

Hepatitis B

Hepatitis B — the virus hides in liver cells; most adults clear it, but some carry it for life and face scarring or liver cancer if left untreated.

THE ONE-LINE TRUTH

Hepatitis B is a virus that attacks liver cells and spreads through blood, sex, or from mother to baby during birth.

The chain — how it unfolds

1. HBV enters the bloodstream through a contaminated needle, unprotected sex, or passage from mother to baby during birth — why: The virus travels in blood and binds to a receptor (NTCP, sodium taurocholate co-transporting polypeptide) on the hepatocyte surface, allowing it to slip inside the cell.
2. Inside the hepatocyte, HBV releases its DNA into the nucleus and hijacks the cell's copying machinery — why: The virus does not kill the cell directly; instead it forces the hepatocyte to churn out new viral DNA and coat proteins, turning one infected cell into a factory that can release thousands of new virus particles into the blood.
3. The immune system's cytotoxic T-cells (CD8+ T-lymphocytes) recognize viral proteins on the hepatocyte surface and attack — why: T-cells release perforin and granzymes that punch holes in the infected cell and trigger apoptosis (programmed cell death), which stops the virus from spreading but also destroys the hepatocyte; when millions of hepatocytes die at once, you get acute hepatitis with fatigue, nausea, dark urine, and yellowing of the skin and eyes (jaundice) (yellow skin and eyes from bilirubin overflow).
4. In 90–95% of adults, the immune system wins and clears every last virus particle within six months — why: Strong T-cell and antibody (anti-HBs) responses eliminate infected cells faster than HBV can spread, the liver regenerates, and you gain lifelong immunity — you will never get hepatitis B again.
5. In 5–10% of adults — and 90% of infants infected at birth — the immune system fails to clear the virus completely — why: Babies' immune systems are immature and tolerate the virus instead of attacking it; some adults have weaker T-cell responses or high viral loads that overwhelm defenses, so HBV hides in hepatocytes and keeps replicating at a low level for years or decades without symptoms (chronic hepatitis B).

6. Chronic low-grade inflammation triggers stellate cells to lay down collagen, turning soft liver tissue into stiff scar tissue (fibrosis) — why: Every time a hepatocyte dies, stellate cells try to patch the wound with collagen, but collagen does not filter toxins or make proteins — it just stiffens the liver; after twenty to thirty years of constant repair, fibrosis becomes cirrhosis, a point where the liver's architecture is so distorted that it cannot do its three main jobs anymore.
7. Cirrhosis leads to portal high blood pressure (hypertension), fluid buildup in the belly (ascites), varices, hepatic encephalopathy, and bleeding risks — why: Scar tissue blocks blood flow through the liver, raising pressure in the portal vein (portal high blood pressure (hypertension)); fluid leaks into the belly (ascites), alternate blood routes open in the esophagus and stomach (varices) that can rupture and bleed, ammonia reaches the brain and causes confusion (encephalopathy), and low clotting factors mean even small cuts bleed dangerously.
8. Hepatocytes under constant viral attack and inflammation accumulate DNA mutations and some turn into hepatocellular carcinoma (HCC) — why: HBV DNA can insert itself into the hepatocyte genome and scramble genes that normally stop cancer (like p53 and RB); chronic inflammation also produces free radicals that damage DNA, and regenerating hepatocytes dividing over and over to replace dead neighbors each risk a copying mistake — eventually one cell loses its brakes and grows into liver cancer, which is why untreated chronic hepatitis B is one of the top causes of HCC worldwide.

Spot it — what you see, and why

- No symptoms at all (most common in chronic infection): The virus quietly damages the liver over many years without triggering pain nerves or obvious signals, so most people feel fine even while the infection is active.
- Fatigue and feeling worn out: The liver isn't processing toxins and making energy molecules as well as it should, so the whole body runs low on fuel and feels tired.
- Loss of appetite and nausea: The inflamed liver releases signals that upset the stomach and turn off hunger, much like when you have the flu.
- Yellow skin and eyes (jaundice): The damaged liver can't clear bilirubin (a yellow waste from old red blood cells), so it builds up in the blood and stains the skin and whites of the eyes yellow.
- Dark urine that looks like tea or cola: Excess bilirubin spills into the urine and turns it dark brown, a sign that the liver isn't filtering waste properly.
- Pale or clay-colored stools: Bilirubin normally gives stool its brown color; when the liver can't send it into the gut, the stool turns very light or gray.

Red flags — act now

- Yellow skin or eyes (jaundice) — means the liver is struggling to clean the blood
- Confusion, extreme sleepiness, or changes in personality — toxins may be building up in the brain
- Easy bruising, bleeding gums, or vomiting blood — the liver isn't making clotting proteins, so bleeding won't stop
- Swollen belly with fluid (ascites) or swollen legs — the liver isn't making enough albumin to hold fluid in the blood vessels
- Severe fatigue with yellowing of the skin and eyes (jaundice) in someone who was recently exposed to the virus — may be acute hepatitis B, which can turn severe quickly

Treat it — in this order

1. Supportive care in acute Hepatitis B: IV fluids for dehydration, rest, nutrition, and close monitoring of mental status, INR, and bilirubin → Keeps the body stable while the immune system clears the virus on its own (which happens in over 95 percent of healthy adults with acute infection). Prevents dangerous dehydration, electrolyte swings, and catches early liver failure.
2. Antiviral therapy with entecavir or tenofovir (TDF or TAF) for chronic Hepatitis B when HBV DNA is above 2,000 IU/mL, ALT is elevated, and there is evidence of fibrosis or cirrhosis → Blocks the enzyme (reverse transcriptase) that Hepatitis B uses to copy its DNA inside liver cells. Viral load drops, liver inflammation calms, and the risk of cirrhosis and liver cancer falls over time.
3. HBIG (Hepatitis B immune globulin) plus the Hepatitis B vaccine series given to newborns of HBsAg-positive mothers within 12 hours of birth → HBIG gives the baby ready-made antibodies that attack the virus immediately. The vaccine trains the baby's own immune system to make lasting antibodies. Together they block vertical transmission (mother to baby during birth) in over 95 percent of cases.
4. Tenofovir (TDF or TAF) started in the third trimester (around 28 to 32 weeks) for pregnant women with HBV DNA above 200,000 IU/mL → Lowers the mother's viral load before delivery, cutting the amount of virus the baby is exposed to during birth and reducing the risk of transmission even when HBIG and vaccine are given.
5. Hepatocellular carcinoma screening with abdominal ultrasound (and sometimes AFP) every 6 months in patients with cirrhosis or chronic Hepatitis B in high-risk groups (Asian men over 40, Asian women over 50, Africans over 20, anyone with a family history of liver cancer) → Catches liver cancer early, when tumors are small and can be removed surgically, ablated, or treated with transplant. Early detection saves lives.

Your top priorities at the bedside

1. Assess mental status every 4 hours using Glasgow Coma Scale and check for asterixis (hand-flapping tremor when arms extended) — why: Hepatic encephalopathy develops when ammonia and toxins build up because the damaged liver cannot filter blood; early detection allows prompt lactulose treatment to prevent coma
2. Monitor coagulation labs (PT/INR and platelet count) and inspect skin, gums, and all puncture sites for bleeding every shift — why: The liver makes clotting factors (proteins that stop bleeding); severe hepatitis reduces these factors and raises bleeding risk, requiring vitamin K or fresh frozen plasma if INR is very high
3. Maintain IV hydration with dextrose-containing fluids and monitor blood glucose every 6 hours — why: The failing liver cannot store glycogen (sugar storage form) or make new glucose, causing low blood sugar (hypoglycemia) (low blood sugar) that can lead to seizures or brain damage if untreated