PULMONARY EDEMA

Take-home points:
1. Elevation of pulmonary wedge pressures helps to differentiate cardiogenic from non-cardiogenic causes of pulmonary edema.
2. CXR, ECG, and ABG are indicated in most patients. History and physical exam can help differentiate causes.
3. For treatment think: L-M-N-O-P (Lasix-Morphine-Nitro-Oxygen-Position/Positive pressure ventilation)

I. Cardiogenic

Pathophysiology: Caused by rapid transudation of fluid into lungs secondary to increased pulmonary wedge pressure without time for compensation of pulmonary bed. Increased wedge pressure translates to increased pulmonary venous pressure and elevated microvascular pressure, leading to transudation of fluid (Starling's forces at work!). Can occur at wedge pressures as low as 18mmHg or not until >25mmHg if chronic condition has resulted in increased lymphatic drainage capacity.

Etiology:
A. Heart muscle:
   1. Systolic dysfunction: Most common cause of pulmonary edema. Can be due to CAD, HTN, valvular disease, idiopathic dilated cardiomyopathy, toxins, hypothyroidism, viral myocarditis. If condition is somewhat chronic, volume overload is exacerbated by renin-angiotensin system upregulation due to decreased forward flow.
   2. Diastolic dysfunction: Increase in ventricular stiffness impairs filling leading to proximal pressure rise. Causes include hypertrophic and restrictive cardiomyopathies, ischemia, HTN crises.

B. Valvular problems:
   1. Mitral stenosis: Usually due to rheumatic heart disease.
   2. Aortic stenosis: Causes pulmonary edema by requiring elevated LVED filling which translates to high pulmonary pressures and cardiac ischemia due to impaired diastolic coronary artery filling.
   3. Aortic regurgitation: Acutely can be seen in infective endocarditis or aortic dissection.

C. Other:
   1. Renal artery stenosis: In some cases, pulmonary edema has been the presenting sign of RAS!
   2. Atrial myxoma, intracardiac thrombus impeding left atrial outflow track, congenital membrane in left atrium (cor triatriatum).

Diagnosis:
   a). HISTORY and physical exam
   b). ECG: Can see ischemic changes consistent with CAD. Can also see negative T waves, global T wave inversions, and marked QT interval prolongation unrelated to ischemia that resolve within 1-7 days.
   c). Echocardiogram

Treatment
1. Supplemental oxygen
2. Diuretics: Lasix or other loop diuretics. Dosage should be at least 40mg IV but often higher doses needed, especially if patient is already on diuretics at home. Peak diuresis in 30minutes. Furosemide initially causes venodilation prior to onset of diuresis. In chronic CHF can occasionally see transient arteriolar vasoconstriction and increased blood pressure due to increase in plasma renin and norepinephrine levels.
3. Morphine: Give 2-5 mg over 3 minutes and repeat in 15 minutes if necessary. Decreases patient anxiety and work of breathing, thereby limiting sympathetic outflow and aiding in arteriolar and venous dilatation.
4. **Vasodilators**: nitroglycerin or nitroprusside. Nitroprusside especially helpful for HTN emergency, acute aortic or mitral regurgitation, acute ventricular septal wall defect.

5. **Position**: sit patient upright

6. **Positive pressure ventilation**: decreases venous return and increases pressure gradient between LV and extrathoracic arteries. To be used with caution as one study showed increased incidence of deterioration requiring intubation when compared with high dose nitrate group.

**II. Noncardiogenic**

**Definition**: Radiographic evidence of alveolar fluid accumulation without elevated pulmonary capillary wedge pressure.

**Pathophysiology**: Alveolar-capillary membrane becomes damaged and leaky, resulting in movement of proteins and water into interstitial space. Note: hypoalbuminemia does NOT cause pulmonary edema.

**Etiologies**:

A. **ARDS** (acute respiratory distress syndrome): Multiple etiologies, including sepsis, DIC, inhaled toxins, radiation pneumonia, inhalation of high oxygen concentrations, severe trauma (thoracic or otherwise). Often occurs within first 2 hours of inciting event but can occur 1-3 days later. Xray shows bilateral alveolar filling pattern. Treat underlying cause. High frequency, low volume ventilation with diuresis proven to be beneficial.

B. **Reexpansion pulmonary edema**: can occur after reexpansion of pneumothorax or following removal of large amounts of pleural fluid (>1.0-1.5 L). Can see within 1 hr in 64%. Ongoing for 24-48hr but sx can last up to 5 days. Pathophysiology unknown but worse in patients with chronic collapse. Supportive treatment. Mortality has been reported as high as 20%.

C. **High altitude pulmonary edema**: etiology unclear but thought due to unequal pulmonary vasoconstriction and overperfusion of remaining vessels. Support patient and move to lower altitudes.

D. **Narcotic overdose**: From overdose of heroin or methadone. Usually occurs within 2 hours of injection. Pathophysiology unknown but believed due to direct toxicity, hypoxia, hyperventilation, or cerebral edema. Supportive measure for patient are indicated.

E. **Pulmonary embolism**: Treatment aimed at anticoagulation and supportive measures.

***Pulmonary edema can be confused with diffuse alveolar hemorrhage or lymphangitic spread of cancer. Not all cases of diffuse alveolar hemorrhage present with hemoptysis, but clues to diagnosis may be in unexplained hematocrit drop. Lymphangitic spread of tumors most often seen with lymphoma or acute leukemia, but solid tumors can behave this way.***

**Diagnosis**:

a). HISTORY and physical exam

b). ABG can be helpful.

**III. Neurogenic**

**Presentation**: hypoxia, tachypnea, diffuse rales, frothy sputum or hemoptysis in setting of neurologic disorders or procedures. Occurs within minutes to hours of severe CNS insult. Can be confused with aspiration pneumonitis/pneumonia.

**Common CNS injuries**: epileptic seizures, head injury, cerebral hemorrhage(subarachnoid or intracerebral). In head injuries, pulmonary edema is seen with elevated intracranial pressures.

**Pathophysiology**: likely due to sympathetic activation causing pulmonary vеноconstriction and increased vascular permeability.

**Treatment**: Supportive measures. Usually resolves within 48-72 hours. Some have tried alpha adrenergic blockers such as phentolamine but no trials done with this yet.