# Talk:Pulmonary Embolization

Pulmonary Embolism

A pulmonary embolism (PE) is the blockage of blood flow in the lungs caused by a blood clot, tissue fragments, fat or air (McCance, Huether, Brashers & Rote, 2010). Most commonly, they occur from the dislodgement of a venous thromboembolism in the pelvis or thigh. Virchow triad is the categories of risks that increase the likelihood of a PE. They include venous stasis, or a slowing or pooling of blood in veins, hypercoagulability, and injuries to the blood vessels (McCance et al., 2010). Prolonged bed rest, sitting during travel, advanced age, pregnancy, obesity, CHF are the most common causes of venous stasis. Damage to vessels can occur from trauma, surgery, IV catheters and medications that are irritating to veins.

Pathophysiology

PE are categorized into four types (McCance et al., 2010). Emboli with infarction are those that cause tissue death in the lung. Emboli without infarction do not cause tissue death. Massive occlusions are those that block a large area of circulation. Multiple PE are those that are frequent or chronic.

Genetics

Individuals are at a greater risk for developing PE if they are genetically predisposed to hereditary anti thrombin deficiency. They are more likely to produce DVT, especially in the legs. These individuals have a 1:2 chance of having a PE in their lifetime. Women with this condition are at a higher risk during and shortly after delivery. Hereditary antithrombin deficiency is believed to be present in about 1 in 2,000 to 3,000 individuals (National institute of health, 2013). 1 in 20 to 200 cases of PE are in individuals that have hereditary antithrombin deficiency (National institute of health, 2013).

The SERPINC1 gene is responsible for this disorder. It is an autosomal dominant, which only requires one copy of the gene for the disorder to be present. This gene provides signals for the production of a protein called antithrombin. Individuals are unable to produce enough antithrombin to block clotting proteins, increasing their chance of developing clots.

Epidemiology

In the United States alone:

• Approximately 1 in 1,000 people per year will have a PE

• The mortality rate from PE decreased over a ten year period of 1979-1998 from 191 deaths per million to 94 per million

• Approximately 60% of individuals who die in a hospitals have a PE, of which 70% were missed

• Women have a 20-30% higher mortality rate due to PE than men

• Blacks are a higher risk than whites, and whites higher others

• Rare in pediatric population

• Higher in elderly population

• Account for 15% of post-op deaths, especially surgeries below the chest

(Ouelette, 2013)

Signs and Symptoms

PE are difficult to diagnose due the non-specific symptoms that may be present. In order to accurately and efficiently diagnose a PE, a thorough history is crucial. Presence of a DVT may increase the possibility, but lack of DVT does not rule out PE. Common symptoms include difficulty breathing, rapid pulse, anxiety, coughing up blood (hemoptysis) and chest pain that is pleuritic in nature. Symptoms generally come on suddenly. Objective symptoms include elevated white count, pleural friction rub, fever and pleural effusion. Massive occlusions result in shock, pulmonary hypertension, and chest pain in addition to above symptoms (McCance et al., 2010).

Diagnosis

After ROS and PMH, if PE is suspected EKG, ABG and chest x-ray should be obtained rapidly. A D-dimer is a blood test that measures degradation of the fibrinolytic system, but is non-specific and often falsely elevated due to other causes (McCance et al., 2010). If it is elevated, a CT angiogram can determine the specific location of a PE if present. If contraindicated due to allergy or elevated renal function lab work, VQ scan can utilized instead of Cat scan.

## Treatment

Treatment can be performed surgically or with medication (Mayo clinic Staff, 2014). Surgical options include removal of the clot via catheter or the placement of a filter to prevent the clot from traveling to the lung. Medications can be used to treat current clots and prevent future clots. Preventive medications include warfarin, low-molecular-weight heparin, fondaparinux (McCance et al., 2010) and enoxaparin. Heparin is generally started immediately after diagnosis, if not contraindicated. Prophylactic therapy may only be necessary for a short period of time if risk factors are short in nature. These include pregnancy and bed rest post-operatively. If risks factors are not modifiable, the patient may be on medication indefinitely.

## Prevention

Prevention of PE include medications and limiting inactivity. Post-operative patients should ambulate as soon as possible or assisted to move while in bed until able to ambulate. The key to prevention is identification of those at risk.

## Reference

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