

Hepatitis D: Recent Advances in Management

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Infection by hepatitis D virus (HDV or delta hepatitis virus) contributes to the most severe form of chronic viral hepatitis and causes progression to liver cirrhosis in a significant proportion of hepatitis B surface antigen (HBsAg) carriers. By itself, without the assistant function of hepatitis B virus (HBV), HDV cannot contribute to liver disease. Only as a co-infection with HBV can HDV cause human liver disease. HBsAg covering for complete virion morphogenesis of HDV nucleoprotein complex occurring in nucleus and secreting into the cytoplasm is needed for HDV self-ligation and HDV-RNA self-cleavage via phosphorylation. Propagation of HDV and HDV cellular attachment to the hepatocytes in chronic hepatitis D (CHD) can be achieved by the HBsAg envelop of the HDV virion. American patients with chronic HBV and possible co-infection with HDV may be accounted for more than 11.8% of them, whereas HDV co-infection among patients with chronic HBV are tested only 4.7% of them. Around 4.3% to 5.7% of chronic hepatitis B carriers is documented among individuals with HDV infection globally. In Mongolia and Pakistan, the World Health Organization (WHO) reported that HDV prevalence was at least 60%. HDV prevalence in chronic hepatitis B-infected patients is higher in certain regions of the world, including Russia, China, Central Asia, Turkey, South America, and Africa.

Epidemiologically, genotype I HDV distributes throughout the world. Genotype II HDV identified in the Far East is associated with a milder form CHD, compared to genotype I. In the Amazon region of South America, genotype III HDV with a specific severe clinical features is identified. In Africa and the Far East (Japan and Taiwan), genotype IV HDV formerly recorded as genotype IIb HDV Exclusively, genotype V to VIII HDV are found in Africa. Possible mild form of African CHD with possible interferon (IFN) response was recently reported. A Brazilian study on HDV viral load comparing to genotype D or F patients demonstrated decrease in patients with genotype A. A study from the US demonstrated that only 499 patients out of 1,191 chronic hepatitis-B patients had been tested for anti-HDV. Positive anti-HDV results was found in 42 (8%) of these 499 patients. Among 42 HDV co-infected patients, 70% of them had liver cirrhosis.

Over 30 years, the management of CHD has not changed that consists of interferon (IFN) treatment. Pegylated IFN, the only modified therapy that switch from conventional IFN treatment. Peg-IFN alpha 2a should be administered as subcutaneous injection at a dose of 180 µgm one weekly whereas peg-IFN alpha 2b should be administered at a dose of 1.5 µgm/kg with the most-studied and -used treatment duration of one year. Interestingly, peg-IFN-adefovir combination therapy leads to the decline of quantitative HBsAg levels with unknown mechanisms and is more pronounced compared to peg-IFN monotherapy. Additionally, a previous study showed that aminoglycosides exert strong suppression effects on HDV ribozyme activities, isoprenylation of large HDAg and glycosylation of HBsAg. Novel vaccine candidates were investigated through both proteins of HDV (ADAg p24 and p27). The protein immunization induced a specific antibody response, nevertheless, there is no protection from HDV superinfection. A previous study from China showed that HBV vaccinating every newborn in China will prevent 1.69 million new infections by 2028, compared to the present 90% HBV vaccination coverage.

In conclusion, new treatment strategies for CHD are urgently needed. Nucleic acid polymers, prenylation inhibitors, and hepatocyte entry inhibitor represent a great hope for CHD patients. The best CHD treatment option is hardly to determine. Possibly, peg-IFN in combination with drugs mentioned above will be implemented. New strategies to develop a candidate protective vaccine for preventing HDV superinfection are urgently needed.

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