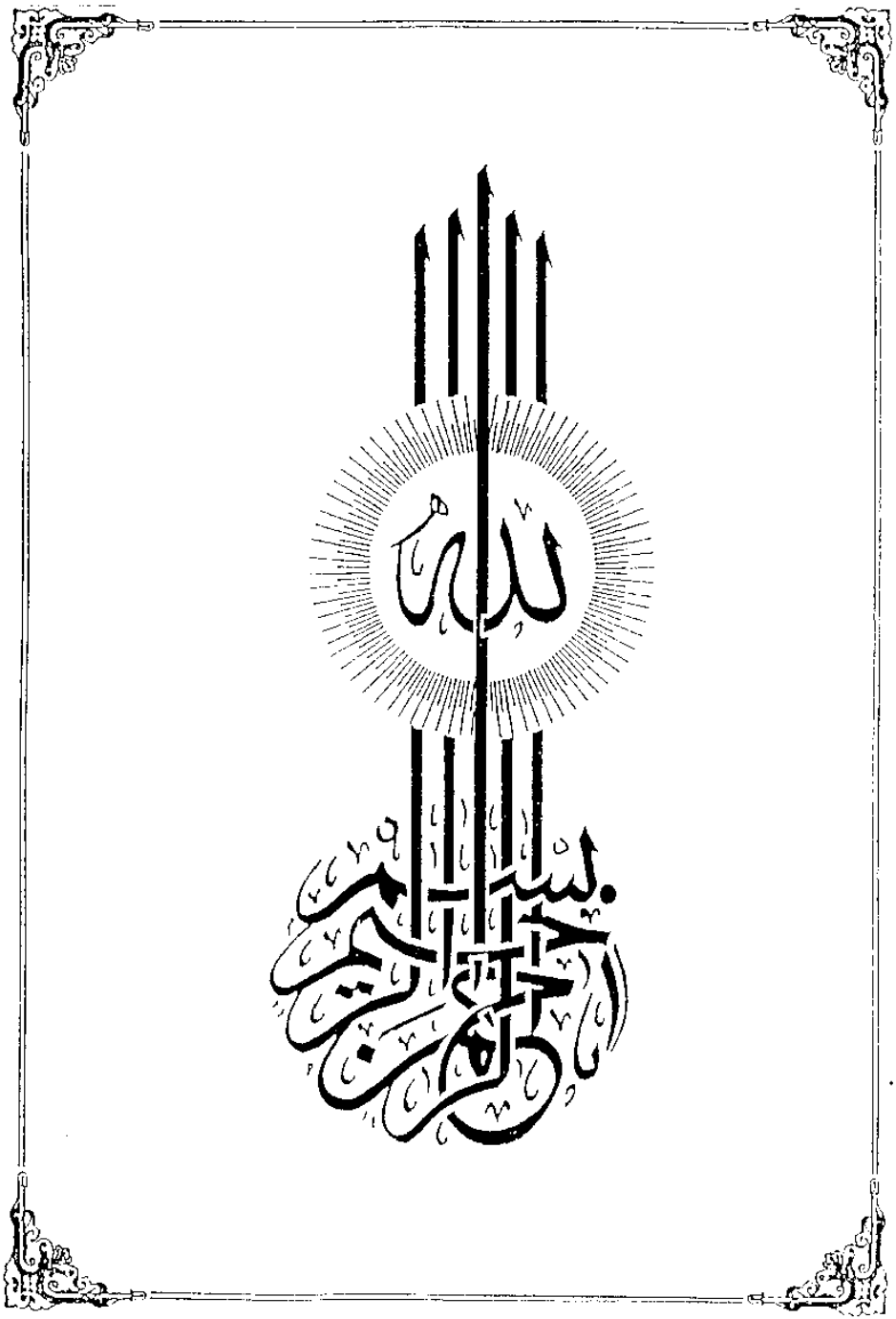

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C O N T E N T S

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Aim of the work

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AIM OF THE WORK

The aim of this work is to study some biochemical and hematological aspects of infectious hepatitis and to follow up the change in these aspects along the course of the disease.

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Review of the literature

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REVIEW OF LITERATURE

* VIRAL HEPATITIS

  A) HISTORICAL REVIEW.

  B) AETIOLOGY AND CLASSIFICATION

  C) CLINICAL FEATURES AND COURSE OF VIRAL HEPATITIS

- BIOCHEMICAL CHANGES IN VIRAL HEPATITIS

- HEMATOLOGICAL CHANGES IN VIRAL HEPATITIS

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A) Historical Review:-

Infective hepatitis was generally known as "catarrhal Jaundice" until just before the second world war. Virchow (1847) described it as an acute inflammation in the duodenum causing blockage of the common bile duct by mucous exudate. Although Eppinger (1937) in the first world war gave a correct description of the disease, which was substantiated by liver function studies, accurate autopsy observations were necessarily few and the old name was Perpetuated until the development of the punch-biopsy method by Rcholm and Iversen (1939), which enabled histological observations to be made at all stages of the disease.

The first refrence to epidemic jaundice has been ascribed to Hippocrates. The earlist record in Western Europe is in a letter Written in AD 751 by Pope Zacharias to St. Bonifac, Archbishop of Mainz . Since then there have been numerous accounts of epidemics, particularly during wars (Gardner, 1950).

B) Aetiology:

Viral hepatitis is caused by at least four different viruses, hepatitis A virus (infective hepatitis), hepatitis B virus (Serum hepatitis), and

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Non A, Non B hepatitis that is caused by more than one virus.

Hepatitis A and B have been characterized, and these infections can now be identified by sensitive laboratory tests for specific viral antigens and antibodies. Non-A, Non-B hepatitis is currently the commentest type of post transfusion hepatitis in areas of the world.

It is also an important cause of sporadic hepatitis in adults, although precise virological criteria and specific laboratory tests are not yet available. (Zuckerman, 1981).

Additional well charecterized viruses that can cause sporadic hepatitis such as yellow fever virus, cytomegalo virus, Epstein Barr virus, (infectious mononucleosis), Herps simplex virus,Rubeola (Measles) and the enteroviruses are mentioned (Jawetz, 1980).

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CLASSIFICATION AND TYPES

Virus A Hepatitis (HAV) - (Infectious Hepatitis):

It was not until 1973 that the virus responsible for hepatitis A was isolated in the stools of volunteer patients who developed acute hepatitis (Feinstone, et al 1973).

The identity of the virus was confirmed in several continents within 3 years.

HAV is non enveloped , 27-nm particles appearing full or empty under the electron microscope.

Inactivation of hepatitis A by RNAase suggests that it is an RNA virus belonging to the enterovirus family, (Feinstone, 1973)..

The RNA genome is a linear, single stranded molecules of 1.9 $x10^{-6}$. Lipid is not an integeral component of HAV, which is stable to treatment with ether, acid and heat (56°C for 30 minutes).

The infectivity can be prevented by autoclaving (121°C for 20 minutes) and the virus is destroyed by boiling in water for one minute or by ultraviolet irradiation and also by the treatment with formaline.

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The virus can be identified in the stool of sufferers from about two weeks before until one week after the onset of jaundice. (Dienstag, 1975).

Electron microscopic examination of infected liver reveals intracytoplasmic localization of virus particles.

Only one serotype is known, and there is no crossing with HBV.

HAV was identified initially in stool and liver preperation by employing immune electron microscopy.

The addition of specific hepatitis A antisera from convalescent patients to faecal specimen obtained from patient early in the incubation period of illness prior to the onset of jaundice, permitted condensation and visibility of virus particles by formation of antigen-antibody aggregates (Hollinger, 1975).

More sensitive serological assay such as the microtiter, solid phase immunoradiometric assay, and immune adherence have made it possible to detect HAV in stool, liver, homogenates, and to measure specific antibody in serum (Locarnini, 1979).

The HAV can grow in tissue culture of human

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human diploid fibroblasts.

The HAV antigen is located exclusively within the cytoplasm of the infected fibroblast (Gauss-Meller 1981), and has been grown in foetal rhesus-cell culture and primary explants of marmoset's liver (Provost, 1979).

A serum antibody (anti-HAV) appears as the stool becomes negative for virus, reaches a maximum in several monthes, and is detectable for many years.

Initially, antibody is predominantly of the Ig M calss, but an IgG antibody soon appear, and after several weeks to a few months the IgM antibody disappears. The IgG antibody persists in serum for, many years.

The presence of IgM indicate recent infection.

IgG anti HAV in the absence of IgM anti HAV implies prior exposure but the time of exposure cannot be estimated (Neimark, & Rogers, 1982).

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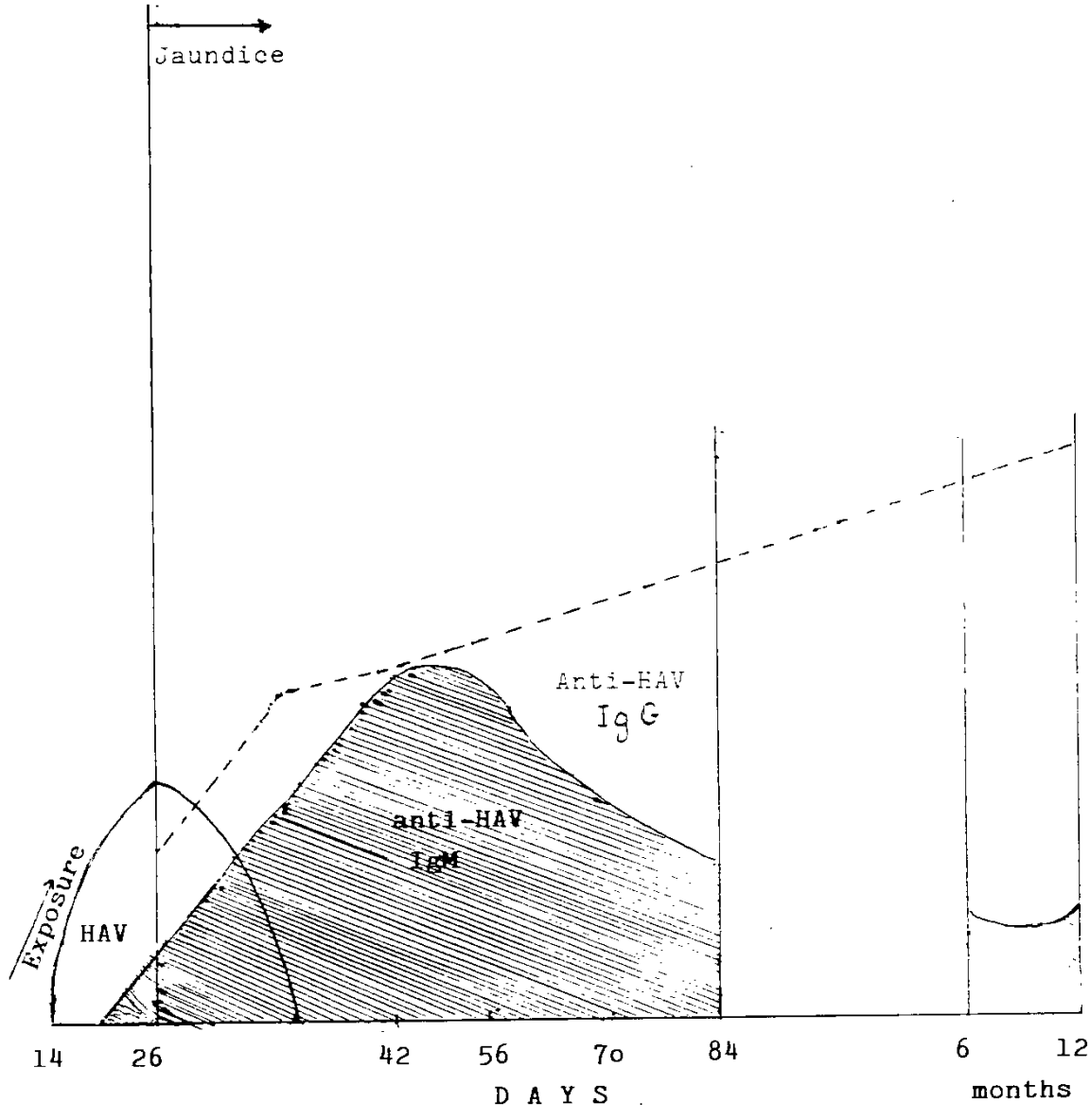

TYPICAL SEROLOGICAL RESPONSE IN HAV

(After Mortimer 1980)

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