

Postpartum Cardiomyopathy

A plain-English guide for you and your family

WHAT IS HAPPENING

During pregnancy, your heart works much harder than usual—it pumps about fifty percent more blood every minute to feed both you and the baby, your blood volume swells, and your heart rate climbs. In postpartum cardiomyopathy, the left ventricle (the heart's main pumping chamber) suddenly weakens in the last month of pregnancy or anytime in the five months after delivery. No one knows exactly why it happens, but leading theories point to a toxic piece of prolactin (the hormone that makes breast milk), the immune system attacking the heart by mistake, inflammation from a virus, or the overwhelming stress pregnancy puts on a heart that was already vulnerable. Because the heart cannot squeeze hard enough anymore, blood backs up into the lungs instead of moving forward to the body, so you feel short of breath when you lie flat, your legs and feet puff up with fluid, you feel exhausted, and your heart races trying to keep up. Early on, these signs can hide inside the normal swelling and faster heartbeat of any pregnancy, which is why postpartum cardiomyopathy is often missed until it gets severe. The good news is that many women recover fully with treatment—diuretics to drain the extra fluid, beta-blockers to slow the heart, blood-pressure drugs that are safe during and after pregnancy, and possibly bromocriptine to block prolactin after delivery. Until the heart heals, you need close monitoring and sometimes even devices like a wearable defibrillator, because the weakened heart can throw dangerous rhythms or form clots. Every weird finding—breathlessness, swelling, racing pulse—is your body heroically trying to keep oxygen and blood moving while the pump is broken. Trace it backward: you cannot breathe flat because blood is backed up in your lungs, and blood is backed up because the left ventricle is too weak to push it forward. Treat the weak pump, and all the dominoes stop falling.

What you might feel

- Shortness of breath, especially when lying flat or during activity
- Swelling in the legs, ankles, and feet
- Extreme tiredness and weakness
- Fast or irregular heartbeat (palpitations)
- Cough that may bring up pink, frothy mucus

- Chest discomfort or pressure
- Waking up at night gasping for air
- Trouble concentrating or feeling confused

Call the doctor right away if...

- Sudden, severe shortness of breath or gasping for air (may mean fluid is flooding the lungs fast)
- Chest pain or pressure that does not go away (could mean the heart itself is starving for oxygen or another emergency)
- Fainting or near-fainting (the brain is not getting enough blood, which can be dangerous)
- Coughing up pink or bloody foam (sign of severe fluid backup in the lungs)
- New confusion, slurred speech, or one-sided weakness (could mean a stroke from a blood clot that formed in the weak heart)

What the care team will do

- Monitor continuous pulse oximetry and keep oxygen saturation above 92 percent with supplemental oxygen if needed
- Place patient in high Fowler position (sitting up at 60 to 90 degrees) or position of comfort
- Measure strict intake and output every shift and weigh patient daily at the same time with same scale
- Place patient on continuous cardiac monitoring and watch for ventricular arrhythmias or atrial fibrillation (an irregular, quivering heartbeat)
- Check blood pressure before giving each dose of ACE inhibitor, ARB, ARNI, or beta-blocker and hold if systolic below 90
- Assess for signs of blood clots including sudden chest pain, one-sided leg swelling, or new confusion
- Monitor potassium and creatinine labs before and during treatment with diuretics and spironolactone
- Educate patient to avoid NSAIDs like ibuprofen for postpartum pain and use acetaminophen instead

Medicines you may be given — and why

Metoprolol or carvedilol (beta-blockers)

These drugs block adrenaline (the body's 'go-faster' hormone) from locking onto special docking sites (beta receptors) on the heart muscle. When adrenaline can't dock, the heart slows down and pumps less forcefully, which gives the tired, stretched heart muscle a chance to rest and recover. Over weeks to months, the heart muscle can remodel itself—shrinking back toward normal size and pumping better. Beta-blockers also lower blood pressure and reduce dangerous irregular heartbeats.

Hydralazine plus isosorbide dinitrate (combination vasodilators, safe during pregnancy and breastfeeding)

These two drugs work as a team to relax and widen blood vessels—like opening more lanes on a highway so blood flows more easily. Hydralazine relaxes the muscle rings around arteries (the vessels that carry blood away from the heart), lowering the pressure the heart has to pump against. Isosorbide dinitrate releases nitric oxide (a natural gas signal in the body) that relaxes veins (the vessels that return blood to the heart), reducing the amount of blood pooling in the lungs and body. Together they cut the heart's workload from both ends. This combination is especially important during pregnancy because the safer long-term drugs (ACE inhibitors and ARBs) can harm the developing baby.

Furosemide or bumetanide (loop diuretics, 'water pills')

These drugs tell the kidneys to pull extra salt and water out of the blood and send it to the bladder as urine—like opening a drain to lower the water level in an overflowing sink. When the weakened heart can't pump well, fluid backs up into the lungs (causing shortness of breath) and into the legs, belly, and liver (causing swelling). Loop diuretics quickly remove that extra fluid, relieving congestion and making it easier to breathe and move. They act on a specific part of the kidney's filtering tube (the loop of Henle) where most salt reabsorption happens.

Digoxin

Digoxin makes each heartbeat a little stronger by shifting the balance of sodium and calcium inside heart muscle cells—like turning up the volume on a weak speaker. It blocks a pump (the sodium-potassium ATPase) on the cell surface, which causes sodium to build up inside the cell. Higher sodium forces another transporter to push out less calcium, so calcium stays in the cell longer. Extra calcium strengthens the squeeze of each heartbeat. Digoxin also slows electrical signals through the heart's relay station (the AV node), which helps control a fast or irregular heart rhythm (such as atrial fibrillation (an irregular, quivering heartbeat)) that often accompanies heart failure.

Enalapril, lisinopril, or other ACE inhibitor (AFTER delivery only—never during pregnancy)

ACE inhibitors block an enzyme (ACE) that turns a weak hormone (angiotensin I) into a powerful one (angiotensin II). Angiotensin II squeezes blood vessels tight, raises blood pressure, tells the adrenal glands to release aldosterone (a hormone that makes the kidneys hold onto salt and water), and over time causes the heart muscle to thicken and scar. By blocking ACE, these drugs let blood vessels stay relaxed, reduce fluid retention, and slow harmful remodeling of the heart. In large studies, ACE inhibitors help people with heart failure live longer, stay out of the hospital, and feel better. However, they can cause severe birth defects and must NEVER be used during pregnancy.

Losartan, valsartan, or other ARB (angiotensin receptor blocker; AFTER delivery only—never during pregnancy)

ARBs block the docking site (the AT1 receptor) where angiotensin II lands to do its work. Even though angiotensin II is still made, it cannot squeeze blood vessels, release aldosterone, or send 'grow and scar' signals to the heart because it is locked out of its receptor. The effect is very similar to an ACE inhibitor—relaxed vessels, lower blood pressure, less fluid retention, and better heart remodeling—but ARBs rarely cause the dry cough seen with ACE inhibitors. They are used when someone cannot tolerate an ACE inhibitor or sometimes combined with one (though combination therapy is less common now that newer drugs are available). Like ACE inhibitors, ARBs improve survival and reduce hospitalizations in heart failure, but they must NEVER be used during pregnancy.

Sacubitril/valsartan (ARNI: angiotensin receptor-neprilysin inhibitor; AFTER delivery only)

This drug combines an ARB (valsartan) with a neprilysin inhibitor (sacubitril) in one pill. Neprilysin is an enzyme that breaks down helpful natural peptides—BNP, ANP, and bradykinin—which relax blood vessels, help the kidneys release salt and water, and protect the heart. By blocking neprilysin, sacubitril lets these good peptides hang around longer and keep working. Valsartan blocks angiotensin II (the harmful hormone), while sacubitril boosts the helpful ones. The result is stronger blood-vessel relaxation, better fluid removal, and improved heart remodeling compared to an ACE inhibitor or ARB alone. Large trials show sacubitril/valsartan reduces death and hospitalization more than enalapril in people with heart failure and low ejection fraction, so current guidelines recommend it as first-line therapy after delivery.

Spironolactone or eplerenone (mineralocorticoid receptor antagonists, MRAs)

These drugs block aldosterone, a hormone made by the adrenal glands that tells the kidneys to hold onto salt and water and dump potassium. In heart failure, aldosterone levels stay high even when they should not be, driving fluid retention, high blood pressure, and scarring

(fibrosis) of the heart muscle. MRAs sit on the aldosterone receptor in the kidney and heart and prevent aldosterone from landing. This lets the kidneys release more salt and water (acting like a mild diuretic), keeps potassium in the body, and—most importantly—reduces harmful remodeling and scarring in the heart. Studies show MRAs improve survival in heart failure when added to ACE inhibitors (or ARBs) and beta-blockers. Spironolactone is older and cheaper but can cause breast tenderness and enlargement in men and women; eplerenone is more selective and causes fewer hormone-related side effects.

Dapagliflozin, empagliflozin, or other SGLT2 inhibitor

SGLT2 inhibitors were first designed to lower blood sugar in diabetes, but researchers discovered they also powerfully protect the heart and kidneys in people with or without diabetes. These drugs block a transporter (SGLT2) in the kidney that reabsorbs glucose and sodium from the urine back into the blood. When SGLT2 is blocked, glucose and sodium spill into the urine and are lost, which lowers blood sugar (if it is high) and causes a gentle, sustained loss of salt and water—like a mild, steady diuretic. This reduces blood pressure and fluid overload. Beyond that, SGLT2 inhibitors shift the heart's fuel use toward more efficient pathways (burning ketones and fat instead of only glucose), reduce inflammation, protect kidney filtering units, and improve the stiffness of the heart muscle. In major trials, they cut heart-failure hospitalizations and death even when added to all the standard drugs. Current guidelines recommend them for everyone with heart failure and low ejection fraction, regardless of whether they have diabetes.

Bromocriptine (sometimes used, especially if prolactin is elevated or breastfeeding is not planned)

Bromocriptine lowers prolactin, the hormone that drives milk production. Some data suggest that high prolactin may contribute to postpartum cardiomyopathy in certain women by affecting heart metabolism or blood vessel function, though the mechanism is not fully understood and remains debated. Bromocriptine binds to dopamine receptors in the pituitary gland (the pea-sized hormone control center at the base of the brain) and mimics dopamine, the brain chemical that naturally suppresses prolactin. When prolactin drops, milk production stops. Some small studies and case reports suggest bromocriptine may speed heart recovery in women with postpartum cardiomyopathy, especially if started early. However, large randomized trials are lacking, and guidelines do not universally recommend it. Bromocriptine is most often considered when the heart is severely weakened, prolactin is unusually high, or the woman chooses not to breastfeed. It is NOT standard for all cases.

Anticoagulation (apixaban, rivaroxaban, warfarin, or enoxaparin) if ejection fraction is very low (below 30%) or if a clot or atrial fibrillation (an irregular, quivering heartbeat) is present

When the heart muscle is very weak and pumps poorly (ejection fraction below 30–35%), blood sloshes slowly in the heart chambers instead of being pushed out efficiently—like water sitting still in a bucket instead of flowing through a hose. Slow-moving blood can clot, forming a blood clot (thrombus) (clot) inside the heart. If a piece breaks off, it can travel to the brain (causing a stroke), lungs (pulmonary a traveling clot lodging in a vessel (embolism)), kidneys, or legs. Anticoagulants interfere with the clotting cascade—a chain of proteins in the blood that normally form clots to stop bleeding. By blocking one or more steps in that chain, these drugs keep the blood thinner and prevent clots from forming. They do NOT dissolve clots that are already there (thrombolytics do that), but they stop new ones. Warfarin blocks vitamin K, which several clotting factors need to be made. Direct oral anticoagulants (DOACs) like apixaban and rivaroxaban directly block specific clotting proteins (factor Xa or thrombin). Enoxaparin (low-molecular-weight heparin) is injected and used short-term or when pills cannot be taken.

Where this information comes from

- ESC Heart Failure Guidelines 2021 (European Society of Cardiology)
- AHA/ACC Heart Failure Guideline 2022
- Cochrane Review on Peripartum Cardiomyopathy
- ACOG Practice Bulletin on Cardiac Disease in Pregnancy

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