

# Myocarditis

Myocarditis means the heart muscle is swollen and irritated — it can be mild or life-threatening.

## THE ONE-LINE TRUTH

Inflammation damages the heart muscle cells, weakening the heart's pump and triggering dangerous rhythms.

## The chain — how it unfolds

1. A virus, bacterium, parasite, autoimmune attack, drug, or toxin enters the myocardium. — why: Coxsackie B and other enteroviruses bind receptors on cardiomyocytes and replicate inside the cells; bacteria release toxins; the immune system in lupus or sarcoidosis mistakenly attacks the heart; checkpoint-inhibitor drugs unleash T cells against myocardial antigens. The trigger varies, but all pathways converge on myocyte injury.
2. Inflammation erupts: immune cells (lymphocytes, macrophages, sometimes eosinophils) flood the muscle; cytokines (interleukin-1, tumor tissue death (necrosis) factor, interferon) amplify the response. — why: The innate immune system detects danger signals from dying cells and calls in reinforcements. The goal is to clear the infection or foreign antigen, but the inflammatory cascade also kills bystander myocytes and disrupts the extracellular scaffold that holds muscle fibers together.
3. Myocytes die (necrosis, apoptosis) and leak their contents — troponin, creatine kinase-MB — into the bloodstream. — why: Cell membranes rupture under immune attack and direct toxin damage. Troponin is a muscle protein normally locked inside the cell; its presence in blood is the biomarker that confirms myocyte death. Because heart muscle cannot regenerate, every dead cell is a permanent loss.
4. The patient feels chest pain, often sharp or pressure-like, sometimes worse when lying flat or breathing deeply. — why: Inflamed tissue is rich in pain nerve endings; stretching the swollen muscle or the overlying pericardium (which is often inflamed at the same time) triggers pain signals. The pattern can mimic a heart attack, but coronary angiography shows open arteries.

5. The weakened left ventricle cannot empty fully; stroke volume drops and fluid backs up into the pulmonary veins and lung capillaries, causing shortness of breath (dyspnea). — why: Systolic dysfunction means less blood is pushed forward per beat. By the law of conservation, the blood that does not go out must stay in — it piles up behind the failing pump, raising pressure in the lungs and forcing fluid across capillary walls into the air sacs (pulmonary swelling (edema)).
6. The heart rate climbs (tachycardia); the patient feels palpitations or a racing, fluttering heartbeat. — why: Baroreceptors in the aorta and carotid arteries sense falling stroke volume and signal the brainstem to release norepinephrine, which speeds the heart rate to try to maintain cardiac output ( $\text{output} = \text{rate} \times \text{stroke volume}$ ). Inflamed muscle is also electrically irritable and fires extra beats (premature ventricular contractions).
7. Fatigue becomes profound; the patient cannot climb stairs or perform usual activities. — why: Low cardiac output means skeletal muscles and the brain receive less oxygen and glucose. Cellular energy production (ATP synthesis) falls, and waste products (lactate) accumulate, causing the sensation of exhaustion and muscle heaviness.
8. The electrocardiogram shows ST-segment elevation or depression, T-wave inversion, or new bundle-branch block — patterns that mimic acute heart attack (myocardial infarction) or pericarditis. — why: Inflammation alters the electrical properties of the myocardium: injured cells depolarize slowly, repolarize abnormally, and conduct impulses erratically. The ECG reflects these electrical disturbances, which look similar to starved blood flow (ischemia) because both involve sick, struggling muscle.

## Spot it — what you see, and why

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- Sharp or tight chest pain that often gets worse when you lie flat or take a deep breath: The outer sac around the heart (the pericardium) often gets inflamed at the same time as the heart muscle, and when you breathe or lie down, the swollen layers rub together and hurt.
- Shortness of breath, especially when lying down or with light activity: The inflamed heart muscle weakens and can't pump blood forward as well, so blood backs up into the lungs and makes them heavy and wet, which makes it hard to breathe.
- Fast or fluttering heartbeat (palpitations): Inflammation irritates the heart's electrical wiring, so the heart may beat too fast or in an uneven rhythm to try to keep up with the body's needs.
- Feeling very tired or weak, even at rest: The weakened heart can't deliver enough oxygen-rich blood to the muscles and organs, so the whole body feels like it's running on low power.
- Swelling in the legs, ankles, or belly: When the heart can't pump well, blood backs up in the veins, and fluid leaks out into the tissues, causing swelling (edema).

- Flu-like symptoms a few days or weeks before (fever, muscle aches, sore throat, diarrhea): Most myocarditis is triggered by a viral infection (like coxsackievirus, adenovirus, or COVID-19), and the flu-like signs are the body fighting that virus before it reaches the heart.

## Red flags — act now

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- 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

## Treat it — in this order

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1. Supportive heart failure therapy — ACE inhibitor (or ARB if ACE not tolerated), beta-blocker, and loop diuretic → ACE inhibitors (like lisinopril or enalapril) relax blood vessels so the weakened heart does not have to push as hard. They also slow down scar formation in the muscle. Beta-blockers (like carvedilol or metoprolol) slow the heart rate so each beat has more time to fill and pump efficiently. Loop diuretics (like furosemide) drain the backed-up fluid from the lungs and legs so the body can breathe and move again. Together, these buy the heart time to heal by reducing the workload and easing the strain.
2. Strict rest — no exercise or exertion for 3 to 6 months → Gives the inflamed heart muscle absolute rest so the inflammation can die down and the muscle can repair itself. Exercise during active myocarditis forces the damaged muscle to work hard, which can trigger arrhythmias or sudden death. Rest prevents the heart from tearing itself apart while it heals.
3. Immunosuppression (corticosteroids like prednisone, or other drugs like azathioprine) — ONLY for giant cell myocarditis, eosinophilic myocarditis, cardiac sarcoidosis, or proven autoimmune myocarditis → Blocks the immune system's overactive attack on the heart muscle in specific autoimmune types. In these rare forms, the immune system itself is the enemy and must be shut down. For viral myocarditis, immunosuppression is contraindicated because it lets the virus spread unchecked and makes things worse.
4. Mechanical circulatory support — ECMO (extracorporeal membrane oxygenation) or percutaneous left ventricular assist device (Impella) → Takes over the job of the heart and lungs when the inflamed muscle is so weak it cannot pump blood to the brain, kidneys, and body (cardiogenic shock or fulminant myocarditis). The machine keeps the patient alive while the heart muscle rests and tries to heal underneath.

5. Antiarrhythmic drugs (amiodarone) and implantable cardioverter-defibrillator (ICD) for life-threatening arrhythmias → Amiodarone stabilizes the chaotic electrical signals that the inflamed muscle fires. An ICD shocks the heart back into rhythm if it slips into a deadly rhythm (ventricular a fast heart rate (tachycardia) or fibrillation). Together, they prevent sudden death while the inflammation heals.

## Your top priorities at the bedside

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1. Place patient on continuous cardiac telemetry monitoring and check rhythm strip every four hours for heart blocks or ventricular arrhythmias — why: Myocarditis damages the conduction system (electrical pathways) and can cause sudden complete heart block (no electrical signals reach lower chambers) or ventricular a fast heart rate (tachycardia) (life-threatening fast rhythm) with no warning
2. Keep crash cart with defibrillator pads and transcutaneous pacing equipment immediately outside the room — why: Inflamed myocardium (heart muscle) is electrically unstable and can develop ventricular fibrillation (chaotic deadly rhythm) or severe a slow heart rate (bradycardia) (very slow heart rate) requiring immediate defibrillation (shock) or pacing (artificial heartbeat)
3. Monitor troponin levels every six to eight hours and report any rise immediately to the provider — why: Rising troponin (a heart enzyme released when muscle cells die) indicates ongoing myocardial injury (continuing damage to heart muscle); worsening troponin suggests fulminant myocarditis (severe rapidly progressing inflammation) that may need mechanical support like ECMO (a machine that pumps blood outside the body)