

Placental Abruption

Placental abruption means the placenta tears away from the uterus before delivery. The baby loses oxygen. The mother can bleed heavily. This is a true emergency.

THE ONE-LINE TRUTH

The placenta peels away from the wall of the uterus before the baby is born, cutting off the baby's oxygen and causing dangerous bleeding.

The chain — how it unfolds

1. A trigger—high blood pressure, preeclampsia, trauma, cocaine, smoking, or rapid fluid loss —damages the decidua or shears the placenta — why: Chronic high blood pressure or preeclampsia makes the tiny spiral arteries brittle and prone to rupture; a sudden blow to the belly tears the placenta loose by inertia; cocaine makes vessels squeeze and burst; smoking weakens the decidua over time; and a sudden gush of amniotic fluid shrinks the uterus so fast it pulls the wall away from the placenta. Any of these breaks the normal attachment.
2. A blood clot (retroplacental a pooled collection of blood (hematoma)) forms behind the placenta and grows, peeling the placenta farther off the wall — why: When maternal vessels in the decidua tear, blood pours into the space between the placenta and the uterine wall, clots, and acts like a wedge—pushing the placenta away and tearing more vessels, so the clot keeps growing and the separation spreads.
3. Dark red or maroon blood leaks out of the vagina, and constant, severe abdominal pain begins — why: Blood from the clot tracks down between the membranes and the uterine wall and drips out the cervix—it looks dark because it has been sitting and clotting. Blood also leaks into the uterine muscle itself (called a Couvelaire uterus), irritating the nerves and causing unrelenting, knife-like pain that does not come and go like contractions.
4. The uterus becomes rock-hard, tender, and does not relax (tetanic contraction) — why: Blood irritating the muscle wall triggers a sustained, tight squeeze—the uterus tries to clamp down on the bleeding vessels like a natural tourniquet. The uterus stays firm and painful to touch instead of softening between contractions.

5. The baby shows signs of distress—heart rate drops, variability disappears, or late decelerations appear — why: The detached portion of the placenta can no longer exchange oxygen, and the tetanic uterine contraction squeezes the spiral arteries and sometimes the umbilical cord, starving the baby of oxygen. The baby's brain senses low oxygen (hypoxia) (low oxygen) and slows the heart to conserve energy, producing late decelerations (heart rate drops after each contraction) or a flat, non-reactive tracing.
6. If bleeding continues, the mother's blood pressure drops, pulse races, skin turns cold and pale, and she goes into bleeding-driven (hemorrhagic) shock — why: Blood loss—either revealed (out the vagina) or concealed (trapped behind the placenta and in the uterine muscle)—shrinks the circulating blood volume. Once the body's compensatory reserve is exhausted, blood pressure falls, organs do not get enough oxygen, and shock begins—confusion, weak pulse, cold extremities, and low urine output.
7. DIC (disseminated intravascular coagulation) develops—clotting factors and platelets are consumed, and the mother bleeds from gums, IV sites, and internal organs — why: The damaged placenta releases tissue factor, a powerful trigger that activates the clotting cascade everywhere in the bloodstream instead of just at the injury site. Tiny clots form throughout the body, using up all the clotting proteins (fibrinogen, factors V and VIII) and platelets faster than the liver can make new ones. Once they are gone, the blood cannot clot anywhere, and the mother oozes blood from every puncture and mucous membrane.
8. Without immediate delivery and resuscitation, the baby dies from low oxygen (hypoxia) and the mother dies from bleeding-driven (hemorrhagic) shock or DIC — why: The placenta cannot reattach once it peels away, so the baby's oxygen supply is permanently cut—if not delivered quickly, the baby will suffer brain damage or death. The mother continues to bleed until the uterus is emptied and can contract fully, and until lost blood and clotting factors are replaced with transfusions. Every minute of delay increases the risk of fetal death, maternal hysterectomy to stop bleeding, kidney failure from shock, or death from DIC.

Spot it — what you see, and why

- Sudden, constant belly pain that does not go away: The placenta (the spongy organ that feeds the baby) tears away from the wall of the uterus (the womb), and the torn edge bleeds into the muscle. Blood trapped inside the uterus stretches and irritates the muscle wall, causing steady pain that does not come and go like a contraction.
- Vaginal bleeding (dark red or bright red): Blood from the torn placenta spills out through the cervix (the opening of the womb) and exits the vagina. The blood may be dark because it took time to leave, or bright red if it flows quickly. Sometimes all the blood stays trapped inside and you see no bleeding at all—called a concealed abruption.

- The uterus feels hard as a board and very tender when you press on it: Blood trapped between the placenta and the uterine muscle irritates the muscle, making it clamp down and stay tight (called tetanic contraction). The constant tightness makes the belly rock-hard instead of soft between contractions.
- The baby's heart rate tracing shows worrying patterns (late decelerations, loss of variability, or a slow heart rate (bradycardia)): The placenta is the baby's only source of oxygen. When part of it tears away, less oxygen reaches the baby through the umbilical cord. Low oxygen stresses the baby's heart, so the heart rate drops after each contraction (late decelerations), stops bouncing up and down (lost variability), or slows dangerously (bradycardia below 110 beats per minute).
- The mother's heart beats fast (tachycardia over 100 beats per minute): When you lose blood, your heart pumps faster to try to keep enough blood flowing to your organs. A fast heart rate (Tachycardia) is the body's first answer to bleeding—it shows up before blood pressure drops.
- The mother's blood pressure drops (hypotension, systolic below 90 mmHg): After losing a large amount of blood—often more than 500 to 1000 mL—the body runs out of tricks to keep pressure normal. Blood vessels have squeezed as tight as they can and the heart is beating as fast as it can, but there is simply not enough blood left in the system to fill the pipes, so pressure falls. This is a late, dangerous sign of severe bleeding.

Red flags — act now

- Heavy vaginal bleeding that soaks through a pad in less than an hour
- The baby's heart rate stays dangerously low (below 110 beats per minute) or shows late drops after every contraction
- The mother feels dizzy, sweaty, or confused—signs that her brain is not getting enough blood
- Blood pressure falls below 90/60 mmHg or the mother's pulse climbs above 120 beats per minute
- The mother's uterus stays rock-hard and does not soften at all between contractions

Treat it — in this order

1. Large-bore IV access and rapid infusion of crystalloid (normal saline or lactated Ringer's) followed by packed red blood cells if hemoglobin is low or patient is in shock → Replaces the blood volume lost behind the placenta and into the vagina, restoring blood flow to the mother's brain, heart, and kidneys. Stops shock before organs shut down.

2. Immediate delivery—vaginal if cervix is favorable and fetal status allows, or cesarean section if fetal distress or maternal instability → Removes the baby from a failing placenta that can no longer deliver oxygen, and stops the source of maternal bleeding by emptying the uterus so it can contract down and squeeze bleeding vessels shut.
3. Transfusion of fresh frozen plasma (FFP), cryoprecipitate, and platelets for documented or suspected DIC → Replaces fibrinogen, clotting factors, and platelets that have been consumed by the runaway clotting triggered by tissue factor from the torn placenta. Stops diffuse bleeding from mucous membranes, IV sites, and surgical incisions.
4. Continuous electronic fetal monitoring from admission until delivery → Tracks the baby's heart rate pattern in real time so you can catch signs of worsening low oxygen (hypoxia) (late decelerations, loss of variability, a slow heart rate (bradycardia)) and decide when delivery must happen immediately.
5. Antenatal corticosteroids (betamethasone 12 mg IM every 24 hours for two doses, or dexamethasone 6 mg IM every 12 hours for four doses) if gestational age is 24 to 34 weeks and delivery is not immediately necessary → Speeds up maturation of the baby's lungs so they can inflate and exchange oxygen after birth, reducing the risk of respiratory distress syndrome in a premature baby forced out early by abruption.

Your top priorities at the bedside

1. Place the patient on continuous electronic fetal heart rate monitoring immediately and watch for late decelerations, minimal variability, or a slow heart rate (bradycardia) — why: The baby depends completely on the placenta for oxygen, so when part of it tears away the baby gets less oxygen and the heart rate pattern shows distress before the baby dies, giving you minutes to act
2. Start two large-bore IV lines, 18-gauge or bigger, and infuse normal saline or lactated Ringer's wide open while you wait for the doctor — why: Abruptio can cause massive bleeding into the uterus and out the vagina, so you need big IV catheters to push fluid and blood fast enough to keep her blood pressure up and her organs perfused with oxygen
3. Draw blood immediately for type and crossmatch, complete blood count, PT, PTT, fibrinogen, and a clot test where you put her blood in a red-top tube and watch if a clot forms in 6 minutes then stays solid — why: Abruptio releases clotting factors from the damaged placenta into her bloodstream, using up all her clotting ability in a condition called DIC (disseminated intravascular coagulation) where she bleeds from everywhere, and the bedside clot test shows this before lab results return