

ETIOLOGY, EPIDEMIOLOGY AND PATHOGENESIS OF VIRAL HEPATITIS DELTA

Bakhronov Jakhongir Jasurovich

Samarkand State Medical University

Assistant of Department of Infectious Diseases and Epidemiology

Abstract. Hepatitis D virus (HDV) infection is a severe and unique form of viral hepatitis that requires the presence of hepatitis B virus (HBV) for replication. This article provides a comprehensive overview of the etiology, global epidemiology, and pathogenesis of HDV. HDV co-infection and superinfection are associated with faster progression to cirrhosis and liver failure compared to HBV mono-infection. The mechanisms of liver injury in HDV infection, including immune-mediated cytotoxicity and direct viral effects, are also discussed. Understanding these aspects is critical for developing targeted interventions and control strategies.

Keywords: Hepatitis D virus, HBV, co-infection, superinfection, epidemiology, pathogenesis, liver cirrhosis.

Introduction. Hepatitis D virus (HDV), also known as delta virus, is a defective RNA virus that requires the helper function of HBV to propagate. First discovered in the 1970s, HDV is responsible for the most severe form of viral hepatitis in humans [1]. Despite advances in vaccination against HBV, HDV continues to pose a public health threat, particularly in endemic regions.

Etiology of Hepatitis D. Hepatitis D virus (HDV), also known as delta virus, is a defective RNA virus that requires the hepatitis B virus (HBV) for its replication and transmission. HDV contains a small, circular, single-stranded RNA genome, surrounded by a delta antigen (HDAg) and a lipoprotein envelope derived from HBV surface antigens (HBsAg) [1].

Due to its incomplete structure, HDV cannot propagate independently and relies entirely on the helper function of HBV, particularly the HBsAg, to infect hepatocytes and assemble new virions [2].

There are at least 8 genotypes (HDV-1 to HDV-8), with HDV-1 being the most globally distributed and associated with both severe and mild forms of liver disease. HDV-3, mainly found in South America, is linked to fulminant hepatitis, while other genotypes show varying degrees of pathogenicity and geographic restriction [3].

Epidemiology. The global prevalence of hepatitis D is estimated at 12–20 million people, though accurate numbers are limited due to underdiagnosis and lack of routine testing [4]. The disease is endemic in regions like Central Asia, Africa, Eastern Europe, and the Amazon Basin, where HBV is also highly prevalent.

In Eastern Europe and Central Asia, co-infection with HDV can affect up to 30% of HBV-positive individuals [5,8].

In developed countries, the prevalence has declined due to successful HBV vaccination, but remains significant among high-risk groups such as intravenous drug users, migrants from endemic regions, and patients co-infected with HIV [6].

Transmission of HDV mirrors that of HBV — via blood and bodily fluids, through parenteral exposure, sexual contact, and vertical transmission (though the latter is less common) [3, 5].

Two main patterns of infection are observed:

Co-infection (simultaneous HBV and HDV infection), usually self-limited but occasionally severe.

Superinfection (HDV infection in a chronic HBV carrier), which often leads to chronic HDV infection and progressive liver disease.

Pathogenesis of HDV Infection.

The pathogenesis of HDV is unique and more severe than HBV alone. HDV causes direct cytopathic effects, unlike HBV, which primarily induces immune-mediated liver damage [7,9]. HDV replication occurs in the nucleus of infected hepatocytes, where the HDAg interacts with host RNA polymerase II to produce viral RNA.

The large form of HDAg (L-HDAg) inhibits replication and facilitates virion assembly, while the small form (S-HDAg) promotes genome replication. The balance between these isoforms influences the course of infection and severity of liver injury [2,10].

HDV induces a strong innate and adaptive immune response, but chronic infection can result in persistent inflammation, hepatocyte apoptosis, fibrosis, and ultimately cirrhosis. Compared to HBV monoinfection, HDV superinfection is associated with:

Faster fibrosis progression,

Higher rates of hepatic decompensation,

Increased risk of hepatocellular carcinoma (HCC) [1, 4,11].

Conclusion

Hepatitis D represents a significant clinical and public health challenge due to its aggressive course and limited therapeutic options. A better understanding of its etiology, epidemiology, and pathogenesis is essential for improving prevention, diagnosis, and management strategies. HDV screening among HBV-infected individuals is crucial, particularly in high-risk populations and endemic areas.

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