Venous Thromboembolism (VTE) or Embolic Disease: Fatal Clot

Venous thromboembolism (VTE) comprises deep vein thrombosis (DVT) and pulmonary embolism (PE), and it is one of the most frequent cardiovascular diseases. The incidence of PE is approximately 60-70 per 100,000, and that of venous thrombosis is approximately 124 per 100,000 of the general population.1

The precise number of people affected by DVT/PE is unknown, although as many as 900,000 people could be affected (1 to 2 per 1,000) each year in the United States. Estimates suggest that 60,000-100,000 Americans die of DVT/PE (also called venous thromboembolism). Ten to 30% of people will die within one month of diagnosis. Sudden death is the first symptom in about one-quarter (25%) of people who have a PE.11

Among people who have had a DVT, one-half will have long-term complications (post-thrombotic syndrome) such as swelling, pain, discoloration, and scaling in the affected limb. One-third (about 33%) of people with DVT/PE will have a recurrence within 10 years. Approximately 5 to 8% of the U.S. population has one of several genetic risk factors, also known as inherited thrombophilias in which a genetic defect can be identified that increases the risk for thrombosis.11

In a study done by Woo-Suk Lee et al; among Asian patients the incidence of symptomatic PE was 0.01%. The incidence of overall DVT, proximal DVT and symptomatic DVT were 40.4%, 5.8% and 1.9% respectively. It was found out that there were no differences in incidence of symptomatic PE among Asian countries and no trends in changes of the incidence overtime.

In a study done by Bernardo et al in 2007 at the Philippine Heart Center, among the 106 patients admitted at the medical and neurological ICU, 2.8% of the subjects developed proven DVT and PE. Seven out of the eight patients with either suspected or proven PE or DVT had Well’s score of more than or equal to two (more than or equal to moderate risk for DVT).
Edema is the most non-specific symptom of DVT. Thrombus that involves the iliac bifurcation, the pelvic veins, or the vena cava produces leg edema that is usually bilateral rather than unilateral.

PE and DVT can result from multiple factors which includes age, previous history of VTE, malignancy, pregnancy, conditions of the heart—congenital or acquired, coagulation disorders, intake of hormone replacement therapy, obesity, major illness, recent surgery or trauma, immobilization, long haul air travel and other pathological lung conditions.

Symptoms of unexplained dyspnea, and sometimes chest pain either pleuritic or atypical are noted on PE. Tachycardia, tachypnea, low grade fever, tricuspid regurgitation and accentuated P2 can be also be seen during physical evaluation.

Leg pain occurs in 50% of patients with DVT, but is entirely non-specific. It can occur on dorsiflexion of the foot (Homans sign). When tenderness is present it is usually confined to the calf muscles or along the course of the deep veins in the medial thigh. Clinical signs and symptoms of PE as the primary manifestation occur in 10% of patients with confirmed DVT.

ECG, D dimer, Lung scanning, Compression Ultrasonography of the legs and echocardiography are some of the diagnostic tests used in patients suspected of pulmonary embolism. Markers of RV dysfunction such as RV dilatation, hypokinesis or pressure overload on echocardiography can be seen. In contrast to the global hypokinesis seen in PAH patients with RV failure, PE patients may exhibit sparing of the RV apex with hypokinesis of the RV free wall and base—a finding termed the “McConnell sign.”

The pathophysiology of PE and DVT centers on thrombus formation, which can induce release of inflammatory mediators resulting in vasoconstriction, vascular obstruction and subsequently Right Ventricular (RV) failure. The pressure overload can be a major cause of death among patients with PE. It decreases preload and Cardiac output, thereby causing hypotension. A silent PE can eventually develop into DVT.¹
Result from CT angiography, BNP and troponin elevation are markers useful for risk stratification in acute Pulmonary embolism.

It can be classified as small to moderate, submassive and massive. Initial treatment is focus on restoration of blood flow. Anticoagulation is the mainstay of treatment.

Chest spiral CT scan with radiocontrast agent showing multiple filling defects both at the bifurcation ("saddle" pulmonary embolism) and in the pulmonary arteries.

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McConnell’s Sign

Image copied from Echocardiography in the diagnosis of Pulmonary Embolism. www.Cardioserv.net

4) Abnormal compressibility in deep vein thrombosis.

In submassive and massive PE addition of thrombolysis or embolectomy or IVC filter are the options of treatment. The primary objectives of the treatment of DVT are to prevent PE and reduce mortality and minimizes the risk of post thrombotic syndrome (PTS). The immediate symptoms of DVT often resolve with anticoagulation alone, and the rationale for intervention is often reduction of the 75% long term risk of PTS.  

Hence, early detection and prompt treatment can aid to lower morbidity and mortality rate.

REFERENCE:

4. Acute Right Ventricular Failure in the Setting of Acute Pulmonary Embolism or Chronic Pulmonary Hypertension: A Detailed Review of the Pathophysiology, Diagnosis, and Management ; Jennifer Cowger Matthews et al; Current Cardiology review Feb 2008
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