Acute Pulmonary Embolism: Causes, Investigations and Management

Abstract

Background: Clinical diagnosis of PE (PE) is not reliable enough to determine treatment, but it is important to classify patients as high, moderate, and low embolism. Laboratory tests can be combined with lung scans to identify groups of patients with a high or low likelihood of PE and to determine constipation or to prevent treatment. About half of PE patients are suspected to fall into one of these categories. Deep vein thrombosis (DVT) can be detected in the legs in approximately 50% of PE patients and double venography in approximately 70% of PE patients, preventing constipation in undiagnosed clinical and some lung patients. What needs to be done to provide the cause. Scan to confirm. If you do not receive DVT, it is less likely, but it does not include the possibility that the patient may be suffering from PE. The majority of patients who combine clinical trials, lung scans, and non-invasive DVT tests can safely be treated without anticoagulation, preliminary evidence suggests that abnormal persistent DVT testing is common within 2 weeks. Pulmonary angiography is important in patients who are not diagnosed with the above tests, (a) the likelihood of PE remaining (eg, 30-80%), (b) lack of cardiac appointments, (c) the possibility of continued follow-up, or (d) future management (eg, post-pregnancy) Is affected by the outcome. D-Dimer measurements are sensitive but not specified in PE and therefore may have a high negative prediction value, making it easier to diagnose PE.

Conclusion: PE remains an important clinical problem with a high mortality rate; Data from ICOPER provides values and highlights negative predictor categories that will help plan future trials for high-risk PE patients.

Keywords*: Pulmonary embolism, thrombosis, venous thromboembolism, diagnosis*

Introduction

Pulmonary embolism (PE) is a common and potentially deadly form of venous thromboembolic disease. It is the third most common cause of cardiovascular death and is associated with multiple inherited and acquired risk factors as well as advanced age. The prognosis from PE depends on the degree of obstruction and hemodynamic effects of PE and understanding the pathophysiology helps in risk-stratifying patients and determining treatment. Though the natural history of thrombus is resolution, a subset of patients have chronic residual thrombus, contributing to the post-PE syndrome. Pulmonary embolism (PE) and deep venous thrombosis (DVT) exist on the spectrum of venous thromboembolic disease (VTE). PE results when thrombus migrates from the venous circulation to the pulmonary vasculature and lodges in the pulmonary arterial system. The clinical presentation of acute PE ranges from asymptomatic and incidentally discovered to massive PE causing immediate death (1).

Causes and Risk Factors

Many medically important PEs begin with VTE in the lower extremities or pelvic arteries. Gradually, elevated thromboembolic events lead to PE. Different conditions lead to the

production of VTE. The trio of hypercoagulability, venous stasis and sinking vortices provide a model for understanding many risk factors. These symptoms are usually inherited or acquired. Overall, the main risk factors for thromboembolic events include recent distress, MI, stroke, surgery, and recent trauma. Other major risk factors include pre-VTE, age, dementia, known thrombophilia and internal venous catheter. Moderate risk factors include family history of VTE, use of estrogen or hormone replacement therapy, smoking, pregnancy, and obesity (figure 1) (2).

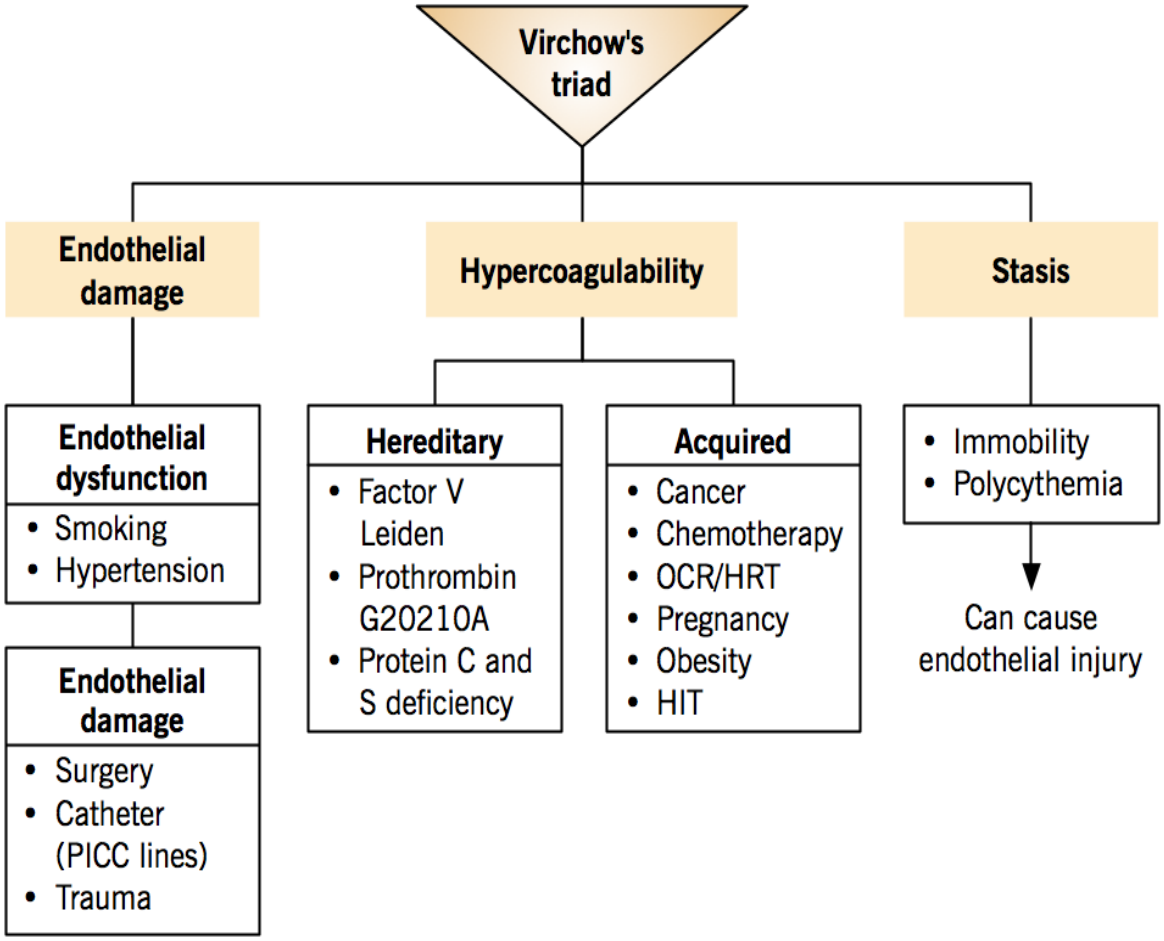

Figure 1 Virchow's triad (2)

Mechanism of Pulmonary Embolism

PE occurs when the deep vein thrombus is broken and connected to the pulmonary circulation. Pulmonary duct closure occurs and disrupts gas exchange and circulation. In the lung, the lower lobe is more affected than the upper lobe, and the involvement of the two lungs is more common. Fetal embolism covers the large artery in the lungs, while the small embolve closes the lateral arteries. External PE can cause pulmonary infarction, manifesting itself as intra-alveolar hemorrhage. Pulmonary infarction occurs in approximately 10% of patients without heart disease. The obstruction of the pulmonary arteries causes the air to enter the dead area, as the alveolar air exceeds the capillary blood flow of the lungs. This leads to a lack of oxygen and clogging of the arteries, which increases the resistance to the

pulmonary arteries. In addition, humoral mediators, such as serotonin and thromboxane, are released from active blood platelets and can cause blood vessels to contract in unaffected areas of the lung. When pulmonary arterial contraction is increased, the cavity rises behind the load, leading to right ventricular failure. If right ventricular failure persists, left ventricular filling may develop. The rapid progression of myocardial ischemia can only occur after coronary insufficiency, which may include hypotension, syncope, electromechanical dissociation, or sudden death (figure 2) (3).

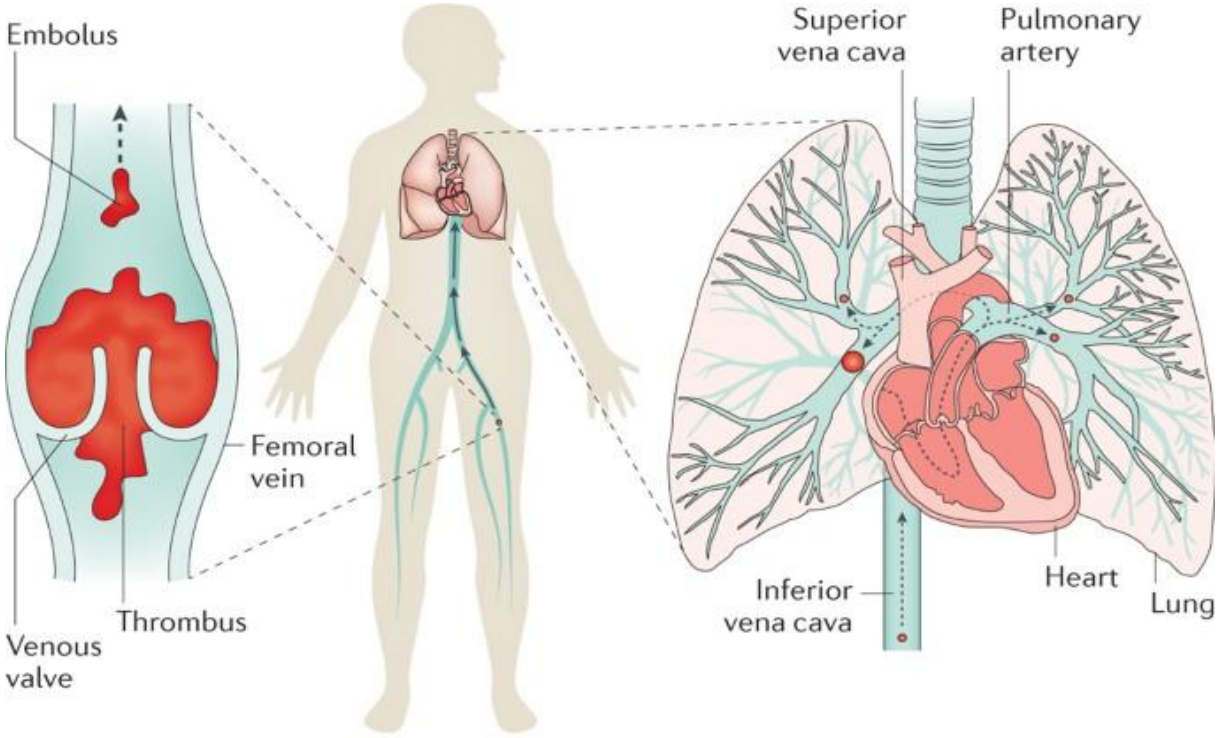

Figure 2 Mechanism of Pulmonary Embolism (3)

Symptoms

Each person may have different symptoms. The most common symptoms include sudden (very fast) breathing, chest pain (usually worse when breathing), restlessness, dizziness, lightheadedness or fainting, irregular heartbeat, pulse, cough or sneezing. , Sweating, low blood pressure, you may also have symptoms of deep vein thrombosis (DVT), such as: pain in the affected leg (possibly only when standing or walking), swelling in the leg, pain, redness, or Warm feet (feet), redness and / or skin changes. If your healthcare provider thinks you have PE, you should examine your foot for signs of deep vein thrombosis. The type and severity of PE symptoms will depend on the size of the embolism and whether you have heart or lung problems. The symptoms of PE can be similar to other diseases or complications. Consult your doctor regularly for diagnosis (4).

Complications

Pulmonary embolism (PE) can cause a lack of blood flow, resulting in damage to lung tissue. This can lead to low oxygen levels in the blood and damage to other organs. PE, especially

large PE or large clots, can quickly lead to serious life-threatening complications and even death. PE is usually treated with anticoagulants or anticoagulants. These drugs can increase the risk of bleeding if they cause heavy bleeding. Heavy bleeding that does not stop after 10 minutes of pressing. Other signs of bleeding that need to be looked at include: Bleeding in the digestive tract Symptoms: Bright red vomiting or vomiting, such as coffee grounds, pale red or black blood in the stool, stools, abdominal pain, bleeding Symptoms: Sudden headache, sudden vision Loss, sudden loss of movement or sensitivity of the legs or arms, memory loss or confusion. If you have any of these, you need to seek immediate treatment (figure 3) (5).

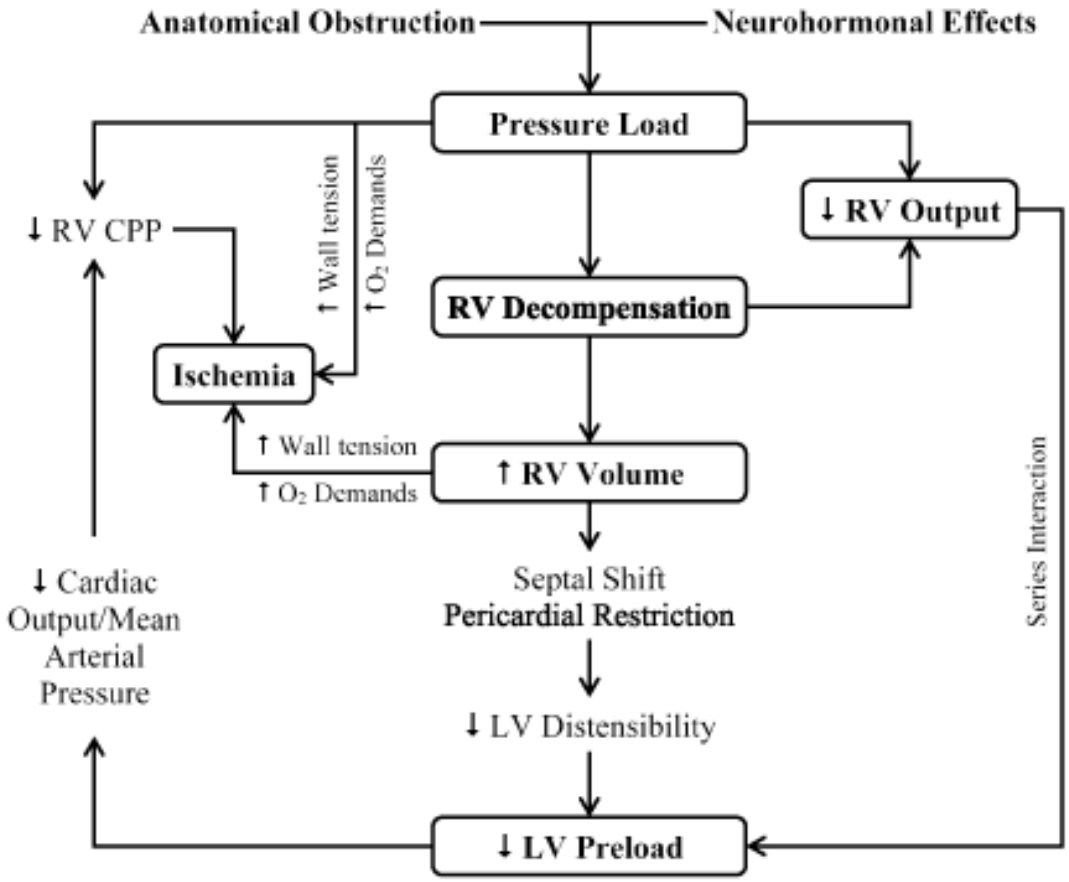

Figure 3 Systemic Pressure decreases as a Complication (5)

Diagnosis and Investigations

Pulmonary embolism can be difficult to diagnose, especially in people with heart or lung disease. For this reason, your doctor will likely review your medical history, examine your body, and coordinate one or more of the following tests (figure 4) (6).

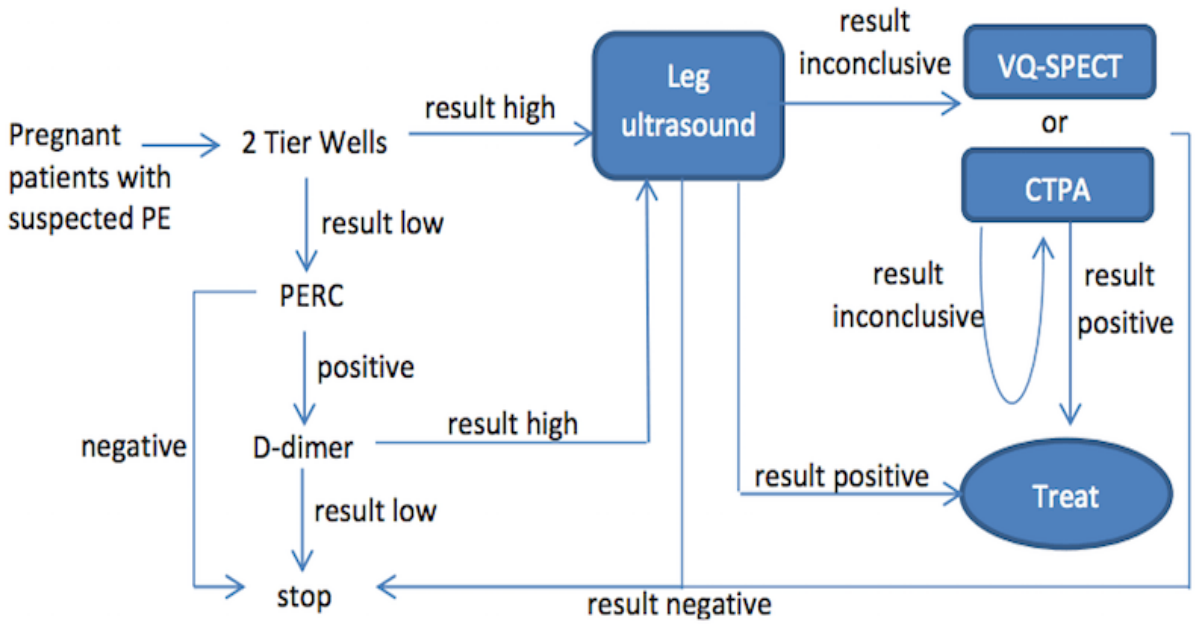

Figure 4 Diagnosis and Investigations (6)

Chest X-ray

This imaging test is used to test the lungs and the heart. The chest x-ray shows information about the size, shape, contour and anatomical location of the heart, lungs, trachea (large airways), aortic and pulmonary arteries, and the mediastinum (figure 5) (7).

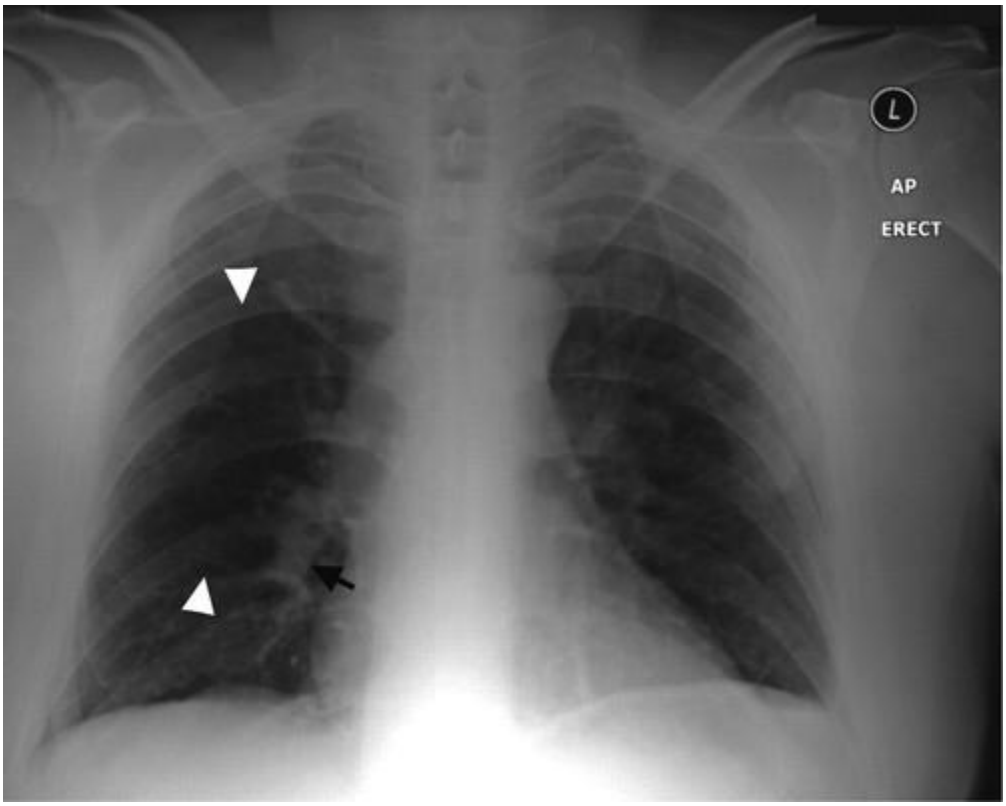

Figure 5 Westermark's and Palla's Signs in Acute Pulmonary Embolism (7)

Ventilation-perfusion scan (V/Q scan)

In this nuclear radiology study, a small amount of radiation is used to help examine the lungs. The ventilation scanner checks the flow of air, or the flow of air entering or leaving the bronchi and bronchioles. An infusion scan checks blood flow to the lungs (figure 6) (8).

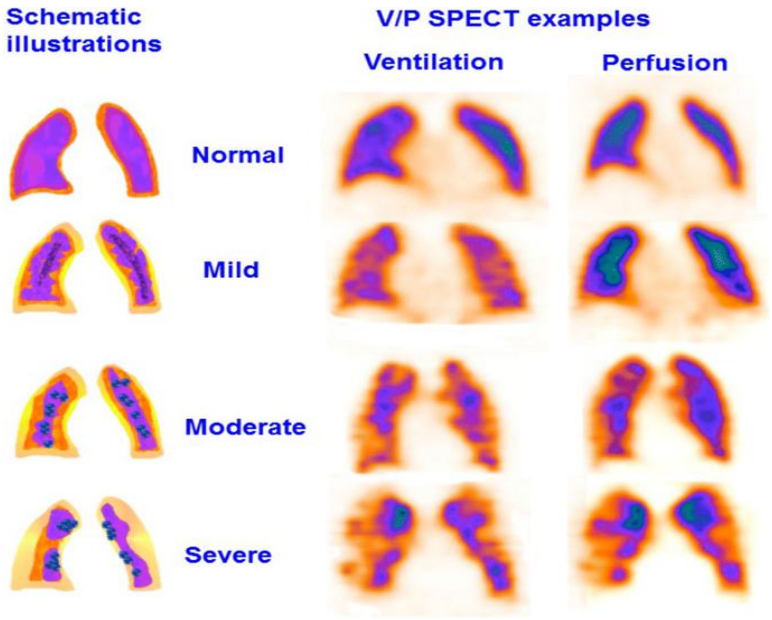

Figure 6 Ventilation/Perfusion Single-Photon Emission Computed Tomography (SPECT) for diagnosis of Acute Pulmonary Embolism (8)

Pulmonary angiogram

This x-ray imaging of blood vessels is used to detect various conditions, such as aneurysm (vascular rupture), stenosis (vascular narrowing) or obstruction. The dye (difference) is injected into a small flexible tube inserted into the vein. This color makes the blood vessels visible in X-rays (figure 7) (9).

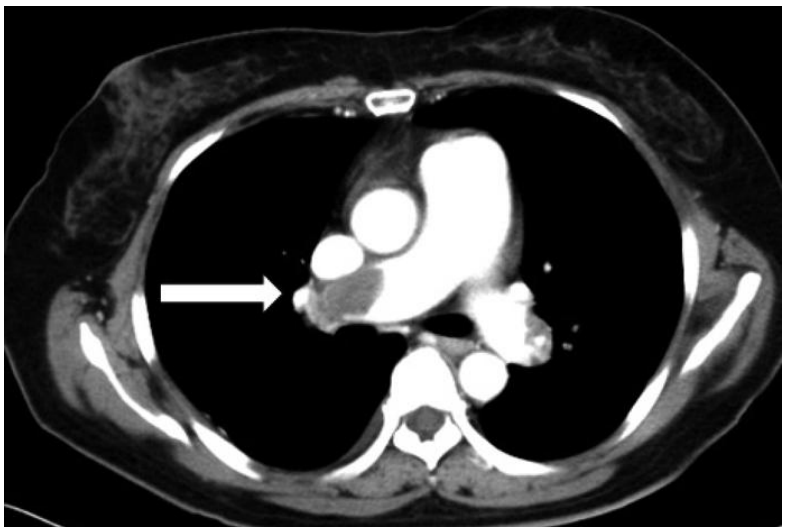

Figure 7 Pulmonary Angiogram for Acute Pulmonary Embolism (9)

CT scan

This is an imaging test that uses X-rays and a computer to take a detailed picture of the body. CT scans show details of bones, muscles, fat and organs. CT improves the image of blood vessels in the lungs. Contrast is a dye-like substance that is inserted into a vein, making it more visible on the organ or tissue under study (figure 8) (10).

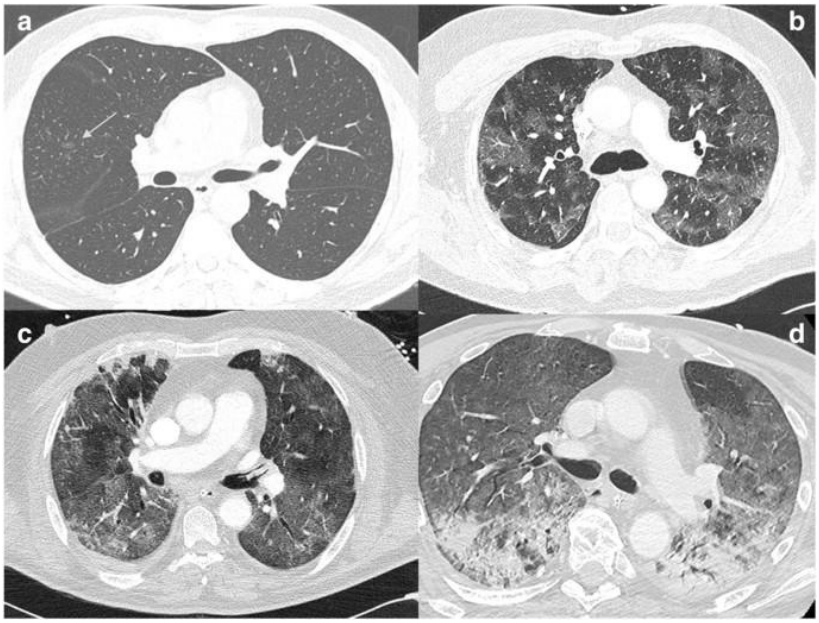

Figure 8 CT Scan for Acute Pulmonary Embolism (10)

MRI

This imaging test uses a combination of magnetic fields, radio waves, and a computer to create detailed images of organs and structures within the body (figure 9) (11).

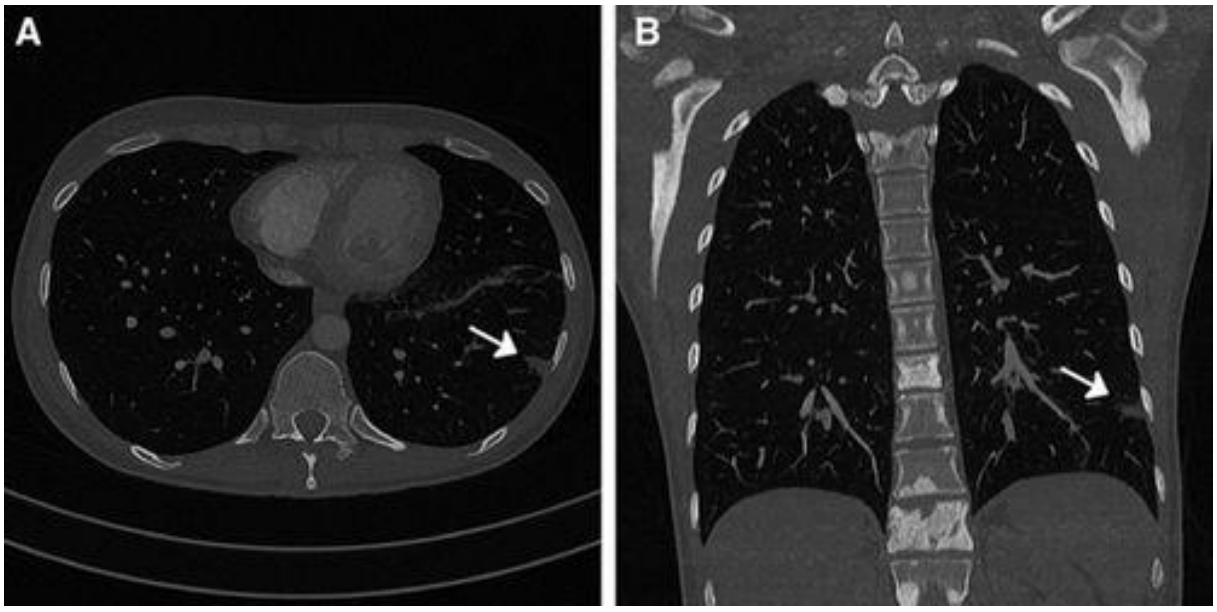

Figure 9 MRI for Acute Pulmonary Embolism (12)

Duplex ultrasound (US)

This type of vascular ultrasound is done to check blood flow and blood vessel formation in the legs. (Blood clots in the legs usually flow and spread to the lungs.) The United States uses high-frequency sound and computers to create images of blood vessels, tissues and organs (figure 10) (13).

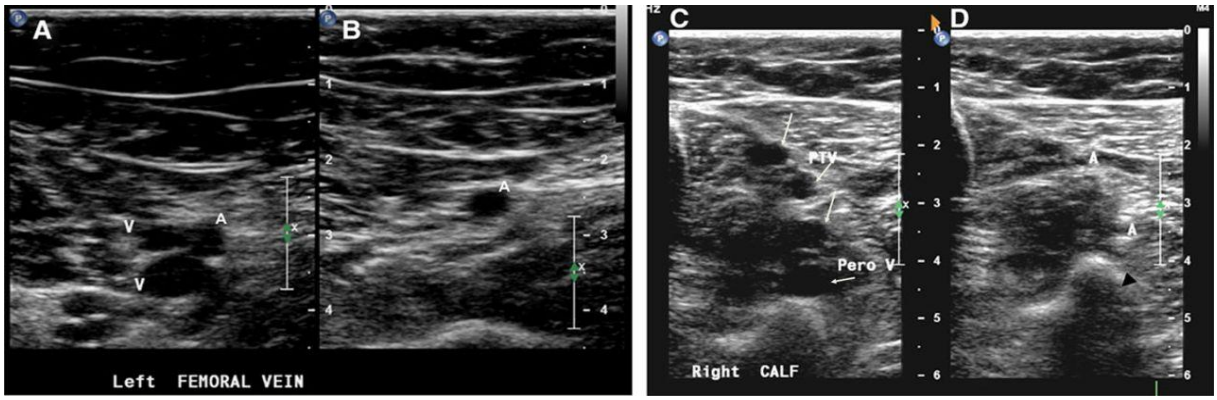

Figure 10 Duplex ultrasound (US) for Acute Pulmonary Embolism (14)

Blood tests

Blood tests are used to check for blood clotting conditions, including tests called D-dimer levels. Other blood tests may include genetic tests that cause abnormal blood pressure. Blood glue can be checked to determine how much oxygen is in the blood (15).

Electrocardiogram (EKG)

This is the fastest and easiest test used for cardiac tests. Electrodes (small, sticky) are placed on certain areas of the chest, arms, and legs. The electrodes are connected to the EKG machine with lead wires. The electrical activity of the heart is measured, interpreted, and printed (figure 11) (16).

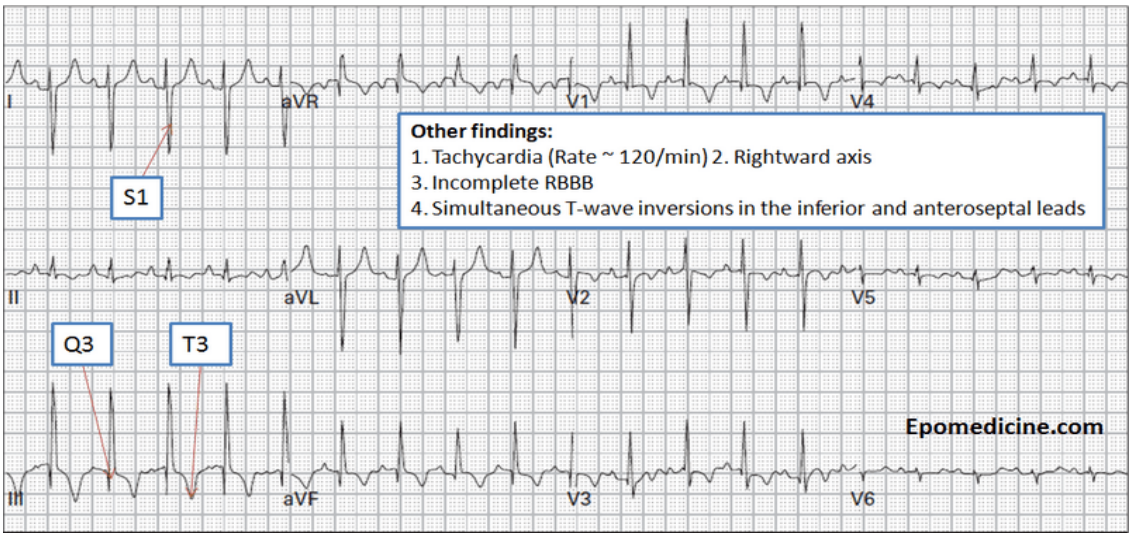

Figure 11 Electrocardiogram (EKG) for Acute Pulmonary Embolism (17)

Prevention of Pulmonary Embolism

You can help prevent PE: regular exercise, eating healthy, eating healthy foods, taking prescription medications, not smoking, you can prevent DVT: socks and socks. These are stretchy socks that tighten or narrow the arteries and prevent blood from flowing back and forth. Air pressure equipment. These are the arms on the legs, which are connected to a machine that alternately applies pressure on the legs to allow blood to flow. He got up. Do this as soon as possible after surgery or illness. Exercise helps prevent blood clots by stimulating blood flow. Tree. Antidepressants and aspirin are often prescribed to prevent DVT. Many people are at risk of developing DVT for a short time after returning from hospital. It is important to continue treatment to prevent DVT until the risk is reduced. It usually takes 3 to 6 months (18).

Important points about pulmonary embolism: Pulmonary embolism (PE) is a blood clot that grows in blood vessels elsewhere in the body (usually in the leg), extends to the lungs, and suddenly forms a blood clot. Abnormal blood clots can form as a result of complications such as “lazy” blood flow to the arteries, malformations, or damage to the artery wall. A variety of conditions and risk factors are linked to PE. Sudden breathing is the most common symptom of PE. PE is often difficult to diagnose because the symptoms and signs of PE are very similar to many other conditions and diseases. Photo imaging and blood tests are used to check for PE. An important aspect of PE treatment is to prevent additional clots. Medications, filters to prevent clots from reaching the lungs, and surgery are used to treat PE. PE, especially large PE or large clots, can quickly cause life-threatening consequences (19).

Medications

Treatment options for Pulmonary Embolism (PE) include: anticoagulants. These drugs are described as antihypertensive. It prevents clots and prevents new clots. Such as warfarin and heparin. Fibrinolytic therapy. These drugs are also known as clotting factors and are given by the intravenous (iv or venous) to break the clot. These drugs are only used in life-threatening situations. Vena cava filter. A metal tube inserted into the vena cava (a large vein that returns blood to the heart) can be used to prevent blood clots from entering the lungs. When you cannot afford anticoagulant therapy (for medical reasons), these filters are most often used if you have additional clots, or if you have bleeding problems with anticoagulant medication. Pulmonary embollectomy. This surgery is rarely used to remove PE. This is usually done in severe cases where your LE is too high, you have not received or responded well to anticoagulant or thrombolytic treatment due to other medical problems, or your condition is stable. Percutaneous thrombectomy. A long, thin, empty tube (catheter) can be attached to the veins at the site of x-ray-guided embolization. Once in place, the catheter is used to break, remove or distribute the emboli with a thrombolytic agent. A major component of PE therapy is the prevention of multiple embolism (figure 12) (20).

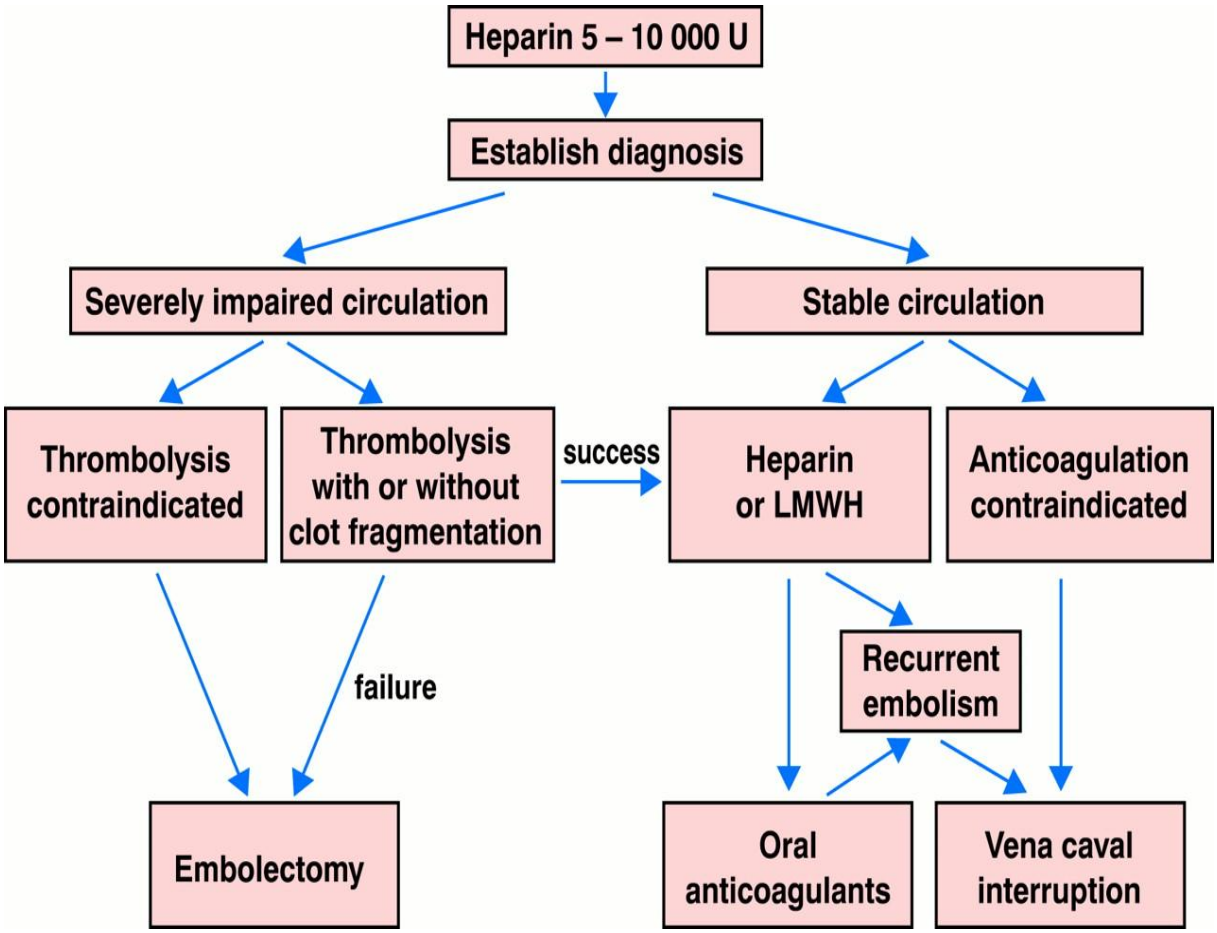

Figure 12 Treatment for Acute Pulmonary Embolism (21)

Results

Venous thromboembolism is a major worldwide burden of disease with ~10 million cases per year and an associated substantial morbidity and mortality. The true incidence of PE is unknown, but in the United States, it is estimated that nearly a third of hospitalized patients are at risk of developing VTE and up to 600,000 cases of VTE are diagnosed per year with 100,000 deaths related to these diseases. In the United States, the estimated incidence of diagnosed VTE is 117 per 100,000, but the true incidence is likely to be more as these diseases are frequently undiagnosed or diagnosed only at autopsy. Based on a review of national inpatient data, the number of admissions for PE increased from nearly 60,000 in 1993 (23 per 100,000) to more than 202,000 in 2012 (65 per 100,000). Despite the increased incidence of PE, there was a decreased incidence of massive PE and hospital mortality over the same time period. Comorbidities associated with PE are also increasing (aging population and medical comorbidities), but the increased incidence in the face of decreased mortality likely reflects increased use of more sensitive CT angiography for diagnosis rather than a true change in prevalence (22).

VTE disproportionately affects the older population and incidence rates of VTE in those older than 70 years are three times higher than those aged 45 to 69 years, which again are three times higher than those aged 20 to 44 years. This age-related increase in incidence in VTE is

largely attributed to a disproportionate increase in PE burden. The reported incidence of VTE is inconsistent with regard to gender, though several studies suggest higher incidence in males. Between 5 and 10% of in-hospital deaths are a direct result of PE. In the United States, PE is responsible for 100,000 deaths per year, though deaths from diagnosed PE have been decreasing. Nevertheless, VTE is associated with significant mortality. The case fatality rate of a VTE event is ~10% at 30 days, which increases to 15% within 3 months, with a further increase up to 20% by 1 year (23).

Discussion

Pulmonary embolism is the third leading cause of death from cardiovascular disease after heart attacks and strokes. Subsequent side effects of venous thromboembolism include high pulmonary thromboembolic blood pressure and post-thrombotic syndrome. Vein thromboembolism and atherothrombosis are common risk factors and share common pathological features of inflammation, hypercoagulation and end-stage injury. Clinical likelihood testing can help identify patients who have a low clinical probability of developing venous thromboembolism with a negative plasma D-dimer test. The diagnosis is usually confirmed by compression ultrasound, which indicates deep vein thrombosis or chest CT, which may indicate pulmonary embolism. Many patients with venous thromboembolism respond to the underlying anticoagulant therapy. Patients with pulmonary embolism face the risk of deciding whether to benefit from additional advanced treatments such as thrombolytic therapy and embolidectomy. Several oral anticoagulants have been developed. In many patients, these drugs can be replaced with vitamin K and heparin antagonists, and require no laboratory monitoring. Although strong clinical trials have reported the efficacy and safety of low-dose anticoagulants and antidepressants, prevention is still frequently used in inpatients with moderate and high risk of venous thromboembolism. In this seminar, we discuss pulmonary embolism and deep vein thrombosis of the lower extremities (24).

Conclusion

VTE and PE remain preventable causes of illness and death. By combining patient presentation, clinical suspicion, and point-to-point programs, diagnosis can be simplified and unnecessary treatments can be reduced. More physicians have the training and access to portable ultrasound devices, which can prevent delays in the recognition and treatment of VTE. Clinical problems persist in hospitalized patients, especially those with serious illnesses. In such patients, diagnostic scores and imaging systems may not be included. The improved precision of helical computed tomography has improved our recognition of PE for many of these patients.

Conflict of Interest

There is nothing to disclose

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