

Pericarditis

A plain-English guide for you and your family

WHAT IS HAPPENING

Your heart sits inside a thin, smooth sac called the pericardium. Normally, this sac holds just a few drops of slippery fluid so the heart can beat freely without friction, and the sac keeps the heart anchored in the center of your chest. When the pericardium gets inflamed — from a virus, an autoimmune disease, kidney failure, a heart attack, heart surgery, cancer, or infection — the inside surfaces rub together painfully and may leak extra fluid. That rubbing causes sharp chest pain that gets worse when you breathe in or lie flat, and better when you sit up and lean forward. The inflammation also creates a scratchy sound doctors can hear with a stethoscope, called a friction rub, and it changes the heart's electrical pattern on an EKG. If inflammation keeps going, more and more fluid collects in the sac. A small amount is usually okay because the sac can stretch, but if fluid builds up faster than the sac can adapt, it starts to squeeze the heart from the outside. When the squeeze gets tight enough, the heart chambers can't fill with blood properly — that's called tamponade, and it's an emergency because blood pressure drops and organs don't get enough oxygen. Treating the inflammation early stops the domino effect before it reaches that dangerous point.

What you might feel

- Sharp chest pain that gets worse when you lie flat and better when you sit up and lean forward
- Chest pain that gets sharper when you take a deep breath, cough, or swallow
- A scratchy, squeaky sound (friction rub) when the doctor listens to your heart
- Low-grade fever (usually under 100.4°F or 38°C)
- Diffuse ST-segment elevation (the line after each heartbeat rises) on the EKG in almost all the heart's electrical zones, plus the PR segment dips down
- Feeling tired and achy, like you have the flu
- Beck triad: low blood pressure, bulging neck veins, and muffled (quiet) heart sounds
- Pulsus paradoxus: your blood pressure drops more than 10 mmHg when you breathe in

Call the doctor right away if...

- Fever above 100.4°F (38°C), especially if it won't come down — suggests infection inside the pericardium
- Large pericardial a fluid collection (effusion) (more than 20 mm of fluid around the heart on ultrasound) — risk of tamponade
- Cardiac tamponade (the heart squeezed by fluid around it) signs: low blood pressure, bulging neck veins, muffled heart sounds, or pulsus paradoxus — the heart is being squeezed and can't pump
- Chest pain that does not improve when sitting forward, or pain that feels crushing like a heart attack — may be myopericarditis (heart muscle is inflamed too) or a different emergency
- Not getting better after one week of anti-inflammatory medicine, or symptoms come roaring back — suggests a tougher cause like tuberculosis, cancer, or autoimmune disease

What the care team will do

- Check blood pressure and heart rate every 15 to 30 minutes until stable, watching for systolic pressure drop greater than 10 mmHg during inspiration (pulsus paradoxus).
- Position the patient sitting upright or leaning forward over a bedside table to reduce chest pain.
- Assess for Beck triad every 2 to 4 hours: listen for muffled or distant heart sounds, check neck veins for distention, and measure blood pressure for low blood pressure (hypotension) (low pressure).
- Administer NSAIDs (ibuprofen 600 to 800 mg three times daily or aspirin 750 to 1000 mg three times daily) with food and colchicine 0.5 mg twice daily as ordered.
- Teach the patient to report new shortness of breath, worsening chest pain, fainting, or inability to lie flat immediately.
- Monitor for signs of NSAID side effects: black or tarry stools, stomach pain, or decreased urine output.
- Obtain and review baseline labs: complete blood count (CBC), inflammatory markers (C-reactive protein and red blood cell (erythrocyte) sedimentation rate), troponin, and kidney function before starting treatment.
- Encourage activity restriction (no heavy lifting, no strenuous exercise) until pain resolves and inflammatory markers normalize.

Medicines you may be given — and why

Ibuprofen

Ibuprofen blocks an enzyme called COX (cyclooxygenase) that makes prostaglandins — chemicals that trigger pain, heat, and swelling. By stopping that enzyme, ibuprofen cools down the inflammation in the pericardium (the sac around the heart) and eases chest pain. Standard dose is 600–800 mg by mouth three times a day, usually for 1–2 weeks, then tapered (slowly lowered) over days to weeks to prevent bounce-back inflammation.

Aspirin

Aspirin works like ibuprofen by blocking COX and stopping prostaglandin production, which lowers inflammation and pain in the pericardium. Usual dose for pericarditis is 750–1000 mg by mouth three to four times a day (high-dose), then tapered over 2–4 weeks. Aspirin is often preferred after a heart attack (post-MI pericarditis or Dressler syndrome) because it also keeps platelets from clumping and forming dangerous clots in the heart arteries.

Indomethacin

Indomethacin is a powerful NSAID that blocks COX enzymes even more aggressively than ibuprofen or aspirin, shutting down prostaglandin production in the pericardium. Standard dose is 25–50 mg by mouth three times a day, then tapered after inflammation cools. It is especially useful when ibuprofen or aspirin is not strong enough or when the patient cannot take aspirin.

Colchicine

Colchicine does NOT block prostaglandins like NSAIDs. Instead, it jams the microtubules — tiny scaffolding rods inside immune cells that help them move and dump out inflammatory chemicals. With microtubules stuck, white blood cells cannot swarm to the pericardium as easily and cannot release as many inflammatory signals. This cools chronic, smoldering inflammation and cuts the chance of pericarditis coming back by about 50 percent. Standard dose is 0.5–0.6 mg once or twice daily for 3 months (sometimes 6 months if the first episode was severe or recurrent).

Prednisone (or other corticosteroids)

Prednisone is a powerful steroid hormone that mimics cortisol (the body's natural stress and anti-inflammatory hormone). It floods into the nucleus of immune cells and turns OFF genes that make dozens of inflammatory chemicals — cytokines, prostaglandins, leukotrienes — all at once. This snuffs out inflammation fast and hard. BUT steroids are NOT first-line for pericarditis because they raise the recurrence rate (the inflammation bounces back when you stop). They are reserved for patients who cannot take NSAIDs (e.g. severe kidney disease),

those with autoimmune pericarditis (lupus, rheumatoid arthritis), or cases that do not respond to NSAIDs plus colchicine. Usual starting dose is 0.25–0.5 mg per kg per day (often 15–25 mg daily), tapered very slowly over weeks to months.

Antibiotics (e.g. vancomycin, ceftriaxone, or culture-directed therapy)

If pericarditis is caused by bacteria (purulent pericarditis), you need antibiotics that kill the specific germ. Empiric (best-guess) therapy often starts with vancomycin (covers MRSA and other tough Gram-positive bacteria) plus a third-generation cephalosporin like ceftriaxone (covers Gram-negative rods and some other bacteria). Once culture results identify the exact bug, therapy is narrowed to the antibiotic that kills it best. Antibiotics work by breaking bacterial cell walls, jamming protein factories, or blocking DNA copying — the exact mechanism depends on the drug class.

Anti-tuberculosis therapy (isoniazid, rifampin, pyrazinamide, ethambutol)

Tuberculous pericarditis is caused by *Mycobacterium tuberculosis*, a slow-growing germ with a waxy, tough coat. You need a cocktail of four drugs for at least 2 months (intensive phase), then two drugs for 4–7 more months (continuation phase) — total 6–9 months. Isoniazid blocks the enzyme that builds the TB germ's cell wall. Rifampin jams the bacterial RNA-copying machine so TB cannot make proteins. Pyrazinamide works best in acidic environments (like inside immune cells) and punches holes in the TB membrane. Ethambutol stops the germ from building its outer armor. Together, they kill TB in the pericardium and prevent it from becoming drug-resistant.

Dialysis intensification or kidney transplant (for uremic pericarditis)

Uremic pericarditis happens when the kidneys fail and toxic waste (urea, creatinine, and other molecules) builds up in the blood and irritates the pericardium. NSAIDs and colchicine help a little, but the real fix is clearing the toxins. That means starting dialysis if the patient is not on it yet, or intensifying (more frequent or longer sessions) if they are already dialyzing. Hemodialysis filters blood through a machine; peritoneal dialysis uses the belly lining as a filter. Either way, waste is pulled out, inflammation cools, and pericarditis usually resolves in days to weeks. Kidney transplant cures severe waste buildup in the blood (uremia) permanently if the patient qualifies.

Chemotherapy or radiation (for malignant pericarditis)

Malignant pericarditis means cancer cells (most often lung, breast, lymphoma, or leukemia) have spread to the pericardium or are pressing on it. Fluid builds up because tumor cells irritate the sac or block lymphatic drainage. The treatment is NOT a pericarditis drug — it is treating the cancer itself. Chemotherapy uses drugs that kill fast-dividing cells (the cancer) by jamming DNA, blocking cell division, or triggering cell suicide. Radiation aims high-energy beams at the tumor to damage its DNA so it cannot grow. If the cancer shrinks, the pericardial

irritation stops, fluid reabsorbs, and pericarditis resolves. Often you also drain the fluid (pericardiocentesis or pericardial window) for immediate relief while chemo or radiation works.

Pericardiocentesis or pericardial window (drainage procedure, not a medication)

Pericardiocentesis is a needle puncture through the chest into the pericardial sac to drain fluid. A pericardial window is a small surgical cut in the sac to let fluid drain continuously into the chest or belly, where it is absorbed. Neither is a drug, but both are critical when fluid squeezes the heart (tamponade) or when a huge fluid collection (effusion) does not respond to medicine. Draining the fluid instantly relieves pressure, lets the heart fill and pump normally again, and often saves the patient's life. Fluid is sent to the lab to look for cancer cells, bacteria, TB, or other clues to the cause.

Where this information comes from

- ESC Guidelines for the diagnosis and management of pericardial diseases (2015)
- AHA/ACC/HRS Guideline on the evaluation and management of patients with bradycardia and cardiac conduction delay (2018, pericarditis EKG patterns)
- American College of Cardiology Expert Consensus Decision Pathway on the evaluation and management of acute pericarditis (2020)
- UpToDate clinical summary on acute pericarditis (2024)

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