

Myocarditis

A plain-English guide for you and your family

WHAT IS HAPPENING

Your heart is a muscular pump. The thick middle layer of muscle (the myocardium) squeezes with every beat to push blood out to your body. Those muscle cells are mature and cannot divide to make new copies, so once they are damaged, the heart has to heal with scar or slowly replace what it can. Normally, the heart muscle stays healthy because small blood vessels feed it oxygen and fuel. When a virus (like coxsackie B, parvovirus B19, influenza, or COVID-19), a parasite (Chagas disease in some regions), a bacterial infection, an autoimmune attack (like giant-cell or eosinophilic myocarditis), a drug (especially some cancer medicines called checkpoint inhibitors), or a toxin breaks into the heart muscle, it sets off inflammation. That inflammation injures the muscle cells directly and calls in the immune system, which can add more damage while trying to clear the invader. The heart muscle swells, weakens, and becomes irritable. You might feel chest pain (because inflamed tissue hurts), shortness of breath (because the weak pump cannot keep up and fluid backs into your lungs), a fast or fluttery heartbeat (palpitations from the irritated muscle firing extra beats), and deep tiredness. Blood tests will show troponin leaking out of damaged cells, and an electrocardiogram may look like a heart attack even though your coronary arteries are open. In mild cases the heart heals on its own with rest. In severe cases — called fulminant myocarditis — the heart can lose almost all its squeeze within hours, triggering cardiogenic shock (the body shuts down because the heart cannot pump enough blood) or sudden death from a dangerous rhythm. Every symptom and test abnormality traces back to one upstream problem: the inflamed, injured heart muscle. The body tries to compensate by speeding the pulse, tightening blood vessels, and holding onto fluid, which can mask how sick you really are until the system tips into crisis. Catching myocarditis early and supporting the heart (sometimes with medicines, sometimes with a mechanical pump) buys time for healing.

What you might feel

- Sharp or tight chest pain that often gets worse when you lie flat or take a deep breath
- Shortness of breath, especially when lying down or with light activity
- Fast or fluttering heartbeat (palpitations)

- Feeling very tired or weak, even at rest
- Swelling in the legs, ankles, or belly
- Flu-like symptoms a few days or weeks before (fever, muscle aches, sore throat, diarrhea)
- Fainting or feeling like you might pass out
- Sudden cardiac arrest (the heart stops beating)

Call the doctor right away if...

- Fainting, near-fainting, or sudden collapse
- New or worsening shortness of breath at rest
- Chest pain that feels crushing or won't go away
- Fast heart rate (over 100 beats per minute at rest) that doesn't slow down
- Signs of shock: cold, clammy skin, confusion, very low blood pressure

What the care team will do

- Place patient on continuous cardiac telemetry monitoring and check rhythm strip every four hours for heart blocks or ventricular arrhythmias
- Keep crash cart with defibrillator pads and transcutaneous pacing equipment immediately outside the room
- Monitor troponin levels every six to eight hours and report any rise immediately to the provider
- Measure strict intake and output every shift and weigh patient daily at same time to detect fluid overload
- Enforce strict bed rest with bedside commode privileges only and no Valsalva maneuvers like straining during bowel movements
- Administer ACE inhibitors or ARBs and beta-blockers exactly on schedule even if blood pressure is low-normal
- Assess lung sounds every four hours and immediately report new crackles or increased work of breathing
- Hold all NSAIDs like ibuprofen and notify provider if patient requests pain medication

Medicines you may be given — and why

ACE inhibitor (angiotensin-converting enzyme inhibitor) — for example, lisinopril or enalapril

An ACE inhibitor blocks an enzyme that makes a chemical called angiotensin II. Angiotensin II squeezes blood vessels tight and makes the heart work harder. By blocking it, the drug lets blood vessels relax and opens them wider, so the heart can pump blood more easily without straining. This lowers blood pressure and helps a weakened heart rest and heal.

ARB (angiotensin receptor blocker) — for example, losartan or valsartan

An ARB blocks the spot where angiotensin II tries to land and work. Even if angiotensin II is made, it cannot squeeze blood vessels because the ARB is sitting in its parking spot. The result is the same as an ACE inhibitor: blood vessels relax, blood pressure drops, and the heart's workload is lighter. Doctors often use an ARB if someone cannot take an ACE inhibitor because of cough.

Beta-blocker — for example, metoprolol, carvedilol, or bisoprolol

A beta-blocker blocks adrenaline (the body's stress hormone) from landing on the heart. Adrenaline usually makes the heart beat faster and harder. By blocking it, the drug slows the heart rate and lowers blood pressure, so the heart does not have to work as hard. A slower, calmer heart uses less oxygen and has more time to fill with blood between beats. Over months, this helps the injured heart muscle heal and get stronger.

MRA (mineralocorticoid receptor antagonist) — for example, spironolactone or eplerenone

An MRA blocks a hormone called aldosterone. Aldosterone normally tells the kidneys to hold onto salt and water, which raises blood pressure and makes the heart work harder. It also causes scar tissue to build up in the heart over time. By blocking aldosterone, the MRA helps the kidneys remove extra fluid, lowers blood pressure, and slows down harmful scarring and remodeling of the heart muscle. This protects the heart as it heals.

Diuretic (water pill) — for example, furosemide (Lasix) or bumetanide

A diuretic tells the kidneys to dump extra salt and water into the urine. When myocarditis weakens the heart, blood can back up into the lungs and body, causing swelling in the legs and shortness of breath. The diuretic removes that extra fluid quickly, which relieves the pressure in the lungs and lets you breathe easier. It also takes strain off the heart by lowering the volume of blood it has to pump.

Inotrope (for severe cases) — for example, dobutamine or milrinone

An inotrope is a powerful drug given through an IV in the hospital when the heart is so weak it cannot pump enough blood to keep the body alive. It works by increasing the calcium inside heart muscle cells or by making the heart more sensitive to the body's own adrenaline. This makes each heartbeat squeeze harder and pump more blood. Inotropes are used as a short-term bridge — they buy time while the heart heals or while doctors prepare for advanced treatments like a mechanical heart pump or transplant.

Vasopressor (for shock) — for example, norepinephrine or vasopressin

A vasopressor is a powerful IV drug used in the ICU when blood pressure drops so low that organs like the brain and kidneys are not getting enough blood. It works by squeezing blood vessels tight all over the body, which raises blood pressure and pushes blood to vital organs. In severe myocarditis with cardiogenic shock (the heart is too weak to keep blood pressure up), vasopressors keep the patient alive while doctors treat the heart and start other support like mechanical pumps.

Immunosuppressive therapy — for example, corticosteroids (prednisone), azathioprine, or mycophenolate

Immunosuppressive drugs calm down the immune system when it is attacking the heart muscle by mistake or overreacting to an infection. They are used in specific types of myocarditis — giant cell myocarditis (a rare, severe autoimmune form), eosinophilic myocarditis (when a type of white blood cell called an eosinophil invades the heart), or myocarditis linked to autoimmune diseases like lupus. These drugs stop the immune attack so the heart muscle can heal. They are NOT used in typical viral myocarditis, because calming the immune system could let the virus grow and cause more damage.

Antiarrhythmic drug (if needed) — for example, amiodarone

An antiarrhythmic drug is used when myocarditis causes dangerous irregular heartbeats (arrhythmias) like ventricular a fast heart rate (tachycardia) or atrial fibrillation (an irregular, quivering heartbeat). The inflamed heart muscle does not conduct electrical signals smoothly, so the heartbeat can become chaotic or dangerously fast. The drug works by slowing down electrical signals in the heart and making the heart cells less excitable, which helps restore a steady, safe rhythm. It does NOT fix the myocarditis itself — it only controls the rhythm problem while the heart heals.

IVIG (intravenous immunoglobulin) — in select cases, especially in children

IVIG is a treatment made from antibodies collected from thousands of blood donors. It is given through an IV and works by calming the immune system and blocking the harmful antibodies or immune cells that may be attacking the heart. It is sometimes used in children with severe

myocarditis or in adults with autoimmune or viral myocarditis when standard treatments are not working. The exact way it works is not fully understood, but it seems to reset the immune response and reduce inflammation. Evidence is mixed, and it is not used routinely — only in select cases.

Where this information comes from

- ESC 2013 Position Statement on Myocarditis (updated 2024)
- AHA/ACC 2022 Guideline for Management of Heart Failure
- Circulation: Cardiovascular Quality and Outcomes 2021 (myocarditis epidemiology)
- CDC (viral myocarditis surveillance and vaccine safety monitoring)

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