

Myocarditis

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

THE STORY IN ONE SENTENCE

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

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

Normal anatomy — the body's job

The heart wall has three layers: a thin inner lining (endocardium), a thick middle muscle (myocardium), and an outer wrap (pericardium). The myocardium does the work — it squeezes to pump blood forward. Heart muscle cells (cardiomyocytes) are mature and cannot divide like skin or liver cells, so the heart's ability to replace dead muscle is very limited. Tiny blood vessels woven through the muscle deliver oxygen and glucose so the cells can make energy and contract. In a healthy heart, the muscle is soft, elastic, and strong; every beat pushes blood smoothly out to the lungs and body.

BODY'S JOB

The myocardium's job is to contract powerfully and rhythmically, creating enough pressure to send oxygenated blood from the left side of the heart out to every organ and tissue. Between beats, the muscle relaxes so the chambers can fill with blood again. This cycle — squeeze, relax, fill — repeats about 100,000 times a day. When the muscle is healthy, the heart meets the body's demands whether you are asleep or running.

1. Blood returns from the body to the right atrium, flows into the right ventricle, then is pumped to the lungs to pick up oxygen.
2. Oxygen-rich blood returns to the left atrium, fills the left ventricle, and the thick left-ventricle myocardium squeezes hard to push it out through the aorta to the whole body.
3. Small coronary arteries branch off the aorta and dive into the heart muscle, delivering the oxygen and fuel the myocardium needs to keep contracting.

4. Between beats, the muscle relaxes and the ventricles refill; low pressure in the chambers lets blood flow in smoothly.
5. Electrical signals travel through specialized pathways in the muscle, telling all the cells to contract together in a coordinated wave.

What breaks

The heart muscle becomes inflamed when a virus, bacterium, parasite, autoimmune attack, drug, or toxin injures the cells and triggers the immune system. The muscle swells, weakens, and becomes electrically unstable, so it cannot pump well and may fire dangerous rhythms.

CLINICAL TERM

Myocarditis is inflammation of the myocardium caused by infectious agents (coxsackie B virus, parvovirus B19, influenza, SARS-CoV-2, HIV, Chagas disease [*Trypanosoma cruzi*], Lyme disease, diphtheria), autoimmune conditions (giant-cell myocarditis, eosinophilic myocarditis, lupus, sarcoidosis), drugs (immune checkpoint inhibitors, anthracyclines, clozapine), or toxins (cocaine, alcohol). Direct cytotoxic injury and immune-mediated damage combine to kill cardiomyocytes, disrupt the extracellular matrix, and provoke a local and systemic inflammatory response. Tissue death (Necrosis) and immune infiltration weaken contractility, impair diastolic relaxation, and create electrical heterogeneity that can trigger arrhythmias. In fulminant cases, widespread myocyte death leads to acute severe systolic dysfunction and cardiogenic shock within hours to days.

How the body compensates

When the heart muscle weakens, the body activates neurohormonal reflexes — faster pulse, tighter blood vessels, more fluid retention — to defend blood pressure and organ blood flow (perfusion). These responses can mask the true severity for hours or days, then suddenly fail, tipping into shock or fatal arrhythmia. Vital signs may look reassuring while the heart is silently decompensating.

1. The sympathetic nervous system detects falling cardiac output and releases norepinephrine (the fight-or-flight chemical), raising the heart rate and squeezing arteries to keep blood pressure up.
2. The kidneys sense low flow and release renin, starting a hormone cascade (renin-angiotensin-aldosterone system) that tells the body to hold salt and water, expanding blood volume to fill the weak pump.
3. The heart itself stretches (Frank-Starling mechanism): a bigger filling volume stretches the muscle fibers, temporarily boosting the force of the next contraction — but only up to a point.

4. The right ventricle may fail first if inflammation is patchy, causing jugular-vein distension, liver swelling, and leg swelling (edema) even while the left side still ejects some blood.
5. Compensatory a fast heart rate (tachycardia) (fast heart rate) shortens the time available for the coronary arteries to fill the muscle with blood, so the heart muscle itself becomes blood-starved (ischemic) (starved of oxygen), worsening pump function.
6. As myocardial swelling (edema) and tissue death (necrosis) progress, stroke volume drops below the point that faster rate and higher vascular tone can offset, and blood pressure falls despite maximal sympathetic drive.
7. The crash point is fulminant myocarditis with cardiogenic shock: the heart cannot generate enough pressure to perfuse the brain, kidneys, or itself; lactate rises, urine output stops, and the patient becomes confused or unresponsive.
8. Ventricular arrhythmias (ventricular a fast heart rate (tachycardia), ventricular fibrillation) or complete heart block can cause sudden death even if hemodynamics were stable minutes before.
9. Giant-cell myocarditis and eosinophilic myocarditis have especially high mortality without prompt immunosuppression and mechanical circulatory support.
10. A patient may look stable — normal blood pressure, talking, walking — while sitting on the edge of collapse. Trend the data (rising troponin, worsening ejection fraction, new arrhythmia) rather than relying on a single snapshot.

Domino effect

1. A virus, bacterium, parasite, autoimmune attack, drug, or toxin enters the myocardium. — 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. — 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. — 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. — 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). — 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. — 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 (output = rate × 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. — 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. — 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.
9. In severe cases, the ejection fraction drops below 30–35 % and the patient develops clinical heart failure: pulmonary edema (fluid flooding the lungs), jugular-vein distension, an enlarged liver (hepatomegaly), peripheral swelling (edema). — When more than half the myocardium is inflamed or dead (necrotic), the ventricle loses the mechanical strength to generate normal pressure. Forward flow plummets; backward congestion overwhelms the lungs and systemic veins. The kidneys retain salt and water in a failed attempt to restore filling pressures, worsening swelling (edema).

10. Fulminant myocarditis triggers cardiogenic shock: blood pressure falls despite maximum sympathetic tone; organs shut down (oliguria, altered mentation, cool extremities, lactic acidosis (acid from oxygen-starved tissues)). — Massive, rapid myocyte death leaves too little functioning muscle to sustain blood flow (perfusion). The brain, kidneys, liver, and gut become blood-starved (ischemic). Without mechanical circulatory support (intra-aortic balloon pump, ventricular assist device, or extracorporeal membrane oxygenation), multiorgan failure and death follow within hours.
11. Ventricular arrhythmias (ventricular a fast heart rate (tachycardia), ventricular fibrillation) or complete heart block cause sudden collapse or cardiac arrest. — Patchy inflammation creates electrical chaos: some areas conduct normally, others slowly, setting up circular wavefronts (re-entry) that spiral into lethal rhythms. If inflammation hits the atrioventricular node or bundle of His, the electrical bridge between atria and ventricles is destroyed, and the ventricles stop beating or beat too slowly to sustain life.
12. Survivors may recover completely if the trigger is cleared and the immune response resolves, or they may develop chronic dilated cardiomyopathy if extensive scarring replaces dead muscle. — Mild inflammation heals with minimal fibrosis; the heart regains near-normal function. Severe or prolonged inflammation kills enough myocytes that scar tissue predominates, leaving the ventricle dilated, thin-walled, and permanently weak — chronic heart failure that requires lifelong medication or eventual transplant.

Symptoms · what the patient feels, and why

- 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.
- Fainting or feeling like you might pass out: Dangerous heart rhythms (ventricular arrhythmias) or a sudden drop in blood pressure can cut off blood flow to the brain for a moment, causing you to black out.
- Sudden cardiac arrest (the heart stops beating): Severe inflammation can scar or electrically scramble the heart muscle so badly that it stops pumping altogether, and this can happen with little or no warning, especially in young, otherwise healthy people.

Focused exam

- New heart murmur or S3 gallop (extra heart sound): In myocarditis, the inflamed heart muscle weakens and the chambers stretch. When the chambers stretch, the valves cannot close all the way and extra fluid sloshes around inside. This makes a third heart sound (S3), like an echo after the normal two beats. It tells you the inflammation has hurt the pump enough to make it weak and boggy.
- Elevated jugular venous pressure (JVP) — bulging neck veins: Myocarditis weakens the right side of the heart so blood backs up into the big veins that drain into the heart. You see the neck veins bulge because blood is stuck there, waiting to get through a pump that is too weak to pull it in. This backup is a red flag that the heart is failing.
- Peripheral swelling (edema) (swollen ankles or legs): When myocarditis damages the heart muscle, the muscle cannot squeeze hard enough to push blood forward. Blood pools in the legs and the pressure pushes water out of the blood vessels into the surrounding tissue. This swelling shows the inflammation has weakened the muscle enough to cause backup all the way down to the feet.
- Fast or irregular heartbeat — a fast heart rate (tachycardia) or arrhythmia: Myocarditis inflames both the heart muscle and the electrical system that runs through it. The inflamed tissue misfires electrical signals, so the heart speeds up or skips beats. The body also tries to speed the heart up to make up for the weak pump — like revving a broken engine to keep the car moving.
- Crackles (popping sounds) at the bases of the lungs: In myocarditis, the inflamed left side of the heart cannot pump blood forward out to the body. Blood backs up into the lungs and water leaks into the tiny air sacs. You hear crackles because air is bubbling through fluid in the lungs — like blowing bubbles through a straw in a drink.

- Low blood pressure (Hypotension) (low blood pressure) or cool, clammy skin on the arms and legs: Severe myocarditis can cause cardiogenic shock — the inflamed muscle is so weak it cannot push enough blood to the body. The body pulls blood away from the skin and arms to keep the brain, heart, and kidneys alive. Cold, sweaty skin means the body is in survival mode.

Diagnostics · every test answers a question

- Troponin I or T (blood test that measures a protein released when heart muscle cells die) → Is the heart muscle being damaged right now?
- BNP (B-type natriuretic peptide) or NT-proBNP (blood tests that measure a hormone the heart releases when it is stretched) → Is the heart muscle so weak that the chambers are stretching and failing?
- 12-lead electrocardiogram (EKG or ECG — measures the electrical activity of the heart) → Does the electrical pattern look like widespread inflammation or a blocked artery?
- Transthoracic echocardiogram (ultrasound of the heart) → Is the heart muscle weak, and is the weakness global or in just one territory?
- Cardiac MRI with late gadolinium enhancement (a special scan that lights up inflamed or scarred heart tissue) → Is there inflammation or injury in the heart muscle, and does the pattern match myocarditis?
- C-reactive protein (CRP) and red blood cell (erythrocyte) sedimentation rate (ESR) — blood markers of inflammation → Is there active inflammation in the body?
- Coronary angiography (dye and X-ray movie of the heart arteries) → Are the coronary arteries blocked, or is the problem in the muscle itself?
- Endomyocardial biopsy (tiny samples of heart muscle taken through a catheter) → What is causing the inflammation — virus, autoimmune attack, or something else?

Treatment logic

- 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.

- 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.
- 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.
- 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.
- 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.
- Avoid NSAIDs (like ibuprofen or naproxen) during acute myocarditis → NSAIDs block inflammation but also interfere with the heart's natural healing process and can worsen heart failure. Avoiding them prevents further damage and gives the muscle a better chance to repair.
- Serial echocardiography every 3 to 6 months to monitor ejection fraction recovery → Tracks whether the heart muscle is healing (ejection fraction rising back toward 50-60%) or scarring down permanently. Tells you when it is safe to return to normal activity and whether long-term heart failure therapy will be needed.

Medications

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

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.

1. The enzyme ACE normally clips a protein into angiotensin II, a powerful squeezer of blood vessels.
2. The ACE inhibitor locks onto ACE and stops it from making angiotensin II.
3. With less angiotensin II around, blood vessels stay relaxed and wide.
4. The heart pumps against less resistance, so it does not have to work as hard.
5. Over weeks and months, the heart muscle can slowly recover and remodel in a healthier way.

SAFETY WATCH

Can raise potassium in the blood — your doctor will check blood tests regularly. · Can cause a dry cough in some people (harmless, but annoying). · Rarely causes swelling of the lips, tongue, or throat (angioedema) — if this happens, stop the medicine and get help right away. · Not safe during pregnancy — tell your doctor if you are pregnant or planning to be. · Can make kidney function change — blood tests will track this.

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

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.

1. Angiotensin II is still made in the body, but the ARB sits on the receptor (the landing dock) where angiotensin II needs to attach.
2. Angiotensin II floats by but cannot work because its spot is blocked.
3. Blood vessels stay relaxed and wide.
4. The heart pumps more easily with less pressure pushing back.
5. Over time, this lighter load helps the injured heart muscle heal and remodel.

SAFETY WATCH

Can raise potassium in the blood — your doctor will check blood tests. · Usually does NOT cause a cough (which is why it is used if ACE inhibitor caused cough). · Rarely causes swelling of the lips, tongue, or throat (angioedema) — stop and get help if this happens. · Not safe during pregnancy — tell your doctor if you are pregnant or planning to be. · Can affect kidney function — blood tests will track this.

Beta-blocker — for example, metoprolol, carvedilol, or bisoprolol · Beta-blocker (beta-adrenergic blocker)

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.

1. Adrenaline (epinephrine) and noradrenaline are released during stress or when the heart is weak.
2. These hormones try to land on beta receptors on heart cells to speed things up.
3. The beta-blocker sits on those receptors and blocks adrenaline from attaching.
4. The heart beats slower and squeezes less forcefully.
5. Over weeks to months, the heart remodels in a healthier shape and pumps better, even though it is going slower.

SAFETY WATCH

Can make you feel tired or dizzy at first as your body adjusts to a slower heart rate. · Not safe if your heart rate is already very slow or if you have certain types of heart block. · Can worsen asthma or severe lung disease — tell your doctor if you have trouble breathing. · Do NOT stop suddenly — the dose must be lowered slowly to avoid rebound fast heart rate or chest pain. · Can mask signs of low blood sugar if you have diabetes — check sugar levels regularly.

MRA (mineralocorticoid receptor antagonist) — for example, spironolactone or eplerenone · MRA (mineralocorticoid receptor antagonist, also called aldosterone blocker)

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.

1. When the heart is weak or injured, the body releases aldosterone to try to keep blood pressure up.
2. Aldosterone lands on receptors in the kidneys and tells them to keep salt and water in the body.
3. The MRA sits on those receptors and blocks aldosterone from working.
4. The kidneys start to let go of extra salt and water in the urine.
5. Less fluid means less strain on the heart, and less aldosterone means less scar tissue forms in the heart muscle over time.

SAFETY WATCH

Can raise potassium in the blood dangerously high — your doctor will check blood tests often, especially if you are also on an ACE inhibitor or ARB. · Can cause breast tenderness or enlargement in men (gynecomastia) with spironolactone; eplerenone has less of this effect. · Can affect kidney function — blood tests will track this. · Not safe if your potassium is already high or if your kidneys are severely damaged.

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

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.

1. Blood filters through the kidneys, and normally most of the salt and water are pulled back into the body in a part called the loop of Henle.
2. The diuretic blocks the pump (the sodium-potassium-chloride cotransporter) in that loop.
3. Salt and water stay in the urine instead of going back into the blood.
4. You urinate more, and the extra fluid leaves the body.
5. With less fluid in the blood vessels and tissues, swelling goes down and the heart has less volume to pump.

SAFETY WATCH

Can lower potassium and magnesium in the blood — your doctor will check levels and may give you supplements. · Can cause dehydration if the dose is too high — watch for dizziness, dry mouth, or feeling very thirsty. · Can make you urinate a lot, especially in the first few hours after taking it — plan bathroom access. · Can affect kidney function and electrolytes — blood tests will track this regularly.

Inotrope (for severe cases) — for example, dobutamine or milrinone · Inotrope (drug that makes the heart squeeze harder)

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.

1. In severe myocarditis, the heart muscle is so damaged it cannot squeeze hard enough to move blood forward.
2. The inotrope is given into a vein and travels straight to the heart.
3. It increases the amount of calcium available to the heart muscle fibers or makes the muscle more responsive to the body's own squeeze signals.
4. Each heartbeat becomes stronger and pumps more blood to the brain, kidneys, and body.
5. This supports the body while the inflammation calms down and the heart begins to recover, or until more advanced support (like ECMO or a heart pump) can be started.

SAFETY WATCH

Can cause dangerous irregular heartbeats (arrhythmias) — the heart is monitored constantly in the ICU. · Can make the heart use too much oxygen and worsen damage if used too long. · Blood pressure and heart rhythm are watched minute by minute. · Only used in the hospital under close supervision — never at home.

Vasopressor (for shock) — for example, norepinephrine or vasopressin · Vasopressor (drug that squeezes blood vessels to raise blood pressure)

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.

1. In cardiogenic shock from severe myocarditis, the heart is so weak that blood pressure falls dangerously low.
2. The vasopressor is given into a vein and travels to receptors on blood vessel walls.
3. It causes the smooth muscle in blood vessel walls to contract and squeeze the vessels narrow.
4. Blood pressure rises, and blood flow to the brain, kidneys, and heart improves.
5. This buys time for other treatments (inotropes, mechanical support, healing) to take effect.

SAFETY WATCH

Can reduce blood flow to the fingers, toes, and intestines if blood vessels squeeze too tight — nurses watch for pale or cold skin. · Can cause dangerous irregular heartbeats. · Blood pressure and heart rhythm are monitored constantly in the ICU. · Can worsen heart strain if used too long. · Only used in the hospital under very close supervision.

Immunosuppressive therapy — for example, corticosteroids (prednisone), azathioprine, or mycophenolate · Immunosuppressant (drugs that calm down the immune system)

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.

1. In giant cell or eosinophilic myocarditis, the immune system is attacking healthy heart muscle.
2. Immune cells release chemicals that cause inflammation and kill heart cells.
3. The immunosuppressant drug blocks the signals that activate these immune cells or stops them from multiplying.
4. The immune attack slows down or stops.
5. Inflammation in the heart goes down, and the muscle can begin to heal.
6. Treatment usually lasts months to years and is tapered slowly under close monitoring.

SAFETY WATCH

Weakens the immune system, so infections become more common and more serious — watch for fever, cough, or any sign of infection and call your doctor right away. · Corticosteroids can raise blood sugar, increase appetite and weight, weaken bones (osteoporosis), cause mood swings, and raise blood pressure. · Other immunosuppressants can lower blood counts (white cells, red cells, platelets) — regular blood tests are essential. · Can cause stomach upset — take with food. · Do NOT stop suddenly — the dose must be lowered slowly to let your body adjust. · Needs close follow-up with a cardiologist and often a rheumatologist or immunologist.

Antiarrhythmic drug (if needed) — for example, amiodarone · Antiarrhythmic (drug that stabilizes the heart's electrical rhythm)

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.

1. Inflammation in the heart disrupts the normal flow of electrical signals, causing irregular or fast heartbeats.
2. The antiarrhythmic drug (like amiodarone) blocks sodium, potassium, and calcium channels in heart muscle cells.
3. This slows down the speed of electrical signals and makes cells less likely to fire off chaotic beats.
4. The heart rhythm becomes slower and more regular.
5. This prevents sudden dangerous rhythms that could cause fainting, cardiac arrest, or sudden death.

SAFETY WATCH

Can cause lung scarring (pulmonary fibrosis) with long-term use — your doctor will check your lungs regularly. · Can affect the thyroid gland — blood tests will monitor thyroid hormones. · Can cause liver problems — blood tests will track liver function. · Can make skin very sensitive to sunlight — use sunscreen and protective clothing. · Can interact with many other drugs — tell every doctor and pharmacist you are taking it. · Can slow heart rate too much or cause other rhythm problems — regular heart monitoring is needed.

IVIG (intravenous immunoglobulin) — in select cases, especially in children · Immunoglobulin therapy (antibodies from donated blood)

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.

1. The immune system is attacking the heart or reacting too strongly to an infection.
2. IVIG is infused into the bloodstream over several hours.
3. The donated antibodies bind to and neutralize harmful antibodies or block receptors on immune cells.
4. The immune attack on the heart slows down.
5. Inflammation goes down, and the heart muscle may begin to recover.
6. The effect lasts weeks to months.

SAFETY WATCH

Can cause headache, fever, chills, or muscle aches during or after the infusion. · Rarely causes blood clots, kidney damage, or allergic reactions. · Can cause fluid overload if the heart is already very weak — given slowly and monitored closely. · Made from human blood, so there is a very small risk of transmitting infections (screened carefully but not zero risk). · Expensive and not always covered by insurance. · Only given in the hospital or infusion center under close supervision.

Red flags

- 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

Nursing priorities

1. Place patient on continuous cardiac telemetry monitoring and check rhythm strip every four hours for heart blocks or ventricular arrhythmias — 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 — 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 — 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)
4. Measure strict intake and output every shift and weigh patient daily at same time to detect fluid overload — Damaged myocardium cannot pump efficiently so fluid backs up into the lungs causing pulmonary edema (fluid flooding the lungs) (water in lung tissue); early detection of volume overload guides diuretic dosing (water pill amounts) before respiratory failure develops
5. Enforce strict bed rest with bedside commode privileges only and no Valsalva maneuvers like straining during bowel movements — Physical exertion increases myocardial oxygen demand (heart muscle needs more oxygen) when the inflamed tissue is already injured and cannot meet those demands; activity can trigger fatal arrhythmias or extend the zone of tissue death (necrosis) (area of dead tissue)
6. Administer ACE inhibitors or ARBs and beta-blockers exactly on schedule even if blood pressure is low-normal — These medicines reduce ventricular remodeling (permanent scarring and enlargement of heart chambers) by blocking neurohormonal activation (stress chemicals like aldosterone and norepinephrine); early use prevents progression to dilated cardiomyopathy (permanent weak stretched heart)
7. Assess lung sounds every four hours and immediately report new crackles or increased work of breathing — Crackles (wet popping sounds) indicate pulmonary edema (fluid flooding the lungs) from acute heart failure; myocarditis can rapidly progress to cardiogenic shock (heart cannot pump enough blood) requiring urgent escalation to intensive care for inotropes (medicines that strengthen heart contractions) or mechanical support

8. Hold all NSAIDs like ibuprofen and notify provider if patient requests pain medication — NSAIDs (non-steroidal anti-inflammatory drugs) worsen myocardial inflammation and increase a patch of dead tissue (infarct) size (area of heart damage) by blocking protective prostaglandins (chemicals that help healing); acetaminophen is the safe alternative for pain or fever

Sources

- 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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