

Hepatitis B

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THE STORY IN ONE SENTENCE

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“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.”

Normal anatomy — the body's job

Your liver is a dark-red, football-sized organ tucked under your right ribs. It is made of millions of tiny cells called hepatocytes, and these cells do three big jobs every minute of every day: they filter toxins out of your blood (like ammonia from digesting protein), they make proteins that stop bleeding (clotting factors), and they store sugar (glycogen) to fuel your body between meals. Hepatocytes are tough — if some die, the liver can grow new ones to replace them, as long as the damage is not constant.

BODY'S JOB

Hepatocytes clean your blood, build the proteins that stop cuts from bleeding forever, and keep a sugar reserve so your brain and muscles have fuel even when you have not eaten. When hepatocytes are healthy, you stay healthy.

1. Blood flows into the liver from two pipes: the hepatic artery (carrying oxygen-rich blood from the heart) and the portal vein (carrying nutrient-rich blood from the intestines).
2. Inside the liver, blood moves through tiny channels lined with hepatocytes, which pull out toxins (especially ammonia), package waste into bile, and release fresh clotting factors and albumin (a protein that holds water in your blood vessels) back into the bloodstream.
3. Clean blood leaves the liver through the hepatic veins and flows back to the heart, while bile drips into tiny tubes that join into the bile duct and empty into the small intestine to help digest fat.

4. If hepatocytes die faster than the liver can replace them, scar tissue fills the gaps, blood flow gets blocked, and all three jobs — detoxifying, clotting, and storing sugar — start to fail.

What breaks

Hepatitis B virus (HBV) sneaks into a hepatocyte, takes over the cell's machinery, and forces it to make millions of new virus copies. Your immune system's T-cells (the soldiers that hunt infected cells) see the virus flag on the hepatocyte surface and kill the infected cell to stop the virus from spreading. In most adults, T-cells clear every infected cell within three to six months and the liver heals completely. But in some people — especially babies and young children whose immune systems are still learning, and some adults with weaker immune defenses — the T-cells cannot finish the job, so the virus hides in hepatocytes for years. That low-level infection keeps triggering inflammation, and chronic inflammation slowly turns liver tissue into stiff scar tissue (fibrosis). Once enough scar builds up, you have cirrhosis, and a cirrhotic liver cannot do its three main jobs anymore. Worse, hepatocytes that keep dividing under constant viral attack and inflammation pile up DNA mistakes, and some of those mistakes turn into hepatocellular carcinoma (liver cancer).

CLINICAL TERM

Chronic hepatitis B infection with immune-mediated hepatocyte injury, progressive fibrosis, and risk of cirrhosis and hepatocellular carcinoma.

How the body compensates

For months or even years, the liver works overtime to keep you feeling normal even as hepatitis B quietly damages cells. The remaining healthy hepatocytes pick up the slack, and you might have no symptoms at all. But once cirrhosis sets in, the liver cannot compensate anymore — toxins build up, bleeding risks rise, and fluid leaks into the belly (ascites). By the time symptoms appear, serious damage is already done, so the key is to catch chronic hepatitis B early with blood tests and start antiviral treatment before cirrhosis or cancer develops.

1. {'step': 'In acute infection, the immune response is strong and fast', 'why': 'Most healthy adults mount a vigorous T-cell and antibody response that clears HBV within three to six months; during that time you might feel tired, lose your appetite, or turn yellow (jaundice) as bilirubin spills into the blood, but the liver regenerates and returns to normal once the virus is gone.'}

2. {'step': 'In chronic infection, the liver silently compensates for years', 'why': 'The virus replicates at a low level, killing hepatocytes slowly; the remaining healthy cells work harder to detoxify blood, make clotting factors, and store sugar, so labs might show only mildly elevated ALT and you feel fine — this is the dangerous window because people skip follow-up and miss the chance to start antivirals early.'}
3. {'step': 'Stellate cells try to patch damage but create scar tissue instead', 'why': 'Every time a hepatocyte dies, stellate cells (normally quiet support cells) wake up and deposit collagen to 'repair' the wound, but collagen is stiff and does not do the liver's jobs, so over time the liver becomes a hard, scarred organ that cannot filter toxins, make proteins, or handle blood flow.'}
4. {'step': 'Portal high blood pressure (hypertension) develops as scar blocks blood flow', 'why': 'Blood from the intestines flows into the liver through the portal vein, but scar tissue acts like a dam, raising pressure upstream; the body opens alternate routes (varices in the esophagus and stomach) that can rupture and bleed, and fluid leaks out of blood vessels into the belly (ascites) because pressure is too high and albumin is too low to hold water inside vessels.'}
5. {'step': 'The liver loses its ability to detoxify ammonia', 'why': 'Healthy hepatocytes convert ammonia (from protein digestion) into urea, which the kidneys flush out; when too many hepatocytes are dead or bypassed by shunts, ammonia reaches the brain and causes confusion, sleepiness, or even coma (hepatic encephalopathy).'}}
6. {'step': 'Clotting factors drop and bleeding risk rises', 'why': 'The liver makes factors II, VII, IX, and X (the proteins that form blood clots); cirrhotic livers cannot keep up, so the INR (a clotting test) rises, and even small cuts or procedures can cause dangerous bleeding.'}
7. {'step': 'Hepatocellular carcinoma can emerge silently', 'why': 'Chronic inflammation and viral DNA integration drive some regenerating hepatocytes to become cancerous; HCC often grows without symptoms until it is large or has spread, which is why guidelines say to screen chronic hepatitis B patients with ultrasound and AFP (a tumor marker) every six months.'}

Domino effect

1. HBV enters the bloodstream through a contaminated needle, unprotected sex, or passage from mother to baby during birth — 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 — 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 — 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 — 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 — 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) — 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 — 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) — 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.

Symptoms · what the patient feels, 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.
- Pain or fullness in the upper right belly: The liver sits under the right ribs and can swell when inflamed, stretching its outer capsule and causing a dull ache or pressure.
- Joint pain and muscle aches: The immune system's fight against the virus releases inflammatory signals (cytokines) that travel through the bloodstream and make joints and muscles sore.
- Confusion or sleepiness that gets worse over hours or days: If the liver is severely damaged, it can't clear ammonia and other toxins from the blood; these build up and fog the brain, a dangerous condition called hepatic encephalopathy.

Focused exam

- Yellowing of the skin and eyes (Jaundice) (yellowing of the skin and the whites of the eyes): The liver breaks down a yellow waste called bilirubin and sends it out of the body. When Hepatitis B damages liver cells, bilirubin piles up in the blood and stains the skin yellow. Yellowing of the skin and eyes (Jaundice) tells you the liver is hurt and cannot do its cleanup job.
- Right upper belly tenderness when you press below the ribs: The liver sits tucked under the right ribs. When Hepatitis B infects liver cells, they swell and the whole organ gets puffy and sore. Pressing that spot tells you the infection is active and the liver is inflamed.
- An enlarged liver (Hepatomegaly) (a liver that feels bigger than normal when you press on the belly or see it on ultrasound): Infected liver cells swell as the immune system fights the virus. A bigger liver means active Hepatitis B or long-term damage piling up inside.

- Spider angiomas (tiny red spider-web patterns of blood vessels on the skin) or palmar redness (erythema) (red palms): A damaged liver cannot clear certain hormones from the blood. These hormones make the body grow extra, fragile blood vessels in the skin. Seeing these signs tells you Hepatitis B has been damaging the liver for a long time and may have caused cirrhosis (deep scarring).
- Asterix (a flapping shake in the hands when the patient holds both arms out straight): When the liver fails, it cannot clean ammonia out of the blood. Ammonia is a poison that reaches the brain and scrambles its signals, causing the shaking. This is a red flag that Hepatitis B has pushed the liver into failure.
- Fluid buildup in the belly (Ascites) (a swollen belly full of fluid): When the liver scars deeply, blood cannot flow through it smoothly. Pressure backs up in the veins and forces water out into the belly. Fluid buildup in the belly (Ascites) means Hepatitis B has caused cirrhosis and the liver is losing the ability to keep the body balanced.

Diagnostics · every test answers a question

- HBsAg (Hepatitis B surface antigen) → Is the Hepatitis B virus in the body right now?
- Anti-HBc IgM (antibody to Hepatitis B core antigen, IgM type) → Is this a brand-new infection or an old one that never cleared?
- HBeAg (Hepatitis B e antigen) and HBV DNA level (viral load) → How active is the virus, and how much is it copying itself inside liver cells?
- ALT (alanine aminotransferase) and AST (aspartate aminotransferase) → How badly are liver cells breaking apart?
- INR (international normalized ratio) or PT (prothrombin time) → Can the liver still make the proteins that stop bleeding?
- Liver elastography (FibroScan) or liver biopsy → How much scarring (fibrosis) has piled up in the liver?
- AFP (alpha-fetoprotein) and abdominal ultrasound every 6 months in chronic Hepatitis B with cirrhosis or high-risk groups → Is liver cancer (hepatocellular carcinoma) starting to grow?

Treatment logic

- 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.

- 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.
- 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.
- 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.
- 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.
- Hepatitis B vaccination for all close contacts, household members, sexual partners, and healthcare workers exposed to the patient → Primes their immune systems to make antibodies against Hepatitis B surface antigen. If they are later exposed to the virus through blood or body fluids, their immune system destroys it before infection takes hold.

Medications

Tenofovir (TDF or TAF) · Nucleotide reverse transcriptase inhibitor (antiviral)

Tenofovir blocks the virus's copy machine. Hepatitis B hijacks liver cells and uses an enzyme called reverse transcriptase (a molecular photocopier) to make new copies of itself. Tenofovir slips into that copier and jams it, so the virus can't multiply. Over time, this lowers the viral load (the number of virus particles in the blood) and lets the liver start to heal.

1. The hepatitis B virus enters a liver cell and releases its DNA.
2. The virus uses reverse transcriptase (its copying enzyme) to make new viral DNA from an RNA template.
3. Tenofovir pretends to be a normal building block (nucleotide) that the enzyme needs.
4. When reverse transcriptase picks up tenofovir, it gets stuck and can't add any more pieces—like a broken chain.

5. Without working copies, the virus can't make new virus particles, so viral load drops.
6. The liver inflammation (hepatitis) calms down because fewer cells are being infected and destroyed.

SAFETY WATCH

Kidney function: Tenofovir can stress the kidneys over time, so a doctor checks blood creatinine and urine protein every 3–6 months. · Bone density: Long-term use can weaken bones slightly; calcium and vitamin D are recommended, and bone scans may be needed in older adults. · Lactic acidosis (acid from oxygen-starved tissues) (rare but serious): A buildup of lactic acid in the blood can occur if liver or kidney problems worsen; seek care immediately for unusual fatigue, fast breathing, or belly pain. · Drug interactions: Tell your doctor about all medications, especially other antivirals or kidney-affecting drugs. · Pregnancy: Tenofovir (especially TAF or TDF) is safe and recommended in the third trimester for mothers with high viral loads to prevent passing the virus to the baby.

Entecavir · Nucleoside analogue (antiviral)

Entecavir is another jammer for the virus's copy machine. Like tenofovir, it sneaks into the hepatitis B virus's DNA-building process and stops reverse transcriptase (the enzyme the virus uses to multiply). By blocking viral replication, entecavir lowers the amount of virus in the blood and reduces liver inflammation, giving the liver a chance to heal.

1. The hepatitis B virus invades a liver cell and begins to copy its DNA using reverse transcriptase.
2. Entecavir mimics a natural DNA building block (a nucleoside) that the enzyme needs.
3. Reverse transcriptase grabs entecavir and tries to add it to the growing viral DNA chain.
4. Once entecavir is added, the chain cannot grow any further—the copier jams.
5. The virus cannot make new copies of itself, so viral load drops in the bloodstream.
6. With fewer viruses attacking liver cells, inflammation decreases and the liver starts to repair.

SAFETY WATCH

Lactic acidosis (acid from oxygen-starved tissues) (rare but serious): A dangerous buildup of lactic acid can occur, especially in people with liver disease; symptoms include unusual tiredness, fast breathing, stomach pain, or muscle aches. · Liver flare on stopping: If entecavir is stopped suddenly, the virus can rebound and cause severe liver inflammation; never stop without a doctor's guidance. · Kidney function: Dose must be adjusted if kidney function is impaired; regular blood tests monitor creatinine. · Drug resistance in HIV: Entecavir should NOT be used in people with untreated HIV, because it can cause HIV to become resistant to other antiviral drugs. · Pregnancy: Entecavir has less safety data in pregnancy than tenofovir; tenofovir is preferred for pregnant women with hepatitis B.

Pegylated interferon alfa (PEG-IFN- α) · Immune modulator (cytokine)

Pegylated interferon is a lab-made version of interferon, a natural alarm protein the immune system uses to fight viruses. It works two ways: it directly slows down the hepatitis B virus inside liver cells, and it wakes up and trains immune cells (T cells and natural killer cells) to recognize and destroy infected liver cells. The 'pegylated' part means a protective coating is added so the drug lasts longer in the body and only needs to be injected once a week.

1. Pegylated interferon is injected under the skin and travels through the bloodstream.
2. It binds to receptors on the surface of liver cells and immune cells.
3. Inside liver cells, interferon switches on antiviral genes that make proteins to stop the virus from copying itself.
4. At the same time, interferon activates immune cells—especially cytotoxic T cells—that hunt down and kill hepatitis B-infected liver cells.
5. Over weeks to months, viral load drops and the immune system learns to control the infection better.
6. In some patients, this can lead to sustained immune control even after treatment stops (functional cure).

SAFETY WATCH

Flu-like symptoms: Fever, chills, muscle aches, and fatigue are very common, especially after the first few injections; acetaminophen can help. · Mood changes and depression: Interferon can cause irritability, sadness, or even suicidal thoughts; mental health must be monitored closely, and treatment stopped if severe. · Bone marrow suppression: White blood cells, red blood cells, and platelets can drop, increasing infection risk, anemia, and bleeding; blood counts are checked every 2–4 weeks. · Thyroid problems: Interferon can trigger overactive or underactive thyroid; thyroid function (TSH) is checked every 3 months. · Autoimmune flares: It can unmask or worsen autoimmune diseases (e.g., lupus, rheumatoid arthritis, psoriasis); it is avoided in people with these conditions. · Liver flare: Sometimes the immune boost causes a spike in liver enzymes (ALT), which usually settles; close monitoring is needed. · Contraindicated in pregnancy: Pegylated interferon can harm a developing baby; reliable contraception is required during treatment.

Hepatitis B immune globulin (HBIG) · Passive immunization (antibody therapy)

HBIG is a concentrated dose of antibodies against hepatitis B, taken from the blood of people who have high levels of protective antibodies. When injected, these ready-made antibodies immediately circulate in the bloodstream and bind to hepatitis B virus particles, tagging them for destruction and preventing them from entering liver cells. HBIG provides instant, short-term protection—like borrowing someone else's immune defense—while the body's own immune system (boosted by the hepatitis B vaccine) learns to make its own antibodies.

1. HBIG is injected into muscle (intramuscular) and the antibodies are absorbed into the bloodstream.
2. The antibodies (called anti-HBs) recognize and stick to the surface protein (HBsAg) of the hepatitis B virus.
3. Once tagged, the virus cannot attach to liver cells, so it cannot start an infection.
4. The immune system clears the antibody-coated virus from the blood.
5. This protection lasts only a few weeks to months (the borrowed antibodies gradually break down), so HBIG is always given alongside the hepatitis B vaccine for long-term immunity.

SAFETY WATCH

Injection site reactions: Pain, redness, or swelling at the injection site are common and usually mild. · Allergic reactions (rare): Hives, itching, shortness of breath, or swelling of the face or throat can occur; severe reactions (anaphylaxis) are very rare but require emergency care. · Transmission risk (extremely low): HBIG is made from screened and treated human blood; modern processing virtually eliminates the risk of transmitting infections, but no blood product is 100% risk-free. · Interference with live vaccines: HBIG can weaken the response to live vaccines (like MMR or varicella); spacing may be needed, though hepatitis B vaccine is not affected. · Use in newborns: HBIG is safe and routinely given to infants born to hepatitis B-positive mothers within 12 hours of birth, along with the first dose of hepatitis B vaccine.

Red flags

- 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

Nursing priorities

1. Assess mental status every 4 hours using Glasgow Coma Scale and check for asterixis (hand-flapping tremor when arms extended) — 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 — 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 — 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

4. Encourage small frequent meals with adequate protein (unless encephalopathy present) and avoid hepatotoxic substances including alcohol and acetaminophen — Nutrition supports liver regeneration (the liver can repair itself if damage stops); alcohol and acetaminophen cause additional hepatocyte (liver cell) death and worsen inflammation
5. Use strict contact precautions with blood and body fluids: gloves for all contact, eye protection during procedures, and safe needle disposal — HBV survives on surfaces for seven days and is 50 to 100 times more infectious than HIV; healthcare workers need protection from percutaneous (through-skin) exposure via contaminated sharps
6. Educate patient to avoid sharing razors, toothbrushes, or nail clippers and to cover open wounds with bandages — HBV spreads through microscopic amounts of blood on personal items; household transmission occurs when infected blood contacts another person's mucous membranes or broken skin
7. Coordinate hepatology referral for patients with chronic HBV to assess need for antiviral therapy (tenofovir or entecavir) based on HBV DNA level, ALT (liver enzyme), and fibrosis stage — Antivirals suppress viral replication (virus copying itself) and prevent cirrhosis (permanent liver scarring) and hepatocellular carcinoma (liver cancer) in patients with active inflammation and high viral loads
8. Teach sexual partners and household contacts to get tested for HBV and receive the three-dose vaccine series if non-immune — The HBV vaccine is 95 percent effective at preventing infection; post-exposure prevention (prophylaxis) with HBIG plus vaccine within 24 hours can stop transmission after known exposure

Sources

- AASLD Hepatitis B Guidance 2018
- CDC Hepatitis B Information
- WHO Guidelines for the Prevention, Care and Treatment of Persons with Chronic Hepatitis B Infection 2015
- ACOG Practice Bulletin on Viral Hepatitis in Pregnancy

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