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CLASSIFICATION AND CHARACTERISTICS OF HEPATITIS VIRUSES

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Abstract

This article provides a comprehensive overview of hepatitis viruses, their classification, and their main biological and clinical characteristics. Hepatitis viruses constitute a group of pathogens that damage liver cells and are characterized by diverse routes of transmission and clinical courses. To date, five major types of viral hepatitis—A, B, C, D, and E—have been identified, each of which differs significantly in etiology, epidemiology, pathogenesis, and clinical manifestations. The abstract emphasizes that hepatitis A and E viruses are primarily transmitted via the fecal–oral route, usually present as acute infections, and do not progress to chronic disease. In contrast, hepatitis B and C viruses are transmitted through blood and other biological fluids and play a major role in the development of chronic hepatitis, liver cirrhosis, and hepatocellular carcinoma. It is also scientifically substantiated that hepatitis D virus infection occurs only in the presence of hepatitis B virus and significantly aggravates the severity of the disease.The article presents general concepts regarding the genomic structure of hepatitis viruses, their effects on the immune response, clinical manifestations, complications, and preventive measures.

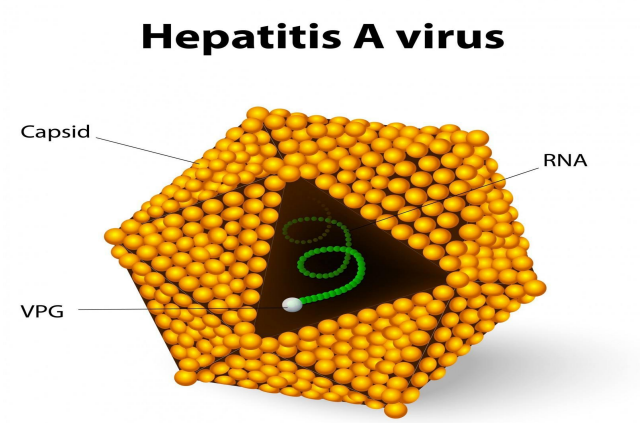
Keywords

Viral hepatitis; Hepatitis A, B, C, D and E viruses; Liver diseases; Classification of viruses; Routes of transmission; Clinical course; Prevention and vaccination.

INTRODUCTION:

Viral hepatitis represents a major danger to public health, and is a globally leading cause of death[1,2]. The five liver-specific viruses: Hepatitis A virus (HAV), hepatitis B virus (HBV), hepatitis C virus (HCV), hepatitis D virus (HDV), and hepatitis E virus (HEV), each have their own unique epidemiology, structural biology, transmission, endemic patterns, risk of liver complications, and response to antiviral therapies. Most of the hepatitis viruses are considered enveloped. Recently, it was reported that the non-enveloped HAV and HEV are, in reality, quasi-enveloped viruses exploiting exosomal-like biogenesis mechanisms for budding. Regardless, all hepatitis viruses use exosomes to egress, regulate, and eventually escape from the host immune system, revealing another key function of exosomes apart from their recognised role in intercellular communication. This review will discuss how the hepatitis viruses exploit exosome biogenesis and transport capacity to establish successful infection and spread. Then, we will outline the contribution of exosomes in viral persistence and liver disease progression.

MAIN PART:

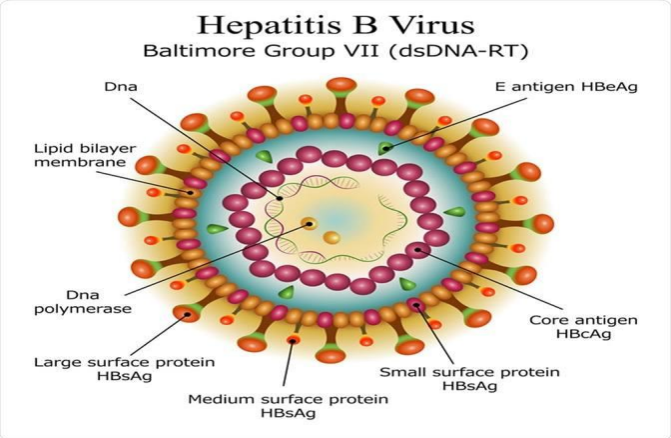


1.1-picture

Figure 1.1 depicts the hepatitis A virus.

Hepatitis A virus (HAV) is an RNA-containing virus that causes acute inflammatory disease of the liver. It belongs to the Picornaviridae family and the Hepatovirus genus and lacks an external lipid envelope. The viral particles are spherical in shape, with a diameter of approximately 27–32 nm. The genome of HAV consists of single-stranded, positive-sense RNA, which plays a key role in viral replication and protein synthesis. HAV is primarily transmitted via the fecal–oral route. Infection most commonly occurs through the consumption of contaminated drinking water and food, as well as through close person-to-person contact in conditions of inadequate hygiene. The virus demonstrates a high level of resistance to environmental factors and can remain viable for prolonged periods in water, food products, and on various surfaces, which contributes to its epidemiological significance. After entering the human body, HAV replicates in hepatocytes and induces liver injury mainly through immune-mediated mechanisms rather than direct cytopathic effects. The incubation period typically ranges from 15 to 45 days. Clinically, hepatitis A manifests with general weakness, loss of appetite, nausea, abdominal discomfort, fever, and jaundice. In most cases, the disease follows a mild to moderate course and resolves spontaneously.

A distinctive feature of HAV infection is that it does not progress to a chronic form. Recovery is usually complete and accompanied by the development of long-lasting, often lifelong immunity, which effectively prevents reinfection. Severe complications are rare but may occur in elderly individuals and patients with pre-existing chronic liver diseases. Prevention of hepatitis A relies on both sanitary-hygienic measures and immunization. Ensuring access to safe drinking water, maintaining proper personal hygiene, and improving food safety standards are essential preventive strategies. In addition, active immunization with inactivated hepatitis A vaccines represents the most effective and reliable method for preventing HAV infection.



1.2-picture.

Figure 1.2 depicts the hepatitis B virus.

Hepatitis B virus (HBV) is a DNA-containing virus that causes infectious inflammatory disease of the liver. It belongs to the Hepadnaviridae family and has a complex structure surrounded by an outer lipid envelope. The complete infectious virion, known as the Dane particle, is spherical in shape and represents the mature form of the virus. The HBV genome consists of partially double-stranded circular DNA, which distinguishes it from other hepatitis viruses. HBV is primarily transmitted via parenteral routes, including exposure to infected blood and other biological fluids. Infection may occur during medical and invasive procedures performed without proper sterilization, blood transfusions, unprotected sexual contact, and vertical transmission from mother to child during childbirth. The virus demonstrates high resistance to environmental conditions and can remain viable in dried blood for extended periods, increasing the risk of transmission. After entering hepatocytes, HBV undergoes a complex replication cycle that involves reverse transcription, a unique feature among DNA viruses. Viral replication occurs within the nucleus and cytoplasm of liver cells. Hepatocellular damage is largely mediated by the host immune response rather than direct viral cytopathic effects, leading to inflammatory changes in liver tissue. Clinically, hepatitis B may present in acute or chronic forms. Acute hepatitis B is commonly characterized by general weakness, fatigue, loss of appetite, nausea, abdominal discomfort, and jaundice, although asymptomatic and subclinical cases are also observed. Chronic hepatitis B is associated with persistent viral replication and inflammation, which may progress to liver fibrosis, cirrhosis, and hepatocellular carcinoma. Prevention of hepatitis B relies heavily on vaccination strategies. Recombinant hepatitis B vaccines induce strong and long-lasting immunity and have significantly reduced the global burden of HBV infection. Additional preventive measures include the use of sterile medical equipment, screening of blood and blood products, and regular monitoring of individuals at high risk of infection.

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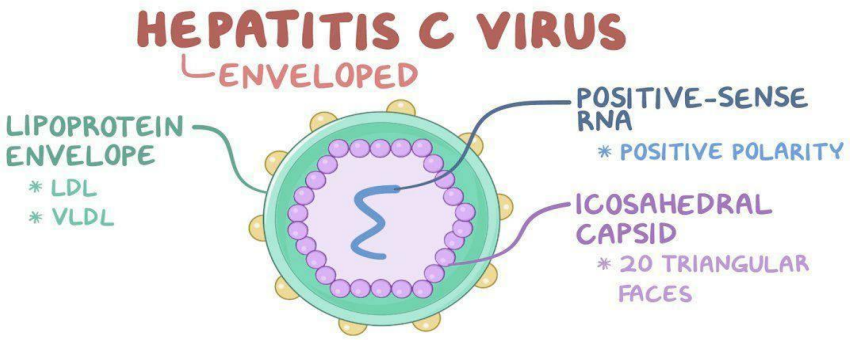


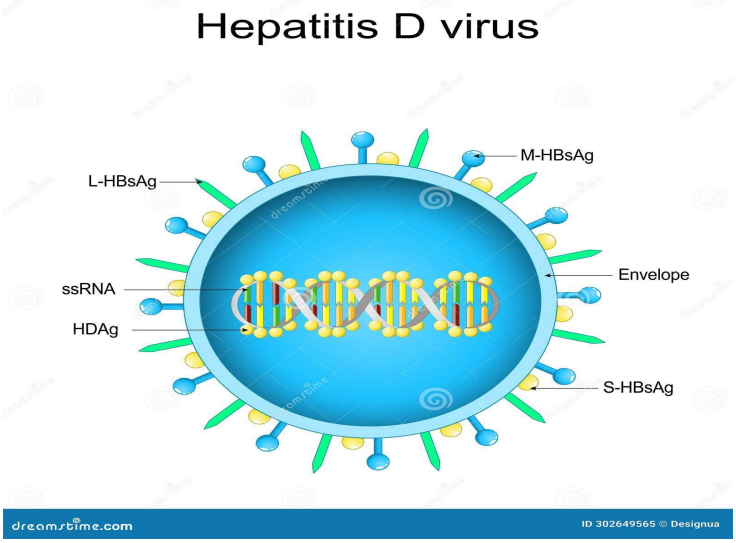
1.3-picture.

Figure 1.3 depicts the hepatitis C virus.

Hepatitis C virus (HCV) is an RNA-containing virus that causes infectious inflammatory disease of the liver. It belongs to the Flaviviridae family and the Hepacivirus genus. The virus is surrounded by a lipid envelope, and its genome consists of single-stranded, positive-sense RNA. A distinctive feature of HCV is its high genetic variability, which plays a critical role in immune evasion and the development of chronic infection. HCV is primarily transmitted through parenteral exposure to infected blood. The most common routes of transmission include medical and invasive procedures performed without adequate sterilization, transfusion of contaminated blood and blood products, sharing of needles and syringes, and hemodialysis procedures. Sexual and vertical transmission from mother to child may occur but are less efficient compared to hepatitis B virus.

After entering the host, HCV replicates mainly in hepatocytes, leading to functional impairment of liver cells. The pathogenesis of hepatitis C is characterized by a prolonged inflammatory process. Acute HCV infection is often asymptomatic or presents with mild, nonspecific clinical manifestations, which contributes to delayed diagnosis. In a significant proportion of infected individuals, the disease progresses to a chronic form.Chronic hepatitis C is associated with progressive liver fibrosis and significantly increases the risk of developing liver cirrhosis and hepatocellular carcinoma. In advanced stages, patients may develop signs of liver failure, which severely affect quality of life and prognosis.

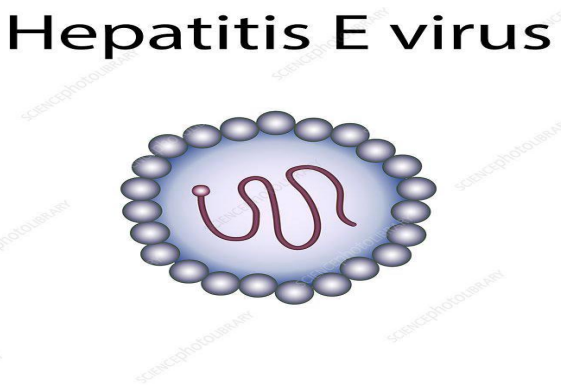
Currently, no effective vaccine against hepatitis C is available. Therefore, prevention relies mainly on reducing the risk of transmission through strict adherence to infection control measures. The use of sterile medical instruments, mandatory screening of blood and blood products, safe injection practices, and early identification of individuals at high risk remain essential strategies in the prevention of HCV infection.



1.4-picture.

Figure 1.4 depicts the hepatitis D virus.

Hepatitis D virus (HDV) is a unique defective RNA virus that requires the presence of hepatitis B virus (HBV) for replication and infectivity. HDV cannot propagate independently, as it relies on HBV surface antigen (HBsAg) to form its outer envelope. Consequently, HDV infection occurs only in individuals who are co-infected with HBV or develop superinfection on top of a pre-existing HBV infection.The HDV genome consists of single-stranded, circular RNA. The virus is small in size, and its envelope is derived from HBsAg of HBV, which facilitates viral entry into host cells and spread within the body. HDV replication differs from other RNA viruses and largely depends on host cellular enzymes to complete its life cycle.HDV is primarily transmitted via parenteral routes, including exposure to infected blood and other biological fluids. Common modes of transmission include unsafe medical procedures, sharing of needles or syringes, and direct contact with contaminated blood. Sexual and vertical transmission are possible but less frequent.mClinically, HDV infection significantly worsens the course of hepatitis B. In co-infection, acute hepatitis symptoms may be severe, while superinfection often leads to rapid progression of chronic liver disease, including cirrhosis and liver failure. HDV is characterized by its ability to induce rapid and destructive changes in liver tissue.Prevention of hepatitis D relies predominantly on hepatitis B vaccination. By preventing HBV infection, the spread of HDV can be effectively controlled. Additionally, strict adherence to infection control measures, including safe medical practices and proper sterilization, is essential to reduce the risk of transmission.



1.5-picture.

Figure 1.5 depicts the hepatitis E virus.

Hepatitis E virus (HEV) is an RNA-containing virus that causes acute inflammatory disease of the liver. It belongs to the Hepeviridae family and possesses a single-stranded, positive-sense RNA genome. The virus is non-enveloped and spherical, with particles measuring approximately 27–34 nm in diameter. The HEV genome contains several genes responsible for viral replication and protein synthesis.HEV is primarily transmitted via the fecal–oral route, meaning that contaminated water and food are the main sources of infection. Outbreaks often occur in areas with poor sanitation, and waterborne transmission represents the principal mode of spread. The virus is relatively stable in the environment and can survive in water for extended periods, which contributes to its epidemiological significance.Hepatitis E typically presents as an acute illness, and chronic infection is rare. The incubation period usually ranges from 2 to 8 weeks. Clinically, HEV infection manifests with general malaise, fever, loss of appetite, nausea, abdominal discomfort, and jaundice. In most cases, the disease follows a mild to moderate course; however, in pregnant women, HEV infection may be more severe and is associated with an increased risk of liver failure.

Prevention of HEV infection relies primarily on adherence to hygiene practices and the provision of safe drinking water. While vaccines against HEV have been developed in some countries, no widely used vaccine is currently available.

CONCLUSION

Hepatitis viruses are a significant group of pathogens that infect liver cells and have considerable impacts on human health. They are classified into five major types—A, B, C, D, and E—each distinguished by its genomic structure, routes of transmission, clinical course, potential for chronic infection, and associated complications.Hepatitis A and E viruses are primarily transmitted via the fecal–oral route, typically cause acute infections, and do not lead to chronic disease. In contrast, hepatitis B and C viruses are transmitted through blood and other biological fluids and are frequently associated with chronic hepatitis, liver cirrhosis, and hepatocellular carcinoma. Hepatitis D virus occurs only in the presence of hepatitis B virus, significantly worsening the severity of liver disease.

Prevention of hepatitis virus infections relies heavily on hygienic practices, access to safe drinking water, use of sterile medical instruments, and available vaccines. Notably, HBV vaccination also effectively prevents HDV infection.

Overall, continued research on hepatitis viruses, their epidemiology, and pathogenesis, along with strengthened preventive and control measures, is essential for reducing the burden of liver diseases and minimizing associated complications.

REFERENCES

1. Brooks G.F., Carroll K.C., Butel J.S., Morse S.A.Jawetz, Melnick & Adelberg's Medical Microbiology. — New York: McGraw-Hill, 2019.
2. Centers for Disease Control and Prevention (CDC). Viral Hepatitis Surveillance and Case Management. — Atlanta, 2021.
3. Dienstag, J. L. (2008). Hepatitis B virus infection. New England Journal of Medicine, 359(14), 1486–1500. https://doi.org/10.1056/NEJMra0801272
4. Kamar, N., Dalton, H. R., Abravanel, F., & Izopet, J. (2014). Hepatitis E virus infection. Clinical Microbiology Reviews, 27(1), 116–138. https://doi.org/10.1128/CMR.00057-13
5. Knipe D.M., Howley P.M.Fields Virology. — 7th ed. — Philadelphia: Wolters Kluwer, 2021.
6. Lemon, S. M., & Thomas, D. L. (2019). Hepatitis viruses (6th ed.). Philadelphia: Elsevier.
7. Murray P.R., Rosenthal K.S., Pfaller M.A. Medical Microbiology. — 9th ed. — Philadelphia: Elsevier, 2020.

8. Rizzetto, M., Ciancio, A., & Bréchot, C. (2018). Hepatitis D: twenty-five years after the discovery. Digestive and Liver Disease, 50(3), 196–202. https://doi.org/10.1016/j.dld.2017.09.014
9. Thomas, D. L. (2019). Global control of hepatitis C: where challenge meets opportunity. Nature Reviews Gastroenterology & Hepatology, 16, 541–558. https://doi.org/10.1038/s41575-019-0165-0
10. World Health Organization (WHO). Hepatitis A. Geneva: WHO; 2024. Available from: https://www.who.int/news-room/fact-sheets/detail/hepatitis-a
11. World Health Organization (WHO). Hepatitis E. Geneva: WHO; 2024. Available from: https://www.who.int/news-room/fact-sheets/detail/hepatitis-e