

Epidemiology & Prevention of Hepatitis

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ABSTRACT

Viral hepatitis, encompassing a spectrum of liver infections primarily caused by hepatitis viruses A, B, C, D, and E, remains a significant global health challenge. These viruses differ considerably in their transmission routes, disease progression, and potential for chronicity. This review provides a comprehensive overview of the epidemiology, pathogenesis, and current therapeutic strategies for each type of viral hepatitis, with a particular emphasis on recent advancements in treatment and prevention.

This review summarizes the pathogenesis of these viruses, explores prevention strategies such as vaccination and public health measures, and discusses current and emerging treatments.

Viral hepatitis is associated with high morbidity and mortality worldwide. Hepatitis A and E viruses are enterally transmitted and typically cause acute self-limited hepatitis. Hepatitis B, C, and D viruses are parenterally transmitted and can cause chronic hepatitis, with potential progression to cirrhosis and hepatocellular carcinoma.

Hepatitis A virus causes acute viral hepatitis, presenting with jaundice, nausea, and vomiting. Symptoms are self-limited and treated symptomatically. Routine vaccination of infants has greatly reduced the incidence in the United States. Vaccination is recommended for all infants and adults at increased risk. Hepatitis B virus can cause acute and chronic viral hepatitis. Acute hepatitis B is usually self-limited, but can develop into a chronic infection. Patients are at highest risk for developing chronic disease if they are infected at a younger age. Routine vaccination and universal screening of pregnant women has decreased the transmission of hepatitis B virus.

Keywords: Hepatitis, Virology, Acute, Chronic, Liver Disease

INTRODUCTION

Hepatitis, an inflammation of the liver, can arise from various causes, including infections, toxins, and autoimmune diseases.¹ Hepatitis is the term for liver inflammation. An injury or infection to the body's tissues results in inflammation. As a result, the liver cannot function correctly. Acute (short-term) or chronic (long term) hepatitis infections are both possible. Only certain forms of hepatitis can produce acute infections, but other types

of infections can cause both acute and chronic illnesses. There are various forms of hepatitis, each with its own etiology. Viral hepatitis is the most prevalent kind and is caused by one of several viruses. These are A, B, C, D, and E hepatitis viruses. Where A, B, and C are the most prevalent.²

The World Health Organization (WHO) 'triple elimination initiative' includes the elimination of mother-to-child transmission HBV, along with of human immunodeficiency virus (HIV) and syphilis.³

Unfortunately, hepatitis is very common, especially in parts of the world where clean drinking water and vaccines aren't readily available. The World Health Organization estimates that 354 million people live with Hepatitis B or C. That's why knowing the symptoms, understanding when to seek medical care, and learning how to prevent your risk of developing hepatitis is essential.¹⁰

Progress in therapeutic strategies:

Therapeutic advancements have transformed the prognosis of viral hepatitis, particularly for HBV and HCV. The shift from interferon-based regimens to targeted antivirals has improved patient compliance and sustained virologic response.⁷

Immunomodulators: Interferon-alpha, once the mainstay, is still used in specific HBV and HDV cases. Its immunostimulatory action offers a finite treatment duration, though with higher adverse effects.⁷

Causes:

The five strains of hepatitis are caused by each of their own individual viruses. You can contract a hepatitis virus from the following transmission routes:³

National Institute of Diabetes and Digestive and Kidney Diseases. Hepatitis (viral).

Digesting unclean food or drinking water

Using contaminated needles or syringes

Exchanging bodily fluids such as blood or semen

Coming into contact with an infected person's urine or stool

Having sex with an infected person

A parent with hepatitis passing on the infection to their newborn during childbirth

Common Symptoms

Each type of viral hepatitis can produce its own unique set of symptoms. However, there are some common symptoms that they share. These symptoms include:

- Jaundice, or the yellowing of the skin and eyes.
- Abdominal pain on the right side of your stomach
- Nausea and vomiting
- Loss of appetite

- Fatigue
- Malaise, or a general feeling of being unwell
- Muscle or joint pain
- Darker urine color than normal

It's worth noting that while these are common signs of hepatitis, you might not experience all of these symptoms.⁹

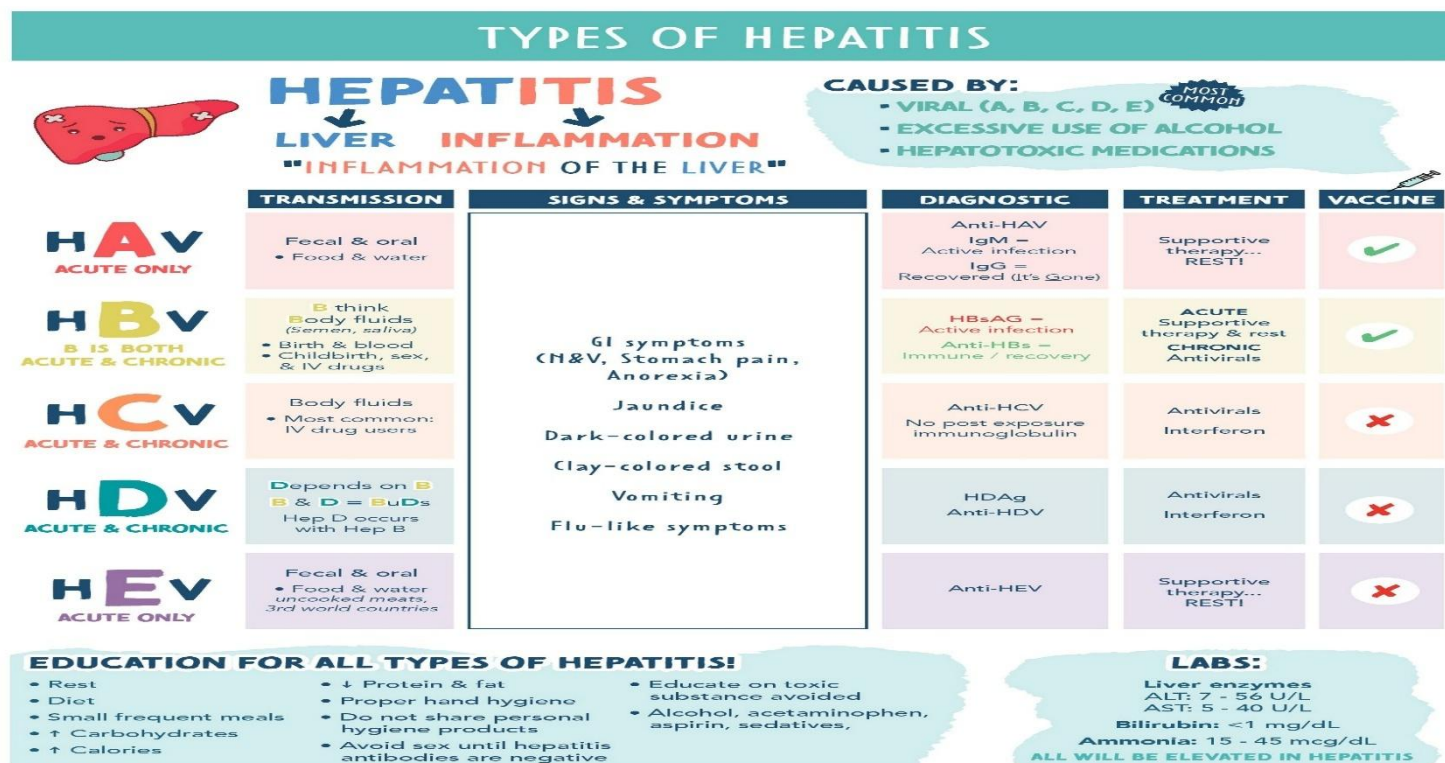


Figure 1: types of Hepatitis

Table 1: Diagnostic Tools for Hepatitis Viruses

Diagnostic Method	Application area	Target virus
RT-PCR / PCR	Viral load detection	HBV,HCV
ELISA	Antibody/antigen detection	All types
Rapid Diagnostic Tests	Field screenig	HAV,HBV,HCV
Fibrosis biomarkers	Disease staging	HBV,HCV

Table 2: Current and Emerging Therapeutic Strategies for Viral Hepatitis

Virus Type	Standard therapy	New /Emerging therapies
HBV	Tenofovir, Entecavir	siRNA, capsid inhibitors, , Bulevirtide
HCV	Sofosbuvir, Velpatasvir	Pan-genotypic DAAs

HDV	Interferon-alpha	Bulevirtide (entry inhibitor)
HAV & HEV	Supportive care	HEV vaccine (Hecolin in China), trials ongoing

Types of Hepatitis:

Hepatitis A:

Positive-strand RNA hepatitis A virus (HAV) can persist in the environment and spread through the fecal-oral pathway because it is stable at low pH and moderate temperature (Martin et al., 2006). Hepatitis A virus (HAV) is still a major cause of acute viral hepatitis globally, despite the development of a highly safe and effective vaccination against the disease in the early 1990s (Tong et al., 1995).²

Epidemiology: The most common cause of HAV from contaminated food and water is a food service employee who neglected to properly wash and sanitize their hands after handling excrement. The amount of hepatovirus A in circulation is lower in developed nations than it is in underdeveloped nations. Since the majority of adults and adolescents in poor nations have already experienced the illness, they are immune. Adults in middle-income nations may be exposed to or at risk for certain diseases.²

Diagnosis of Hepatitis A:

HAV is eliminated in the feces at the conclusion of the incubation period, but the identification of HAV-specific IgM antibodies in the blood allows for a precise diagnosis. The blood only contains IgM antibodies after an acute hepatitis A infection. It appears 1-2 weeks following the original illness and lasts for up to 14 weeks. IgG antibodies in the blood indicate that the sickness has progressed past its acute phase and that the patient is no longer susceptible to infection. After vaccination, IgG antibodies to HAV are also detected in the blood, and the detection of these antibodies serves as the basis for testing for viral immunity.²

Prevention of Hepatitis A: Sanitation, hygiene, and vaccination can all help to prevent hepatitis A. Injectable vaccinations are administered. Two to four weeks after vaccination, the first dosage offers protection for a year; six to twelve months later, the second booster dose offers protection for more than twenty years.²

Hepatitis B:

Hepatitis B is a form of viral hepatitis that damages the liver and is an infectious disease brought on by the Hepatitis B virus (HBV). Both acute and chronic infections may result from it. Exposure to contaminated blood or bodily fluids spreads the infection. Holding hands, sharing cutlery, kissing, hugging, coughing, sneezing, or nursing cannot transfer the hepatitis B virus. After exposure, the infection might be identified 30 to 60 days later.²

Virology and pathophysiology of HBV

HBV, a non-cytopathic DNA virus, belongs to the *Hepadnaviridae* family. It has been proposed that HBV is not cytotoxic and that the adaptive immune response mediates both viral clearance and liver damage. Virus-specific CD8⁺ T cells are crucial in determining the clinical progression of HBV infection.

In individuals with chronic HBV, the failure to control the virus correlates with impaired adaptive immune responses, which include diminished functioning and coordination of virus-specific T and B cells. Understanding the unique mechanisms underlying HBV-specific CD8⁺ T cell dysfunction provides a foundation for creating suitable immunotherapies that restore antiviral efficacy while reducing hepatic damage and facilitating individualized treatment approaches.⁵

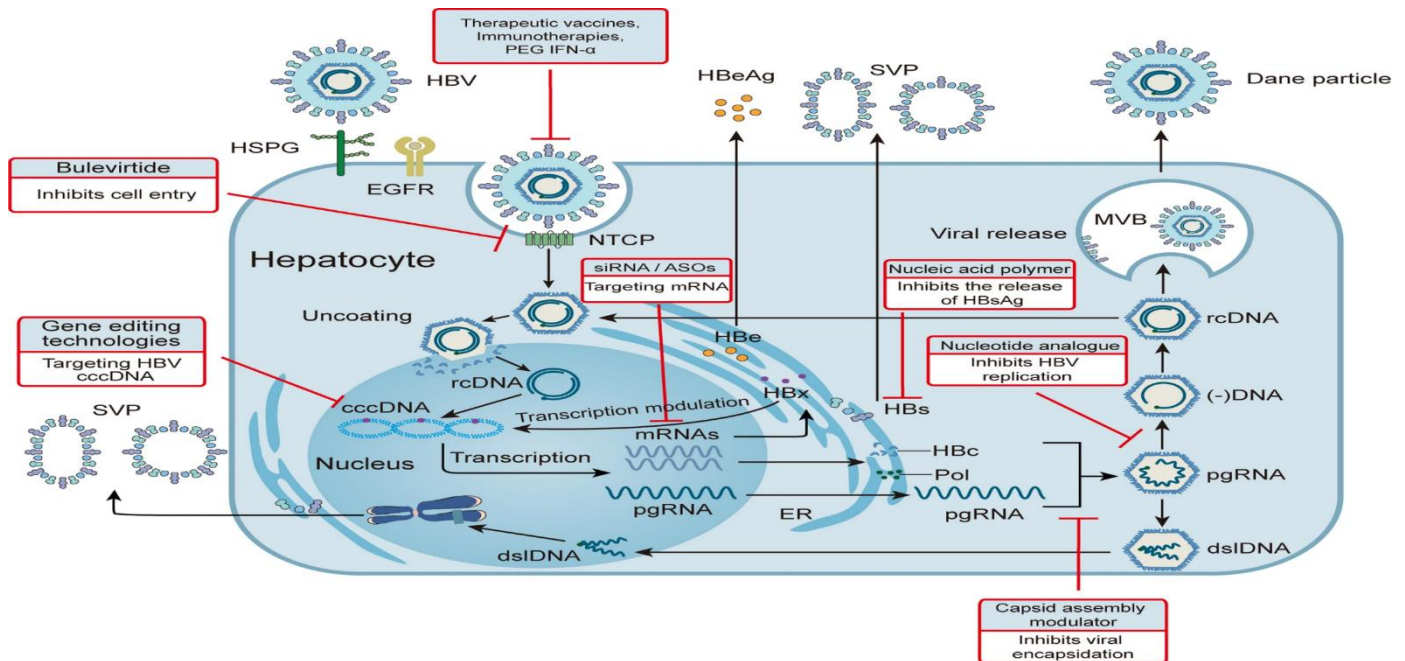


Figure 2

Epidemiology: As of 2019, there were at least 296 million cases of chronic HBV infection worldwide, or 3.8% of the total population. That year, there were an additional 1.5 million instances of acute HBV infection. Globally, regional prevalence's vary from approximately 7.5% in Africa to 0.5% in the America. Where there is a 2–7% chronic infection rate in places with moderate incidence, the disease primarily spreads horizontally, frequently among youngsters, although it can also spread vertically. As of 2018, the estimated proportion of people living with HBV infection in the US was 0.26%.²

Diagnosis of Hepatitis B: Assays, or tests for the identification of hepatitis B virus infection, are performed on serum or blood samples to identify the presence of either host-generated antibodies or viral antigens, which are the virus's manufactured proteins. These assays require complicated interpretation. The most common test for this illness is the hepatitis B surface antigen (HBsAg). It is the initial detectable antigen produced by the virus following infection. But this antigen might not be present in the early stages of an infection, and it might not be noticeable in the latter stages when the host is eliminating it. An internal "core particle" that encloses the viral genome is present in the infectious virion. The 180 or 240 copies of the core protein, also referred to as the hepatitis B core antigen, or HBcAg, make up the icosahedral core particle. IgM antibodies specific to the hepatitis B core antigen (anti-HBc IgM) may be the only serological evidence of disease. Therefore, most hepatitis B diagnostic panels contain HBsAg and total anti-HBc (both IgM and IgG)²

Prevention of Hepatitis B: Since 1991, the United States has routinely recommended vaccinations for babies to prevent hepatitis B. The first dose of the vaccine is typically advised within a day of birth. The hepatitis B vaccine was the first to be capable of preventing cancer, specifically liver cancer.²

In recent years, the world has made impressive progress in a number of areas, including innovations in hepatitis treatment and the expansion of HBV vaccine immunization to prevent new HBV infections.⁸

Hepatitis C :

The hepatitis C virus (HCV) is the infectious agent that causes hepatitis C, an illness that mostly affects the liver. During the initial infection period, patients frequently have minimal or no symptoms. Early signs can include fever, black urine, abdominal pain, and yellow colored skin.²

Virology and pathophysiology of HCV

Hepatitis C is caused by the HCV, an RNA virus belonging to the *Flaviviridae* family. HCV particles, which have a diameter of 50–80 nm, contain the glycoproteins E1 and E2 and core, as well as a single-stranded RNA genome. HCV virions exist as lipovirions (LVPs) due to interactions with low-density lipoproteins (LDL) and very-low-density lipoproteins (VLDL) in the infected host, exhibiting pleomorphism and heterogeneity that varies with growth conditions. E1 and E2 both form non-covalent heterodimers that stabilize the viral particle through disulfide bonds. It has been shown that several cellular proteins are involved in the multistep process of HCV entrance. A series of co-interactions involving the low density lipoprotein receptor (LDLR), the Scavenger Receptor class B type I (SR-BI), the tight junction proteins claudin-1 (CLDN1), occludin (OCLN), and the human Cluster of Differentiation 81 (CD81) have been reported. In addition, the Niemann-Pick C1-like 1 (NPC1L1) cholesterol uptake receptor and the epidermal growth factor receptor are probably implicated in the HCV entry.⁵

After attachment, virus particles are internalized by the cell through the endocytic pathway, and the fusing of membranes relies on endosomal acidification. Positive-strand genomic RNA is released from the viral nucleocapsid and enters the cytoplasm, serving as mRNA for polyprotein production. The viral RNA has highly structured untranslated regions on either side of a single open reading frame (ORF). It is translated as a single polyprotein that is co- and post-translationally processed by both host and viral proteases, producing ten different proteins. The Core, E1, and E2 proteins are synthesized from the N-terminal region, while seven non-structural proteins are produced from the polyprotein's C-terminal region. The 5' untranslated region of HCV includes an internal ribosome entrance site that regulates the translation of the ORF. The internal ribosome entrance site in the HCV 5' UTR initiates translation linked with the endoplasmic reticulum. The NS2-NS3 and NS3-NS4A proteases, as well as cellular proteases, cleave the resulting HCV polyprotein, releasing ten proteins. By interacting with NS3 and attaching to the membrane, NS4A functions as a cofactor. NS5B is the RNA-dependent RNA polymerase, which is the key enzyme responsible for RNA synthesis. The phosphorylated protein NS5A is capable of binding itself to RNA. It appears to have a significant role in regulating viral replication and assembly. The NS3 helicase is essential for the separation of newly formed and template RNA strands, the unwinding of RNA secondary structures, and the displacement of RNA-binding proteins. The hydrophobic protein NS4B causes changes in vesicular membranes that are also crucial in the process of RNA replication. P7 is classified as a viroporin and plays many roles in the formation and release of virions.⁵

In addition to viral proteins, various cellular proteins, including diacylglycerol acyltransferase-1 (DGAT1) and the endosomal sorting complexes required for transport (ESCRT) proteins, have been identified as crucial components in HCV assembly.

Viral particles budding at the endoplasmic reticulum acquire their envelope. In addition, virus morphogenesis is closely associated with the assembly pathway of VLDL. The trans-Golgi Network-to-Endosome pathway has been shown to facilitate the trafficking and release of infectious HCV.⁵

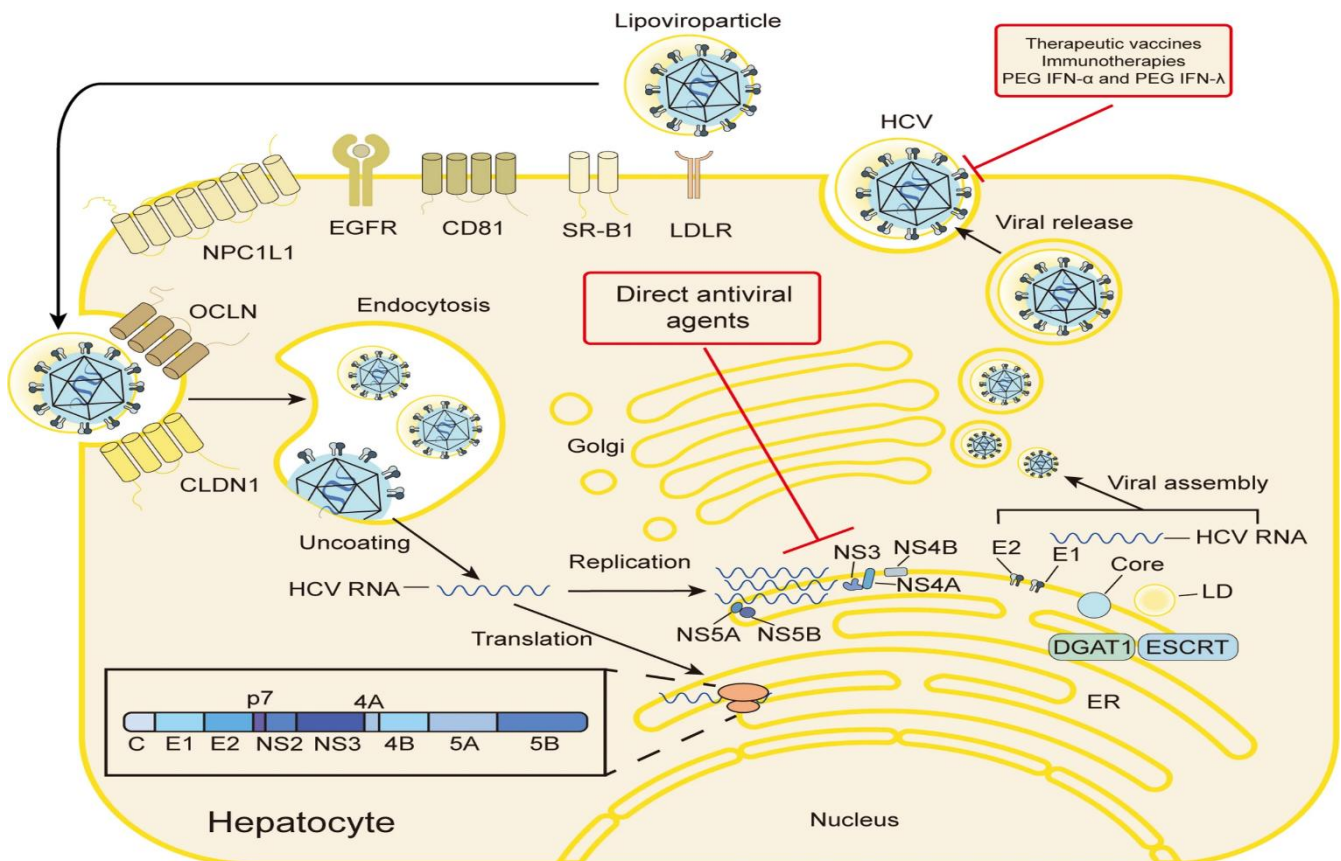


Figure 3

Epidemiology: In a 2021 report, the World Health Organization predicted that, as of 2019, 58 million people worldwide have chronic hepatitis C. An estimated 1.5 million people contract hepatitis C year, and 290,000 people pass away from conditions connected to the virus, primarily cirrhosis and liver cancer.²

Diagnosis of Hepatitis C: Hepatitis C can be diagnosed using a variety of methods, such as the quantitative HCV RNA polymerase chain reaction (PCR), recombinant immunoblot assay, and HCV antibody enzyme immunoassay (ELISA). One to two weeks after infection, HCV RNA can be found by PCR, but it can take much longer for antibodies to develop and become visible.²

Prevention of Hepatitis C:

When possible, medicine should be administered orally rather than by injection in nations with limited access to sterilized syringes (Alter et al., 2007). Recent research also suggests that treating people with active infection, thereby reducing the potential for transmission, may be an effective preventive measure.²

Hepatitis D:

It is a viral hepatitis which is generally caused by the Hepatitis Delta Virus (HDV). HDV can spread through co-infection (concurrent HBV infection) or superinfection (superimposition of HDV on chronic HBV-positive or HBV-carrying individuals). Because of the severity of its effects, HDV infection infected individuals with chronic hepatitis B (superinfection) are regarded as having the most dangerous form of viral hepatitis.²

Virology and pathophysiology of HDV

Hepatitis D is the result of infection with HDV, a small RNA virus. The HDV genome is composed of a circular, single-stranded negative RNA molecule that is about 1700 nucleotides long. It is enclosed within a particle that is coated with HBsAg, but it does not have a nucleocapsid structure. The secondary RNA structure of HDV interacts with approximately 40 to 220 subunits of the hepatitis delta antigen (HDAg), resulting in the formation of a ribonucleoprotein (RNP) complex. The circular RNA of HDV has the ability to fold by pairing around 70% of its nucleotides, resulting in the formation of an unbranched, rod-like structure. It is the smallest identified pathogenic virus that infects humans.⁵

The mechanisms by which HBV and HDV enter the hepatocyte are similar. On the cell surface, HDV particles are concentrated by HSPGs. Moreover, HDV also utilizes the NTCP receptor to gain access to hepatocytes. Upon entering the hepatocyte, the HDV RNP is delivered to the nucleus, where it uses the host's cellular machinery to replicate HDV genomic RNA. Two positive-strand RNAs are produced by transcription of the genomic RNA: a full-length antigenome and a subgenomic mRNA with about 800 nucleotides. HDV sub-genomic mRNAs encode a single ORF, which facilitates the synthesis of L-HDAg and S-HDAg. The transcription of S-HDAg mRNA changes to L-HDAg mRNA due to antigenomic RNA editing caused by adenosine deaminase acting on RNA 1 (ADAR1).⁵

Replication and mRNA synthesis are dependent on host polymerases, as the virus lacks an RNA-dependent RNA polymerase. It has been demonstrated that both RNA polymerase I and II are involved in this process. Subsequently, the mRNA encoding S-HDAg is transcribed and translated. The expression of S-HDAg is necessary for the maintenance of nuclear RNA replication. L-HDAg, an essential part of viral assembly, exerts an inhibitory influence on replication. Post-translational changes are essential to the specific functions of both HDAg proteins. The phosphorylation of S-HDAg facilitates the binding of RNA polymerase II, which in turn enables the replication of viral RNA. L-HDAg undergoes prenylation by the cellular farnesyltransferase. This prenylation occurs at a specific Cys residue located inside the L-HDAg's C-terminal extension. L-HDAg in its farnesylated form is vital for virion assembly and suppresses RNA replication.⁵

HDV is dependent on HBsAg for its release from hepatocytes, even though it is capable of replication and the synthesis of new RNPs without the assistance of HBV. The interaction between HBsAg and farnesylated L-HDAg is essential for HDV assembly. The prenylation inhibitors stop L-HDAg from interacting with HBsAg, which consequently limits HDV virion release. Some studies have shown that HDV utilizes HBs SVPs, while HBV SVPs are released via the Golgi. Therefore, HDV particles may be excreted via the Golgi.⁵

Epidemiology: Globally, HDV is widely distributed. Hepatitis B vaccine campaigns, however, are helping to lower the prevalence in many higher income nations (though rates are still high in particular populations, such as drug injectors and immigrants from HDV-endemic regions). Wherever there is high HBV incidence, low-income areas are plagued by a significant medical problem due to HDV infection.²

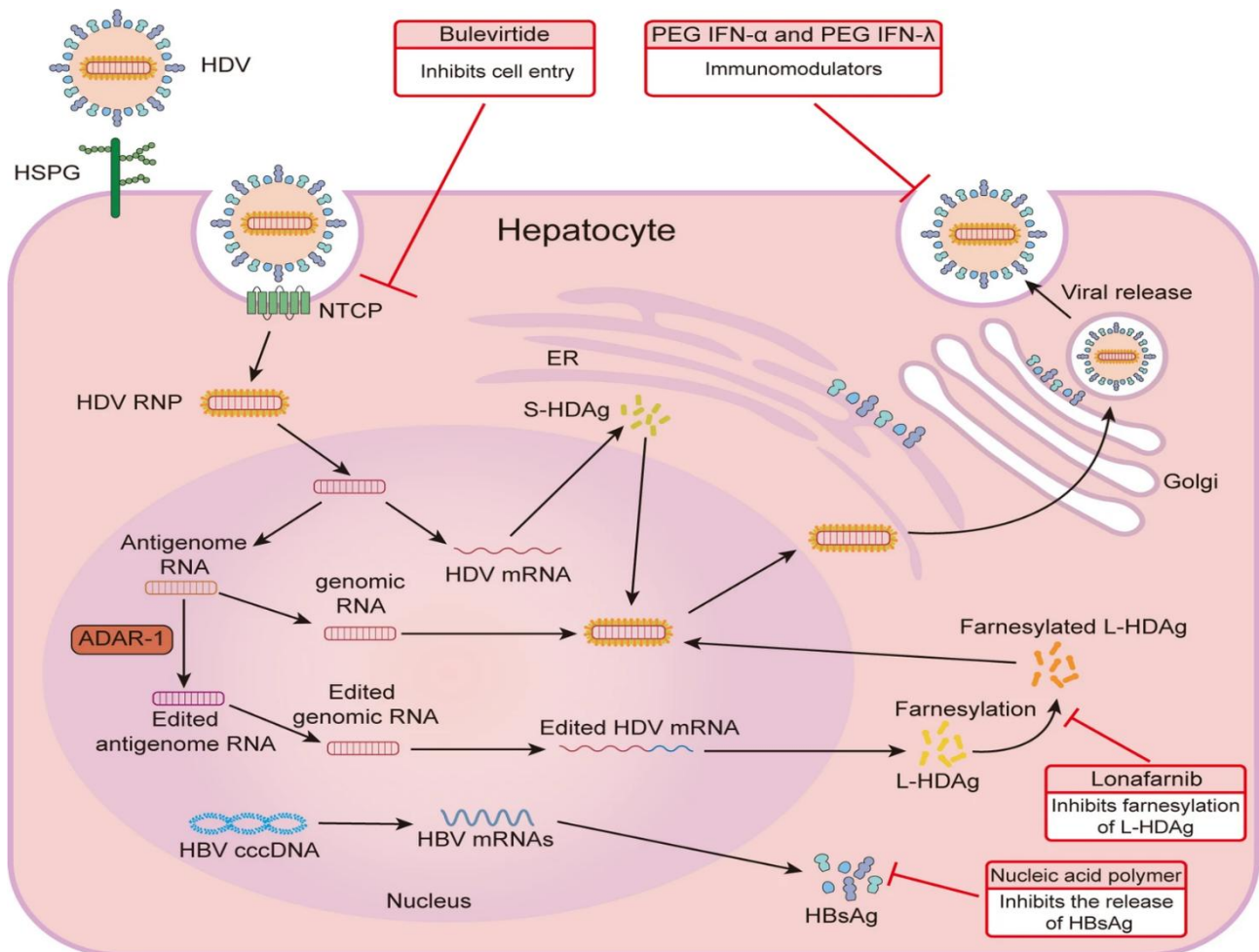


Figure 4

Diagnosis of Hepatitis D: Testing for anti-HDV antibodies, which show previous viral exposure or active infection, is necessary for hepatitis D screening. Hepatitis D RNA levels are used to demonstrate active HDV infection in the presence of anti-HDV antibodies. As HDV requires a hepatitis B virus infection to infect humans, testing for HDV is only recommended in individuals who are positive for the hepatitis B surface antigen (i.e., those who have had or are currently infected with hepatitis B). Non-invasive liver imaging techniques like transient elastography, commonly referred to as the Fibro Scan, or biomarker-based Fibro Test have not been proven to be reliable quantitative indicators of liver fibrosis in people with long-term hepatitis D infection. A liver biopsy is typically required in patients with suspected liver cirrhosis or fibrosis.²

Prevention of Hepatitis D: Since hepatitis D needs an active hepatitis B virus to infect and multiply in humans, Hepatitis E vaccination against hepatitis B protects against hepatitis D viral infection. The World Health Organization advises that everyone have a hepatitis B immunization. To prevent viral infections of hepatitis B and D, the hepatitis B vaccine is usually administered within 24 hours of birth. In order to avoid hepatitis B and D

spreading through bodily fluids, people getting tattoos or body piercings should use sterile equipment. In addition, contaminated needles can spread the disease, so people who inject drugs should seek help to cease using drugs, use sterile needles, and refrain from sharing needles with others.²

Hepatitis E: Hepatitis E is an infection with the hepatitis E virus (HEV) that results in liver inflammation. Similar to hepatitis A, hepatitis E typically has an acute and self-limiting course of illness (the illness is transient and the person recovers), with low death rates in areas with abundant resources; on the other hand, it can be more severe in individuals who are pregnant or have compromised immune systems, with significantly higher death rates. The condition is more common in pregnant women, especially in the third trimester, and is linked to fulminant liver failure, a clinical state that has a 20% fatality rate.

Epidemiology: HEV infection can cause issues for pregnant women, who can develop an acute form of the disease that is deadly in at least 30% of cases. In the underdeveloped world, HEV is a leading source of disease and mortality, and it accounts for a disproportionate share of deaths among expectant mothers. Although there have been outbreaks in the Middle East and Central America, hepatitis E is prevalent in Central Asia.²

Diagnosis of Hepatitis E: Only a laboratory blood test that verifies the existence of HEV RNA or IgM antibodies to HEV may be relied upon for the diagnosis of hepatitis E. The Food and Drug Administration has never approved any serologic tests in the US for the diagnosis of HEV infection. To detect and quantify HEV RNA, the World Health Organization has created an international standard strain. Three weeks following the onset of symptoms, the viremic window for HEV RNA detection in acute infection closes.²

Prevention of Hepatitis E: The most crucial step in preventing hepatitis E is sanitation, which includes better personal hygiene practices, hygienic food preparation, increased standards for public water supplies, and appropriate handling and disposal of human waste. As a result, the disease's prevention techniques are comparable to those of many other illnesses that afflict underdeveloped countries. The hepatitis E virus is killed by cooking meat at 71 °C (159.8 °F) for five minutes; however, other temperatures require varied amounts of time to inactivate the virus.²

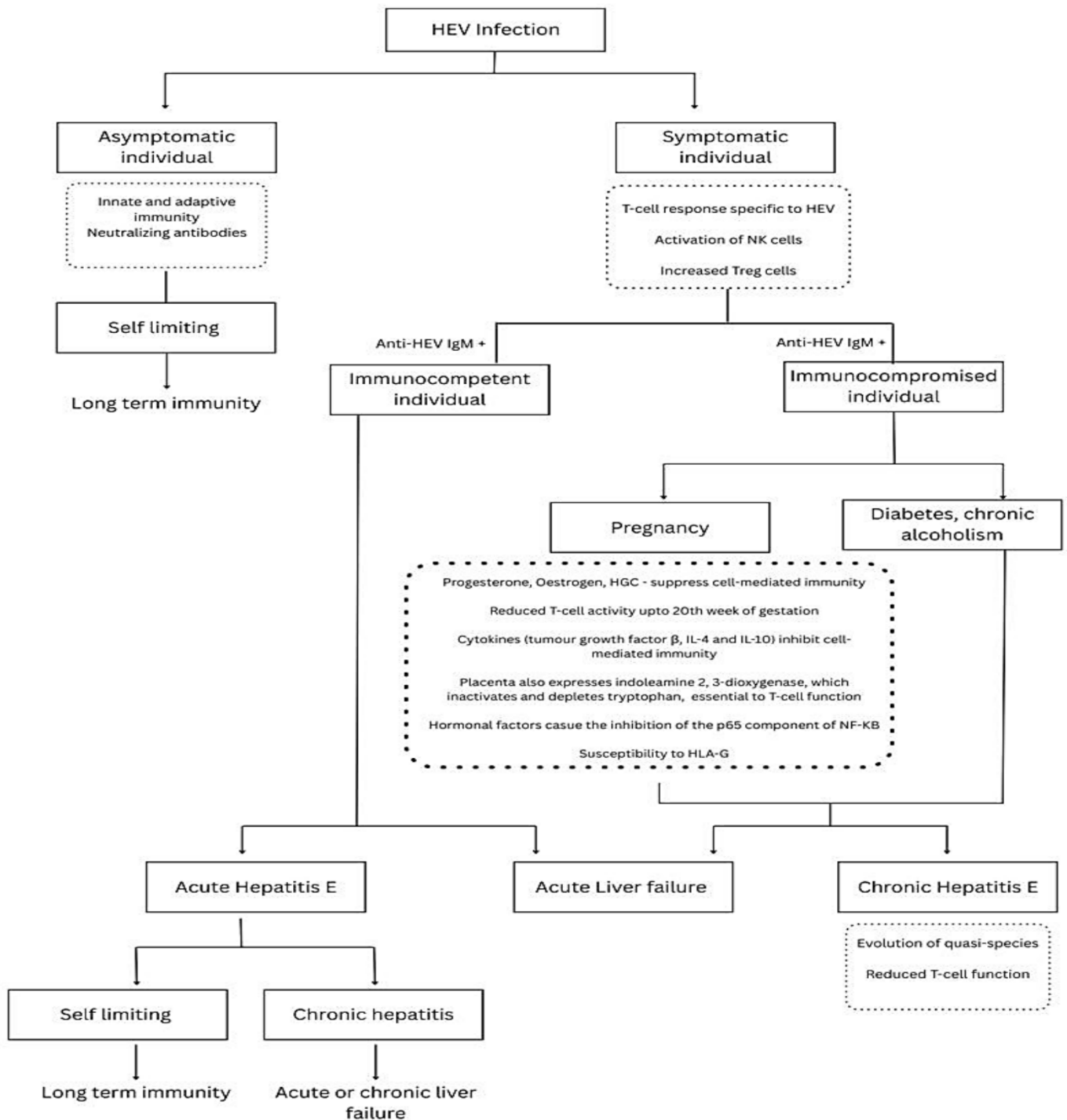


Figure 5: HEV Infection

Viral hepatitis affects nearly 300 million people. Across the globe, most people with viral hepatitis are not diagnosed, nor get the treatment they need. Worldwide, an estimated 1.3 million people die each year from liver disease caused by viral hepatitis.^[1] Safe and effective vaccines, prevention, and treatments can avert liver disease and cancer caused by viral hepatitis. Increasing intervention and prevention strategies can save money and lives.⁴

FUTURE PERSPECTIVES

Artificial intelligence (AI) and machine learning algorithms are being developed for early detection, treatment prediction, and drug discovery based on real-world data and electronic health records. Personalized medicine approaches, integrating patient-specific genetic, proteomic, and clinical data, can improve treatment outcomes and reduce side effects.⁷

Discussion

Hepatitis remains a major global public health challenge despite significant advances in vaccination, antiviral therapy, and public health interventions. Viral hepatitis, caused primarily by hepatitis A, B, C, D, and E viruses, affects hundreds of millions of people worldwide and contributes substantially to liver cirrhosis, hepatocellular carcinoma, and liver-related mortality. The epidemiology of hepatitis varies geographically depending on socioeconomic conditions, sanitation, healthcare infrastructure, vaccination coverage, and behavioral risk factors.

Hepatitis A and E are mainly transmitted through the fecal–oral route and are more common in developing countries with poor sanitation and contaminated food or water. Improved hygiene, access to clean drinking water, and effective vaccination programs have significantly reduced the incidence of hepatitis A in many regions. Hepatitis E remains a concern in low-resource settings and among pregnant women, where infection can lead to severe complications.

Hepatitis B and C account for the majority of chronic hepatitis cases worldwide. These viruses are transmitted through exposure to infected blood and body fluids, unsafe injections, transfusion of unscreened blood, sharing contaminated needles, and mother-to-child transmission. Although universal hepatitis B vaccination has markedly reduced new infections, chronic HBV infection continues to be a major health burden, particularly in Asia and sub-Saharan Africa. Unlike HBV, there is currently no vaccine for HCV, making prevention dependent on infection control practices, blood screening, harm reduction strategies, and early diagnosis.

Prevention of hepatitis requires a comprehensive, multi-level approach. Universal immunization against hepatitis B, vaccination of high-risk populations against hepatitis A, routine screening of blood products, implementation of standard infection control measures, safe injection practices, and public awareness campaigns are fundamental preventive strategies. Screening of pregnant women for HBV and administration of hepatitis B vaccine and immunoglobulin to newborns are highly effective in preventing vertical transmission. For hepatitis C, early detection through screening and treatment with direct-acting antivirals (DAAs) can reduce disease transmission and prevent progression to chronic liver disease.

The World Health Organization (WHO) has set ambitious targets to eliminate viral hepatitis as a public health threat by 2030, aiming for a 90% reduction in new infections and a 65% reduction in hepatitis-related deaths. Achieving these goals requires expanding vaccination coverage, improving access to affordable diagnostics and antiviral therapies, strengthening surveillance systems, and integrating hepatitis services into primary healthcare. Special attention should be given to vulnerable populations such as people who inject drugs, healthcare workers, prisoners, migrants, and individuals with HIV co-infection.

Conclusion

The global burden of hepatitis remains substantial, but many infections are preventable through vaccination, improved sanitation, safe healthcare practices, early diagnosis, and effective antiviral treatment. Strengthening public health policies, increasing awareness, and ensuring equitable access to preventive and therapeutic interventions are essential for achieving the WHO hepatitis elimination targets. Continued research, surveillance, and international collaboration will play a critical role in reducing the incidence, morbidity, and mortality associated with viral hepatitis.

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