Summary

Pulmonary embolism occurs when a foreign body, such as a blood clot or an air bubble, travels through the blood stream before becoming lodged in an artery of the lung (pulmonary artery) and blocking the flow of blood.

The most common type of emboli that travel to the lungs is a blood clot, thromboembolism, which usually forms in a leg or pelvic veins when blood flow slows down or stops. Far less often, blood clots form in the veins of the arms or in the right side of the heart. Once a clot breaks free into the bloodstream, it usually travel to the lungs.

Although PE can occur in patients without any identifiable predisposing factors, one or more of these factors are usually identified (secondary PE). The proportion of patients with idiopathic or unprovoked PE was about 20% in the International Cooperative Pulmonary Embolism Registry **(ICOPER).**

There is little information on epidemiology of thrombosis in Africa. Venous thromboembolism is predominantly a disease of older age. Incidence rates increase exponentially with age for both men and women and for both deep vein thrombosis and pulmonary embolism.

VTE is currently regarded as the result of the interaction between patient-related (genetic) and setting-related (acquired) risk factors. Patient-related predisposing factors are usually permanent, whereas setting-related predisposing factors are more often temporary.

The type of anesthesia (general or regional) also contributes to the incidence rate and should be considered when assessing a patient's risk for postoperative DVT. Patients who receive epidural or spinal anesthesia have a more than 50% decreased incidence of postoperative VTE after total hip or knee replacement compared to those having these surgeries under general anesthesia.

The very high risk associated with orthopedic surgery results from a number of factors that contribute to venous stasis, including the supine position on the operating table, the anatomic positioning of the extremity, and in patients undergoing total knee replacement, inflation of a thigh tourniquet to obtain a bloodless field. In addition, intimal injury may result from positioning of the extremity, and compression of the femoral vein may occur due to flexion and adduction of the hip during surgery on this joint.

The consequences of acute PE are primarily haemodynamic and become apparent when more than 30–50% of the pulmonary arterial bed is occluded by thromboemboli. The contribution of reflex or humoral pulmonary vasoconstriction, documented in experimental PE, is less important in humans. Secondary haemodynamic destabilization may occur, usually within first 24–48 h, as a result of recurrent emboli and/or deterioration of RV function.

CLINICAL FORMS OF PULMONARY EMBOLISM

Acute minor pulmonary embolism: A small embolus often produces no symptoms. If symptoms do develop the commonest is dyspnoea on exertion.

Acute massive pulmonary embolism: When > 50% of the pulmonary circulation is suddenly obstructed, there is a substantial increase in right ventricular afterload**.** The patient becomes acutely distressed, severely short of breath, and may be syncopal due to the combination of hypoxaemia, and low cardiac output. The combination of hypotension, hypoxaemia, and increased cardiac work may cause anginal chest pain.

Subacute massive pulmonary embolism

This is caused by multiple small or moderately sized emboli that accumulate over several weeks. Because the obstruction occurs slowly, there is time for the right ventricle to adapt; consequently the main symptoms are increasing dyspnoea and falling exercise tolerance. The blood pressure and pulse rate are usually normal because the cardiac output is well maintained.

Evaluating the likelihood of PE in an individual patient according to the clinical presentation is of utmost importance in the interpretation of diagnostic test results and selection of an appropriate diagnostic strategy. In 90% of cases, suspicion of PE is raised by clinical symptoms such as dyspnoea, chest pain and syncope, either singly or in combination. In several series, dyspnoea, tachypnoea, or chest pain were present in more than 90% of patients with PE.

Electrocardiography is no specific and its main value is to exclude other potential diagnoses, such as myocardial infarction or pericarditis. also Chest radiography findings are non specific but may be helpful Pleural effusions, atelectasis, elevation of a hemidiaphragm, and pulmonary infiltrates may be detected. In arterial blood gases the characteristic changes are a reduced PaO_2, and a normal or reduced arterial carbon dioxide pressure ($PaCO_2$) because of hyperventilation. so, clinical signs, symptoms and routine laboratory tests do not allow the exclusion or confirmation of acute PE but increase the index of its suspicion.

A negative D-dimer result in a highly sensitive assay safely excludes PE in patients with a low or moderate clinical probability, while a moderately sensitive assay excludes PE only in patients with a low clinical probability.

searching for a proximal DVT in patients with PE by compression venous ultrasonography yields a positive result in around 20% of patients. It can be used either as a backup procedure to reduce the overall false-negative rate when using single-detector CT or it can be performed to avoid CT when positive in patients with contraindications to contrast dye and/or irradiation.

A single-detector CT or multi-detector CT showing a thrombus up to the segmental level can be taken as adequate evidence of PE in most instances, whereas the necessity to treat isolated subsegmental thrombi in a patient without a DVT is unclear.

Pulmonary angiography is a reliable but invasive test and is currently useful when the results of non-invasive imaging are equivocal. Whenever angiography is performed, direct haemodynamic measurements should be performed.

Echocardiography, both transthoracic and transesophageal, is particularly helpful in emergency management decisions. In a patient with shock or hypotension, the absence of echocardiographic signs of RV overload or dysfunction practically excludes PE as a cause of haemodynamic compromise.

Chronic thromboembolic pulmonary hypertension (CTEPH)

CTEPH is a relatively rare complication of PE. The diagnostic strategy is based on echocardiography, perfusion scintigraphy, CT, right heart catheterization and pulmonary angiography. Medical therapy aims to treat right heart failure and to lower pulmonary artery resistance.

Pulmonary endarterectomy provides excellent results and should be considered as a first-line treatment whenever possible. Drugs targeting the pulmonary circulation in patients in whom surgery is not feasible or has failed are currently being tested in clinical trials.

There are two approaches to the prevention of fatal pulmonary embolism.

Primary prophylaxis is carried out using either drugs or physical methods that are effective for preventing DVT. which is preferred in most clinical circumstances.

Secondary prevention involves the early detection and treatment of subclinical venous thrombosis by screening postoperative patients with objective tests that are sensitive for venous thrombosis.

The prophylactic measures most commonly used include both pharmacologic and mechanical approaches.

The pharmacologic agents presently include

- Low dose unfractionated heparin
- Low molecular weight heparin
- Oral anticoagulants (International Normalized Ratio [INR] of 2.0 to 3.0)
- Aspirin
- Use of the substituted pentasaccharide fondaparinux

The mechanical devices include

- compression stockings
- Intermittent Plantar Compression
- intermittent pneumatic compression (IPC)

In Europe, LMWH prophylaxis is generally started 10–12 h before surgery, in practice usually the evening before the surgery. In North America, prophylaxis with LMWH is usually started 12–24 h after surgery, to both minimize the risk of bleeding and to simplify same-day hospital admission for elective surgery. There is a widespread under-use of thromboprophylaxis in medical and surgical patients. Previous studies conducted in different countries have shown that only 35–42% of patients in the highest risk groups receive adequate thromboprophylaxis , with a lowest score of 16% for hospitalized medical patients

The goals of venous thromboembolism (VTE) treatment are the prevention of clot propagation, prevention of pulmonary embolism, and prevention of recurrent thrombosis as well as the development of late complications, such as the postphlebitic syndrome and chronic thromboembolic pulmonary hypertension. The mainstay of therapy is anticoagulation. Venous thrombosis usually occurs in a normal vessel wall, with stasis or hypercoagulability as predisposing factors. Because platelets are of lesser importance in the pathogenesis of venous thrombosis, drugs that prevent thrombin formation or lyse fibrin clots are of major importance.

At first, hemodynamically unstable PE or acute RV failure with resulting low systemic output is the leading cause of death in patients with high-risk PE. Therefore, respiratory and hemodynamic supportive treatment is of vital importance in patients with PE and RV failure.

Patients with DVT or pulmonary embolism should be treated acutely with LMW heparin, fondaparinux, unfractionated intravenous heparin, or adjusted-dose subcutaneous heparin. When unfractionated heparin is used, the dose should be

sufficient to prolong the activated partial thromboplastin time (aPTT) to 1.5 - 2.5 times the mean of the control value, or the upper limit of the normal aPTT range. Treatment with LMW heparin, fondaparinux, or unfractionated heparin should be continued for at least five days and oral anticoagulation should be overlapped with LMW heparin, fondaparinux, or unfractionated heparin for at least four to five days.

For most patients, warfarin should be initiated simultaneously with the heparin, at an initial oral dose of approximately 5 mg per day. In elderly patients and in those at high risk of bleeding or who are undernourished, debilitated, or have heart failure or liver disease, the starting dose should be reduced. The heparin product can be discontinued on day five or six if the INR has been therapeutic for two consecutive days.

Oral anticoagulation with warfarin should prolong the INR to a target of 2.5 (range: 2.0 to 3.0). If oral anticoagulants are contraindicated or inconvenient, long-term therapy can be undertaken with either adjusted-dose unfractionated heparin, low-molecular-weight heparin, or fondaparinux. Because of ease of use, especially in the outpatient setting, LMW heparin or fondaparinux is preferred to unfractionated heparin.

Treatment with heparin is usually followed by at least a three to six month period of anticoagulation to prevent recurrent disease. Warfarin therapy is highly effective for this purpose and is preferred in most patients. In patients with a VTE, long-term therapy with warfarin reduces the frequency of objectively documented recurrent venous thromboembolism from 47 to 2 %.

Thrombolysis clearly is indicated in patients with massive PE and associated hemodynamic instability . However, the role of thrombolysis in patients with submassive PE remains controversial. Inferior vena cava filters are used to prevent the pulmonary embolization of a thrombus. Surgical embolectomy is rarely performed. It should be reserved for patients who have a massive pulmonary embolism and hemodynamic instability despite heparin and cardiopulmonary support, who either fail thrombolytic therapy or have a contraindication to it. Even in the hands of an experienced surgical team, postoperative mortality is high.

In pregnancy, heparin is the mainstay of therapy for acute VTE. Heparin does not cross the placenta, and so does not carry risks of teratogenesis or fetal hemorrhage, although bleeding at the uteroplacental junction is a possibility . Like unfractionated heparin, **LMWHs** do not cross the placenta, and so are not felt to carry increased risk of fetal hemorrhage or teratogenesis. **Coumarin** derivatives cross the placenta, and are relatively contraindicated in pregnancy.

Most patients with acute pulmonary embolism who receive adequate anticoagulant therapy survive. Shock at presentation is associated with an increase in mortality rate ; a majority of the deaths among patients presenting in shock occur within the first hour after presentation. Both chronic leg pain and swelling (the post-thrombotic syndrome) and chronic thromboembolic pulmonary hypertension are possible long term sequelae of acute pulmonary embolism.

10% of patients with symptomatic PE die within 1 hour of onset of symptoms. Among patients who are diagnosed with PE, the mortality rate is about 10% at 2 weeks and 25% at 1 year. However, only 20% of deaths during the first year after PE are a direct consequence of PE; most are due to malignancy and underlying cardiorespiratory disease.

Finally, Pulmonary embolism (PE) remains one of the leading causes of morbidity and mortality in the emergency and cardiovascular setting, especially when associated to hemodynamic instability.