

Antepartum Triage and Urgent Maternal Warning Signs

Three pregnancy emergencies you must never miss: preeclampsia (high blood pressure damaging organs), placental abruption (the placenta tears off the uterus wall too early), and pulmonary embolism (a clot lodging in a lung artery) (a blood clot blocking the lung arteries).

THE ONE-LINE TRUTH

Pregnancy reshapes how the body works, so normal changes can look like sickness, and real emergencies can hide behind normal-looking vital signs until the mother suddenly crashes.

The chain — how it unfolds

1. Abnormal placental implantation and spiral artery remodeling failure occur in early pregnancy, before symptoms appear. — why: In normal pregnancy, fetal trophoblast cells (cells from the developing placenta) invade the uterine lining and spiral arteries (the coiled arteries in the uterus that feed the placenta) during the first trimester, replacing the muscular artery walls with a loose matrix, transforming them into high-flow, low-resistance channels that can deliver massive blood flow without clamping down. In preeclampsia, this remodeling fails—spiral arteries keep their muscle layer and stay narrow and reactive—so the placenta receives far less blood than it needs and becomes blood-starved (ischemic) (starved of oxygen).
2. The blood-starved (ischemic) placenta releases anti-angiogenic factors (proteins that damage blood vessels) into maternal circulation. — why: The oxygen-starved placenta tries to signal for more blood supply by secreting proteins called sFlt-1 (soluble fms-like tyrosine kinase-1) and soluble endoglin. These proteins act as decoys—they bind and neutralize VEGF (vascular endothelial growth factor) and PlGF (placental growth factor), which are essential for maintaining healthy endothelial cells lining blood vessels. Without VEGF and PlGF, endothelial cells throughout the mother's body become damaged and dysfunctional.

3. Endothelial dysfunction causes systemic vasoconstriction (blood vessels tighten everywhere) and increased vascular permeability (vessel walls leak). — why: Damaged endothelium loses its ability to produce nitric oxide (a molecule that keeps vessels relaxed and wide). The vessels constrict, and the body must raise blood pressure to force blood through the narrowed channels. At the same time, the damaged vessel walls become leaky, allowing protein and fluid to spill out into tissues (causing swelling (edema), or swelling) and into urine (proteinuria). The kidneys develop glomerular endotheliosis (swelling and damage to the glomeruli—the kidney's tiny filters), which restricts blood flow and makes the kidneys release even more renin (a hormone that triggers a cascade raising blood pressure) and aldosterone (a hormone that tells the kidneys to hold on to salt and water), driving blood pressure higher and causing salt and water retention.
4. High blood pressure (Hypertension) and endothelial damage injure multiple organ systems in a predictable pattern. — why: The liver develops periportal tissue death (necrosis) (liver cells die in zones around the portal veins—the vessels bringing blood into the liver) and, in severe cases, a subcapsular a pooled collection of blood (hematoma) (bleeding forms a pocket under the liver's outer capsule), causing right upper quadrant or epigastric pain (pain under the right ribs or in the upper middle belly) and elevated AST and ALT (liver enzymes spilling into blood from dying cells). Kidneys lose filtration capacity as glomeruli clog with swollen endothelial cells, causing proteinuria (300 milligrams or more in twenty-four hours, or a protein-to-creatinine ratio of 0.3 or higher) and rising creatinine (above 1.1 mg/dL, which is high for pregnancy). Platelets stick to damaged endothelium throughout the body and are consumed faster than the bone marrow can make new ones, causing a low platelet count (thrombocytopenia) (platelet count below 100,000 per microliter). In HELLP syndrome (Hemolysis, Elevated Liver enzymes, Low Platelets), red blood cells are sheared apart as they squeeze through damaged small vessels (microangiopathic red blood cell destruction (hemolysis)—mechanical destruction of red cells), releasing LDH (lactate dehydrogenase, an enzyme from inside broken cells) and unconjugated bilirubin (a yellow waste product from red cell breakdown) and dropping haptoglobin (a protein that normally binds free hemoglobin; low levels signal red blood cell destruction).

5. Cerebral swelling (edema) (brain swelling) and posterior reversible encephalopathy syndrome (PRES) develop when severe high blood pressure (hypertension) overwhelms the brain's protective mechanisms. — why: The brain normally autoregulates its blood flow—when systemic blood pressure rises, cerebral arteries constrict to keep brain blood flow (perfusion) (blood flow to brain tissue) constant and protect delicate capillaries from high pressure. But when blood pressure climbs too high or too fast, autoregulation fails. High-pressure blood forces fluid across the blood-brain barrier (the tight seal between blood vessels and brain tissue) into brain tissue, especially in the occipital lobes (the back of the brain, which process vision) and parietal lobes (the sides of the brain, which process sensation and spatial awareness). This causes vasogenic swelling (edema) (fluid swelling around brain cells, not inside them), which shows up clinically as severe headache (unrelieved by acetaminophen, or Tylenol), visual changes (blurry vision, scotomata—dark spots in vision—flashing lights, or cortical blindness—the eyes work but the brain can't process the images), and overactive reflexes (hyperreflexia) (overactive deep tendon reflexes, like an exaggerated knee-jerk, with rhythmic muscle jerking (clonus)—rhythmic muscle jerking when you stretch the ankle—a sign of central nervous system irritability). If untreated, the swelling triggers an eclamptic seizure (generalized tonic-clonic seizure—the whole body stiffens, then jerks—often followed by in the groggy phase after a seizure (postictal) confusion or coma, the drowsy, confused state after a seizure).
6. The systemic inflammatory and prothrombotic state increases the risk of venous thromboembolism and can trigger placental abruption. — why: Endothelial damage, platelet activation (platelets become sticky and clump), and elevated clotting factors create a prothrombotic state—blood clots too easily. A clot in a deep leg vein (Deep vein thrombosis) (DVT—a clot in a leg or pelvic vein) can form; if a clot breaks off, it travels to the lungs as a pulmonary embolism (a clot lodging in a lung artery). At the same time, poor placental blood flow (perfusion), vessel fragility (weakened blood vessels), and high blood pressure (hypertension) increase the risk that a vessel in the decidua basalis (the uterine lining under the placenta) will rupture, causing blood to dissect (spread) between the placenta and uterus (placental abruption). The bleeding and endothelial injury consume clotting factors and activate the coagulation cascade (the chain reaction that forms clots) everywhere, triggering DIC (disseminated intravascular coagulation), in which the body forms clots in small vessels throughout the body and simultaneously bleeds uncontrollably because clotting factors and platelets are used up.

7. Delivery of the placenta removes the source of anti-angiogenic factors and begins the resolution process. — why: Once the blood-starved (ischemic) placenta is removed—either by vaginal delivery or cesarean section—the mother's blood levels of sFlt-1 and soluble endoglin drop rapidly. Without these anti-angiogenic proteins attacking endothelial cells, the blood vessel linings start to heal. Blood pressure normalizes over days to weeks, proteinuria resolves (though it may take months), and platelet counts and liver enzymes return to normal. Kidney function usually recovers, though some women are left with chronic kidney disease and a higher long-term risk of cardiovascular disease (high blood pressure, stroke, heart failure) because the endothelial injury leaves lasting damage. This is why preeclampsia is not just a pregnancy problem—it is a window into future cardiovascular risk.
8. If delivery is delayed in severe preeclampsia, the risk of maternal death from stroke, liver rupture, eclampsia, pulmonary edema (fluid flooding the lungs), DIC, or acute kidney injury climbs steeply, and the fetus faces low oxygen (hypoxia), growth restriction, and stillbirth. — why: The only cure for preeclampsia is delivery. Temporizing measures—antihypertensives (medicines that lower blood pressure—labetalol, hydralazine, nifedipine—given to keep blood pressure below 160/110 and prevent stroke) and magnesium sulfate (given to raise the seizure threshold and prevent eclampsia)—buy time to give corticosteroids (betamethasone or dexamethasone shots) for fetal lung maturity if gestational age is less than thirty-four weeks, but they do not stop the underlying disease. If the pregnancy continues, organ damage accumulates, and the risk of catastrophic complications rises. Delivery is indicated (must happen) immediately if there are signs of maternal instability (uncontrolled severe high blood pressure (hypertension), eclampsia, pulmonary edema (fluid flooding the lungs), DIC, renal failure, hepatic a pooled collection of blood (hematoma)—blood pooling under the liver capsule—or rupture, or placental abruption) or fetal compromise (non-reassuring fetal heart tracing—late decelerations, loss of variability, or a slow heart rate (bradycardia); severe growth restriction—the baby is much smaller than expected; reversed end-diastolic flow on umbilical artery Doppler—blood flow in the cord actually moves backward during part of the heartbeat, a sign the placenta is failing; or oligohydramnios—too little amniotic fluid, a sign the baby's kidneys aren't making enough urine because of poor blood flow).

Spot it — what you see, and why

- Severe headache that does not go away with acetaminophen (Tylenol): Very high blood pressure pushes extra fluid through the walls of blood vessels in the brain. This fluid builds up inside the skull and swells the brain tissue, squeezing the nerves that sense pain. The pounding headache is a warning that the brain is under dangerous pressure and a seizure could happen soon.

- Vision problems (blurry vision, seeing spots or flashing lights, blind spots, bright light hurts your eyes, or complete loss of vision): Very high blood pressure makes the tiny blood vessels in the back of the eye and the occipital cortex (the part of the brain that reads what you see) squeeze tight or leak fluid. This swelling in the seeing center of the brain scrambles the signals from your eyes. The vision changes usually go away after delivery, but they are a danger sign that the brain is swollen.
- Sharp pain in the right upper belly or just below the ribs in the middle (the epigastric area): The liver swells and tiny spots of liver tissue die because the blood vessels inside the liver are damaged and blood flow is poor. Sometimes blood pools under the liver's outer covering (the capsule), stretching it like an overfilled water balloon. This stretching irritates the nerves around the liver and causes sharp, stabbing pain. It means the liver is in serious trouble.
- Overactive reflexes when the doctor taps your knee or ankle, sometimes with rhythmic twitching (clonus) of the foot: The brain and spinal cord become jumpy because swelling and low magnesium make the nerves fire too easily. The nervous system is on high alert, so reflexes are too strong—the knee jerks higher and faster than normal, and the foot may twitch several times instead of once. This tells the doctor that a seizure could happen at any moment.
- Sudden trouble breathing or chest pain that feels different from the normal shortness of breath in late pregnancy: Fluid leaks through damaged blood vessel walls into the air sacs of the lungs (pulmonary swelling (edema)), so oxygen cannot cross into the blood—like trying to breathe through a wet sponge. Or a blood clot travels to the lungs (pulmonary a traveling clot lodging in a vessel (embolism)) and blocks oxygen from getting to the body. Either way, you feel like you are drowning or suffocating. This is a life-threatening emergency.
- Blood pressure of 140 over 90 or higher that starts after 20 weeks of pregnancy: The lining of blood vessels (the endothelium) is damaged during pregnancy by toxins released from the placenta. The vessels squeeze tight (vasoconstriction) instead of staying relaxed. This makes the heart pump harder to push blood through narrower tubes. The blood pressure climbs because the body is trying to deliver enough blood to all the organs through these tight vessels.

Red flags — act now

- Blood pressure of 160 over 110 or higher that stays high for 15 minutes or more—the brain can have a stroke (bleeding or blood vessel rupture), so blood pressure medicine (labetalol or hydralazine IV, or nifedipine by mouth) must be given within 30 to 60 minutes

- A seizure (eclampsia) that happens for the first time during pregnancy or in the first few days after delivery—this means the brain is severely swollen; the airway must be protected (turn her on her side so she does not choke), magnesium sulfate given through the IV to stop more seizures, and the baby delivered as soon as it is safe
- Platelet count below 100,000 per microliter or platelets dropping fast—this means the blood cannot clot properly and dangerous bleeding could happen during delivery or surgery
- Liver enzyme levels (AST or ALT) more than twice the normal limit, especially with sharp right upper belly pain—the liver could rupture (liver a pooled collection of blood (hematoma) rupture) and cause life-threatening internal bleeding into the belly
- Fluid in the lungs (pulmonary swelling (edema)) or oxygen level below 95 percent on room air—the heart and lungs are failing and intensive care with oxygen, diuretics, or even a breathing tube is needed immediately

Treat it — in this order

1. IV labetalol 20 mg over 2 minutes, then double the dose every 10 minutes (20 mg, then 40 mg, then 80 mg, max total 300 mg); OR IV hydralazine 5 to 10 mg every 20 minutes; OR oral immediate-release nifedipine 10 to 20 mg every 20 minutes until blood pressure is sustained below 160 systolic and below 110 diastolic within 60 minutes → Lowers systolic and diastolic pressure by relaxing the smooth muscle layer inside blood vessel walls, which makes the vessels widen. This reduces shear stress (the scraping force of blood flowing against the inner lining) on the endothelium and lowers the risk of maternal stroke, bleeding inside the brain (intracerebral bleeding (hemorrhage)), and worsening kidney and liver injury. The goal is to bring pressure down to a safer range (140 to 150 systolic, 90 to 100 diastolic) without dropping it so low that the placenta gets even less blood.
2. IV magnesium sulfate: 4 to 6 grams loading dose given over 15 to 20 minutes, followed by continuous infusion at 1 to 2 grams per hour for seizure prevention in preeclampsia with severe features, or for treatment if an eclamptic seizure occurs → Raises the seizure threshold by blocking NMDA receptors (gates on nerve cells in the brain that normally let calcium rush in and fire the cell) and stabilizing neuronal membranes, which prevents eclamptic seizures. Magnesium sulfate reduces seizure risk by more than 50 percent compared to no treatment and works better than phenytoin or benzodiazepines for eclampsia prevention (Magpie Trial, Lancet 2002; Cochrane 2010; ACOG 2020). It does not treat high blood pressure.

3. Expedited delivery by labor induction (if the cervix is favorable and the fetus is stable) or cesarean section (if cervix is unfavorable, fetus is in distress, or maternal condition is critical) after maternal stabilization with magnesium sulfate and blood pressure medication; or immediate delivery without delay if eclampsia, placental abruption, non-reassuring fetal status, or HELLP syndrome is present → Removes the diseased placenta, which is the source of antiangiogenic factors (sFlt-1 and soluble endoglin, proteins that damage the endothelium and cause blood vessels to narrow and leak). Within 24 to 48 hours after delivery, blood pressure begins to fall, platelet count rises, liver enzymes drop, and kidney function starts to recover. Delivery is the only definitive treatment for preeclampsia.
4. Betamethasone 12 mg intramuscular injection every 24 hours for 2 doses, OR dexamethasone 6 mg intramuscular injection every 12 hours for 4 doses, if gestational age is 24 weeks 0 days to 36 weeks 6 days and delivery is expected within the next 7 days → Speeds up fetal lung maturation by stimulating type II pneumocytes (specialized cells lining the air sacs in the lungs) to produce surfactant (a soapy substance that keeps air sacs open and prevents them from collapsing when the baby exhales). This reduces neonatal respiratory distress syndrome by 30 to 50 percent, lowers bleeding inside the baby's brain (intraventricular bleeding (hemorrhage)), and cuts neonatal death (Cochrane 2020, ACOG 2021). Maximum benefit happens if delivery occurs 24 hours to 7 days after the first dose.
5. Therapeutic-dose low-molecular-weight heparin (enoxaparin 1 mg per kilogram subcutaneously every 12 hours, or dalteparin 100 units per kilogram every 12 hours) if a clot in a deep leg vein (deep vein thrombosis) or pulmonary embolism (a clot lodging in a lung artery) is confirmed on imaging; OR preventive (prophylactic)-dose enoxaparin (40 mg once daily or 30 mg twice daily) if high clinical suspicion exists before imaging or if risk factors are present → Blocks factor Xa (a key enzyme in the clotting cascade, the step-by-step chemical chain that forms blood clots), which stops existing clots from growing and prevents new clot formation. Pregnancy raises venous blood clot risk five to six times because of hypercoagulability (the blood clots more easily because clotting factors go up and natural anticoagulants go down), venous stasis (blood pools in the legs because the uterus presses on the inferior vena cava, the big vein returning blood to the heart), and endothelial injury (ACOG 2018, ASH 2018).

Your top priorities at the bedside

1. Obtain blood pressure manually in both arms within 2 minutes of arrival, then repeat in 15 minutes if the first reading is 140 over 90 or higher. — why: Sustained severe high blood pressure (hypertension) (blood pressure 160 systolic or 110 diastolic lasting 15 minutes) causes cerebral autoregulation to fail (brain blood vessels cannot protect themselves), leading to stroke, so antihypertensive medicine must start within 60 minutes.

2. Start continuous fetal heart monitoring and uterine contraction monitoring immediately. — why: The placenta (organ feeding the baby) is very sensitive to high maternal blood pressure and can stop delivering enough oxygen, causing late decelerations (the baby's heart rate drops after contractions) or prolonged a slow heart rate (bradycardia) (slow heart rate below 110 for more than 2 minutes), signaling fetal distress that may require emergency delivery.
3. Establish large-bore IV access (18-gauge or bigger) in each arm and draw labs: complete blood count, comprehensive metabolic panel, liver enzymes (AST and ALT), and platelet count. — why: HELLP syndrome (hemolysis, elevated liver enzymes, low platelets) can develop rapidly; a low platelet count (thrombocytopenia) (low platelet count below 100,000) increases bleeding risk during delivery, and two IV lines allow simultaneous magnesium sulfate infusion and antihypertensive bolus dosing without delay.