

Viral Hepatitis

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THE STORY IN ONE SENTENCE

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“Five different viruses (A, B, C, D, E) can attack the liver; some leave fast, others hide for years and slowly scar it.”

Normal anatomy — the body's job

The liver is a chemical factory the size of a football, sitting under your right ribs. Every minute, about a quarter of your blood flows through it to get cleaned, restocked, and balanced before heading back to your heart.

BODY'S JOB

Hepatocytes—the liver's working cells—do three giant jobs: (1) filter toxins, drugs, and waste out of blood, (2) build essential proteins like albumin (keeps fluid in blood vessels) and clotting factors (stop bleeding), and (3) break down old red blood cells into bilirubin, a yellow pigment that gets dumped into bile, flows to your gut, and leaves in your poop (giving stool its brown color).

1. Blood from your intestines, loaded with nutrients and some toxins, enters the liver through the portal vein.
2. Hepatocytes pull out poisons (ammonia, alcohol, drug leftovers) and convert them into safer chemicals or send them to the kidneys to pee out.
3. Hepatocytes build albumin to hold water in your blood, clotting factors so cuts stop bleeding, and bile to digest fats.
4. Old red blood cells break into bilirubin; hepatocytes grab it, attach a tag (conjugation), and dump it into bile ducts.
5. Bile flows to your gallbladder, then squirts into your small intestine when you eat, helping digest fat and carrying bilirubin out in poop.

6. Clean, restocked blood leaves the liver through the hepatic vein and returns to your heart to feed the whole body.

What breaks

A hepatitis virus invades liver cells, your immune system attacks the infected cells to kill the virus, and that battle damages the liver's ability to filter blood and make proteins.

CLINICAL TERM

Viral hepatitis: direct cytopathic injury (the virus itself breaking cell machinery) plus immune-mediated hepatocyte destruction (cytotoxic T cells killing infected cells) cause hepatocellular tissue death (necrosis), inflammation, and impaired synthetic and excretory function.

How the body compensates

In acute viral hepatitis, the liver is big and resilient—most of it keeps working while the immune system fights, so vital signs stay stable and many people never even know they're infected. The danger is missing the rare severe case (fulminant hepatic failure) or the slow scar buildup in chronic infection.

1. The liver has huge reserve: you can lose 70% of hepatocytes and the remaining 30% will cover the basic jobs, so blood pressure, heart rate, and oxygen levels look normal even during the acute battle.
2. Your body ramps up bilirubin excretion through the kidneys (dark urine) to offload what the liver can't handle, buying time while hepatocytes regenerate.
3. Fatigue and loss of appetite are protective—your body shuts down non-essential activity to funnel all energy into the immune fight and liver repair.
4. In hepatitis A and E, the virus usually clears in 2–8 weeks, hepatocytes regrow, and function returns to 100% with no lasting damage.
5. In hepatitis B, C, and D, the virus can dodge the immune system by hiding in hepatocytes, turning the infection chronic—compensation keeps you feeling fine for years while silent scarring accumulates.
6. The crash point to watch for acutely is fulminant hepatic failure (massive hepatocyte death in days to weeks), signaled by confusion (encephalopathy), easy bruising (coagulopathy, INR >1.5), and falling albumin—this is a medical emergency needing ICU care and possible liver transplant.

7. The crash point in chronic hepatitis is decompensated cirrhosis (ascites, variceal bleeding, encephalopathy) or hepatocellular carcinoma, both showing that scar tissue has choked off too many working cells.

Domino effect

1. Virus enters liver cells — Hepatitis A and E spread fecal-oral (contaminated food, water) and land in the gut, then hitch through the portal vein into hepatocytes. Hepatitis B, C, and D spread through blood or body fluids (needle-stick, sex, childbirth) and enter the bloodstream directly, docking onto hepatocyte surface receptors and injecting their genetic code inside.
2. Virus hijacks the cell and copies itself — Once inside, the virus uses the hepatocyte's ribosomes and enzymes to make thousands of new virus particles, which bud off to infect neighboring cells—this direct hijacking damages the cell's normal protein-making and detox machinery.
3. Immune system spots the invader and attacks — Infected hepatocytes display viral protein fragments on their surface (via MHC class I). Cytotoxic T cells (CD8+ T cells) recognize these flags and release perforin and granzymes to punch holes in the infected cell and trigger apoptosis (controlled cell death), killing the virus but also the hepatocyte.
4. Inflammation swells the liver; cytokines flood the body — Dying hepatocytes release damage signals (DAMPs); macrophages and natural killer cells rush in, releasing cytokines like TNF-alpha and interleukin-6, which cause fever, fatigue, muscle aches, and loss of appetite—the 'flu-like' early-warning phase (prodrome) of acute hepatitis—and swell the liver capsule, stretching it and causing right upper quadrant pain.
5. Bilirubin backs up, causing yellowing of the skin and eyes (jaundice) and dark urine — Damaged hepatocytes cannot conjugate bilirubin or pump it into bile, so both unconjugated (indirect) and conjugated (direct) bilirubin spill into blood. Unconjugated stains the skin and sclera yellow; conjugated is water-soluble, so kidneys filter it into urine, turning it dark brown or tea-colored.
6. Acute hepatitis resolves (A, E) or becomes chronic (B, C, D) — In hepatitis A and E, the immune system clears the virus completely in weeks to months, hepatocytes regenerate, and you're immune for life. In hepatitis B and C (and D, which needs B), the virus can evade immune surveillance—B integrates into hepatocyte DNA, C mutates rapidly—so the infection persists, the low-grade immune attack continues, and repeated rounds of cell death and repair lay down collagen scar tissue (fibrosis).

7. Chronic inflammation scars the liver into cirrhosis — Over 10–30 years, fibrosis bands harden into cirrhosis—thick scar walls choke blood flow through the portal vein, raising pressure (portal high blood pressure (hypertension)), which forces blood to detour through fragile veins in the esophagus (varices that can bleed) and belly (ascites fluid), and leaves too few working hepatocytes to clear toxins (encephalopathy) or prevent cancer (5% annual risk of hepatocellular carcinoma in cirrhosis).
8. Treatment or vaccination breaks the domino chain — Hepatitis A and B vaccines train your immune system to block the virus before it ever infects a cell. Direct-acting antiviral pills (sofosbuvir, ledipasvir, glecaprevir/pibrentasvir) cure >95% of hepatitis C by stopping viral replication, letting the immune system mop up leftovers. Nucleos(t)ide analogs (tenofovir, entecavir) suppress hepatitis B replication for life, halting fibrosis and dropping cancer risk. Caught early, even significant fibrosis can partially reverse when the virus is gone.

Symptoms · what the patient feels, and why

- Feeling tired all the time, even after rest: The liver is working overtime to fight the virus and clear damaged cells, so less energy is left for the rest of the body. Also, the liver makes less glycogen (stored sugar fuel), leaving muscles low on power.
- Yellow skin and eyes (jaundice): The damaged liver cannot properly process bilirubin (a yellow waste product from broken-down red blood cells), so it builds up in the blood and spills into the skin and whites of the eyes.
- Dark urine, like cola or strong tea: Excess bilirubin in the blood gets filtered by the kidneys and dumped into the urine, turning it dark brown or amber.
- Pale or clay-colored stools: The liver is not releasing enough bile (which contains bilirubin) into the intestines, so the stool loses its normal brown color.
- Nausea, loss of appetite, or feeling sick to the stomach: Inflammation in the liver releases inflammatory signals (cytokines) that travel to the brain's vomiting center (the area postrema in the brainstem) and slow down digestion.
- Pain or tenderness in the upper right belly, just below the ribs: The liver is swelling because it is inflamed and filled with immune cells fighting the virus. The liver's capsule (the thin outer wrapping) stretches and triggers pain nerves.
- Mild fever and body aches, like a flu: The immune system releases pyrogens (fever-causing chemicals such as interleukin-1) to raise body temperature and fight the virus. The same signals also make muscles ache.
- Confusion, sleepiness, or trouble thinking clearly: A severely damaged liver cannot filter ammonia (a toxic waste from protein breakdown), so ammonia builds up in the blood and crosses into the brain, poisoning nerve cells and causing confusion—a condition called hepatic encephalopathy.

- Easy bruising or bleeding that does not stop quickly: The liver makes most of the clotting factors (proteins that help blood clot). When the liver is badly damaged, clotting-factor levels drop, so even small cuts bleed longer and bruises appear with minor bumps.
- Swelling in the belly (ascites) or legs (edema): A failing liver makes less albumin (the main protein that holds fluid inside blood vessels), so fluid leaks out into the belly and tissues. Also, scar tissue in the liver (cirrhosis) blocks blood flow, raising pressure in the portal vein and pushing fluid into the abdomen.

Focused exam

- Yellow skin and whites of eyes (jaundice): The liver cells (hepatocytes) are damaged by the virus and cannot clean bilirubin (a yellow breakdown product from old red blood cells) out of the blood fast enough, so it stains the skin and eyes yellow.
- Dark urine that looks like tea or cola: Bilirubin is spilling into the blood because damaged liver cells cannot package it into bile, so the kidneys filter it out and turn the urine dark brown.
- Tenderness when you press the right upper belly under the ribs: The liver is swollen because virus-infected cells are inflamed and the capsule (the thin stretchy covering around the liver) is being pulled tight, which causes pain when pressed.
- Pale or clay-colored stool: Bile (which contains bilirubin and gives stool its brown color) is not flowing into the intestines because the damaged liver cells cannot make or release it normally, so the stool turns pale or white.
- Fever and body aches in the first week: The immune system detects the virus and releases inflammatory chemicals (cytokines) to fight it, which cause fever, muscle aches, and fatigue just like the flu.
- Liver edge enlarged and firm when you feel below the rib cage: Inflamed and swollen hepatocytes make the liver bigger than normal, so you can feel the edge sticking out below the ribs instead of being tucked up under them.

Diagnostics · every test answers a question

- Alanine aminotransferase (ALT) and aspartate aminotransferase (AST) → Are liver cells breaking open and leaking their inside enzymes into the blood?
- Total and direct bilirubin → Is bilirubin backing up in the blood because the liver cannot process it?
- Hepatitis A IgM antibody → Is this acute hepatitis A (spread by contaminated food or water)?
- Hepatitis B surface antigen (HBsAg), core IgM antibody (anti-HBc IgM), and surface antibody (anti-HBs) → Is this acute hepatitis B (spread by blood, sex, or birth)?

- Hepatitis C antibody (anti-HCV) and HCV RNA by PCR → Is this hepatitis C (spread mainly by blood exposure)?
- Prothrombin time (PT) and INR → Is the liver still making clotting proteins, or is it failing?
- Albumin → Is the liver still making the main protein that keeps fluid inside blood vessels?
- Hepatitis D antibody (anti-HDV) and HDV RNA (if HBV is positive) → Does this person with hepatitis B also have hepatitis D (a virus that only infects people who already have HBV and makes the liver damage worse)?

Treatment logic

- Supportive care: rest, hydration, nutrition, and serial liver function tests every few days to weeks → Keeps the body strong and hydrated while the immune system fights and clears the virus; monitoring ALT, AST, bilirubin, and INR tells you if the liver is recovering or failing.
- Hepatitis A vaccine and hepatitis B vaccine for unvaccinated close contacts within 2 weeks of exposure → Trains the immune system to recognize and block hepatitis A or B virus before infection takes hold, preventing illness in exposed people.
- Hepatitis B immune globulin (HBIG) within 24 hours of exposure (e.g., needle stick, newborn of HBsAg-positive mother) → Gives ready-made antibodies that neutralize hepatitis B virus immediately, before the person's immune system can make its own, preventing or reducing infection.
- Antiviral therapy for chronic hepatitis B: entecavir or tenofovir daily, often for years or lifelong → Blocks the reverse transcriptase enzyme (the tool HBV uses to copy its DNA inside liver cells), stopping viral replication and reducing inflammation and scarring; lowers risk of cirrhosis and liver cancer.
- Direct-acting antivirals for hepatitis C: sofosbuvir/velpatasvir or glecaprevir/pibrentasvir for 8–12 weeks → Blocks the proteins HCV needs to copy itself (NS5A, NS5B polymerase), stopping viral replication completely and curing > 95% of people (undetectable HCV RNA 12 weeks after finishing treatment = sustained virologic response = cure).
- Pegylated interferon-alpha or bulevirtide for chronic hepatitis D (in people with HBV co-infection) → Pegylated interferon boosts the immune system's antiviral response and suppresses HDV replication in some people; bulevirtide blocks the receptor HDV uses to enter liver cells, lowering viral load and inflammation.
- Liver transplant for acute liver failure (INR > 6.5, encephalopathy, multi-organ failure) or end-stage cirrhosis from chronic viral hepatitis → Replaces the scarred, failing liver with a healthy donor liver that can clean the blood, make proteins, and support life again.

Medications

Hepatitis A vaccine (given to close contacts) · Inactivated virus vaccine

The vaccine contains killed hepatitis A virus pieces. When injected, it trains the immune system to recognize hepatitis A so the body can block infection before it starts. This stops the spread to people who were exposed.

1. The killed virus pieces enter the body and travel to lymph nodes.
2. Immune cells called B cells read the virus signature and learn to recognize it.
3. B cells make antibodies (protective proteins) that stick to hepatitis A virus.
4. The antibodies stay in the blood for years, ready to grab and destroy any hepatitis A virus that enters the body.
5. When real hepatitis A virus arrives, the antibodies block it from infecting liver cells.

SAFETY WATCH

Soreness, redness, or swelling at the injection site for 1-2 days · Mild fever or tiredness for 1-2 days · Very rare: severe allergic reaction (hives, trouble breathing, fast heartbeat) within minutes to hours — call 911 if this happens

Entecavir · Nucleoside analog antiviral (blocks virus copying)

Entecavir is a fake building block that looks like the real DNA piece hepatitis B virus needs to copy itself. When the virus tries to use entecavir, the copying machine jams and the virus cannot multiply. This lowers the number of virus particles attacking the liver, so liver damage slows or stops.

1. Hepatitis B virus invades liver cells and starts copying its DNA to make more virus.
2. The virus polymerase (copying enzyme) grabs building blocks to build new DNA strands.
3. Entecavir enters the liver cell and looks like a real DNA building block.
4. The polymerase accidentally uses entecavir instead of the real block.
5. The fake block jams the polymerase so it cannot finish copying the virus DNA.
6. Fewer new virus particles are made, so viral load drops and liver inflammation calms down.

SAFETY WATCH

Lactic acidosis (acid from oxygen-starved tissues) (too much acid in the blood) — rare but serious; watch for extreme tiredness, fast breathing, belly pain, nausea · Liver damage flare if you stop the drug suddenly — hepatitis can come roaring back, so never stop without your doctor's plan · Kidney problems if taken long-term — check kidney blood tests regularly · Headache, tiredness, or dizziness — usually mild

Tenofovir (TDF or TAF) · Nucleotide analog antiviral (blocks virus copying)

Tenofovir is a fake DNA building block that hepatitis B virus accidentally uses when copying itself. Once the virus polymerase (the copying machine) grabs tenofovir, the chain stops growing and the virus cannot finish making new copies. Fewer virus particles means less liver damage.

1. Hepatitis B virus enters liver cells and begins copying its DNA.
2. The virus polymerase picks up building blocks to assemble new DNA strands.
3. Tenofovir (after the liver activates it) looks like a real building block.
4. The polymerase inserts tenofovir into the growing DNA chain.
5. The chain cannot grow any further — tenofovir has no hook for the next block.
6. Virus copying stops, viral load drops, and liver inflammation decreases.

SAFETY WATCH

Kidney damage — TDF (the older form) can hurt kidney tubules over time; check kidney function every 6-12 months. TAF (the newer form) is gentler on kidneys. · Bone thinning (lower bone density) — TDF can weaken bones over years; TAF is safer for bones. · Lactic acidosis (acid from oxygen-starved tissues) (too much acid in the blood) — rare but serious; watch for deep tiredness, fast breathing, belly pain. · Liver flare if you stop suddenly — hepatitis can surge back; never stop without doctor guidance. · Headache, nausea, or tiredness — usually mild

Sofosbuvir-velpatasvir · Direct-acting antiviral combination (blocks two virus enzymes)

This pill combines two drugs that attack hepatitis C virus at two different steps. Sofosbuvir jams the virus copying machine (the polymerase) so the virus cannot make new RNA. Velpatasvir blocks the NS5A protein that the virus needs to assemble new particles. Together they stop the virus from multiplying and the immune system clears it out — this cures more than 95 percent of people.

1. Hepatitis C virus invades liver cells and starts copying its RNA genetic code.
2. Sofosbuvir enters the liver cell and looks like a real RNA building block.
3. The virus polymerase (NS5B enzyme) accidentally uses sofosbuvir, which jams the copying chain.
4. At the same time, velpatasvir grabs onto the NS5A protein and locks it in a shape that cannot help assemble or release new virus.
5. The virus cannot copy its RNA or build new particles, so no new virus is made.
6. The immune system clears the remaining virus over 8-12 weeks, and the infection is cured.

SAFETY WATCH

Headache or tiredness — common but usually mild · Reactivation of hepatitis B — if you also have chronic hepatitis B (even if silent), hepatitis C treatment can wake it up and cause liver damage; always test for hepatitis B before starting. · Slow heart rate (bradycardia) — rare, mostly if you also take amiodarone (a heart drug); can be dangerous. · Drug interactions — many medicines (especially seizure drugs, stomach-acid reducers, and HIV drugs) can interfere; tell your doctor every drug and supplement you take.

Glecaprevir-pibrentasvir · Direct-acting antiviral combination (blocks three virus enzymes)

This pill combines two drugs that hit hepatitis C virus at three weak spots. Glecaprevir blocks the NS3/4A protease (a virus scissor enzyme that cuts big virus proteins into working pieces). Pibrentasvir blocks the NS5A protein (needed to package and release new virus). Together they stop the virus from making the parts it needs and from assembling new particles. The virus dies out and the infection is cured in 8-12 weeks in more than 95 percent of people.

1. Hepatitis C virus invades liver cells and makes a long chain of virus proteins.
2. The NS3/4A protease (virus scissor enzyme) normally cuts this chain into the separate working pieces the virus needs.
3. Glecaprevir jams into the protease active site and locks it shut, so the scissor cannot cut.
4. At the same time, pibrentasvir binds the NS5A protein and freezes it in a shape that cannot help assemble or release new virus.
5. The virus cannot make working proteins or build new particles, so replication stops.
6. The immune system clears the remaining virus over 8-12 weeks, and the infection is cured.

SAFETY WATCH

Headache, tiredness, or nausea — common but usually mild · Liver enzyme rise (ALT/AST elevation) — rare but can be serious; do NOT use if you have advanced cirrhosis (Child-Pugh B or C). · Reactivation of hepatitis B — if you also have chronic hepatitis B (even if silent), hepatitis C treatment can wake it up; always test for hepatitis B before starting. · Drug interactions — this combination is very sensitive to other medicines; tell your doctor every drug and supplement (it interacts with many common drugs like statins, blood thinners, and seizure meds).

Pegylated interferon alfa · Immune-system booster (cytokine)

Pegylated interferon is a lab-made copy of interferon alfa, a natural alarm signal your immune system uses to fight viruses. The 'pegylated' part means it is wrapped in a coating (polyethylene glycol) so it lasts longer in the body. When injected, it wakes up the immune

system, tells infected cells to resist the virus, and helps T cells and natural killer cells destroy virus-infected liver cells. This is the main treatment for chronic hepatitis D (which comes only with hepatitis B). It also used to be used for hepatitis B and C, but newer drugs are now easier and more effective for those.

1. Pegylated interferon is injected under the skin and enters the bloodstream.
2. It binds to interferon receptors on the surface of liver cells and immune cells.
3. Inside these cells, the signal turns on hundreds of antiviral genes — this is called the 'interferon response.'
4. Infected liver cells make proteins that block virus copying and flag themselves for destruction.
5. T cells and natural killer cells recognize the flags and kill the infected cells.
6. The virus load drops and liver inflammation decreases.

SAFETY WATCH

Flu-like symptoms — fever, chills, muscle aches, headache, and extreme tiredness after each injection (very common). · Depression, anxiety, or mood swings — can be severe; tell your doctor if you feel hopeless or have thoughts of hurting yourself. · Low blood counts — the drug can lower white blood cells, red blood cells, and platelets; regular blood tests are needed. · Thyroid problems — interferon can damage the thyroid gland, causing it to make too much or too little hormone. · Lung, kidney, or eye damage — rare but serious. · Autoimmune flares — can trigger the immune system to attack your own tissues.

Bulevirtide · Virus entry blocker (hepatitis D-specific)

Bulevirtide is a lab-made copy of a piece of the receptor (the door lock) that hepatitis D virus uses to enter liver cells. When injected, bulevirtide plugs the lock (called NTCP, the sodium-taurocholate co-transporting polypeptide) so the virus cannot get in. This stops new liver cells from being infected. It is used for chronic hepatitis D, often combined with pegylated interferon.

1. Bulevirtide is injected under the skin and travels through the blood to the liver.
2. It binds tightly to the NTCP receptor on the surface of liver cells.
3. Hepatitis D virus particles (which travel with hepatitis B) try to dock at the NTCP receptor but find it blocked.
4. The virus cannot enter the liver cell, so it cannot replicate.
5. Virus levels drop and liver inflammation decreases.

SAFETY WATCH

Injection site reactions — redness, swelling, or itching · Rise in bile acids in the blood — because the drug blocks NTCP, which also transports bile salts; this can cause itching. · Liver enzyme rise (ALT/AST) — monitor liver tests regularly. · Long-term safety is still being studied — this drug was approved recently (2020 in Europe, investigational in the U.S.).

Ribavirin · Nucleoside analog antiviral (virus mutation promoter and immune modulator)

Ribavirin is a fake building block that looks like the RNA pieces hepatitis E virus (and some other RNA viruses) use to copy themselves. When the virus polymerase uses ribavirin, it makes mistakes — so many mistakes that the virus falls apart. Ribavirin also shifts the immune response to help T cells fight the virus. This is used for hepatitis E only in people with weak immune systems (organ transplant, HIV, chemotherapy), because in healthy people the body clears hepatitis E on its own.

1. Ribavirin enters liver cells and is activated into a form that looks like a real RNA building block.
2. The virus polymerase accidentally uses ribavirin when copying virus RNA.
3. Ribavirin causes copying errors — the virus makes so many mistakes that the new virus particles cannot work.
4. Ribavirin also boosts the immune system's Th1 response (the branch that fights viruses inside cells).
5. The combination of virus errors and immune help lowers the virus load.

SAFETY WATCH

Severe birth defects — NEVER use if pregnant or planning pregnancy (for both men and women); use two forms of birth control during treatment and for 6 months after. · Destroying red blood cells (Hemolytic) anemia (red blood cells break apart) — very common; watch for pale skin, tiredness, shortness of breath; regular blood counts are needed. · Cough, shortness of breath — can worsen lung disease. · Gout or rash — ribavirin raises uric acid. · Depression or irritability

Hepatitis B vaccine (Recombivax HB, Engerix-B) · Recombinant protein vaccine

The vaccine contains a lab-made copy of the hepatitis B surface protein (the outer coat of the virus). When injected, the immune system sees this protein, recognizes it as foreign, and learns to make antibodies against it. These antibodies stay in the blood for decades. If hepatitis B

virus enters the body later, the antibodies grab it and destroy it before it can infect liver cells. The vaccine is given as a series of 2-3 shots (depending on the brand) to build strong, lasting protection.

1. The vaccine protein (hepatitis B surface antigen) is injected into a muscle.
2. Immune cells called dendritic cells pick up the protein and carry it to nearby lymph nodes.
3. In the lymph nodes, dendritic cells show the protein to B cells.
4. B cells that recognize the protein multiply and start making antibodies (protective proteins).
5. Memory B cells are created — these cells remember the hepatitis B surface antigen for decades.
6. When real hepatitis B virus enters the body, memory B cells quickly make antibodies that stick to the virus and block it from infecting liver cells.

SAFETY WATCH

Soreness, redness, or swelling at the injection site for 1-2 days · Mild fever or tiredness for 1-2 days · Very rare: severe allergic reaction (hives, swelling of face or throat, trouble breathing, fast heartbeat) within minutes to hours — call 911 if this happens. · Very rare: Guillain-Barré syndrome (temporary nerve weakness) — reported in fewer than 1 in a million doses

Red flags

- Confusion, extreme sleepiness, or slurred speech (sign the liver is failing and toxins are reaching the brain)
- Vomiting blood or black, tarry stools (sign of bleeding from swollen veins in the esophagus or stomach)
- Severe belly pain with rapid swelling (could mean infection of fluid in the belly, called spontaneous bacterial peritonitis)
- Trouble breathing or very fast heart rate with yellowing of the skin and eyes (jaundice) (sign of liver failure affecting the whole body)
- New bruising, nosebleeds, or bleeding gums without injury (sign clotting factors are dangerously low)

Nursing priorities

1. Monitor for signs of hepatic encephalopathy every 4 to 6 hours: check orientation to person, place, and time; look for hand flapping tremor called asterixis; ask family if personality changed — The liver normally removes ammonia (a toxin) from blood. When the liver fails, ammonia builds up in the brain causing confusion, coma, and death. Early detection allows treatment with lactulose (a laxative that removes ammonia through stool) before brain damage occurs.
2. Check for bleeding every shift: inspect skin for new bruises or pinpoint bleeding spots (petechiae) (tiny red dots), test stool for blood, watch IV sites for oozing, measure any vomit or stool blood loss — The liver makes clotting factors (proteins that stop bleeding). A damaged liver cannot make enough, so patients bleed easily. The international normalized ratio or INR (a blood test measuring clotting) rises above 1.5 when the liver is failing. Portal high blood pressure (hypertension) also causes varices in the esophagus (food tube) that can bleed massively.
3. Give IV fluids at ordered rate if patient is vomiting or cannot drink, and measure urine output every hour; notify provider if urine drops below 30 milliliters per hour — Viral hepatitis causes nausea and vomiting, leading to dehydration (low body water). The kidneys need good blood flow to work. Dehydration plus liver failure can cause hepatorenal syndrome (kidney failure from liver disease). Maintaining hydration protects the kidneys and helps the liver heal.
4. Place patient on contact and standard precautions for HAV and HEV: use gloves and gown for all care, place sign on door, use dedicated equipment, wash hands with soap for 20 seconds after touching patient or environment — HAV and HEV live in stool and spread through the fecal-oral route (poop to mouth). Healthcare workers can carry the virus on hands to other patients. These precautions stop hospital spread. HBV, HCV, and HDV need standard precautions only because they spread through blood, not casual contact.
5. Hold all hepatotoxic medicines (drugs that harm the liver) until provider reviews: acetaminophen, statins, some antibiotics, herbal supplements; check medication reconciliation list with pharmacist — An inflamed liver cannot process medicines normally. Drugs build up to toxic levels or cause more liver cell death. The alanine aminotransferase or ALT (a liver enzyme in blood) and aspartate aminotransferase or AST (another liver enzyme) are already elevated from viral hepatitis. Adding hepatotoxic drugs increases risk of fulminant hepatic failure (sudden complete liver shutdown).

6. Provide small frequent meals high in calories and protein every 2 to 3 hours; avoid fatty foods; give anti-nausea medicine 30 minutes before meals as ordered — The liver stores glucose (blood sugar) as glycogen (a starch) and makes albumin (a protein). A sick liver cannot do this, so patients lose muscle and weight fast. Small meals are easier to tolerate with nausea. Adequate nutrition helps liver cells regenerate (regrow). However, if encephalopathy develops, protein must be restricted because the liver cannot process it.
7. Teach patient and family strict hand hygiene after bathroom use and before eating; explain the virus type, how it spreads, and that close contacts may need vaccination or immunoglobulin — HAV and HEV spread easily in households through shared bathrooms and kitchens. Post-exposure prevention (prophylaxis) (prevention after exposure) with hepatitis A vaccine or immune globulin (antibodies from donated blood) within 2 weeks can stop infection in contacts. For HBV, unvaccinated household and sexual contacts need hepatitis B immune globulin plus vaccine series immediately. Education stops community spread.
8. Schedule and ensure completion of follow-up lab work: repeat ALT, AST, bilirubin (a yellow waste product from red blood cells), INR, and viral load (amount of virus in blood) in 1 to 2 weeks as ordered — Most patients with HAV and HEV recover in 4 to 6 weeks. However, HBV and HCV can become chronic (permanent) infections. Tracking lab trends shows if the liver is healing or worsening. Rising bilirubin above 10 milligrams per deciliter or INR above 2 suggests acute liver failure and may need transplant evaluation. Viral load guides antiviral treatment decisions for HBV and HCV.

Sources

- AASLD Hepatitis B Guidance 2018
- AASLD-IDSA HCV Guidance 2023
- CDC Viral Hepatitis Surveillance and Prevention
- WHO Guidelines on Hepatitis B and C Testing and Treatment 2023

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