

DUAL INFECTION OF HEPATITIS B AND C

Essay

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HDV	: Hepatitis D virus
HEV	: Hepatitis E virus
HPCE	: High performance capillary electrophoresis
IFN	: Interferon
IgG	: Immunoglobulin G
IgM	: Immunoglobulin M
IVDA	: Intravenous drug addict
LKMA	: Liver-kidney microsomal antibody
LSP	: Liver specific protein
M U	: Million Unit
NANBH	: Non-A, non-B hepatitis
OFLX	: Ofloxacin
ORFs	: Open reading frames
PBMcs	: Peripheral blood mononuclear cells
PCR	: Polymerase chain reaction
PCT	: Porphyria cutanea tarda
PDA	: Parenteral drug abuser
PHSA	: Polymerized human serum albumin
PTH	: Post transfusion hepatitis
RIBA	: Recombinant immunoblot assay
RNA	: Ribonucleic acid.
SGOT	: Serum glutamic oxaloacetic transaminase
SGPT	: Serum glutamic pyruvic transaminase
SMA	: Smooth muscle antibody
SOD	: Superoxide dismutase
TUDCA	: Tauroursodeoxycholic acid
UDCA	: Ursodeoxycholic acid
WHO	: World Health Organization
YDV	: Yeast derived vaccine

Viral Hepatitis

History of Viral Hepatitis

Outbreaks of jaundice, were described as early as the 5th century in babylonia, and the term epidemic jaundice appeared in writing of Hippocrates in Greece. Viral etiology of hepatitis was not postulated until the first decade of the 20th century (*McDonald, 1908*).

Early clinical experience suggested that infectious jaundice was transmitted by person-to-person contact and that the incubation period was several weeks long (*Pickles, 1930*).

Transmission to a laboratory worker who worked with serum from a known case was reported in 1931. Apparent transmission by a blood transfusion was observed in 1938. The first experimental transmission may have been achieved by Japanese workers in 1940 (*Yoshibumi and Shigemoto, 1941*).

The availability of specific serological markers to diagnose HAV and HBV infections did not solve the diagnostic problem of acute and chronic hepatitis, particularly that developing after blood transfusion [post-transfusion hepatitis (PTH)]. A third, major category has always been suspected but, in the absence of a diagnostic test, had been termed non-A, non-B hepatitis (NANBH), which is now recognized as hepatitis C (*Knodell et al., 1975*).

However, epidemiologic studies established the occurrence of NANBH more frequently outside the transfusion settings, intravenous drug users have displayed particular risk and comprised the largest known group of infected individuals (*Zuckerman, 1989*). Additionally direct

person to person transmission, by intimate or sexual contact, had been implicated (*Esteban et al., 1989*).

Definition of Viral Hepatitis

Viral hepatitis is defined as a systemic viral infection affecting the liver predominantly, in which hepatic cell necrosis and hepatic inflammation lead to characteristic clinical, biochemical, immunoserological, and morphological features. It is caused by at least 5, and possibly 6 viral agents. The etiologically identified forms are called: Hepatitis A Virus (HAV), Hepatitis B Virus (HBV), Hepatitis C Virus (HCV), Hepatitis D Virus (HDV), and Hepatitis E Virus (HEV) (*Edwards and Bouchier, 1991*). A sixth agent had been postulated (*Koff, 1993*).

Phillips et al. (1991), lately described, a viral particles associated with hepatitis named "G:" virus.

Hepatitis A Virus (HAV) :

Hepatitis A virus is a small, non enveloped 27 nm, heat, acid and ether-resistant RNA virus in the picornavirus family, it had been classified as enterovirus type 72 (*Dienstag and Isselbacher, 1994*). Its virion is composed of four polypeptides referred to as VP I to VP 4 (*Cohen, 1989*).

Only a single serotype had been identified. However, the genome has been cloned and characterized and a number of minor differences had been identified among isolates from different parts of the world (*Robertson et al., 1991*).

Inactivation of viral activity can be achieved by boiling for one minute, by contact with formaldehyde and chlorine, or by ultraviolet irradiation (*Dienstag and Isselbacher, 1994*).