

Prevention, Diagnosis, and Treatment Strategies of Hepatitis: A Comprehensive Review

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Abstract

Hepatitis is a leading cause of liver morbidity and mortality across the globe and continues to be a public health concern. Hepatitis includes a set of liver inflammation illnesses caused by infections of viruses, autoimmunity, alcohol use, metabolism disturbances, drugs, and toxins in the environment. Among these, hepatitis viruses A, B, C, D, and E are the main causes of viral hepatitis cases. Chronic hepatitis B and C infections may lead to liver fibrosis, cirrhosis, hepatic insufficiency, and hepatocellular carcinoma, posing a great burden of disease on a global scale. While vaccines against certain strains of the disease, antivirals, and advanced diagnostics have made significant strides in disease prevention and treatment, there are still many people who are undiagnosed and untreated due to a lack of access to health care services, especially in low- and middle-income countries.

Recent advances in serology and molecular diagnostics, such as PCR, qPCR, dPCR, next-generation sequencing technologies, and biomarkers, helped detect and monitor hepatitis infections at an early stage. Alongside with this, preventive measures like vaccination for all, blood transfusion safety, infection prevention practices, maternal screening, public education, and harm reduction programs are critical in minimizing disease spread.

This review presents current knowledge on the epidemiology, classification, pathophysiology, diagnosis, prevention, and treatment of hepatitis. Recent developments in diagnostic methods, antiviral drugs, immunotherapy, and other preventive measures have been outlined with regard to the World Health Organization's objective of eliminating viral hepatitis as a public health issue by 2030. This review can be used as an update for students, researchers, healthcare workers, and policy makers on preventing hepatitis.

Keywords: Hepatitis, Viral hepatitis, Hepatitis B virus, Hepatitis C virus, Diagnosis, Prevention, Antiviral therapy, Vaccination, Liver diseases, Hepatocellular carcinoma

Introduction

Hepatitis is characterized by an inflammation of the liver that results from either an infectious or non-infectious source. Liver is an essential organ that contributes to metabolism, detoxification, nutrient storage, bile secretion, immune responses, and synthesis of plasma proteins and coagulation factors. Therefore, when the liver becomes inflamed, there is potential disruption of many physiological processes, leading to acute and chronic liver diseases. Hepatitis could be either asymptomatic or become serious liver disease with fibrosis, cirrhosis, liver failure, and hepatocellular carcinoma (HCC). The clinical presentation differs greatly based on etiology, host immune response, and the time of the infection.

Worldwide, hepatitis continues to be one of the biggest contributors to death caused by infectious diseases. The World Health Organization estimated that, in 2022, there were about 304 million people living with either hepatitis B or hepatitis C infections. Deaths caused by viral hepatitis are higher than those caused by tuberculosis, malaria, or HIV in certain parts of the world. Even though vaccinations against hepatitis A and hepatitis B and also curative treatment for hepatitis C are available, millions of people have not been remain undiagnosed or untreated, especially in resource-limited settings.

There are five types of hepatotropic viruses that constitute the major causes of viral hepatitis. They are hepatitis A virus (HAV), hepatitis B virus (HBV), hepatitis C virus (HCV), hepatitis D virus (HDV), and hepatitis E virus (HEV). These viruses are highly diverse in terms of genomic organization, modes of transmission, course of infection, epidemiology, and management of disease. While HAV and HEV infections are mainly associated with the development of acute non-persistent hepatitis and are transmitted via fecal-oral route, HBV, HCV, and HDV infections are mostly transmitted sexually, parenterally, or vertically and can cause either acute or chronic hepatitis. Chronic hepatitis B and C infections are considered the main reasons for the development of liver cirrhosis and hepatocellular carcinoma throughout the world.

There are approximately million cases of chronic HBV infection in the world, although there are very efficacious vaccines against HBV and it is still considered one of the leading causes of liver-related deaths.

In comparison, HCV is a positive-stranded RNA virus that belongs to the family Flaviviridae. In contrast to HBV, HCV does not insert itself into the genome of the host but is very genetically diverse due to high mutation rates.

Finally, HDV is the smallest human RNA virus known to science that needs hepatitis B virus for its reproduction as it uses hepatitis B surface antigen for viral assembly. HDV co-infection/superinfection leads to the faster development of liver diseases and increases the chance of developing cirrhosis and hepatocellular carcinoma. New therapies, such as entry inhibitors, for example, bulevirtide, were recently introduced for HDV infection treatment.

Early diagnosis is fundamental for reducing hepatitis-related complications. Conventional serological assays detecting viral antigens and antibodies continue to play a central role in screening; however, molecular techniques including quantitative polymerase chain reaction (qPCR), transcription-mediated amplification, digital PCR, loop-mediated isothermal amplification (LAMP), and next-generation sequencing have significantly enhanced diagnostic sensitivity and specificity. Biomarkers such as hepatitis B core-related antigen (HBcrAg), hepatitis B RNA, quantitative HBsAg, liver stiffness measurements using transient elastography, and circulating micro RNAs are increasingly being investigated for disease monitoring and therapeutic decision-making.

Prevention remains the cornerstone of hepatitis control. Universal childhood vaccination against HBV has dramatically reduced new infections worldwide. Additional preventive measures include screening of blood donors, implementation of standard infection control precautions, safe injection practices, harm reduction strategies for people who inject drugs, maternal screening during pregnancy, antiviral prophylaxis to prevent mother-to-child transmission, health education, and public awareness initiatives

The therapeutic approach towards hepatitis has come through much progress in the last few decades. The currently available treatment options for HBV infection include long-term treatment with effective nucleotide analogues like entecavir and tenofovir that are able to suppress the viral replication, and HCV infection can nowadays be successfully treated and cured in the majority of patients with the use of pangenotypic direct-acting antivirals. Novel approaches in research include such therapeutic methods as vaccine therapies, RNA interference, genome editing with CRISPR technology, immune checkpoint therapy, capsid assembly inhibitors, and gene editing methods to develop functional or full cures for chronic HBV infection.

2. Classification of Hepatitis

Hepatitis refers to a collection of inflammatory conditions affecting the liver, which may be caused either by infectious or non-infectious mechanisms. Classification of hepatitis is important in order to understand pathophysiology, select appropriate tests, develop treatment modalities, and predict prognosis. There are several ways to classify hepatitis based on etiology, duration of illness, and pathology.

Table 1:

Type	Cause	Examples
Viral Hepatitis	Infection by hepatotropic viruses	HAV, HBV, HCV, HDV, HEV
Alcoholic Hepatitis	Excessive alcohol consumption	Alcohol-induced liver inflammation
Drug-Induced Hepatitis	Medications, herbal products, toxins	Paracetamol overdose, Isoniazid, Methotrexate, Amoxicillin-clavulanate
Autoimmune Hepatitis	Autoimmune destruction of liver cells	Type 1 and Type 2 autoimmune hepatitis
Metabolic Hepatitis	Metabolic disorders	Wilson's disease, Hemochromatosis, Alpha-1 antitrypsin deficiency
Non-alcoholic Steatohepatitis (NASH)	Fat accumulation and inflammation associated with metabolic syndrome	NASH (now often included under MASLD/MASH terminology)
Ischemic Hepatitis	Reduced blood supply to the liver	Shock liver
Toxic Hepatitis	Chemical toxins and poisonous substances	Industrial chemicals, poisonous mushrooms

2.1 Classification Based on Etiology

2.1.1 Viral Hepatitis

Viral hepatitis is the most common cause of hepatitis globally and is caused by five hepatotropic viruses: hepatitis A virus (HAV), hepatitis B virus (HBV), hepatitis C virus (HCV), hepatitis D virus (HDV), and hepatitis E virus (HEV).

Hepatitis A Virus (HAV)

HAV is a non-enveloped, positive-sense single-stranded RNA virus belonging to the *Picornaviridae* family. It is primarily transmitted through the fecal–oral route via contaminated food, water, or close personal contact. Clinical symptoms include fever, fatigue, anorexia, nausea, abdominal pain, and jaundice.

Hepatitis B Virus (HBV)

HBV is a partially double-stranded DNA virus belonging to the *Hepadnaviridae* family. Transmission occurs through exposure to infected blood, sexual contact, contaminated needles, and vertical transmission from mother to infant during childbirth.

Hepatitis C virus (HCV)

HCV is an enveloped, positive-sense, single-stranded RNA virus belonging to *Flaviviridae* family. This virus is primarily transmitted by blood exposure especially in individuals who inject drugs, recipients of untested blood products, and unsafe medical practices. Most of the infections have no symptoms while 55-85% of the infected develop chronic hepatitis.

Hepatitis D virus (HDV)

This is a defective RNA virus that depends on HBV for multiplication since HDV cannot replicate independently due to the requirement of the surface antigen (HBsAg) to package infectious particles. The infection is possible both during coinfection with HBV or superinfection when an individual with chronic HBV gets infected with HDV.

Hepatitis E virus (HEV)

This is a positive-sense, single-stranded RNA virus that is transmitted mainly through drinking contaminated water. While HEV infection is usually self-limiting, there is the possibility of severe acute hepatitis in pregnant women, immunosuppressed patients, and chronic liver diseases

2.1.2 Autoimmune hepatitis

Autoimmune hepatitis (AIH) is an autoimmune liver disease that involves destruction of hepatocytes by the immune system. The condition occurs because of lack of immunity tolerance to hepatic antigens. Autoimmune hepatitis usually affects females, and is associated with increased serum IgG and specific autoantibodies.

2.1.3 Alcoholic hepatitis

Alcoholic hepatitis occurs as a result of chronic abuse of alcohol and involves oxidative stress, release of inflammatory cytokines, cell death, and fibrosis. Clinical presentation of the disorder varies from mild liver enzymes elevation to more severe alcoholic hepatitis that is accompanied with jaundice, ascites, hepatic encephalopathy, and liver failure.

2.1.4 Drug-induced hepatitis

Drug-induced liver injury (DILI) is considered one of the major causes of acute liver failure in the world. Many drugs, herbal medicines, and environmental substances may be responsible for hepatocellular, cholestatic or mixed liver injury patterns

2.1.5 Metabolic and Genetic Hepatitis

There are several genetic metabolic diseases that lead to hepatitis which include Wilson disease, hereditary hemochromatosis, alpha-1 antitrypsin deficiency, and non-alcoholic fatty liver disease (NAFLD), which is now known as metabolic dysfunction associated steatohepatitis (MASLD). These diseases are characterized by excessive deposits of either copper, iron or fats within hepatocytes leading to fibrosis and cirrhosis.

2.2 Classification by Duration of Infection

Acute Hepatitis

Acute hepatitis involves the presence of inflammation of the liver for a period of less than six months and usually occurs due to HAV, HEV, HBV infections, medications, and alcohol induced injury. The symptoms include fatigue, nausea, vomiting, anorexia, abdominal pain, jaundice, dark urine, and increased serum aminotransferases.

Chronic Hepatitis

Chronic hepatitis refers to persistent liver inflammation for more than six months. The condition is mainly linked to chronic infection by HBV, HCV, HDV, autoimmune hepatitis and metabolic liver conditions. Persistent inflammation causes progressive fibrosis, cirrhosis, portal hypertension and hepatocellular carcinoma requiring constant clinical management and treatment.

3. Pathogenesis of Hepatitis

3.1 General Information

Pathogenesis of hepatitis is a process which involves multiple factors including infecting agent, immune response, genetic susceptibility and environmental influence. Even though hepatitis can be caused by viral infection, autoimmune conditions, alcohol, medications or metabolic conditions, damage to the liver occurs due to immune response of the host and not as a result of cell death due to pathogenic factors. Factors influencing the course of the disease include viral persistence, inflammation, oxidative stress and balance between fibrosis and repair.

Continuous inflammation causes liver damage, activation of hepatic stellate cells (HSCs), matrix deposition, fibrosis, cirrhosis and eventually hepatocellular carcinoma (HCC).

3.2 Viral Infection and Replication

Each of the viruses that cause hepatitis has different modes of infection for hepatocytes.

Hepatitis A Virus (HAV)

The HAV infects hepatocytes via receptor-mediated endocytosis. Once inside the cell, the viral RNA is released into the cytoplasm, functioning as the messenger RNA for protein synthesis. Viral replication takes place in the cytoplasm to form new viral particles, which get released into the bile and expelled in the feces.

Hepatitis B Virus (HBV)

The HBV infects hepatocytes via interaction with the NTCP receptor located on the surface of the hepatocyte. Following infection, the viral nucleocapsid releases relaxed circular DNA, which gets converted into covalently closed circular DNA (cccDNA) in the nucleus. The cccDNA serves as a template for transcription of the virus.

Hepatitis C virus (HCV)

This virus enters hepatocytes by binding to several cellular receptors, namely CD81, scavenger receptor class B type I (SR-BI), claudin-1, and occludin. Then viral RNA is translated to a polyprotein which is further cleaved into structural and non-structural proteins. Replication takes place in replication complexes in the cytoplasm.

Hepatitis D virus (HDV)

This virus depends fully on HBV because it needs hepatitis B surface antigen (HBsAg) for its assembly and spread. Therefore, HDV infection can occur only after HBV infection. This virus promotes liver inflammation and dramatically increases the risk of cirrhosis and liver failure.

Hepatitis E virus (HEV)

This virus infects hepatocytes after consuming infected food and water. Replication takes place in the cytoplasm leading to acute and self-limited hepatitis. However, in immunocompromised patients, chronic HEV infection may occur.

3.3 Mechanisms of Liver Damage

Unlike what was believed previously, liver injury mainly results from immune reactions and not because of viral replication itself.

Inflammatory substances produced by activated immune cells include:

- Tumor necrosis factor alpha (TNF- α)
- Interleukin-1 beta (IL-1 β)
- Interleukin-6 (IL-6)
- Interferon-gamma (IFN- γ)
- Reactive oxygen species (ROS)

Inflammatory factors lead to cell death, necrosis, mitochondrial impairment, oxidation, and lipid peroxidation resulting in gradual liver cell injury.

Ultimately, repeated inflammation-repair cycles trigger hepatic stellate cells which initiate fibrosis.

3.4 Mechanisms of Hepatocellular Carcinoma Development

Chronic hepatitis B virus infection and hepatitis C virus infection are among the top causes of hepatocellular carcinoma.

The following factors are associated with hepatocellular carcinoma development:

- Continuous inflammation
- Oxidative damage to DNA
- Chronic cell regeneration
- Genomic instability

Oncogenic characteristics of HBV can be attributed to its ability to integrate into the host DNA, interfering with tumor suppressor genes and leading to uncontrolled cell proliferation.

Although HCV does not integrate into host DNA, inflammation and oxidative stress elevate the risks of developing cancer.

3.5 Pathogenesis of Non-Viral Hepatitis

- Autoimmune Hepatitis

Autoimmune hepatitis arises when tolerance of the immune system towards hepatocytes becomes lost, leading to an autoimmune attack on normal liver cells and causing inflammation and fibrosis. Genetic predisposition with HLA-DR3 and HLA-DR4 alleles plays an important role in the development of the disease.

- Alcoholic Hepatitis

Metabolism of alcohol leads to the formation of acetaldehyde and reactive oxygen species, thus causing oxidative stress, mitochondrial dysfunction, lipoperoxidation and release of inflammatory cytokines.

- Drug-Induced Hepatitis

Some of the drugs and their metabolites have a direct effect on the liver or cause immunological reaction. The drug-induced liver damage may be hepatocellular, cholestatic, or both depending on the mode of action.

4. Diagnosis of Hepatitis

4.1 Introduction

The proper diagnosis of hepatitis is critical in order to decrease the risk of transmission, initiate treatment, prevent complications like cirrhosis and HCC, and improve the patient's outcome. The diagnosis of hepatitis requires a thorough evaluation, which includes the clinical diagnosis, liver function tests, serology, molecular diagnosis, and imaging modalities.

A holistic approach to the diagnosis allows for identifying the pathogen, liver damage, viral load, degree of fibrosis, and the efficacy of treatment.

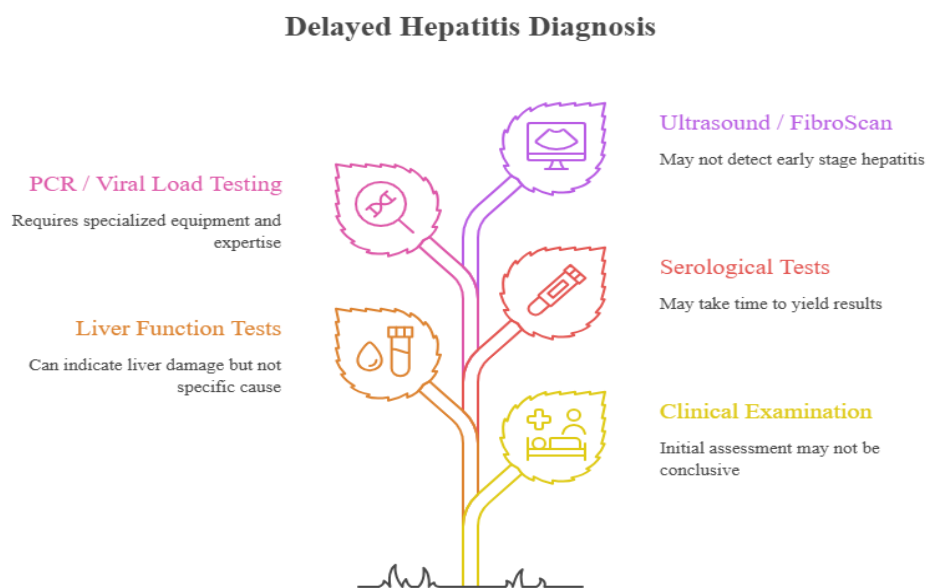


Figure 1: Delayed Hepatitis Diagnosis

4.2 Clinical Assessment

The assessment starts with a comprehensive patient's history and physical exam. The history related to the recent travel, alcohol intake, medications, occupation, blood transfusion, IV drug usage, tattooing, sexual activity and any liver disease history in the patient's family gives clues to the etiology of hepatitis.

The patients with acute hepatitis show constitutional symptoms like fever, tiredness, poor appetite, nausea and vomiting prior to becoming jaundiced. However, in patients with chronic hepatitis, there might be no symptoms until the development of cirrhosis or liver failure.

The identification of risk factors and symptoms is helpful in further investigation process

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4.3 Laboratory Investigation

Laboratory investigation is the mainstay in the diagnosis of hepatitis. This test evaluates hepatocellular damage, liver synthetic function, cholestasis, and viral infection. It can also help differentiate between acute and chronic hepatitis.

Liver function testing involves the following lab tests:

- Liver function test (LFT)
- Complete blood count (CBC)
- Coagulation studies
- Serum albumin
- Renal function test

Among them, LFT is most common investigation for hepatitis

4.4 Liver Function Tests (LFTs)

Liver function tests yield significant insights into the degree of hepatocellular damage and hepatic function. While elevated levels of liver enzymes do not help to determine the cause of hepatitis, they provide useful markers of inflammation of the liver.

Alanine Aminotransferase (ALT)

ALT is found mainly in hepatocytes, and it is believed to be the best biochemical marker of hepatocellular damage. Increased levels of ALT are an indicator of hepatocyte damage and are usually seen during viral hepatitis.

Aspartate Aminotransferase (AST)

AST is found in the liver, muscle, heart, kidney, and red blood cells. An increased level of AST is associated with hepatocellular damage; however, it is less liver-specific compared to ALT.

The AST/ALT ratio helps in diagnosing various liver diseases.

- AST/ALT <1 is typical of viral hepatitis.
- AST/ALT >2 is indicative of alcoholic liver disease

Alkaline Phosphatase (ALP)

Elevated in patients with cholestatic liver diseases owing to poor bile movement. Slightly elevated in case of viral hepatitis with biliary involvement.

Gamma-Glutamyl Transferase (GGT)

GGT is a very useful indicator of hepatobiliary disease and alcohol induced liver damage. High GGT together with ALP indicate hepatobiliary disease.

Serum Bilirubin

It gives an indication of the excretory function of the liver.

High serum bilirubin is seen when there is a problem in conjugation or excretion of bilirubin and leads to jaundice.

Table 2. Major Liver Function Tests in Hepatitis

Test	Clinical Significance
ALT	Most specific marker of hepatocellular injury
AST	Indicates hepatocyte injury; AST/ALT ratio assists diagnosis
ALP	Marker of cholestasis
GGT	Indicates hepatobiliary disease and alcohol use
Bilirubin	Reflects hepatic excretory function
Albumin	Assesses hepatic synthetic capacity
PT/INR	Evaluates coagulation and liver function

5 Serological Diagnosis

Serological tests serve as the foundation of hepatitis diagnosis since they detect viral antigens, antibodies, and immune reactions. These tests are cheap, easily accessible, and highly sensitive.

- Diagnosis of Hepatitis A

The diagnosis of HAV infection is done using anti-HAV IgM antibody detection. This is because anti-HAV IgM antibodies become detectable after a few days and last for about three to six months.

Anti-HAV IgG antibodies indicate past infection or vaccine success and offer lifelong immunity.

- **Diagnosis of Hepatitis B**

Diagnosis of HBV depends on several serological indicators.

Hepatitis B Surface Antigen (HBsAg)

This is the main serological indicator used to diagnose an active HBV infection.

If the indicator persists for more than six months, then there is chronic hepatitis B.

Hepatitis B Surface Antibody (Anti-HBs)

This appears after recovery from the infection or vaccine success.

- **Diagnosis of Hepatitis C**

The test for initial diagnosis is anti-HCV antibody ELISA.

Since the antibodies stay positive even after the virus is cleared, a positive result for ELISA should be confirmed through HCV RNA PCR.

Detection of HCV RNA indicates active infection.

- **Diagnosis of Hepatitis D**

HDV infection is diagnosed through:

- Anti-HDV antibodies
- HDV RNA through PCR test

It is advisable to do testing in those who are positive for HBsAg.

5.6 Molecular Diagnosis

Molecular techniques for diagnosis have brought about an evolution in hepatitis diagnosis through direct detection of viral nucleic acids with high sensitivity and specificity. Such methods are extremely necessary for confirmation of active infection, replication of virus, antiviral therapy monitoring, and antiviral resistance.

Polymerase Chain Reaction (PCR)

The conventional PCR assay is used to diagnose the presence of viral DNA (HBV) or RNA (HCV, HDV, HEV). PCR is extremely sensitive and enables the identification of virus infection prior to the development of serologic markers, thus being useful especially at the beginning of infection.

5.7 Imaging Techniques

Imaging studies play an important role in assessing liver structure, complications, and staging of chronic liver diseases. While imaging studies do not provide any direct evidence about the viral cause of hepatitis, they allow the evaluation of fibrosis, cirrhosis, portal hypertension, and HCC.

Ultrasonography (USG)

USG is the primary method of imaging since it is non-invasive, inexpensive, and widely available. USG allows one to find out hepatomegaly, fatty liver, cirrhosis, splenomegaly, ascites, and focal lesions of the liver.

Computed Tomography (CT)

CT allows obtaining detailed cross-sectional images of the liver and is very useful for identifying liver tumors and other pathologies.

Magnetic Resonance Imaging (MRI)

MRI provides excellent soft tissue contrast and high sensitivity for detection of liver fibrosis, iron excess, and small liver masses. Magnetic resonance elastography (MRE) is an advanced imaging modality for the assessment of liver stiffness without biopsy.

5.8 Transient Elastography (FibroScan)

Transient elastography (FibroScan) is a non-invasive technique, which uses ultrasound-based elastography to measure liver stiffness. High liver stiffness indicates the extent of fibrosis. FibroScan is widely applied to stage liver fibrosis and monitor the progress of disease in chronic hepatitis B and C.

5.9 Liver Biopsy

Liver biopsy is a reference method for evaluation of the presence of inflammation and fibrosis in the liver. This test gives complete histopathological information about the degree of necrosis, fibrosis, steatosis, and cirrhosis in the liver. Nevertheless, being an invasive procedure, it can be associated with complications, such as pain and bleeding.

5.10 Artificial Intelligence in Hepatitis Diagnosis

Artificial intelligence (AI) and machine learning are new technologies in hepatology. Using AI algorithms, laboratory results, imaging, and electronic health records can help detect liver fibrosis, disease development, and risk of hepatocellular carcinoma early. Also, AI-assisted image analysis increases the accuracy of interpreting ultrasound, CT, and MRI results.

6. Prevention Strategies of Hepatitis

Prevention is the best strategy for managing the worldwide problem of hepatitis. Public health, immunization, screening, and education activities have managed to reduce the number of cases of viral hepatitis.

6.1 Vaccination

Vaccination is the key preventive measure against hepatitis. There are vaccines against hepatitis A (HAV) and hepatitis B (HBV). According to the WHO, universal immunization against HBV should be implemented and administered in the first 24 hours of life to prevent transmission from the mother. Immunization against HBV also helps protect against hepatitis D virus (HDV) because HDV needs HBV to replicate

6.2 Blood Transfusion and Injection

Screening for hepatitis B virus (HBV), hepatitis C virus (HCV), human immunodeficiency virus (HIV), and other blood-borne pathogens is now common and prevents the spread via transfusions. Sterile needle and syringe use in healthcare institutions is crucial.

6.3 Good Hygiene and Food Safety

Sanitation, availability of safe drinking water, proper hand hygiene, and appropriate handling of food products prevent the transmission of HAV and HEV. Public health education on hygiene is important in preventing the spread of HAV and HEV.

6.4 Screening

The screening of high-risk groups, including pregnant mothers, healthcare personnel, blood donors, drug addicts, and those undergoing hemodialysis helps in early diagnosis and treatment of hepatitis.

6.5 Public Health Education

Community education on vaccination, safe sex, not sharing syringes, and signs of hepatitis is important in preventing the disease.

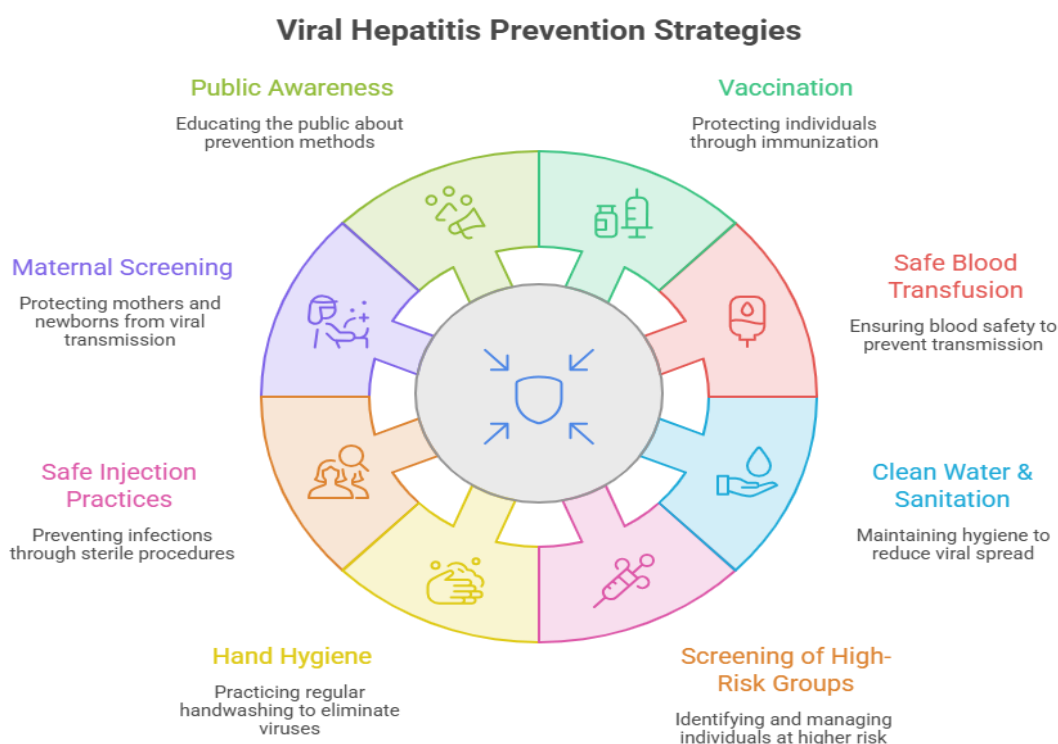


Figure 2: Viral Hepatitis Prevention Strategies

7. Treatment Modalities of Hepatitis

Treatment modalities depend on the form of hepatitis, severity of disease and presence or absence of complications.

7.1 Hepatitis A

HAV infection in acute stages is a self-limiting condition requiring only supportive therapy that includes hydration, nutrition, and rest. Antiviral treatment is unnecessary.

7.2 Hepatitis B

Suppression of virus replication for long-term period is the main target in treating chronic HBV infection and prevention of cirrhosis and hepatocellular carcinoma.

First-line drugs are:

- Tenofovir disoproxil fumarate (TDF)
- Tenofovir alafenamide (TAF)
- Entecavir

7.3 Hepatitis C

Incorporation of DAAs has led to revolution in HCV treatment. Nowadays pangenotypic treatment options have SVR rate of more than 95% and allow to cure almost all the patients within 8-12 weeks.

DAA combinations are:

- Sofosbuvir/Velpatasvir
- Glecaprevir/Pibrentasvir
- Sofosbuvir/Ledipasvir

7.4 Hepatitis D

Hepatitis D management includes the treatment of the primary hepatitis B virus infection. The recently approved drug bulevirtide, which acts as an entry inhibitor, seems to be efficient against the chronic HDV infection.

7.5 Hepatitis E

The acute HEV infection is usually treated by supportive care. In some immunosuppressed patients with a chronic HEV infection, ribavirin can be used.

7.6 Management of Advanced Liver Disease

People suffering from cirrhosis should undergo regular screening for hepatocellular carcinoma by means of ultrasound and AFP tests every six months. The definitive therapy for end-stage liver disease and acute liver failure is liver transplantation.

8. Future Perspectives

The recent developments in molecular biology and biotechnology have led to the increased rate of novel therapies for hepatitis. Some of the emerging technologies are CRISPR-Cas9 gene editing, RNA interference (RNAi), vaccine, and immune-checkpoint inhibition, which are currently being explored in order to provide a functional cure for chronic HBV infection.

AI and machine learning are anticipated to help with the early diagnosis of the disease, as well as its prognosis and individual treatment plans. The discovery of new biomarkers such as HBV core-related antigen (HBcrAg), circulating microRNAs, and cell-free DNA will add to monitoring and prevention of the disease.

The WHO target of eliminating viral hepatitis as a major public health concern by 2030 is only possible with enhanced vaccination rates, screening efforts, affordable antiviral treatment, and improved healthcare infrastructure.

Emerging Approaches in Hepatitis Diagnosis and Treatment

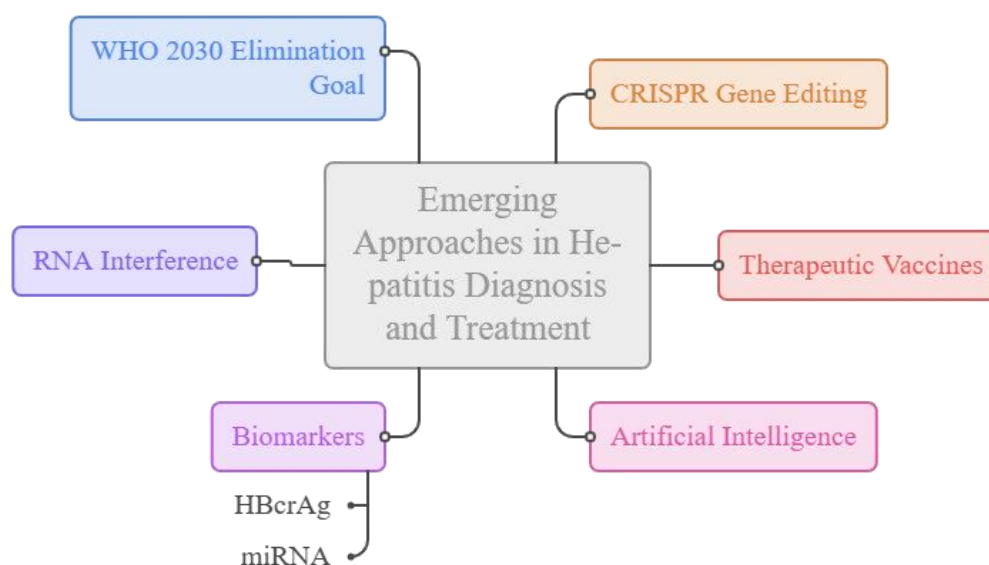


Image 3. Emerging Approaches in Hepatitis Diagnosis and Treatment

Conclusion

Despite advancements in the prevention, detection, and treatment of hepatitis, it is still a challenging problem in global health. Hepatitis B and C viruses cause most cases of liver cirrhosis and hepatocellular carcinoma worldwide. Early detection using serological and molecular methods, as well as efficient treatment and regular follow-up of patients, have improved the prognosis considerably.

Vaccination, safe blood transfusion, infection control measures, public awareness, and early testing of at-risk populations are some of the prevention methods that should be implemented to minimize the spread of hepatitis. New technologies in the field of molecular diagnostics, non-invasive fibrosis assessment, artificial intelligence, and antiviral medications provide great hope for future hepatitis treatment. Further studies and public health interventions are needed to reach the WHO goal of eliminating hepatitis by 2030.

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