

Hepatitis C

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THE STORY IN ONE SENTENCE

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“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.”

Normal anatomy — the body's job

The liver is a chemical factory in the upper-right belly that cleans toxins from your blood, makes proteins to stop bleeding, stores sugar for energy, and breaks down old red blood cells. Hepatocytes (liver cells) do these jobs around the clock and are built to resist most viruses.

BODY'S JOB

Hepatocytes filter every drop of blood coming from your intestines, pulling out nutrients to store and poisons to neutralize. They make albumin (a protein that keeps fluid inside your blood vessels), clotting factors (so you don't bleed too much), and bile (a green liquid that helps digest fat). A healthy liver can lose half its cells and still keep you alive because the remaining cells work overtime and new ones grow back.

1. Blood flows from your intestines through the portal vein into the liver, carrying nutrients and also any toxins or germs you swallowed.
2. Inside the liver, blood moves slowly through tiny channels lined with hepatocytes, so each cell can reach into the blood and grab what it needs to process.
3. Hepatocytes pull out sugar (glucose) and store it as glycogen, break down ammonia (a poison from protein digestion) into urea (which your kidneys can safely remove), and neutralize alcohol and drugs.
4. They release albumin and clotting factors into the bloodstream, keeping fluid balanced and bleeding under control.

5. Cleaned blood leaves the liver through the hepatic vein and flows back to the heart, while bile drains through tiny ducts into the intestine to help digest fat.
6. Healthy hepatocytes resist most viruses because their membranes are hard to crack and their internal defenses chop up viral genes quickly.

What breaks

Hepatitis C virus (HCV) sneaks into liver cells through contaminated blood. Once inside, it hijacks the cell's machinery to make thousands of new virus copies. Your immune system sees the infected cells and attacks them, but it can't kill the virus completely. This slow battle destroys liver cells one by one, and the liver tries to patch the damage with scar tissue. Over many years, scar tissue piles up, stiffens the liver (cirrhosis), blocks blood flow, and raises the risk of liver cancer.

CLINICAL TERM

Chronic hepatitis C infection causes persistent hepatocyte injury and immune-mediated inflammation, leading to progressive hepatic fibrosis, cirrhosis, portal high blood pressure (hypertension), hepatocellular carcinoma, and decompensated liver failure.

How the body compensates

For years — even decades — the liver quietly absorbs damage, and most people feel completely normal. Blood tests might be normal or only mildly off. This silent phase is dangerous because there's no alarm bell; by the time symptoms appear, cirrhosis or cancer may already be present. The key is to catch hepatitis C early with screening, treat it with antiviral pills, and monitor for fibrosis before the liver reaches the point of no return.

1. {'what': 'Hepatocyte regeneration', 'how': 'Healthy liver cells multiply to replace dead ones, so liver size and basic function stay near-normal for years. This is why blood tests (ALT, AST, bilirubin, albumin) can look okay even when viral load is high and inflammation is simmering.'}
2. {'what': 'Gradual fibrosis accumulation', 'how': '"Scar tissue builds slowly — often taking 20 to 30 years to progress from mild fibrosis (stage F1) to cirrhosis (stage F4). The liver keeps working during this climb, so symptoms like fatigue, yellowing of the skin and eyes (jaundice), or confusion don't appear until late. This silent march is why screening people at risk (born 1945–1965, history of injection drug use, recipients of blood products before 1992, people on hemodialysis, HIV-positive individuals) is guideline-recommended."}

3. {'what': 'Collateral circulation', 'how': 'When portal vein pressure rises (portal high blood pressure (hypertension)), blood reroutes through smaller veins in the esophagus, stomach, and abdominal wall. This keeps blood flowing back to the heart and buys time, but those collateral veins are thin-walled and can rupture, causing life-threatening bleeding (variceal bleeding (hemorrhage)).'}
4. {'what': 'Splenic sequestration', 'how': 'The spleen swells (splenomegaly) because backed-up blood pools there. It traps platelets and white blood cells, so platelet count drops (thrombocytopenia) and infection risk rises. This is the body's overflow reservoir, not a solution — it just spreads the problem.'}
5. {'what': 'The danger of silent stability', 'how': 'A person can feel fine, work full-time, and have near-normal labs while fibrosis silently climbs from F2 to F3. Once cirrhosis (F4) and decompensation hit — fluid buildup in the belly (ascites), variceal bleed, encephalopathy — the window to reverse damage is closed. Antiviral cure at that point stops further harm but can't undo advanced scarring. This is why trend matters more than one snapshot: rising ALT, falling platelet count, or increasing stiffness on FibroScan are red flags even when the patient feels well.'}
6. {'what': 'Watch for the tipping point', 'how': 'Decompensation events — first variceal bleed, new fluid buildup in the belly (ascites), or hepatic encephalopathy — mark the transition from compensated to decompensated cirrhosis. Median survival without transplant drops to 2 years. Hepatocellular carcinoma can emerge suddenly. Screen with ultrasound and AFP (alpha-fetoprotein) every 6 months once cirrhosis is confirmed, and refer for transplant evaluation early.'}

Domino effect

1. Hepatitis C virus enters the bloodstream through needle-sharing, unsafe tattoos, contaminated medical equipment, or blood transfusions before 1992. — 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. — 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.
— 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). — 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. — 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. — 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). — 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. — 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.

Symptoms · what the patient feels, 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.
- Swollen belly (ascites): Scar tissue in the liver (cirrhosis) blocks blood flow through the liver like a clogged pipe, so pressure rises in the veins and pushes fluid out into the belly. The liver also stops making albumin (a sticky protein that holds water in the blood), so water leaks out even faster.
- Easy bruising and bleeding: The liver makes clotting factors (proteins that plug holes in blood vessels). When the liver is severely scarred, it cannot make enough clotting factors, so even a small bump causes a bruise or a nosebleed that is hard to stop.
- Confusion or sleepiness (hepatic encephalopathy): A badly damaged liver cannot clean ammonia (a toxic waste from digesting protein) out of the blood. Ammonia travels to the brain and poisons the nerve cells, making thinking slow and foggy, and sometimes causing deep sleepiness or strange behavior.

Focused exam

- Press gently on the right upper belly under the ribs to check liver size and tenderness: Hepatitis C virus grows inside hepatocytes (liver cells that clean your blood and make proteins). When the virus multiplies, the liver swells, making it bigger and sore to the touch. A tender, enlarged liver tells you active inflammation is happening right now.

- Look at the whites of the eyes and the skin for yellow color (jaundice): When hepatitis C damages enough liver cells, they cannot move bilirubin (a yellow waste chemical from old red blood cells) into bile. Bilirubin backs up into the blood and turns the skin and eyes yellow. Yellowing of the skin and eyes (Jaundice) means the liver is struggling to do its job and the damage is real, not just a quiet infection.
- Feel the liver edge for a hard, bumpy, or nodular texture: After 20 to 30 years of hepatitis C, the liver tries to heal itself over and over. Healing leaves scars (collagen fibers that are stiff). When scars replace most of the liver, you feel a hard edge. A hard, bumpy liver means cirrhosis (heavy scarring), which raises the risk of liver cancer and liver failure.
- Look for dilated veins on the belly and chest (caput medusae and venous collaterals): Hepatitis C scarring (cirrhosis) squeezes the portal vein (the main blood highway into the liver). Blood cannot flow through, so it backs up and finds new paths through small veins in the belly wall and chest. These veins swell and show through the skin. This is portal high blood pressure (hypertension)—high pressure in the liver's blood system—and it signals advanced disease.
- Check for confusion, slow answers, or sleepiness (hepatic encephalopathy): A healthy liver cleans ammonia (a poison made when your gut bacteria break down protein) out of your blood. When hepatitis C causes severe cirrhosis, ammonia builds up and crosses into the brain, poisoning neurons. The person becomes confused or sleepy. This is a medical emergency because ammonia can cause coma.
- Look for spider angiomas (tiny red spider-shaped blood vessels) on the chest, face, or arms: When hepatitis C causes cirrhosis, the liver cannot break down estrogen (a hormone) as well. Extra estrogen makes tiny arteries in the skin branch out and swell, forming red spider shapes. Spider angiomas are a sign of chronic liver damage, not just infection.

Diagnostics · every test answers a question

- Anti-HCV antibody (hepatitis C antibody test) → Has this person ever been exposed to hepatitis C virus?
- HCV RNA (hepatitis C viral load by PCR) → Is the hepatitis C virus actively multiplying in the liver right now?
- ALT and AST (alanine aminotransferase and aspartate aminotransferase) → Are liver cells dying right now?
- Hepatitis C genotype test → Which strain of hepatitis C does this person have?
- FibroScan or liver ultrasound with elastography → How much scarring (fibrosis) has hepatitis C caused in the liver?
- Platelet count → Is the liver so scarred that blood flow is backing up and trapping platelets in the spleen?

- Albumin and INR (international normalized ratio) → Can the liver still make the proteins it is supposed to make?
- Abdominal ultrasound with Doppler (if cirrhosis is present) → Is there a liver tumor, fluid in the belly, or reversed blood flow in the portal vein?

Treatment logic

- 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.
- 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.
- 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.
- 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.
- 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.
- Avoid hepatotoxic medications (acetaminophen > 2 grams per day, NSAIDs, certain antibiotics, and supplements) → When the liver is already inflamed from hepatitis C, adding drugs that stress liver cells (like high-dose acetaminophen, which makes a toxic byproduct called NAPQI, or NSAIDs like ibuprofen, which reduce blood flow to the liver) can tip the liver into failure. Avoiding these drugs protects the remaining healthy liver tissue.

Medications

Sofosbuvir-velpatasvir · Direct-acting antiviral (DAA) combination

This pair stops the hepatitis C virus from copying itself inside liver cells. Sofosbuvir blocks the NS5B polymerase (the virus's copy machine that builds new viral RNA), and velpatasvir blocks the NS5A protein (a helper the virus needs to assemble and release new copies). Without these tools, the virus cannot multiply, so your immune system clears out the infection over 8 to 12 weeks.

1. Sofosbuvir enters the liver cell and is turned into an active form that mimics one of the building blocks of viral RNA.
2. When the NS5B polymerase (the virus's copy enzyme) tries to use it, the chain stops growing—like a broken link in a chain—so no new virus RNA is made.
3. Velpatasvir locks onto the NS5A protein, which normally helps package and release new virus particles, freezing that step.
4. With both copying and assembly blocked, the virus cannot spread to new cells.
5. Over weeks, the amount of virus in your blood drops to zero, and the infection is cured—your body clears the last traces and the liver can start healing.

SAFETY WATCH

Tell your doctor about ALL other medicines and supplements; velpatasvir levels drop dangerously if you take antacids, proton-pump inhibitors (like omeprazole), or the tuberculosis drug rifampin at the wrong time. · If you take amiodarone (a heart-rhythm drug), sofosbuvir can cause a severe slow heartbeat (bradycardia)—this combination is usually avoided. · Rarely, in people with advanced cirrhosis, liver function can worsen during treatment; your doctor will check labs before and during therapy. · No major food restrictions, but take the pill at the same time every day with or without food to keep steady levels.

Glecaprevir-pibrentasvir · Direct-acting antiviral (DAA) combination

This pair also stops hepatitis C from reproducing, using a slightly different attack. Glecaprevir blocks the NS3/4A protease (a pair of scissors the virus uses to cut its long protein into working pieces), and pibrentasvir blocks the NS5A protein (the assembly helper). With both cut off, the virus cannot mature or spread, and the infection clears in 8 to 16 weeks depending on your situation.

1. Glecaprevir jams the NS3/4A protease, the enzyme that acts like molecular scissors to chop the virus's long protein chain into functional pieces.

2. Without working scissors, the virus cannot assemble the tools it needs to copy itself or build new particles.
3. Pibrentasvir binds to the NS5A protein, which normally helps the virus package its RNA and bud out of the cell, stopping that step cold.
4. With both assembly and maturation blocked, no infectious virus is released.
5. The immune system mops up the remaining virus, and blood levels fall to undetectable—meaning cured—within weeks.

SAFETY WATCH

Do NOT use if you have severe cirrhosis (Child-Pugh class B or C) because drug levels can build up and harm the liver further. · Tell your doctor if you take atazanavir or rifampin—these drugs interact and can raise or lower glecaprevir levels dangerously. · Take with food; absorption is much better with a meal or snack. · Mild headache, fatigue, or nausea can happen in the first weeks but usually fades. · Rarely, itching (pruritus) can occur; let your doctor know if it becomes bothersome.

Hepatitis A vaccine · Inactivated (killed) viral vaccine

This shot trains your immune system to recognize and block the hepatitis A virus before it can infect your liver. It contains pieces of killed virus—completely unable to cause disease—that teach your B cells (immune cells that make antibodies) to build a memory. If the real hepatitis A virus enters your body later, those memory cells wake up fast and flood your blood with antibodies that trap and destroy the virus before it harms you. Because people with hepatitis C already have one liver infection, adding hepatitis A on top can cause sudden, dangerous liver failure—so this vaccine is essential protection.

1. The vaccine delivers inactivated hepatitis A virus particles into your muscle.
2. Dendritic cells (immune scouts) grab the particles and carry them to nearby lymph nodes.
3. In the lymph nodes, B cells inspect the viral pieces, learn their shape, and start making antibodies (proteins that lock onto that virus).
4. Some B cells become memory cells that stay in your body for decades, ready to respond instantly.
5. If hepatitis A virus enters your body in the future, memory B cells multiply in hours and release a flood of antibodies that glue to the virus and mark it for destruction.
6. The virus is cleared before it can infect liver cells and cause hepatitis.

SAFETY WATCH

Two doses are needed, six to twelve months apart, for long-lasting protection. · Soreness, redness, or swelling at the injection site is common and fades in a day or two. · Mild fever, tiredness, or headache can happen; rest and drink fluids. · Severe allergic reactions (anaphylaxis) are extremely rare—stay in the clinic for fifteen minutes after the shot just in case. · Safe during pregnancy if needed, and safe to give with other vaccines at the same visit.

Hepatitis B vaccine · Recombinant subunit vaccine

This shot contains a lab-made piece of the hepatitis B virus's outer coat (the surface antigen), which cannot cause infection but trains your immune system to recognize and attack the real virus. Your B cells learn to make antibodies against that coat, so if hepatitis B virus enters your bloodstream later, the antibodies immediately lock onto it and stop it from infecting liver cells. People with hepatitis C are at higher risk for hepatitis B because both spread through blood and other body fluids, and catching both at once (coinfection) can lead to severe liver damage, so this vaccine is strongly recommended.

1. The vaccine delivers a harmless piece of the hepatitis B virus surface protein into your muscle.
2. Dendritic cells capture the protein and present it to B cells in nearby lymph nodes.
3. B cells recognize the foreign protein, begin making antibodies, and turn into memory cells that persist for decades.
4. When real hepatitis B virus enters your body, memory B cells activate in hours and produce massive amounts of antibody.
5. The antibodies bind to the virus's surface, blocking it from attaching to and entering liver cells.
6. The tagged virus is destroyed by other immune cells, preventing infection and liver damage.

SAFETY WATCH

Three doses are needed: the first shot, then one month later, then six months after the first—all three are required for full, long-lasting protection. · Soreness, redness, or mild swelling at the injection site is common and goes away in a day or two. · Low-grade fever, tiredness, or mild body aches can occur; treat with rest and fluids. · Severe allergic reactions are extremely rare; you will be watched for fifteen minutes after the shot. · Safe during pregnancy and can be given at the same visit as other vaccines, including hepatitis A.

Red flags

- 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

Nursing priorities

1. Check for signs of bleeding including vomit that looks like coffee grounds, black tarry stools, bruising, or bleeding gums — 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 — 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 — 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
4. Screen for alcohol use and connect the patient with addiction counseling or support groups — Alcohol directly poisons liver cells and speeds up fibrosis (scarring), and continuing to drink while infected with Hepatitis C increases cirrhosis risk by ten times
5. Teach the patient to take direct-acting antivirals at the same time every day without missing doses — Missing doses lets the virus multiply and develop resistance (ability to survive the medicine), which causes treatment failure and limits future cure options
6. Educate the patient never to share razors, toothbrushes, nail clippers, or any item that might have blood on it — Hepatitis C spreads through blood-to-blood contact, and even tiny amounts of dried blood on shared items can carry live virus for up to three weeks
7. Verify the patient received Hepatitis A and Hepatitis B vaccines or check immunity with blood tests — Getting a second liver infection on top of Hepatitis C causes fulminant hepatitis (sudden total liver failure) and greatly increases the risk of dying from liver disease

8. Schedule six-month ultrasound surveillance if cirrhosis is present and explain why screening for liver cancer is lifelong — Cirrhosis increases the risk of hepatocellular carcinoma (liver cancer) by one hundred times, and ultrasound every six months catches tumors early when transplant or surgery can still cure the cancer

Sources

- AASLD/IDSA HCV Guidance 2024
- CDC Hepatitis C Guidelines
- WHO Hepatitis C Fact Sheet
- U.S. Preventive Services Task Force (USPSTF) HCV Screening 2020

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