1. Preeclampsia

Preeclampsia begins with abnormal development of the placenta during early pregnancy. Normally, the spiral arteries of the uterus widen to provide enough blood supply to the placenta. In preeclampsia, these arteries remain narrow, causing decreased placental perfusion and placental ischemia. Because the placenta is deprived of oxygen, it releases inflammatory substances into the maternal circulation that damage the endothelial lining of blood vessels.

Endothelial injury causes generalized vasospasm and increased vascular permeability. Vasospasm increases systemic vascular resistance, leading to hypertension. Increased permeability allows plasma proteins and fluid to leak into tissues, causing edema. Damage to kidney glomeruli decreases filtration and allows protein to leak into urine, resulting in proteinuria. Reduced blood flow to organs can also affect the liver, brain, and placenta, causing complications such as headache, visual disturbances, elevated liver enzymes, and fetal growth restriction.

---

2. Eclampsia

Eclampsia is the progression of severe preeclampsia in which cerebral involvement leads to seizures. Severe vasospasm and endothelial damage decrease blood flow to the brain and increase capillary permeability. This results in cerebral edema, ischemia, and irritation of brain tissue.

As cerebral edema worsens, neurons become unstable and excessively excitable, triggering generalized tonic-clonic seizures. During seizures, oxygen delivery to both mother and fetus decreases, increasing the risk of hypoxia, placental abruption, and maternal organ failure.

---

3. Gestational Hypertension

Gestational hypertension develops after 20 weeks of pregnancy without proteinuria. Pregnancy normally increases blood volume and cardiac output. In some women, vascular sensitivity to vasoconstrictors becomes exaggerated, causing increased peripheral resistance.

The blood vessels constrict, increasing arterial pressure. Unlike preeclampsia, endothelial damage and proteinuria are absent. However, if placental perfusion becomes impaired, gestational hypertension may progress to preeclampsia.

---

4. Chronic Hypertension

Chronic hypertension exists before pregnancy or develops before 20 weeks gestation. Persistent vasoconstriction increases systemic vascular resistance, forcing the heart to pump harder to maintain circulation.

Over time, constant high pressure damages blood vessels and decreases organ perfusion. During pregnancy, the already compromised vessels may not adapt well to increased blood volume, increasing the risk for placental insufficiency, fetal growth restriction, and superimposed preeclampsia.

---

5. HELLP Syndrome

HELLP syndrome is a severe form of preeclampsia characterized by hemolysis, elevated liver enzymes, and low platelets. Endothelial injury activates the coagulation cascade and causes fibrin deposition in small blood vessels.

As red blood cells pass through narrowed vessels containing fibrin strands, they become fragmented and destroyed, resulting in hemolysis. Platelets are consumed during abnormal clotting, causing thrombocytopenia. Reduced liver blood flow and microvascular injury lead to hepatic ischemia and liver cell damage, elevating liver enzymes. Severe liver swelling may even cause hepatic rupture.

---

6. Iron Deficiency Anemia in Pregnancy

During pregnancy, maternal blood volume expands significantly to support fetal growth. Iron demand increases because iron is needed for fetal development and increased maternal RBC production.

If dietary iron intake is inadequate, hemoglobin synthesis decreases. Red blood cells become smaller and contain less hemoglobin, reducing oxygen-carrying capacity. Tissue hypoxia develops, causing fatigue, pallor, dizziness, and weakness. The fetus may also receive less oxygen and nutrients.

7. Megaloblastic Anemia

Megaloblastic anemia is usually caused by folate or vitamin B12 deficiency. These vitamins are essential for DNA synthesis during RBC production in the bone marrow.

Without adequate DNA synthesis, cell division is delayed while cytoplasmic growth continues. This produces abnormally large, immature red blood cells called megaloblasts. These defective cells have shortened survival and poor oxygen-carrying capacity, leading to anemia and tissue hypoxia.

---

8. Gestational Diabetes Mellitus (GDM)

Placental hormones such as human placental lactogen, cortisol, estrogen, and progesterone create insulin resistance to ensure glucose availability for the fetus. In some women, the pancreas cannot produce enough insulin to overcome this resistance.

As a result, maternal blood glucose rises. Excess glucose crosses the placenta and stimulates fetal insulin production. Fetal hyperinsulinemia promotes excessive fat storage and growth, leading to macrosomia. After birth, the newborn may develop hypoglycemia because maternal glucose supply suddenly stops while insulin remains elevated.

---

9. Consumptive Coagulopathy (DIC)

DIC occurs when the coagulation cascade becomes excessively activated, often due to placental abruption, sepsis, or severe preeclampsia. Massive clot formation develops throughout small blood vessels.

Clotting factors and platelets become rapidly consumed. As supplies are depleted, the body can no longer form effective clots, leading to widespread bleeding. Microthrombi also obstruct blood flow, causing tissue ischemia and organ damage.

---

10. Placenta Previa

Placenta previa occurs when the placenta implants in the lower uterine segment near or over the cervical opening. As pregnancy advances and the lower uterine segment stretches, placental attachment becomes disrupted.

Maternal blood vessels tear, causing painless bright red bleeding. Because the placenta is implanted abnormally low, normal fetal descent may also become obstructed.

---

11. Abruptio Placenta

Abruptio placenta occurs when the placenta prematurely separates from the uterine wall. Hypertension, trauma, or smoking may damage maternal vessels supplying the placenta.

Bleeding occurs into the decidua basalis, forming a hematoma behind the placenta. The accumulating blood separates the placenta further, decreasing oxygen and nutrient delivery to the fetus. Maternal bleeding and uterine irritation cause painful contractions and abdominal rigidity.

---

12. Subinvolution

Subinvolution occurs when the uterus fails to return to its normal pre-pregnant size after delivery. Retained placental fragments or infection interfere with uterine contraction and healing.

Because the uterus remains enlarged and relaxed, uterine blood vessels at the placental site continue to bleed. Persistent lochia and postpartum hemorrhage may result.

---

13. Uterine Atony

After delivery, uterine muscle fibers normally contract strongly to compress blood vessels at the placental site. In uterine atony, the uterus fails to contract effectively.

Overdistention from multiple pregnancy, prolonged labor, or retained placenta weakens uterine muscles. Without contraction, blood vessels remain open, leading to massive postpartum hemorrhage and hypovolemic shock.

14. Postpartum Hemorrhage

Postpartum hemorrhage occurs when blood loss after delivery exceeds the body's compensatory ability. Common causes include uterine atony, retained placental tissue, genital tract trauma, or coagulation disorders.

Failure to control bleeding decreases circulating blood volume, reducing oxygen delivery to tissues. Severe hypovolemia may lead to tachycardia, hypotension, shock, and organ failure.

---

15. Cystitis

Cystitis usually occurs when bacteria from the perineal area ascend through the urethra into the bladder. E. coli is the most common organism.

The bacteria adhere to the bladder wall and trigger inflammation. Inflammatory mediators irritate the bladder mucosa, causing dysuria, urinary frequency, urgency, and suprapubic pain. Pregnancy increases risk because progesterone relaxes urinary smooth muscles, causing urinary stasis.

16. Asymptomatic Bacteriuria

Asymptomatic bacteriuria occurs when bacteria colonize the urinary tract without causing symptoms. During pregnancy, progesterone relaxes the ureters and bladder, causing urinary stasis and incomplete bladder emptying. The enlarging uterus may also compress the ureters, slowing urine flow.

Stagnant urine allows bacteria, commonly E. coli, to multiply. Although inflammation is minimal and symptoms are absent, untreated infection may ascend to the kidneys and develop into pyelonephritis.

---

17. Pyelonephritis

Pyelonephritis develops when bacteria from the lower urinary tract ascend through the ureters into the kidneys. Pregnancy increases susceptibility because urinary stasis and ureteral dilation promote bacterial growth.

The bacteria invade the renal pelvis and kidney tissue, triggering inflammation and edema. White blood cells infiltrate the kidney, impairing renal function. Systemic inflammatory response causes fever, chills, flank pain, nausea, and vomiting. Severe infection may lead to sepsis and preterm labor.

---

18. Vaginitis

Vaginitis occurs when the normal vaginal flora becomes disrupted. Factors such as antibiotics, hormonal changes, poor hygiene, or sexual transmission alter the balance between protective lactobacilli and pathogenic organisms.

As harmful bacteria, fungi, or protozoa multiply, they irritate the vaginal mucosa and trigger inflammation. Increased blood flow and immune response cause redness, itching, burning, and abnormal discharge.

---

19. Genital Warts

Genital warts are caused by human papillomavirus (HPV), usually types 6 and 11. The virus enters through tiny breaks in the skin or mucous membrane during sexual contact.

HPV infects epithelial cells and stimulates abnormal cell growth and proliferation. This excessive growth produces soft, flesh-colored wart-like lesions on the genital area. The virus may remain dormant for long periods before visible lesions appear.

---

20. Mastitis

Mastitis commonly occurs during breastfeeding when milk stasis develops due to incomplete breast emptying. Cracked nipples allow bacteria, usually Staphylococcus aureus, to enter breast tissue.

Bacterial invasion triggers inflammation of the breast ducts and surrounding tissue. Increased blood flow and immune response cause breast pain, swelling, warmth, redness, and fever. If untreated, an abscess may form.

---

21. Morbidly Adherent Placenta (Placenta Accreta Spectrum)

Normally, the placenta attaches only to the decidua basalis. In placenta accreta spectrum, defective decidual formation allows placental villi to invade too deeply into the uterine wall.

Depending on depth of invasion:

- Accreta = attached to myometrium
- Increta = invades into myometrium
- Percreta = penetrates through uterus

Because the placenta is abnormally attached, it cannot separate normally after delivery. Attempted removal tears maternal blood vessels, causing severe hemorrhage.

---

22. Peritonitis

Peritonitis occurs when bacteria or irritating substances enter the peritoneal cavity, commonly from bowel perforation, infection, or rupture of abdominal organs.

The peritoneum becomes inflamed and releases inflammatory mediators. Capillary permeability increases, causing fluid to shift into the abdominal cavity. This leads to abdominal pain, rigidity, decreased bowel movement, fever, and possible hypovolemic shock due to fluid loss.

---

23. Uterine Inversion

Uterine inversion occurs when the uterus turns inside out after delivery, usually due to excessive traction on the umbilical cord or fundal pressure while the uterus is relaxed.

The inverted uterus pulls blood vessels downward, causing severe hemorrhage. Sudden blood loss and vagal stimulation may rapidly produce shock, even when bleeding seems minimal.

---

24. Umbilical Cord Prolapse

Umbilical cord prolapse occurs when the cord slips below the presenting fetal part after membrane rupture. The descending fetus compresses the cord against the pelvis.

Compression decreases or completely obstructs blood flow through the umbilical vessels, reducing oxygen delivery to the fetus. Prolonged compression causes fetal hypoxia and distress.

---

25. Fetal Macrosomia

In maternal diabetes, excess maternal glucose crosses the placenta. The fetal pancreas responds by producing large amounts of insulin.

Fetal insulin acts as a growth hormone, increasing fat deposition and protein synthesis. This leads to excessive fetal growth, especially in the shoulders and trunk. Macrosomia increases the risk for shoulder dystocia and birth trauma.

---

26. Toxic Shock Syndrome

Toxic shock syndrome is caused by toxins released by Staphylococcus aureus or Streptococcus pyogenes. These toxins act as superantigens, triggering massive activation of T-cells and cytokine release.

The overwhelming immune response causes widespread vasodilation, capillary leakage, fever, rash, hypotension, and multi-organ dysfunction. Reduced blood pressure decreases tissue perfusion and may lead to shock.

---

27. Threatened Abortion

Threatened abortion occurs when slight placental separation or hormonal insufficiency causes uterine bleeding during early pregnancy while the cervix remains closed.

The embryo is still attached and viable, but uterine irritation may stimulate mild contractions and spotting. If placental attachment stabilizes, pregnancy may continue normally.

---

28. Hydramnios (Polyhydramnios)

Hydramnios occurs when amniotic fluid production exceeds removal. Normally, the fetus swallows amniotic fluid, which is absorbed through the gastrointestinal tract.

Conditions such as fetal GI obstruction or neurologic defects impair fetal swallowing, causing fluid accumulation. Maternal diabetes may also increase fetal urination, adding excess fluid. The enlarged uterus may trigger preterm labor and respiratory discomfort.

---

29. Oligohydramnios

Oligohydramnios occurs when amniotic fluid volume becomes abnormally low. Since fetal urine is the major source of amniotic fluid in later pregnancy, conditions that reduce fetal urine output decrease fluid levels.

Placental insufficiency decreases fetal kidney perfusion, while renal abnormalities impair urine production. Membrane rupture may also cause fluid loss. Reduced cushioning compresses the fetus and may impair lung development.

---

30. Ectopic Pregnancy

An ectopic pregnancy occurs when a fertilized ovum implants outside the uterine cavity, most commonly in the fallopian tube.

Tubal damage from infection or scarring slows ovum transport. As the embryo grows, the narrow tube stretches and eventually ruptures because it cannot accommodate the expanding pregnancy. Rupture causes internal bleeding and severe abdominal pain.

---

31. Cephalopelvic Disproportion (CPD)

CPD occurs when the fetal head is too large or the maternal pelvis is too small for vaginal delivery.

During labor, the fetal head cannot descend properly through the birth canal. Ineffective descent prolongs labor, increases uterine exhaustion, and may compromise fetal oxygenation due to prolonged compression.

---

32. Hypotonic Uterine Dysfunction

Hypotonic uterine dysfunction occurs when uterine contractions become weak and ineffective, usually during active labor.

Uterine muscle fibers lose strength from overdistention, exhaustion, or excessive sedation. Weak contractions fail to dilate the cervix and move the fetus downward effectively, prolonging labor and increasing infection risk.

---

33. Hypertonic Uterine Dysfunction

Hypertonic uterine dysfunction occurs when contractions become excessively frequent, painful, and uncoordinated.

The uterus fails to relax adequately between contractions, decreasing uteroplacental blood flow. Despite severe pain, contractions are ineffective for cervical dilation because they lack proper coordination.

---

34. Hirschsprung Disease

Hirschsprung disease results from failure of neural crest cells to migrate into portions of the colon during fetal development.

Without ganglion cells, the affected bowel segment cannot relax or perform peristalsis. Stool accumulates above the obstructed segment, causing bowel dilation, abdominal distention, and constipation.

---

35. Meconium Aspiration Syndrome

Fetal distress causes passage of meconium into the amniotic fluid. The fetus may inhale meconium before or during birth.

Meconium obstructs airways and causes chemical irritation of lung tissue. Air trapping, inflammation, and surfactant inactivation impair gas exchange, leading to respiratory distress and hypoxia.

---

36. Ophthalmia Neonatorum

Ophthalmia neonatorum occurs when a newborn is exposed to infectious organisms during passage through the birth canal, commonly Neisseria gonorrhoeae or Chlamydia trachomatis.

The organisms infect the conjunctiva, causing intense inflammation, redness, edema, and purulent discharge. Severe gonococcal infection may rapidly damage the cornea and lead to blindness.

---

37. Hyaline Membrane Disease

Premature infants often lack adequate surfactant because type II alveolar cells are immature.

Without surfactant, alveoli collapse during exhalation due to high surface tension. Repeated collapse causes widespread atelectasis and poor gas exchange. Protein-rich fluid lines the alveoli, forming “hyaline membranes” that further impair oxygenation.

---

38. Physiologic and Pathologic Jaundice

Jaundice occurs when bilirubin accumulates in the blood and tissues.

In physiologic jaundice, newborn RBC breakdown increases bilirubin production while the immature liver cannot conjugate bilirubin efficiently. Temporary unconjugated hyperbilirubinemia develops.

In pathologic jaundice, excessive hemolysis, infection, liver disease, or blood incompatibility causes bilirubin to rise rapidly and excessively. Very high bilirubin levels may cross the blood-brain barrier and damage brain tissue (kernicterus).

39. Necrotizing Enterocolitis (NEC)

NEC commonly occurs in premature infants because their intestines are immature and poorly perfused. Reduced blood flow to the bowel causes intestinal ischemia and weakens the intestinal lining.

When enteral feeding begins, bacteria can easily invade the damaged intestinal wall. The inflammatory response causes edema, ulceration, and tissue necrosis. Gas produced by bacteria may enter the bowel wall and bloodstream. Severe necrosis may lead to bowel perforation, peritonitis, sepsis, and shock.

---

40. Pertussis

Pertussis is caused by Bordetella pertussis infection of the respiratory tract. The bacteria attach to the ciliated epithelial cells lining the airways and release toxins.

These toxins damage and paralyze the cilia, preventing normal mucus clearance. Thick mucus accumulates in the airways, triggering repeated severe coughing fits. During coughing episodes, oxygen intake decreases, causing cyanosis and the characteristic “whooping” sound when the child gasps for air.

---

41. Congenital Diaphragmatic Hernia

Congenital diaphragmatic hernia occurs when the diaphragm fails to close completely during fetal development.

Abdominal organs such as the intestines and stomach move into the thoracic cavity through the defect. The herniated organs compress the developing lungs, preventing normal lung growth (pulmonary hypoplasia). After birth, the small lungs cannot exchange oxygen effectively, causing severe respiratory distress and hypoxia.

---

42. Laryngotracheobronchitis (Viral Croup)

Croup is usually caused by a viral infection, commonly parainfluenza virus. The virus infects the larynx, trachea, and bronchi, causing inflammation and edema.

Swelling is greatest in the narrow subglottic region of the airway. Because children have small airways, even mild swelling significantly narrows airflow. Air moving through the narrowed airway produces inspiratory stridor and the characteristic barking cough.

---

43. Acute Epiglottitis (Supraglottitis)

Acute epiglottitis is usually caused by bacterial infection, historically Haemophilus influenzae type B.

The infection causes rapid inflammation and swelling of the epiglottis and surrounding tissues. The swollen epiglottis can suddenly obstruct the airway, making breathing difficult. The child develops high fever, drooling, muffled voice, and respiratory distress. Complete airway obstruction may occur if untreated.

---

44. Kawasaki Disease

Kawasaki disease is an acute systemic vasculitis of unknown cause, likely triggered by an abnormal immune response to infection in genetically susceptible children.

Inflammation affects medium-sized blood vessels throughout the body, especially the coronary arteries. Endothelial damage weakens vessel walls and may cause coronary artery aneurysms. Widespread inflammation also produces prolonged fever, mucosal redness, rash, lymph node enlargement, and swelling of hands and feet.

---

45. Acyanotic Congenital Heart Disease

In acyanotic defects such as ventricular septal defect (VSD), atrial septal defect (ASD), or patent ductus arteriosus (PDA), blood flows from the left side of the heart to the right side because left-sided pressure is higher.

Oxygenated blood recirculates through the lungs instead of going to the body. Increased pulmonary blood flow overloads the lungs and right heart, causing pulmonary congestion and heart failure over time. Since oxygenated blood still reaches systemic circulation, cyanosis is absent.

---

46. Cyanotic Congenital Heart Disease

In cyanotic defects such as Tetralogy of Fallot, deoxygenated blood bypasses the lungs and enters systemic circulation through a right-to-left shunt.

Because blood is not adequately oxygenated, tissues receive insufficient oxygen, producing cyanosis. Chronic hypoxia stimulates increased RBC production, leading to polycythemia. Severe hypoxia may also cause clubbing and “tet spells.”

---

47. Congenital Adrenal Hypoplasia

Congenital adrenal hypoplasia occurs when the adrenal cortex fails to develop properly.

The adrenal glands cannot produce adequate cortisol and aldosterone. Cortisol deficiency causes hypoglycemia and poor stress response, while aldosterone deficiency leads to sodium loss, dehydration, hyperkalemia, and hypotension. Severe electrolyte imbalance may result in adrenal crisis.

---

48. Esophageal Fistula (Tracheoesophageal Fistula)

Tracheoesophageal fistula develops when the trachea and esophagus fail to separate properly during fetal development.

An abnormal connection forms between the airway and esophagus. Milk or saliva may pass into the lungs during feeding, causing choking, coughing, cyanosis, and aspiration pneumonia. Air may also enter the stomach through the fistula, causing abdominal distention.

---

49. Hypertrophic Pyloric Stenosis

Hypertrophic pyloric stenosis occurs when the pyloric muscle between the stomach and duodenum progressively hypertrophies and thickens.

The thickened muscle narrows the gastric outlet, preventing stomach contents from passing into the intestine. The stomach contracts forcefully to overcome the obstruction, resulting in projectile non-bilious vomiting. Persistent vomiting causes dehydration and electrolyte imbalance.

---

50. Down Syndrome

Down syndrome is caused by an extra copy of chromosome 21 (trisomy 21). The additional genetic material alters normal growth and development.

Abnormal protein production affects multiple organ systems, particularly the brain and heart. Delayed neuronal development leads to intellectual disability, while abnormal organ formation causes congenital anomalies such as congenital heart defects and GI abnormalities.

---

51. Neural Tube Defect

Neural tube defects occur when the neural tube fails to close completely during early embryonic development, often due to folic acid deficiency.

Incomplete closure exposes portions of the brain or spinal cord. Depending on the location and severity, nerve damage may impair movement, sensation, bladder control, and cognitive function.

---

52. Cleft Lip and Palate

Cleft lip and palate occur when facial structures fail to fuse properly during embryonic development.

Incomplete fusion leaves an opening in the lip, palate, or both. This disrupts normal feeding, speech development, and ear drainage. Milk may enter the nasal cavity during feeding because the oral and nasal cavities are connected.

---

53. Talipes Equinovarus (Clubfoot)

Clubfoot develops due to abnormal musculoskeletal positioning during fetal development.

The bones, muscles, tendons, and ligaments of the foot become abnormally aligned. The foot turns inward and downward because of muscle imbalance and shortened connective tissue. Without correction, walking becomes difficult.

---

54. Gastroschisis

Gastroschisis occurs when the abdominal wall fails to close completely beside the umbilicus during fetal development.

The intestines protrude directly into the amniotic fluid without a protective membrane. Continuous exposure to amniotic fluid irritates and inflames the bowel, leading to edema and impaired intestinal function after birth.

---

55. Fetal Alcohol Syndrome

Alcohol freely crosses the placenta and enters fetal circulation. Because the fetal liver is immature, alcohol remains in the fetal body longer.

Alcohol disrupts cell growth, migration, and brain development during organ formation. Neuronal injury and impaired tissue formation cause growth restriction, facial abnormalities, cognitive impairment, and behavioral problems.

---

56. Intestinal Intussusception

Intussusception occurs when one segment of the intestine telescopes into another segment.

The invaginated bowel pulls its blood vessels along with it, compressing circulation. Venous congestion develops first, followed by edema and ischemia. As swelling worsens, bowel obstruction and tissue necrosis may occur. Currant jelly stool results from sloughed mucosa mixed with blood and mucus.

---

57. Abortion

Abortion refers to termination or loss of pregnancy before fetal viability.

Common mechanisms include chromosomal abnormalities, hormonal insufficiency, infection, or maternal illness. These conditions impair fetal development or placental attachment, leading to fetal death and uterine contractions that expel pregnancy tissue.

---

58. Septic Abortion

Septic abortion occurs when retained products of conception become infected, usually after unsafe abortion or incomplete miscarriage.

Bacteria invade the uterus and surrounding tissues, causing intense inflammation and tissue necrosis. Infection may spread into the bloodstream, causing sepsis, shock, and disseminated intravascular coagulation.

---

59. Inevitable Abortion

Inevitable abortion occurs when uterine contractions and cervical dilation progress to the point that pregnancy cannot continue.

Placental separation stimulates prostaglandin release and uterine contractions. The cervix opens, allowing products of conception to descend. Because the cervix is dilated, continuation of pregnancy is no longer possible.

---

60. Incomplete Abortion

Incomplete abortion occurs when some products of conception are expelled while others remain inside the uterus.

Retained tissue prevents full uterine contraction, so bleeding continues. The retained tissue also maintains uterine irritation and contractions, causing abdominal pain and persistent hemorrhage.

---

61. Complete Abortion

Complete abortion occurs when all products of conception are expelled from the uterus.

After expulsion, the uterus contracts effectively, compressing blood vessels and reducing bleeding. The cervix gradually closes, and symptoms improve.

---

62. Missed Abortion

Missed abortion occurs when fetal death happens but the products of conception remain inside the uterus for an extended period.

Because the pregnancy tissue is retained, the cervix remains closed and bleeding may be minimal. Hormone levels gradually decline, causing pregnancy symptoms to disappear. Prolonged retention may eventually trigger coagulation problems or spontaneous expulsion.