

Hepatitis C

Hepatitis C hides in your liver cells for decades, slowly turning healthy tissue into scar — but today's antiviral pills can cure it before that happens.

THE ONE-LINE TRUTH

Hepatitis C is a virus that spreads through blood and quietly damages the liver over many years.

The chain — how it unfolds

1. Hepatitis C virus enters the bloodstream through needle-sharing, unsafe tattoos, contaminated medical equipment, or blood transfusions before 1992. — why: The virus is fragile outside the body but survives in tiny amounts of blood on needles or equipment. Even a microscopic trace is enough to infect someone when that blood enters their vein. Sexual transmission and mother-to-baby spread can happen but are less common.
2. The virus binds to receptors on the surface of hepatocytes and slips inside. — why: Hepatitis C uses the CD81 receptor and other proteins as doorways into liver cells. Once inside, it releases its RNA genome and hijacks the cell's ribosomes to make thousands of new virus particles. The infected cell becomes a virus factory.
3. Your immune system detects infected liver cells and sends cytotoxic T cells to destroy them. — why: T cells recognize viral proteins displayed on the infected cell's surface and punch holes in the cell membrane, killing it. This immune attack releases inflammatory chemicals (cytokines) that call in more immune cells, creating chronic inflammation. The goal is to stop the virus, but hepatitis C mutates so quickly that the immune system can't catch up — the infection becomes chronic in about 75 to 85% of people.
4. Dead and dying hepatocytes trigger stellate cells to lay down collagen, forming scar tissue (fibrosis). — why: The liver tries to patch each injury with scar. At first, this holds the organ together. But the immune battle never stops, so new scar keeps piling up. Over 20 to 30 years, fibrosis advances from mild (F1) to moderate (F2), severe (F3), and finally cirrhosis (F4), where scar tissue replaces most healthy liver and blocks blood flow.

5. Cirrhosis raises pressure in the portal vein (portal high blood pressure (hypertension)), forcing blood to detour through fragile collateral veins in the esophagus and stomach. — why: Scar tissue is stiff and dense, so blood can't flow through the liver's normal channels. Pressure in the portal vein shoots up (normal is 5–10 mmHg; portal high blood pressure (hypertension) is ≥ 10 mmHg). Blood reroutes through esophageal and gastric varices — thin-walled veins that can swell, rupture, and bleed massively. The spleen also swells and traps platelets, raising bleeding risk further.
6. The cirrhotic liver can't make enough albumin or clotting factors, and can't detoxify ammonia. — why: With most hepatocytes replaced by scar, synthetic function drops. Low albumin lets fluid leak into the belly (ascites) and legs (edema). Low clotting factors cause easy bruising and prolonged bleeding (high INR). Ammonia — normally broken down into urea — accumulates in the blood, crosses into the brain, and causes confusion, drowsiness, and coma (hepatic encephalopathy). This late stage is decompensated cirrhosis.
7. Chronic inflammation and repeated cell turnover raise the risk of liver cancer (hepatocellular carcinoma). — why: Every time a hepatocyte divides to replace a dead neighbor, there's a chance for a DNA copying mistake. In a cirrhotic liver, cells divide constantly, and inflammatory chemicals damage DNA. Over decades, mutations pile up until a cell escapes normal growth control and becomes cancer. Hepatocellular carcinoma risk is 1 to 4% per year once cirrhosis is established.
8. Direct-acting antiviral pills (DAAs) can cure hepatitis C in 8 to 12 weeks, stopping this entire cascade. — why: DAAs block the viral proteins that copy RNA and assemble new virus particles. Without new virus being made, viral load drops to undetectable in weeks. Once the virus is gone (sustained virologic response, or SVR), immune attack stops, inflammation cools, and fibrosis progression halts. Early-stage fibrosis can even regress. But advanced cirrhosis and existing cancer won't reverse — so the earlier you treat, the more liver you save.

Spot it — what you see, and why

- No symptoms at all for years (most common): The hepatitis C virus (the germ) quietly damages liver cells bit by bit, but the liver keeps working well enough that you feel fine — sometimes for 20 or 30 years — even while scar tissue is slowly building up inside.
- Feeling tired all the time: The liver cleans the blood and makes energy fuel for the body. When the virus damages liver cells, waste builds up in the blood and less fuel gets made, so you feel worn out even after rest.
- Belly pain or feeling full on the right side under the ribs: The liver sits on the right side under your ribs. When the virus causes swelling (inflammation) inside the liver, the covering around it stretches and sends pain signals.

- Yellow skin and eyes (jaundice): The liver normally breaks down bilirubin (a yellow waste from old red blood cells). When liver cells are badly damaged, bilirubin spills into the blood and stains the skin and the whites of the eyes yellow.
- Dark urine (tea-colored or cola-colored): When bilirubin builds up in the blood because the liver cannot clear it, the kidneys filter it out into the urine, turning it dark brown or orange.
- Itchy skin all over: Bile salts (chemicals the liver makes to digest fat) build up under the skin when the liver is damaged, and they tickle nerve endings, causing an itch that feels like it is coming from deep inside.

Red flags — act now

- Yellow skin or eyes (jaundice) — the liver may be failing
- Swollen belly that keeps growing — fluid is leaking out because scar tissue is blocking blood flow
- Confusion, strange sleepiness, or trouble waking up — ammonia is poisoning the brain
- Vomiting blood or black tarry stools — a vein in the food pipe has burst because of high pressure from liver scarring
- Easy bruising or bleeding that does not stop — the liver cannot make clotting proteins anymore

Treat it — in this order

1. Sofosbuvir-velpatasvir or glecaprevir-pibrentasvir (pangenotypic direct-acting antivirals) for 8 to 12 weeks → These pills block the enzymes hepatitis C virus needs to copy its RNA and build new virus particles inside liver cells. Over 95 percent of people reach sustained virologic response (SVR)—undetectable virus 12 weeks after finishing treatment—which is a cure. The virus dies, the liver stops being attacked, and inflammation drops to zero.
2. Hepatitis A and hepatitis B vaccination → The vaccines teach your immune system to recognize and kill hepatitis A virus (spread by contaminated food or water) and hepatitis B virus (spread by blood or sex) before they can infect your liver. If you catch a second hepatitis virus on top of hepatitis C, the combined damage can cause sudden liver failure. Vaccination blocks that second hit.
3. Ultrasound screening for hepatocellular carcinoma every 6 months (if cirrhosis is present) → Hepatitis C cirrhosis raises liver cancer risk to 3 to 5 percent per year. Ultrasound every 6 months catches tumors when they are smaller than 2 cm. Small tumors can be removed with surgery, burned with radiofrequency ablation, or treated with a liver transplant. Early detection saves lives because big tumors cannot be cured.

4. Stop all alcohol use completely and permanently → Alcohol is broken down in the liver into acetaldehyde (a toxin that kills hepatocytes directly). When you drink alcohol on top of hepatitis C, you add a second source of liver cell death. Stopping alcohol prevents that second hit and slows the progression from inflammation to cirrhosis by 50 percent or more.
5. Test for HIV and treat with antiretroviral therapy if positive → HIV weakens CD4 T cells (the immune cells that coordinate your defense against viruses). When HIV and hepatitis C infect the same person (coinfection), hepatitis C scarring happens twice as fast because the immune system cannot control the virus. Treating HIV with antiretroviral therapy restores CD4 counts and slows hepatitis C progression.

Your top priorities at the bedside

1. Check for signs of bleeding including vomit that looks like coffee grounds, black tarry stools, bruising, or bleeding gums — why: Cirrhosis stops the liver from making clotting factors (proteins that stop bleeding), and portal high blood pressure (hypertension) (high pressure in liver blood vessels) causes varices (swollen veins) in the esophagus that can burst and bleed to death
2. Assess mental status every shift for confusion, sleepiness, or personality changes — why: Hepatic encephalopathy happens when the damaged liver cannot remove ammonia from the blood, and ammonia crosses into the brain causing confusion, coma, and death if untreated
3. Measure abdominal girth daily at the same spot and weigh the patient every day at the same time — why: Cirrhosis causes fluid buildup in the belly (ascites) when the liver cannot make albumin to hold fluid in blood vessels, and tracking belly size and weight shows if fluid is building up and diuretics (water pills) are working