Fifth International Symposium on Hepatitis Delta Virus and Liver Disease held on the 28-29 August 1995, The Marriott Surfers Paradise Resort, Queensland.

Report on International Symposium

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Hepatitis Delta virus (HDV) is a small RNA virus (1.7kb) which only infects individuals with co-existing hepatitis B virus (HBV) infection. The HDV particle consists of an outer envelope of HBV origin and inner ribonucleoprotein complex made of a circular single-stranded RNA genome and Two HDV encoded delta antigens (HDAg).

HDV causes severe chronic hepatitis and fulminant hepatitis especially among drug users with human immunodeficiency viris (HIV) and hepatitis C virus (HCV) infection. The high morbidity and mortality of HDV justifies the use of interferon therapy (though not always successfully!) in order to modify the natural course of the disease.

Three genotypes of Hepatitis Delta virus have been described. Genotype 1 is the predominant HDV genotype in most areas of the world. Two broadly defined subtypes of this genotype could be resolved with 1A in the USA and Western Europe and 1B in Eastern Asia with subtype 'mixing' in Southern Europe and the Middle East. Genotype 1 is found in Japan/Taiwan only and genotype 3 in South America only.

A new generation of Enzyme Immunoassay (EIA) for determination of IgM antibodies of Hepatitis Delta antigen, using recombinant Delta antigen from E coli, a murine monoclonal anti-Delta antibody and a chimeric (mouse + human) IgM anti-HDAg as positive control has been developed. Thus the presence of infectious Delta virus particles is avoided resulting in a 'safer' diagnostic assay.

However as the virus is found in patients only with HBV infection, and Hepatitis B vaccine is now in widespread use, there will be a decreasing incidence of the disease in those world regions with health resources able to immunise a large proportion of their population against HBV.

3rd International Meeting on Hepatitis C Virus and Related Viruses 30 August-3 September 1995, The Marriott Surfers Paradise Resort Queensland: Summary Report

The Centers for Disease Control (CDC) in the USA reports 100,000 new cases of hepatitis C virus (HCV) per year. The majority appear to be in intravenous drug users. Sexual transmission is inefficient but more research is required to identify the relative risk of acquisition.

The RNA genome of HCV contains a single open reading frame encoding a polyprotein of about 3000 amino acids. Processing of this viral precursor polyprotein produces 12 viral proteins. The structural region of the HCV polyprotein consists of a nucleocapsid core followed by two envelope glycoproteins, E1 and E2. Work presented later in the meeting showed 10 major genotypes (including 52 subtypes) of HCV.

Results showed that current serological assays used for the detection of HCV infection have different sensitivities for different virus genotypes. A bias towards genotype 1 exists. Antibody to E2 represents the most common serologic response in HCV infected individuals and its diagnostic potential was shown. Clearance of E2 was shown to be associated with complete recovery.

Serological HCV typing assays were shown to be convenient but their sensitivity is significantly reduced compared with molecular typing methods (PCR based).

Nosocomial HCV outbreaks in a paediatric oncology ward in Sweden were discussed. Between 1990-1993 nine unexplained cases occurred of HCV genotype 3a. Hygienic procedures were scrutinised and improved. In 1993 one child, infected with genotype 1b abroad, started oncology treatment. There were 9 cases of HCV genotype 1b in the ward in 1994 despite the awareness of nosocomial infection risks.

Sustained alpha interferon responders can be identified before therapy by liver histology, genotype and viral quantity, or during therapy by an early HCV-RNA response. Viraemia levels now appears to be the critical factor with respect to non-response to interferon therapy. The question of treatment following needlestick exposure to HCV was raised and the consensus was to wait for ALT levels to rise and then treat with alpha-interferon.

Hepatitis G virus (HGV) is a novel flavivirus-like agent and its 'discovery' is a result of collaboration twixt Steven Foung, Genelabs, Boehringer Mannheim, laboratories at CDC, London and Greece. It is a human virus and is found worldwide. The search for another 'agent' was because 18% of community acquired hepatitis were non A,B,C,D,E and 12% of patient cases were non A,B,C,D,E! HGV was shown to have only 26% identity with HCV at the amino acid level. It is a transfusion transmitted, usually mild disease which can be transmitted, coexist with HCV, persist and result in chronic hepatitis. The prevalence of HGV in blood donors is higher than that of HCV and unrelated to the ALT status of the donor. A nucleic acid based assay will be available by the end of 1995 for the agent.

Abbott Laboratories described 'their' newly discovered flavi-like viruses GBV agents (GB being the initials of the surgeon who developed hepatitis and whose serum was used for inoculation of tamarinds). The size of GBV are similar to HCV and they are positive strand RNA viruses. Three agents were 'discovered' GBV-A, GBV-B and GBV-C. The GBV-A agent is an endogenous tamarind virus, the GBV-B agent causes a viraemia with ALT elevation and induces protective immunity and there are 4 genotypes of GBV-C. Some patients with fulminant hepatitis also had GBV.

HGV and GBV-B, GBV-C may prove to be the same agent and they do not appear to be a form of HCV.