Hepatitis A, B, C, D, and E and Complications

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Purpose

The purpose of this course is to provide an overview of the course of infection and treatment options for hepatitis A, B, C, D, and E and to outline the primary complications.

Goals

Upon completion of this course, the healthcare provider should be able to:

- List at least 6 types of hepatitis.
- Describe two different mechanisms of transmission.
- List at least 8 symptoms common to all main types of hepatitis.
- Describe at least 5 different liver function tests.
- Discuss primary features of hepatitis A, B, C, D, and E, including transmission and symptoms.
- Discuss treatment options for each type of hepatitis.
- Discuss how genotype affects treatment for hepatitis C.
- Discuss available hepatitis vaccinations.
- Discuss risk factors and prevention for each type of hepatitis.
- Discuss nursing precautions to prevent transmission.
- Discuss dietary management.
- Discuss fulminant hepatic failure.
- Describe at least 5 symptoms of cirrhosis.
- Describe at least 3 symptoms that indicate that chronic hepatitis may have progressed to hepatocellular cancer.

Introduction

Hepatitis, inflammation of the liver, can be caused by viruses, bacteria, other microorganisms, toxic chemicals, alcohol, and other drugs, but viral hepatitis is the most common cause of hepatitis. Viral hepatitis is a systemic infection in which virus infects the liver cells, causing biochemical and cellular changes and interfering with liver function.

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Hepatic liver

There are a number of different hepatitis viruses (not all identified), but the primary viruses are types A (HAV), B (HBV), C (HCV), D (HDV), and E (HEV). HAV and HEV are spread by the fecal-oral route while types HBV, HCV, and HDV are bloodborne and have similar characteristics. Researchers estimate that only 10 to 40% of hepatitis cases are correctly diagnosed as many people have subclinical disease or non-specific symptoms attributed to other disorders, such as the flu.

In the United States, HAV, HBV, and HCV account for about 500,000 infections annually, and about 5 million people have chronic infections. Hepatitis is classified as foodborne (HAV and HEV) or bloodborne. Food-borne hepatitis viruses do not cause chronic liver disease. HBV and HCV are of primary concern because they can lead to permanent damage of the liver and death. Immunizations are only available for HAV and HBV.

Another hepatitis virus, G (HGV) has been identified, but it rarely causes hepatitis. HGV is bloodborne and produces a chronic viremia that lasts about 10 years. Researchers believe that HGV may prolong survival in those coinfected with HIV and may reduce fibrosis in those with HCV-HIV coinfection.

About 7.5% of blood donors have a DNA virus designated TT (TTV) that is readily transmitted through blood transfusions, but as yet no association between this virus and liver disease has been detected. Another virus, known as SEN-V has been found in about 2% of blood donors and is also transmitted through transfusion. Researchers

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believe that SEN-V may account for some cases of non-ABCDE hepatitis.

While symptoms vary depending upon viral load, immune system, and type of hepatitis, some symptoms are commonly seen with all types of symptomatic hepatitis. About 50% develop mild to moderate hepatomegaly and 15% splenomegaly.

Symptoms common to hepatitis A, B, C, D, and E
• Jaundice and light or clay-colored stools, resulting from impaired excretion of bilirubin excretion. • Dark yellow-green urine, resulting from urobilinogen being excreted in the urine. • Abdominal pain, caused by stretching of Glisson's capsule surrounding the liver, especially in the right upper quadrant. • Anorexia, nausea, vomiting, diarrhea, and constipation, caused by alterations in digestion • Fatigue and weakness, because of reduced energy metabolism by the liver • Fever, flu-like symptoms, muscle or joint pains, caused by the general body response to inflammation. • Pruritis, caused by accumulation of bile salts in the skin. • Increased bruising and bleeding tendencies, because of decreased prothrombin synthesis and reduced Vitamin K absorption.

Liver Function Tests: Normal values	
Bilirubin	Indicates the ability of the liver to conjugate and excrete bilirubin: direct 0.0-0.3 mg/dL, total 0.0-0.9 mg/dL, and urine 0.
Total protein	Indicates whether the liver is producing protein in normal amounts: 7.0-7.5 g/dL: • Albumin: 4.0-5.5 g/dL. • Globulin: 1.7=3.3 g/dL. • Serum protein electrophoresis is done to determine the ratio of proteins. • Albumin/globulin (A/G) ratio: 1.5:1 to 2.5:1. (Albumin should be greater than globulin.)
Prothrombin time (PT)	• 100% or clot detection in 10 to 13 seconds. PT increases with liver disease. • **International normalized ratio (INR)** (PT result/normal average): <2 for those not

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	receiving anticoagulation and 2.0 to 3.0 for those receiving anticoagulation. Critical value: >3 in patients receiving anticoagulation therapy.
Alkaline phosphatase	17-142 adults. (Normal values vary with method.) Indicates biliary tract obstruction if no bone disease.
AST (SGOT)	10-40 units. (Increases with liver cell damage from inflammation.)
ALT (SGPT)	5-35 units. (Increases with liver cell damage from inflammation.)
GGT, GGTP	5-55 μ/L females, 5-85 μ/L males. (Increases with alcohol abuse and may indicate liver or bile duct damage.)
LDH	100-200 units. (Increases with alcohol abuse and liver damage.)
Serum ammonia	150-250 mg/dL (Increases in liver failure.)
Cholesterol	Total increases with bile duct obstruction and decreases with parenchymal disease. • <200 Optimal • 200-239 Borderline high • ≥240 High

The primary tests that indicate hepatitis are alanine transaminase (ALT) and aspartate transaminase (AST). These liver enzymes elevate in the presence of inflammation and damage to liver cells. Alkaline phosphatase also increases with liver damage. ALT is more specific than AST (and usually higher) and is often used to monitor the progress of treatment. ALT levels may increase to 10 times the normal level during acute hepatitis, staying elevated for 1 to 2 months and returning to normal within 6 months. ALT levels tend to be less elevated with chronic liver disease, usually to <4 times normal values. Because many drugs can affect ALT levels, people should be questioned about all prescription and non-prescription drugs when tested.

Because hepatitis can interfere with liver function, levels of albumin and total protein may fall, indicating that liver cannot produce adequate amounts of albumin. Additionally, bilirubin levels may increase because the liver cannot adequately filter bilirubin, which results from the breakdown of red blood cells.

In some cases, especially if tests are not conclusive, a liver biopsy may be indicated to determine the severity of hepatitis and the degree of liver damage.

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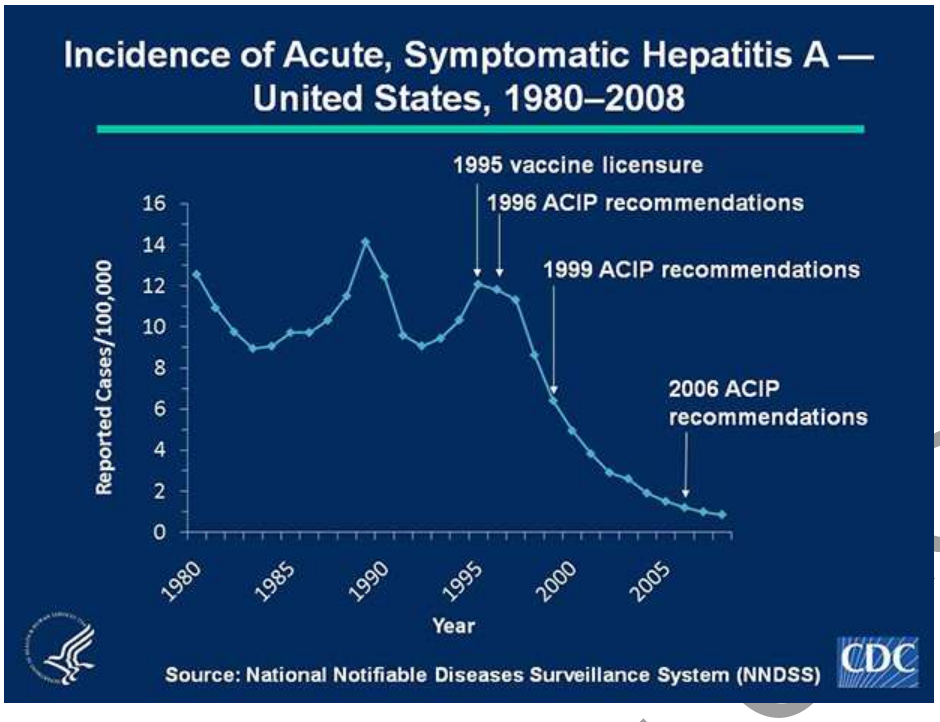
Hepatitis A

Hepatitis A, caused by an RNA virus of the Enterovirus family, is foodborne hepatitis, spread by the fecal-oral route, most commonly from contaminated food, water, or shellfish, or through oral-anal sexual practices. Hepatitis A virus (HAV) may cause individual infections or epidemics. Outbreaks have occurred in daycare centers, probably from diaper contamination of surfaces, such as changing tables, and facilities with people with developmental disabilities. A large outbreak occurred in Pennsylvania in 2003, traced to fecally-contaminated onions from Mexico.

Incubation period	15 to 50 days (mean 28 to 30)
Illness duration	4 to 8 weeks. Duration is longer in those >40.
Carrier state	No carrier state exists.
Mortality	Rarely fatal.
Laboratory	Abnormal liver function tests. Increased ALT. HAV antigen is present in stool for 7 to 10 days prior to onset of illness and for 14 to 21 days after symptoms subside.
Vaccination	Available. Recommended for: • All children 1 to 2 years in 2 doses and those 24 months to 18 years at risk in 2 doses, with the second dosage 6 months after the first (Mandatory for infants in some states.) • Adults ≥18. Two doses with the second dose 6 to 12 months after the first. Also recommended for those with risk factors as well as those with clotting factor disorders or chronic liver disease and those working in laboratories where they may have contact with HAV.

In 1992, the advisory committee on immunization practices (ACIP) recommended the vaccination only for those at risk, expanding this advisory in 1996 to include children in 11 states with high rates of HAV and then in 2006 to include all children. Immunization has had a significant impact on the number of people infected.



Symptoms

Symptoms are more severe in those >40, and morbidity increases in the presence of previous liver disease. Most patients are asymptomatic or have mild symptoms, which include:

- Low-grade fever.
- Anorexia, nausea.
- Fatigue, malaise.
- Myalgia.
- Dark urine.
- Clay-colored stools
- RUQ abdominal discomfort.
- Indigestion.
- Aversion to taste or cigarettes or other strong odors.

Jaundice and hepatomegaly may occur. By the time jaundice occurs, the person is usually no longer infectious to others. Jaundice usually peaks in about 10 days after onset. The virus lasts only a short time in the serum, so chronic infection does not occur, but about 15% of people with HAV develop cholestatic hepatitis with relapsing symptoms and jaundice for about 6 to 9 months.

Treatment

Individuals who are exposed to HAV and who have not received HAV vaccine should receive prophylactic doses of immune globulin, which provides passive immunity for 2-3 months.

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Treatment comprises bedrest initially with gradual increase in activity based on liver function tests and nutritious diet to prevent weight loss. People must avoid alcohol.

Risk factors	• Household contact with infected persons. • Sexual contact with infected persons. • Traveling to counties where HAV is common. • Living in endemic areas during 1987-1997. • Males having sex with males. • Using injection and non-injection drugs.
Prevention	• Careful handwashing before eating or preparing foods. • Handwashing after changing diapers or touching fecally-contaminated surfaces. • Avoiding shellfish (especially raw and undercooked) from contaminated waters.

Hepatitis B

Hepatitis B is the most common bloodborne hepatitis, with infection through percutaneous or permucosal routes. The virus is present in bodily fluids, including blood, semen, vaginal fluids, tears, breast milk, and saliva. HBV may also be transmitted from an infected mother to her newborn during delivery. HBV is more infectious than HIV. Outbreaks in healthcare facilities (assisted living and skilled nursing) have resulted from the use of multi-dose vials and contaminated blood-sampling and hemodialysis equipment.

The HBV is composed of a number of antigenic particles to which the infected person develops antibodies. Antigenic particles include:

- HbcAg (core antigen).
- HbsAg (surface antigen).
- HbeAg (independent protein circling in the blood stream).
- HBxAg (gene product of X gene of HBV DNA).

Each antigen elicits production of a specific antibody at different stages of the disease. The presence of these antibodies helps to identify and predict the course of the disease. There are 8 different genotypes of HBV, affecting the course of the illness and response to treatment.

Incubation period	1 to 6 months.
Illness duration	Most people recover within 6 months of onset of symptoms, but 3-4 months of convalescence is

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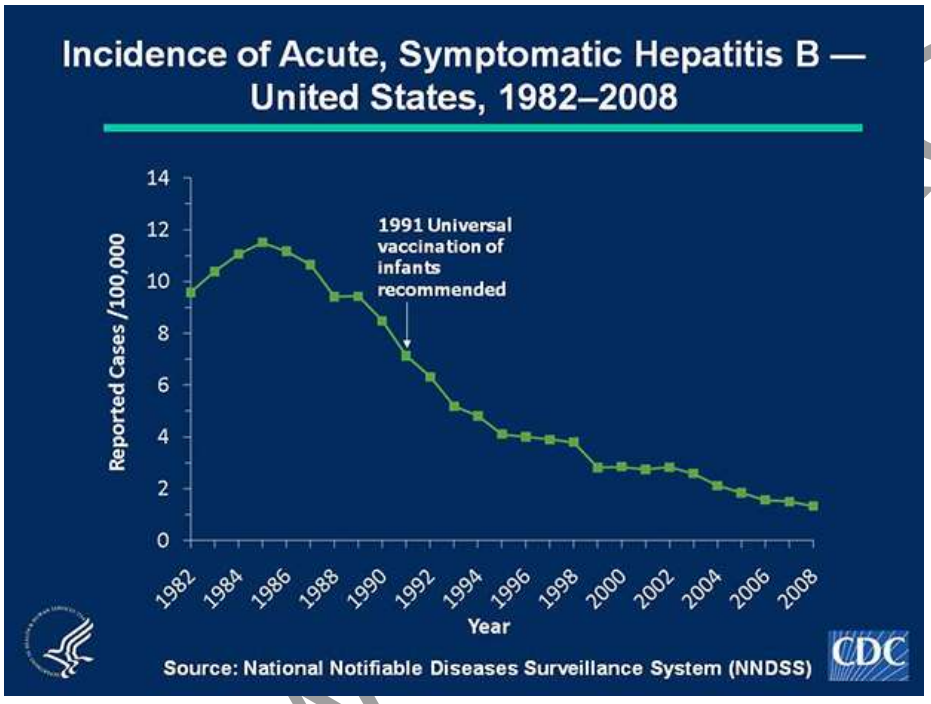
	common. Those whose disease lasts >6 months are more likely to develop chronic illness.
Carrier state	10% may develop a carrier state. About 1.2 million Americans are HBV carriers.
Mortality	10%
Laboratory	Abnormal liver function tests. ALT levels are higher than with HAV. Antibody tests: • Anti-HBc: Usually present during acute phase and may indicate continuing presence of HBV in the liver. • Anti-HBs: Usually present during recovery stage late in convalescence, indicating developing immunity. • Anti-HBe: Usually present with reduced infectivity. • Anti-HBxAg: Usually indicates ongoing chronic replication of HBV. If this persists for ≥6 months, the person is considered a carrier.
Vaccination	Available and given in 3 doses with second dose 1 month after first and last dose 6 months after first. Universal vaccination is recommended for all infants with catch-up vaccinations for all those ≤19 or those at risk. **Active immunization:** Two vaccines are available: • Yeast-recombinant HBV vaccine. • Plasma-derived (rarely used) except for those who are immunodeficient or allergic to other vaccine. Recommended for those at risk, including healthcare workers, those with STDs, those with high-risk sexual activity, and IV drug users to provide immunity for up to 5 years. OSHA requires health care facilities offer HBV vaccine to employees with jobs that require exposure to blood, blood products, or other potentially infectious materials. **Passive immunization:** Hepatitis B immune globulin (HBIG) protects those exposed to HBV and who have never had HBV or received a previous vaccination. HBIG is used for post-exposure from needlestick, splashes that contact mucous membranes, sexual

	contact with infected people, and perinatal exposure. Note: Some people may be advised to have both HBIG and vaccination. In that case, the vaccines should be given at separate sites and in separate syringes.

The ACIP's recommendation for universal vaccination of infants has been effective in lowering transmission of HBV.



Symptoms

Onset of symptoms is usually quite gradual and about 30% are essentially asymptomatic. Symptoms can include:

- Rash.

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- Arthralgia, myalgia.
- Malaise, generalized weakness.
- Clay-colored stools.
- Dark urine.
- Anorexia.
- Dyspepsia.
- Hepatosplenomegaly. The liver may be enlarged vertically to 12 to 14 cm.
- Enlargement of posterior cervical lymph nodes.

People are usually contagious throughout the incubation period and for up to 15 weeks after onset of symptoms. Those who develop chronic HBV infection remain infectious. About 10% develop chronic HBV infection and are at risk for hepatocellular necrosis, fulminant hepatitis, cirrhosis, and hepatocellular carcinoma.

Treatment

Infants born to infected mothers should receive hepatitis B immune globulin (HBIG) and a vaccination ≤ 12 hours after birth to prevent infection. Post-exposure prophylaxis (PEP) for HBV includes HBIG and initiating the HBV vaccine series to those unvaccinated. Treatment options for chronic HBV include:

- Interferon-alpha 2a or 2b requires 4-6 months of daily injections of 5 million units or 3 times weekly injections of 10 million notes.

- Pegylated interferon alpha 2a is indicated for adults with HBeAg-positive and HBeAg-negative chronic hepatitis B disease with compensated liver disease and evidence of viral replication and liver inflammation. Dosage is 180mg sc weekly for 48 weeks.

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- Lamivudine may be used as primary HBV treatment or with advanced decompensated cirrhosis (while people wait for transplant). It is usually taken 100 mg orally for 12 months, which results in suppression of viral reproduction in about 30% and most liver function tests will become normal. Lamivudine may be used initially but may also be used if interferon-alpha was not successful, but there is no advantage to using both treatments simultaneously.

- Other antivirals include:
    - Tenofovir disoproxil fumarate (Viread®) is an antiviral usually taken in one 300 mg oral dose daily for 12 months.
    - Adefovir dipivoxil (Hepsera®) may be used as a primary treatment for HBV or if lamivudine is not successful. Dosage varies.
    - Telbivudine (Tyzeka) may be used for those who do not respond to or can't tolerate interferon. Dosage varies.

Patients may be on bedrest initially with activity slowly increased as tolerated. Adequate nutrition is important, but protein may be restricted if the liver is unable to adequately metabolize proteins.

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Risk factors	The following are at increased risk: • People who work in fields that bring them in contact with bodily fluids, including nurses, physicians, respiratory therapists, and laboratory workers, are at increased risk. • Both staff and patients in hemodialysis and oncology units. • People who have multiple sexual partners, especially those engaging in oral/anal sex, or whose partner is infected. • Those with HIV infection (7% are co-infected with HBV). • IV injection drug users, especially those sharing needles (over 50% of cases arise from injection drug use). • Those with household contact with chronically infected persons. • Infant born to infected mother. • Children of immigrants from endemic areas. • Those who receive tattoo/ body piercing with contaminated needles/equipment.

	• Those who are incarcerated (approximately half of patients with HBV in the US have been previously incarcerated or treated for an STD).
Prevention	Interrupt transmission through vaccinating those at high risk, ensure safe practices, promote condom use, and provide passive immunization to those exposed to HBV. Specific measures include: Continued screening of blood donors. • Use of disposable needles. • Use of gloves when handling blood or contaminated materials. • Prohibition on eating or smoking in laboratory or areas where one may encounter blood or other secretions.

Hepatitis C

Hepatitis C (HCV) is the most common bloodborne viral infection in the United States. HCV can be transmitted to the fetus during pregnancy if the mother's viral RNA blood levels are extremely high, but almost all infections occur during delivery, infecting about 4% of infants born to HCV positive mothers.

About 8,000 to 10,000 deaths each year are directly attributable to HCV, and it is the primary reason for liver transplantation. Highest incidence is in ages 40 to 59, especially in African Americans. HCV was first identified in 1988. Before that, it was designated as "non-A, non-B" hepatitis. Outbreaks have occurred in healthcare facilities (endoscopy, dialysis, out-patient cardiac) related to reuse of syringes, multi-use vials, and breaches in infection control.

HCV is an RNA virus that can mutate quickly, so treatment can be difficult. Additionally, HCV infections can be caused by a number of different genotypes. Genotype 1 is most common in the United States, but this genotype is more resistant to treatment than types 2 or 3, and immigrants or travelers may present with other genotypes. In addition to genotypes, there are about 50 subtypes:

- 1a: Primarily in North & South America; common in Australia.
- 1b: Primarily in Europe and Asia.
- 2a: Most common genotype 2 in Japan and China.
- 2b: Most common genotype 2 in the U.S. and Northern Europe.

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- 2c: Most common genotype 2 in Western and Southern Europe.

- 3a: Highly prevalent in Australia (40% of cases) and South Asia.

- 4a: Highly prevalent in Egypt
- 4c: Highly prevalent in Central Africa.
- 5a: Highly prevalent only in South Africa 6a - restricted to Hong Kong, Macau and Vietnam.
- 7a and 7b: Common in Thailand.
- 8a, 8b & 9a: Prevalent in Vietnam.
- 10a & 11a: Found in Indonesia.

Incidence has fallen sharply since testing of the blood supply was instituted. The high point was 1992 with 6010 active symptomatic cases diagnosed (rate of 2.4/100,000 population), dropping to 878 active cases in 2008 (rate of 0.3/100,000 population) although the last few years have shown a slow increase in cases.

Despite the low numbers of confirmed diagnoses, the CDC estimates that 17,000 new HCV infections occur each year, and 3.2 million people are chronically-infected with highest rates in those born between 1945 and 1965 and infected in the 1970s and 1980s when rates were high. Many people remain undiagnosed until they present with severe liver disease, usually about 20 years after initial infection.

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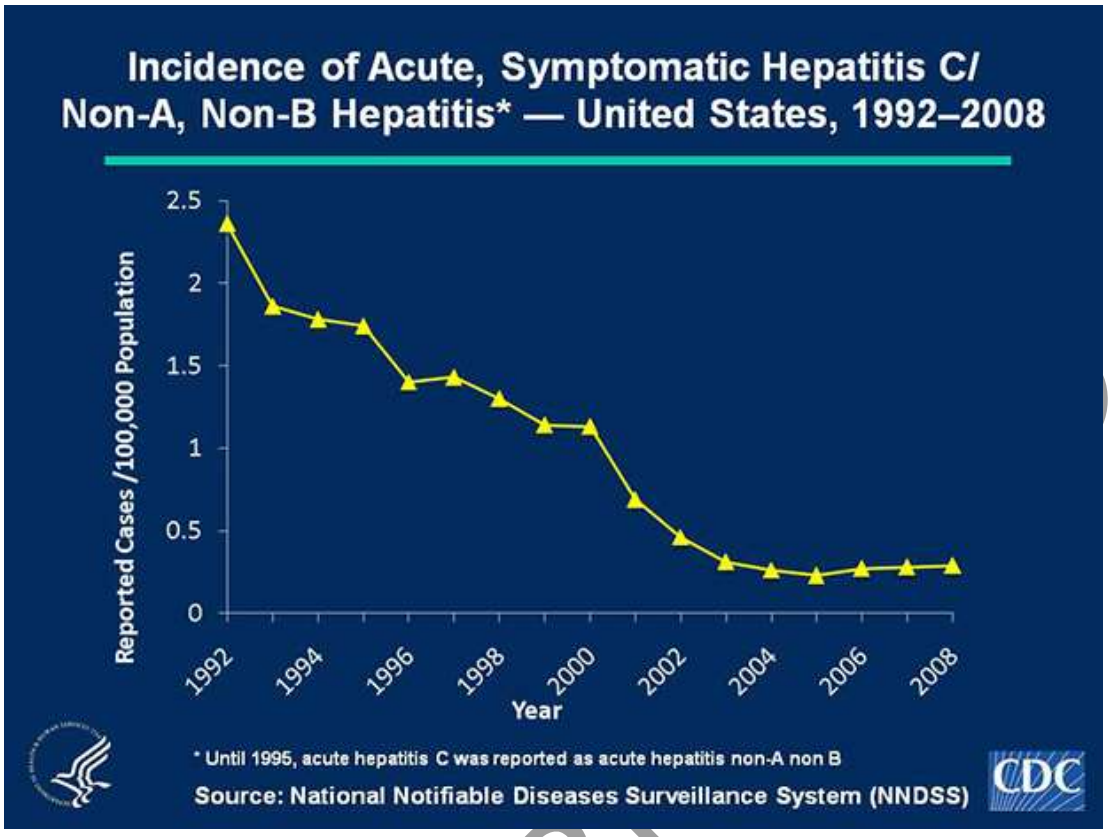

Incubation period	2 to 24 weeks (most at 4-12 weeks)
Illness duration	Varies from a few months to 20 years
Carrier state	Occurs frequently (75-85% develop chronic disease).
Mortality	1-5%
Laboratory	Anti-HCV screening test with positive findings verified by anti-HCV supplemental tests (RIBA and HCV RNA).
Vaccination	None available.

Symptoms

Many people are asymptomatic, but symptoms, which occur in 20 to 30%, are similar to other forms of hepatitis and can include:

- Fever
- Fatigue
- Dark urine
- Clay-colored stool
- Abdominal pain
- Loss of appetite
- Nausea
- Vomiting

- Joint pain
- Jaundice
- Slight neurocognitive impairment (with chronic disease).

Progression to cirrhosis is most common in those who are older, obese, co-infected with HIV, and consume >50 g of alcohol daily. Cirrhosis may progress to hepatocellular necrosis and carcinoma, but symptoms are slow and insidious.

HCV can also impact other organs than the liver, resulting in increased incidence of a variety of illnesses:

- Diabetes mellitus, which occurs three times more frequently in HCV-infected persons
- Glomerulonephritis, a type of kidney disease caused by inflammation of the kidney
- Essential mixed cryoglobulinemia, a condition involving the presence of abnormal proteins in the blood
- Porphyria cutanea tarda, an abnormality in heme production that causes skin fragility and blistering
- Non-Hodgkins lymphoma, slight increased incidence.

Treatment

Response to treatment varies according to genotype. Genotypes 2 and 3 are three times more likely to respond to treatment than genotype 1. The goal of treatment is to achieve **sustained virological response** (SVR), absence of HCV RNA 24 weeks after discontinuation of therapy. In some cases undetected virus may be present, so SVR cannot be guaranteed. If a **rapid virological response** (RVR), which is undetectable levels of HCV RNA, occurs at the 4th week of treatment, this is a good indication that SVR can be achieved. Those who fail to achieve an **early virological response** (EVR), a ≥2 log reduction or absence of HCV RNA by 12 weeks of therapy are at risk of not achieving SVR.

Current treatment recommendation is for combination therapy with pegylated interferon and ribavirin, as this has proven more effective than monotherapy.

HCV Treatment	
Genotype 1 and 4	• Peginterferon alpha-2a 180 μg sc per week with ribavirin 1000 mg for those ≤75 kg and 1200 mg for those >75 kg for 48 weeks OR • Peginterferon alpha-2b 1.5 μg/kg /sc per week with ribavirin 800 mg for those <65 kg, 1000 mg for ≥65 to 85 kg, 1200 mg for >85

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	to 105kg, and 1400 mg for >105 kg for 48 weeks **Note**: if viral clearance occurs slowly during weeks 12 to 24, treatment may be extended to 72 weeks. Note: See below for information regarding new treatment.
Genotype 2 and 3	Peginterferon as above with ribavirin 800 mg for 24 weeks. **Note:** see below.

On May 13, 2021, the FDA approved use of a new drug for hepatitis C, boceprevir (Victrellis®). This drug is added to the existing drug regimen after the first 4 weeks of therapy. Studies show the cure rate increased from 20-40% overall to 60% with the addition of the new drug. A similar drug, telaprevir, by another pharmaceutical company is awaiting FDA approval. Studies with boceprevir indicate that 44% were able to clear the virus within 28 weeks instead of the usual 48 weeks, suggesting that protocols for treatment may be shortened. Side effects of boceprevir include increased risk of anemia and neutropenia. The medication is taken orally with 4 capsules 3 times daily

Treatment may be discontinued in those who fail to achieve EVR by week a2 because 97 to 100% do not achieve SVR. For those with some response but failure to achieve complete EVR, the person should be retested at week 24 and if failure to achieve EVR persists, no further treatment is indicated as the patient is resistant.

It is extremely important that people with HCV be cautioned against the use of any alcohol as alcohol increases the risk that HCV will become chronic and increases liver damage. Healthcare providers should review all over-the-counter drugs as many may be contraindicated as well. Diet and rest seems to have little effect on the outcome of the disease. There is no effective treatment for those who do not respond to the current drugs although chronic disease may be treated with the addition of polymerase and protease inhibitors. Contraindications to treatment include pregnancy, autoimmune disease, and active drinking or injection drug use.

Risk factors	• Injection drug use (currently the most common cause). • Long-term hemodialysis.

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	• Chronic liver disease. • Transfusions or organ transplants before 1992. • Percutaneous or permucosal exposure to HCV-positive blood. • Born to an HCV-positive woman. • Sex with an infected person. • Multiple sex partners, males having sex with males. • Living with an infected person and sharing personal items. • HIV co-infection.
Prevention	• Continued screening of blood donors. • Dialysis center precautions. • Use of disposable needles. • Use of gloves when handling blood or contaminated materials. • Prohibition on eating or smoking in laboratory or areas where one may encounter blood or other secretions. • Safe sex practices. • Drug therapy programs. • Increased testing for those at risk.

Hepatitis D

Hepatitis D is caused by the hepatitis D virus (HDV) also known as the delta virus, an incomplete bloodborne RNA virus that requires HBV surface antigens to replicate, so it only occurs as a co-infection or super infection with hepatitis B (HBV), putting individuals with HBV at increased risk. Three different genotypes have been identified. Genotype 1 occurs worldwide, genotype 2 in Asia, and genotype 3 in South America. In the United States, HDV infection is most common in adults although it occurs in children in developing countries per breaks in the skin.

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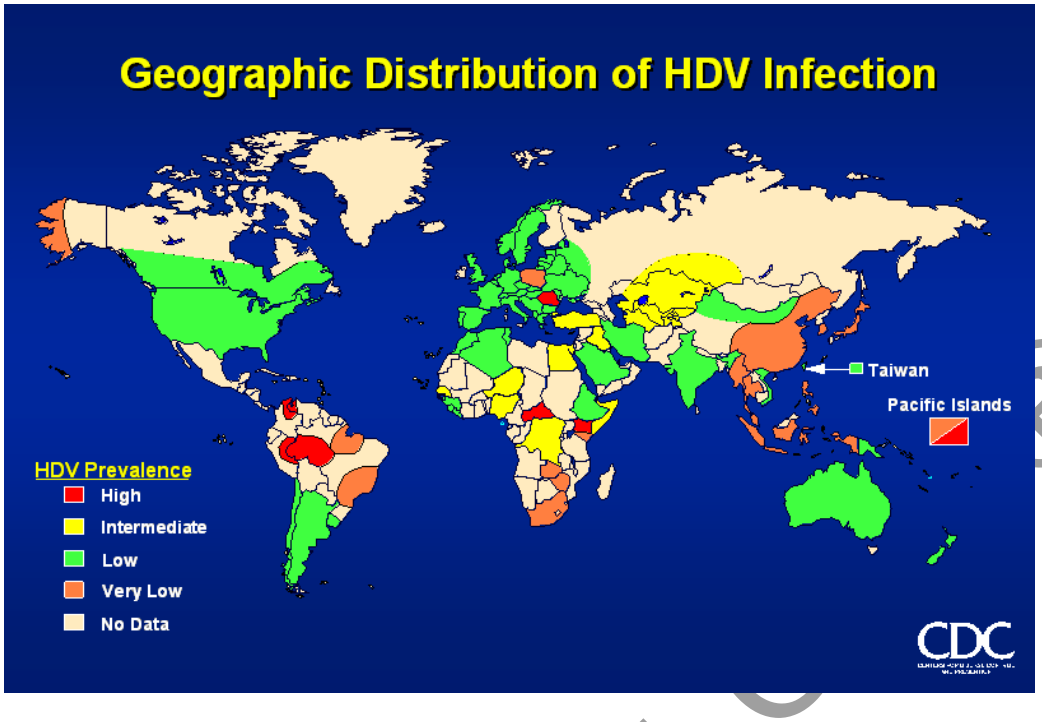

Because the symptoms are the same as for HBV, people may have co-infection without symptomatic evidence; however, co-infection places the person at additional risk of developing fulminant hepatitis with liver failure, and superinfection increases the risk of progression to cirrhosis:

- **Co-infection** is defined as HDV occuring when a patient has a simultaneous infection with both HDV and HBV. Of these, <5% develop chronic HDV infection. Most people clear both infections.
- **Superinfection** is defined as infection with HDV in someone who is already HbsAg positive. This is much more serious as fulminant liver failure occurs in 5% and 80 to 90% develop chronic infection. These patients also progress more quickly to cirrhosis and hepatocellular carcinoma.

Fulminant hepatitis is a rare occurrence, but incidence in those with HDV is 10 times more common with HDV than with other types of hepatitis. Researchers believe there are 3 phases to HDV infection:

1. Early active phase during which HDV replicates and HBV is suppressed.
2. Moderately active phase during which HDV decreases and HBV reactivates.
3. Chronic condition with development of cirrhosis and hepatocellular carcinomas.

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Incubation period	21 to 45 days (shorter if occuring as a superinfection.
Illness duration	Varies.
Carrier state	Occurs with chronic infection.
Mortality	2 to 20%. Up to 80% in those with fulminant hepatitis.
Laboratory	HDV antigen positive in 20%. HDV RNA is positive in 90%. Abnormal liver function tests.
Vaccination	No specific vaccination is available for HDV, but preventing HBV through hepatitis B vaccination also serves to prevent HDV infection.

Symptoms
About 90% are essentially asymptomatic, or symptoms are not distinguishable from those of HBV. Typical symptoms include:

- Jaundice
- Dark urine
- Abdominal pain
- Nausea with vomiting
- Confusion, bruising, and bleeding (rare)
- Pruritus
- Scleral icterus
- Fever
- Abdominal pain, usually right upper quadrant
- Dark urine
- Encephalopathy (rare)

Complications include autoimmune manifestations, liver failure, and hepatocellular carcinoma.

Treatment
Chronic infection in adults is treated with interferon alpha-2a. This has not proven effective in the treatment of children and is not recommended for co-infection as HDV usually clears spontaneously. Lamivudine and ribavirin have not proven effective to treat HDV:

- Interferon alpha-2a 10 million U sc 3 times weekly for a year. Dosage is reduced by half if platelet level falls to <50,000/μL or granulocyte level falls to <750/μL. Treatment must be discontinued if platelet levels falls to <30,000/μL or granulocyte level falls to <500/μL

Risk factors	• Injection drug use. • Hemodialysis.

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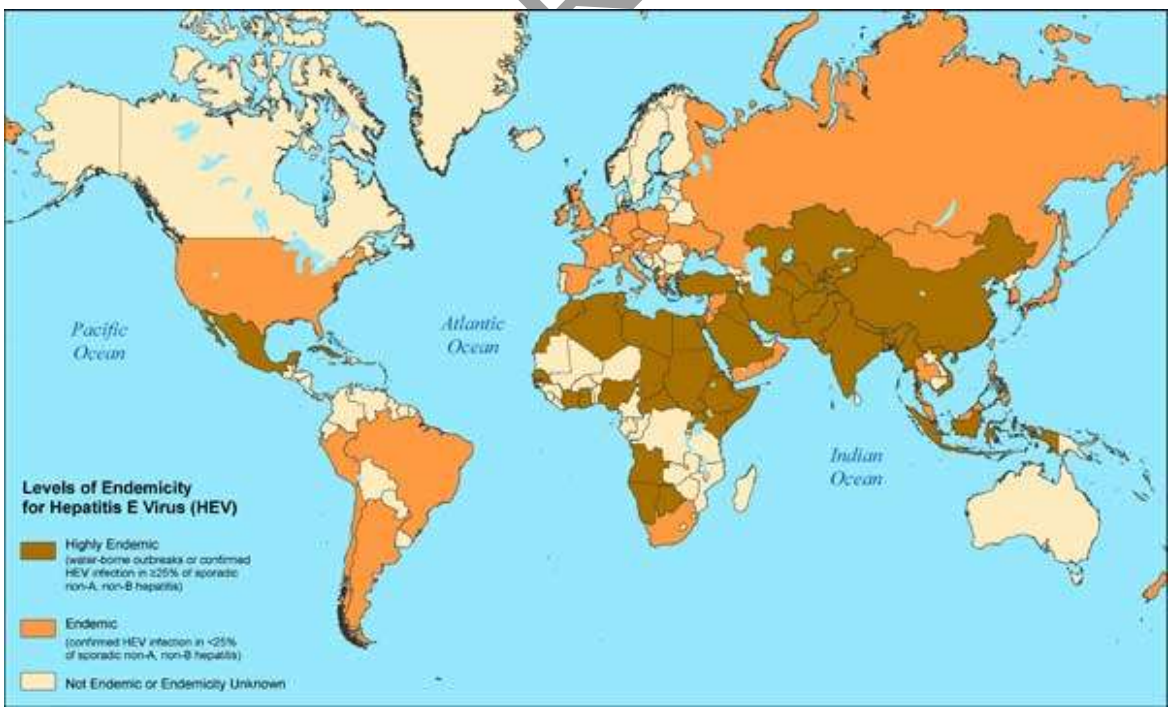
	• Sexual contact with infected persons. (Sexual transmission is less effective than with HBV.) • High-risk professions, such as healthcare and public safety workers. • Multiple blood transfusions.
Prevention	• HBV vaccination. • Safe handling of blood and blood products. • Safe sex practices. • Pre or post exposure prophylaxis for co-infection includes both HBIG and HBV vaccine series.

Hepatitis E

Hepatitis E (HEV), a foodborne RNA virus first identified in 1980, is rarely found in the United States, where sewage and sanitation systems are usually adequate, but HEV may occur in travelers from developing countries where the disease occurs, such as South Asia and North Africa. Large waterborne outbreaks affecting hundreds of thousands of people have occurred in Asia, Africa, and Central America.



HEV is most common in those 15-40 years of age and is contracted by the oral-fecal route by ingestion of contaminated water or food. HEV is an acute disorder and does not produce a chronic infection or carrier

state. However, women who are pregnant may develop liver failure. People remain contagious up to 2 weeks after symptoms occur.

Incubation period	14 to 60 days (mean of 40 days).
Illness duration	Usually about 2 weeks after onset of symptoms.
Carrier state	None.
Mortality	0.5 to 4%. Fulminant hepatitis in pregnant women causes a 20% mortality rate during the 3rd trimester.
Laboratory	Antibody tests (not widely available).
Vaccination	None available.

Symptoms
Many are asymptomatic, especially children. Symptoms, when they occur are similar to those of HAV, appearing abruptly, and may include:

- Jaundice.
- Abdominal discomfort.
- Hepatomegaly.
- Anorexia.
- Fatigue.
- Dark urine.
- Diarrhea.
- Clay-colored stools.
- Nausea and vomiting.
- Fever.
- Myalgia and arthralgia.

Treatment
There is no treatment, but this acute infection is usually self-limiting.

Risk factors	Travel to endemic areas ingesting contaminated food or water. Japan: Eating undercooked venison, boar meat, or pig liver.
Prevention	When traveling to endemic areas: Drink bottled water. Avoid uncooked shellfish. Avoid raw, unpeeled fruits and vegetables. Avoid swimming in contaminated waters

Nursing considerations for hepatitis

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Nursing precautions
Patients with hepatitis do not require strict isolation. Standard precautions should be used with all patients with hepatitis and contact precautions, including thorough handwashing, after changing diapers or contacting potentially contaminated utensils, bedding, or clothing. Healthcare providers should use care when handling disposable needles and avoid recapping and should not use multi-dose vials.

While much treatment is supportive, dietary management is a concern because anorexia is common. Research indicates there may be benefit from a high protein, high calorie diet, but people should be advised not to try to force food or to limit fat. Dietary management includes:

- Small, frequent meals.
- Intake of 2000 to 2000 kcal/day during acute illness.
- Enteral feedings if nausea and vomiting severe.
- Adequate fluid balance.
- Avoidance of alcohol during recovery and for at least 6 months afterward.

Stools and urine should be observed for color, consistency and amount. Patients and families should receive instruction in reducing risk, such as through good hygiene, careful handwashing (especially after bowel movements and before eating or preparing food) and sanitation.

Patients with pruritis should be advised to keep nails trimmed and to avoid the use of drying soaps and alcohol-based lotions. Emollients may relieve itching.

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Complications

Fulminant hepatitic failure

Fulminant hepatitic hepatitis is a severe life-threatening acute liver infection that usually occurs suddenly within 8 weeks after the first indication of jaundice and leads to hepatocellular necrosis and fulminant liver failure. While various toxic substances and diseases can cause fulminant hepatitic failure, viral hepatitis (especially HCV) is a common cause. The disease progresses very rapidly with onset of jaundice, hepatic necrosis, encephalopathy, and death often occurring within days:

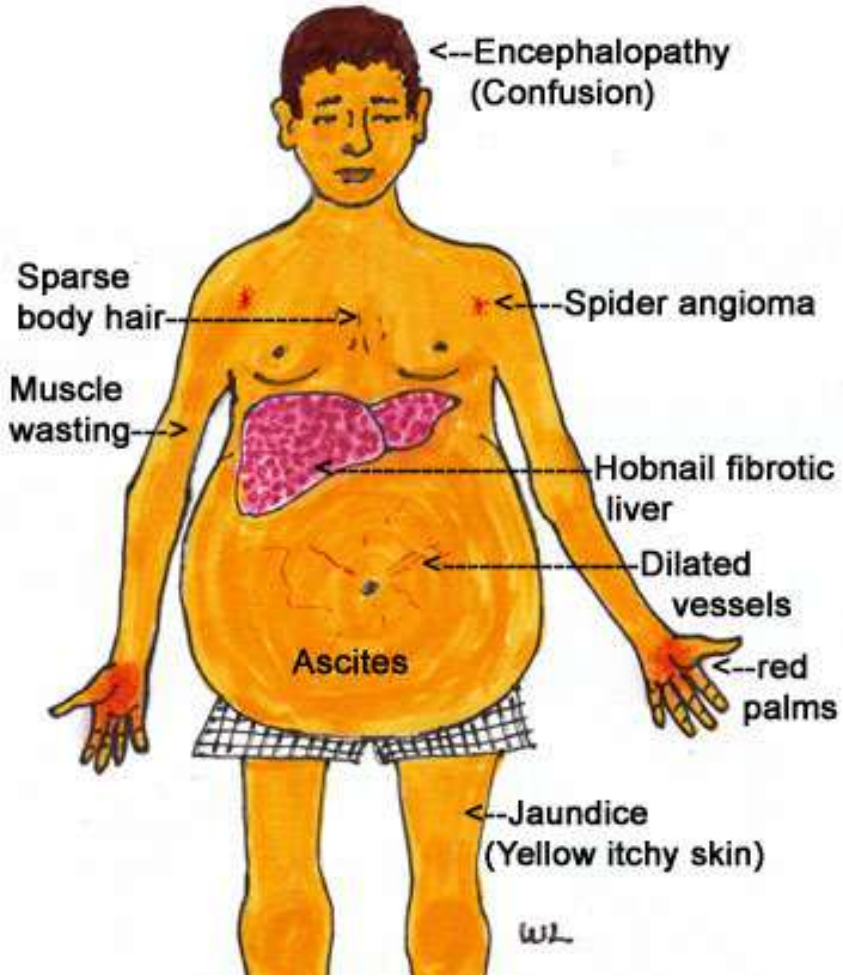
- Hyperacute: 0-7 days.
- Acute: 8-28 days.

- Subacute: 28-72 days.

Initial symptoms often include severe anorexia and jaundice. As encephalopathy with cerebral edema develops, intracranial pressure increases. Other abnormalities include coagulopathies, renal failure, hepatocellular necrosis, and electrolyte imbalances. Mortality rates are up to 80%, resulting from gastrointestinal bleeding, sepsis, brainstem compression from cerebral edema, or multisystem failure.

Initial treatment includes identifying and treating the specific cause, if possible, as well as monitoring intracranial pressure, and providing diuretic therapy. With severe liver damage, liver transplantation may be the only definitive treatment.

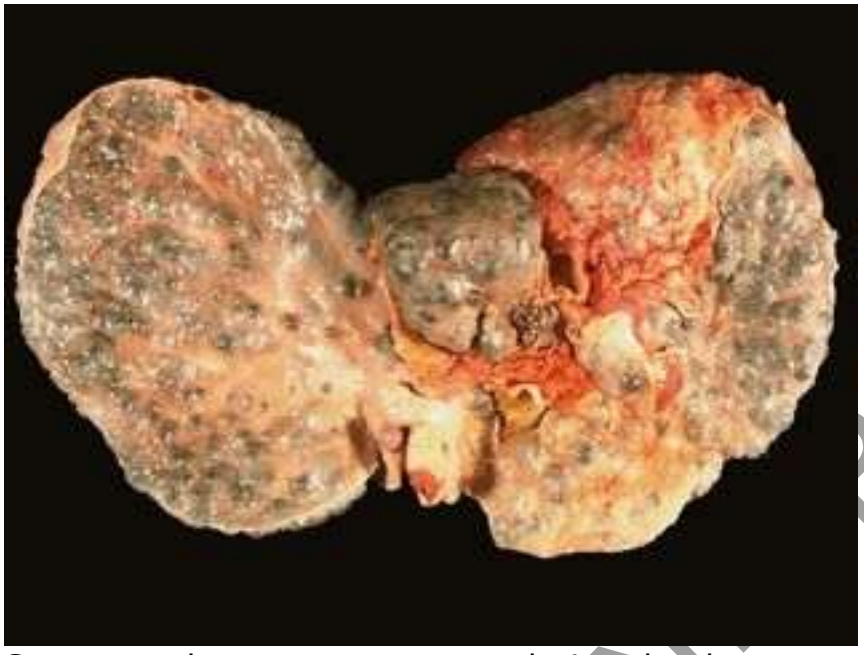
Cirrhosis



Because it has a rich blood supply, the liver can recover from infection in many cases and can compensate for damage until a threshold is reached. The three types of cirrhosis include alcoholic, biliary, and post-necrotic. Hepatitis causes post-necrotic cirrhosis in which the damaged liver cells are replaced with fibrotic tissue, and the liver exhibits broad bands of fibrosis. In the early stages of compensated

cirrhosis, symptoms may be non-specific and can include intermittent fever, peripheral edema, nausea, anorexia, and hepatosplenomegaly.

Post-necrotic cirrhosis



Symptoms become more acute during the decompensated stage when the liver is too damaged to function adequately:

- Portal hypertension.
- Esophageal varices.
- Spider angiomas.
- Hepatomegaly and splenomegaly
- Thrombocytopenia occurs with resulting purpura, bruising, and epistaxis.
- Peripheral and presacral edema (from decreased plasma albumin).
- Clubbing of fingers may result from poor systemic oxygenation.
- Ascites (sometimes complicated by peritonitis).
- Amenorrhea in women, gynecomastia in men, and gonadal atrophy (because of altered hormones).
- Vitamin deficiency of A, C, and K.
- Anemia.
- Hypotension (from vasodilation in advanced cirrhosis).

Once fibrotic changes occur, they cannot be reduced, so treatment becomes supportive to try to halt or slow the progress of the disease. Liver transplantation is the only definitive treatment for advanced cirrhosis. Supportive treatment includes:

- Nutritional support with supplements and vitamins.

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- Potassium-sparing diuretics, such as Aldactone® and Dyrenium® (to relieve ascites).
- In some cases, a shunt may be inserted to divert ascitic fluid from the abdominal cavity into the superior vena cava.

Hepatocellular carcinoma

Hepatocellular carcinoma most commonly occurs in those with a history of chronic hepatitis, especially HBV and HCV. While primary cancerous tumors may originate in either hepatocytes or bile duct cells, hepatocytes comprise 80% of liver tissue, so up to 95% of tumors arise in hepatocytes, resulting in hepatocellular carcinoma.

Hepatocellular carcinoma occurs more often in males than females, but prognosis is uniformly poor with only 5% surviving to 5 years. One problem is that damage from chronic hepatitis occurs slowly over up to 20 years and symptoms may be evident only after cancer is advanced. Initial symptoms may include pain in the right upper quadrant, loss of weight, increased weakness, hepatomegaly, and a bruit in up to 50% over the liver from increased blood flow and turbulence in the hepatic artery.

For patients previously diagnosed with cirrhosis, initial indications of liver cancer include sudden onset of complications, such as jaundice and ascites. Metastasis may occur before diagnosis with lesions to the regional lymph nodes and lungs firs although liver cancer may metastasize to the bones or brain.

Because so much liver damage has already occurred with chronic hepatitis, treatment is very difficult, but may chemotherapy,

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chemoembolization, radiofrequency ablation, and proton beam therapy, but treatment is primarily palliative. Liver transplantation is the only definitive treatment.

Summary

Hepatitis, inflammation of the liver, can be caused by a wide range of organisms and toxic substances but viral hepatitis is the most common. Common symptoms include fever, malaise, jaundice, dark urine, clay-colored stools, abdominal pain, pruritis, and increased bruising. A variety of liver function tests may be used to diagnose and monitor viral hepatitis, but ALT is used most commonly as levels increase with hepatitis.

The primary hepatitis-causing viruses include:

- HAV: Foodborne hepatitis spread by oral-fecal route is rarely fatal and has no carrier state. Vaccination is recommended for all children and those at risk. Treatment comprises rest and adequate diet.
- HBV: Bloodborne hepatitis may require up to 6 months convalescence and 10% may develop carrier state. Vaccination is available for active immunization and is recommended for all infants and those <19 as well as those at risk. Passive immunization with HBIG is recommended post-exposure. Treatment options include pegylated interferon alpha 2a or antivirals, such as lamivudine.
- HCV: Bloodborne hepatitis is the primary cause of chronic disease and liver transplantation. Genotype 1 is most common in the United States but is less responsive to treatment than types 2 and 3. No vaccination is available. Treatment aims to achieve sustained virological response (SVR). Combination treatment with pegylated interferon and ribavirin is recommended.
- HDV (delta virus): Bloodborne hepatitis requires HBV surface antigen to replicate. HDV may occur as co-infection or superinfection and increases risk of developing fulminant hepatitis. No vaccination is available. Adults are treated with interferon alpha-2a.
- HEV: Foodborne hepatitis occurs where sanitation is poor. This acute disorder has no carrier state but may cause fulminant hepatitis in pregnant women.

Complications of hepatitis result from chronic infection and liver damage: fulminant hepatitic failure, cirrhosis, and hepatocellular cancer.

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