

Learn simply

Acute Shortness of Breath



- 1. Ask about acute-onset shortness of breath (dyspnea), pleuritic chest pain, cough, and/or hemoptysis.
- 2. On examination, look for low-grade fever, tachypnea, tachycardia, diminished oxygen saturation, diminished breath sounds, audible crackles, and/or evidence of pleural effusion on pulmonary examination.
- 3. Laboratory tests may reveal acidosis and an elevated A-a gradient on arterial blood gas analysis (ABG), and evidence of right-heart strain (S1Q3T3 pattern with or without right axis deviation, T wave inversion) on EKG.
- 4. CXR may be normal or show evidence of multiple peripheral wedge-shaped areas of consolidation, pulmonary edema, or pleural effusion.
- 5. However, if ABG reveals an arterial pO2 > 80 mm-Hg, the diagnosis of pulmonary embolism (PE) is highly unlikely.
- 6. Measurement of D-dimer levels in pregnancy is generally unhelpful in making the diagnosis of PE.

- 1. The differential diagnosis of acute shortness of breath includes pulmonary embolism, amniotic fluid embolism (AFE), pneumonia (including aspiration pneumonitis (Mendelson's syndrome)), pneumothorax, congestive cardiac failure, pericarditis, pulmonary edema, venous air embolism (rare, associated with ruptured uterus, placenta previa, and persistent atrial septal defect), eclampsia, drug overdose/withdrawal, and rib fracture.
- 2. Amniotic fluid embolism (AFE) is an obstetric emergency with 80-90% maternal and perinatal mortality.
- 3. It accounts for 10% of maternal deaths in the United States. AFE is seen most commonly during labor, delivery, and in the immediate postpartum period. Risk factors include cesarean delivery, chorioamnionitis, multiparity, preeclampsia, prolonged labor, fetal demise, amniotomy, intrauterine pressure catheter, intrauterine saline injection (abortion), and placental abruption.
- 4. It is characterized by acute-onset dyspnea, hypotension, and hypoxemia.
- 5. Prodromal symptoms may include sudden chills, sweating, or anxiety.
- 6. Physical examination may reveal acute-onset respiratory distress, cyanosis, hypotension, tachycardia, hypoxemia, neurologic manifestations (seizures, coma), and/or hemorrhage.
- 7. Disseminated intravascular coagulopathy (DIC) is usually acute and severe.
- 8. AFE is a clinical diagnosis. CXR and V/Q scan is of little value in the acute setting. Components of amniotic fluid (fetal squames, mucin) may be identified in the pulmonary vasculature at postmortem, but this is not pathognemonic.



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- 1. Therapy is primarily supportive. Cardiovascular support should be optimized. This includes maintaining O2 saturation >90%, arterial PO2 > 60 mmHg, systolic BP >90 mmHg, and urine output >25 mL/h. Ionotropic support (dopamine) and mechanical ventilation should be considered, if needed. Treat bronchospasm (terbutaline, aminophylline, steroids) as needed. CPR and cardiopulmonary bypass may be required.
- 2. DIC is typically a predominant clinical feature of AFE. Serial CBC and coagulation studies should be followed, and aggressive blood product replacement initiated. Avoid heparin in established DIC.
- 3. The fetus is at risk of hypoxic ischemic cerebral injury and/or IUFD. Urgent delivery may be necessary regardless of gestational age. Regional anesthesia is contraindicated in the acute setting. General endotracheal anesthesia may be needed for cesarean.



Kawita Bapat



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