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Review Article

Pulmonary Embolism

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Abstract

Pulmonary embolism (PE) is a relatively common cardiovascular pathology (approximately 1 case per thousand population per year). In the United States, PE affects approximately 650,000 people and results in 350,000 deaths per year. PE occurs more often in the elderly (In the United States, venous thromboembolism occurs for the first time in about 100 out of 100,000 people per year and increases exponentially from less than 5 cases per 100,000 people in children under 15 years of age to 500 cases per 100,000 people at age 80 years).

Keywords: pulmonary embolism (PE); European Society of Cardiology; hypoxia; embolism; pulmonary infarction

Summary

Pulmonary embolism (PE) is a relatively common cardiovascular pathology (approximately 1 case per thousand population per year). In the United States, PE affects approximately 650,000 people and results in 350,000 deaths per year. PE occurs more often in the elderly (In the United States, venous thromboembolism occurs for the first time in about 100 out of 100,000 people per year and increases exponentially from less than 5 cases per 100,000 people in children under 15 years of age to 500 cases per 100,000 people at age 80 years) [1]. Data on the association of gender with PE vary, but an analysis of a US national database found that men have a 20% to 30% higher mortality rate than women.[1] The incidence of venous thromboembolism among the elderly is higher in men, but in patients under 55 years of age it is higher in women. The disease is based on thrombus formation, which is promoted by three factors (Virchow's triad): impaired blood flow (blood stasis), damage to the endothelium of the vascular wall, hypercoagulation (as well as inhibition of fibrinolysis). Impaired blood flow is caused by varicose veins, compression of vessels from the outside (tumor, cyst, bone fragments, enlarged uterus), destruction of venous valves after phlebothrombosis, as well as immobilization, which disrupts the function of the muscular-venous pump of the lower extremities.

Polycythemia, erythrocytosis, dehydration, dysproteinemia, and increased fibrinogen levels increase blood viscosity, which slows blood flow. When the endothelium is damaged, the subendothelial zone is exposed, which triggers a cascade of blood clotting reactions. The reasons for this are direct damage to the vessel wall during the installation of intravascular catheters, filters, stents, vein replacement, trauma, and surgery. Hypoxia, viruses, and endotoxins also cause damage to the endothelium. During a systemic inflammatory reaction, leukocytes are activated, which, by attaching to the endothelium, damage it.

The source of blood clots in PE most often are the veins of the lower extremities (lower extremity venous thrombosis), much less often - the veins of the upper extremities and the right side of the heart. The likelihood of venous thrombosis increases in pregnant women, in women who have taken oral contraceptives for a long time, in patients with thrombophilia.

According to the 2008 recommendations of the European Society of Cardiology, patients are stratified into high and low risk groups. The latter is also divided into moderate and low risk subgroups. High risk - the risk of early death (in hospital or within 30 days after pulmonary embolism) is more than 15%, moderate - no more than 15%, low - less than 1%. To determine it, three groups of markers are taken into account - clinical markers, markers of right ventricular dysfunction and markers of myocardial damage.

Embolization is caused by blood clots freely located in the lumen of the vein, attached to its wall only in the area of its base (floating blood clots). The detached thrombus flows through the right side of the heart into the pulmonary artery, obstructing its lumen. The consequences of this depend on the size, number of emboli, the reaction of the lungs and the activity of the body's thrombolytic system.

With small emboli there are no symptoms. Large emboli impair the perfusion of segments or even entire lobes of the lung, which leads to impaired gas exchange and the development of hypoxia. In response to this, the lumen of the vessels of the pulmonary circulation reflexively narrows, and the pressure in the pulmonary arteries increases. The load on the right ventricle increases due to high pulmonary vascular resistance caused by obstruction and vasoconstriction. Thromboembolism of small branches of the pulmonary artery is not accompanied by hemodynamic disorders and in 10% of cases pulmonary infarction and secondary infarction pneumonia develop.

Clinically, PE is classified into the following types:

• massive - more than 50% of the volume of the vascular bed of the lungs is affected (embolism of the pulmonary trunk and/or main pulmonary arteries) and the disease manifests itself in shock and/or systemic hypotension;

• submassive - 30-50% of the volume of the vascular bed of the lungs is affected (embolism of several lobar or many segmental pulmonary arteries) and is manifested by symptoms of right ventricular failure;

• non-massive - less than 30% of the volume of the vascular bed of the lungs is affected (embolism of small distal pulmonary arteries), manifestations are absent or minimal (pulmonary infarction). Embolism of small branches of the pulmonary arteries may be asymptomatic or have nonspecific symptoms (slight increase in body temperature, cough).

Massive PE manifests as acute right ventricular failure with the development of shock and systemic hypotension (a decrease in blood pressure <90 mmHg or a drop of ≥40 mmHg, which is not associated with arrhythmia, hypovolemia, or sepsis). Shortness of breath, tachycardia, and fainting may occur. With submassive PE, there is no arterial hypotension, and pressure in the pulmonary circulation increases moderately. In this case, signs of dysfunction of the right ventricle of the heart and/or myocardial damage are detected, which indicates increased pressure in the pulmonary artery. With non-massive pulmonary embolism, there are no symptoms and after a few days a pulmonary infarction occurs, which is manifested by pain in the chest when breathing (due to irritation of the pleura), fever, cough and, sometimes, hemoptysis and is detected x-ray (typical triangular shadows).

During auscultation of the heart, an increase and accentuation of the second tone over the tricuspid valve and pulmonary artery is detected, as well as a systolic murmur at these points. Splitting of the second tone and gallop rhythm are possible, which is considered a bad prognostic sign. In the area of pulmonary infarction, weakened breathing, moist rales and pleural friction noise are heard.

Diagnosis is difficult because symptoms of PE are nonspecific and diagnostic tests are imperfect. Standard testing (routine laboratory tests, electrocardiography (ECG), chest x-ray) is useful only to exclude other pathologies (eg, pneumonia, pneumothorax, rib fractures, myocardial infarction, pulmonary edema).

Sensitive and specific methods for diagnosing pulmonary embolism include:

• massive - more than 50% of the volume of the vascular bed of the lungs is affected (embolism of the pulmonary trunk and/or main pulmonary arteries) and the disease manifests itself in shock and/or systemic hypotension;

• submassive - 30-50% of the volume of the vascular bed of the lungs is affected (embolism of several lobar or many segmental pulmonary arteries) and is manifested by symptoms of right ventricular failure;

• non-massive - less than 30% of the volume of the vascular bed of the lungs is affected (embolism of small distal pulmonary arteries), manifestations are absent or minimal (pulmonary infarction).

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Sensitive and specific methods for diagnosing PE include d-dimer determination, echocardiography, computed tomography (CT), ventilation-perfusion scintigraphy, pulmonary vascular angiography, as well as methods for diagnosing deep vein thrombosis of the lower extremities (ultrasonography, CT venography).[2]

D-dimer is a fibrin breakdown product; its elevated level suggests recent thrombus formation. Determination of the level of d-dimers is a highly sensitive (more than 90%) but not specific method for diagnosing pulmonary embolism. This means that increased levels of d-dimers occur in a wide variety of other pathological conditions (eg, infection, inflammation, necrosis, aortic dissection). However, a normal level of d-dimers (<500 μg/L) allows one to exclude PE in patients with a low and intermediate probability [2].

Signs of pulmonary embolism on the electrocardiogram are nonspecific and absent in most patients [2]. Sinus tachycardia and a tall and pointed P wave (P-pulmonale, a sign of right atrium overload) are often detected [3]. Approximately 20% of patients may exhibit signs of acute cor pulmonale (right ventricular overload): deviation of the electrical axis of the heart to the right; SIQIIITIII syndrome (McGinn-White syndrome [4]) - deep S wave in lead I, pronounced Q wave and negative T wave in lead III; new right bundle branch block; deep S waves in V5-6 combined with negative T waves in V1-4[5]. However, acute cor pulmonale can also occur in other acute pathologies of the respiratory system (massive pneumonia, severe attack of bronchial asthma) [6].

X-ray of the chest organs

Chest x-ray reveals signs of pulmonary hypertension of thromboembolic origin: high standing of the dome of the diaphragm on the affected side, dilation of the right heart and roots of the lung, Pall's sign (dilation of the right descending pulmonary artery), Westermarck's sign (local depletion of the vascular pattern of the lung), discoid atelectasis. In case of pulmonary infarction - Hampton's triangle (a cone-shaped compaction with the apex facing the hilum of the lungs), pleural effusion on the affected side.

Echocardiography

Using echocardiography for pulmonary embolism, it is possible to detect dysfunction of the right ventricle (its dilatation and hypokinesis, bulging of the interventricular septum towards the left ventricle), signs of pulmonary

hypertension, and tricuspid regurgitation. Sometimes it is possible to detect blood clots in the heart cavity (for this, transesophageal echocardiography is more informative) [6]. Also, using this method, they exclude other heart pathologies and can identify a patent foramen ovale, which can affect the severity of hemodynamic disorders and cause paradoxical embolism of the arteries of the systemic circulation [7].

Spiral computed tomography

CT angiopulmonography can detect blood clots in the pulmonary artery. With this method, the sensor rotates around the patient, who is previously injected intravenously with a contrast agent. As a result, a three-dimensional picture of the lungs is created. However, the patient must be able to hold his breath during the procedure (several seconds). The method is less invasive and safer than angiography [8]. According to experts from the European Society of Cardiology, in high-risk patients, single-detector CT is sufficient to confirm or exclude PE. In low-risk patients, multidetector CT is recommended, as it allows more clearly identifying thrombi in the branches of the pulmonary artery [8].

Ultrasound examination of the deep veins of the lower limb

Ultrasound examination (ultrasound) of peripheral veins can detect blood clots in the veins of the lower extremities. In most cases, they are the source of thromboembolism. For this, two methods are used. The first is compression ultrasound; in this case, in B-mode, a cross-section of the lumen of arteries and veins is obtained. Then the ultrasound sensor is pressed on the skin in the projection of the blood vessels. If there is a blood clot in the vein, its lumen does not decrease (compression does not occur). Another technique is Doppler ultrasound; at the same time, using the Doppler effect, the speed of blood flow in the vessels is determined. A decrease in the speed of blood flow in the veins is a possible sign of blockage by a blood clot. The combination of compression and Doppler ultrasound is called duplex ultrasonography.

Ventilation-perfusion scintigraphy

Ventilation-perfusion scintigraphy can identify areas of the lung that are ventilated but not perfused (as a result of obstruction by a thrombus). A normal lung scintigram can exclude pulmonary embolism with 90% accuracy. However, perfusion deficiency can occur in a variety of other lung pathologies. Typically, this method is used when CT is contraindicated.

Angiography of pulmonary vessels

Angiography of pulmonary vessels is the most accurate method for diagnosing pulmonary embolism, but at the same time it is invasive and has no advantages over CT. The criteria for a reliable diagnosis are a sudden break in the branch of the pulmonary artery and the contours of a blood clot; the criteria for a probable diagnosis are a sharp narrowing of the branch of the pulmonary artery and slow washout of contrast.

Treatment

Treatment of patients with pulmonary embolism should be carried out in intensive care units.

In case of cardiac arrest, cardiopulmonary resuscitation is performed. If hypoxia occurs in a patient with pulmonary embolism, oxygen therapy is administered (masks, nasal catheters); Artificial ventilation is less often required. To eliminate hypotension, saline solutions or vasopressors (adrenaline, dobutamine, dopamine) are administered intravenously [9].

Anticoagulant therapy

Timely anticoagulant therapy reduces the risk of death and recurrent thromboembolism, so it is recommended to start it not only with a confirmed diagnosis, but also during the diagnosis process if there is a high probability of pulmonary embolism. For this purpose, unfractionated heparin (intravenously), low molecular weight heparin is used: enoxaparin, dalteparin (subcutaneous) or fondaparinux (subcutaneous).

The dosage of unfractionated heparin is adjusted based on the patient's weight and activated partial thromboplastin time (aPTT) [19]. To do this, prepare a solution of sodium heparin - 20,000 IU/kg per 500 ml of physiological solution. First, 80 IU/kg is administered intravenously, and then an infusion is carried out at a rate of 18 IU/kg/h. 6 hours after the bolus injection, the APTT is checked and the rate of heparin administration is adjusted as indicated in the table. APTT is determined 3 hours after each speed change; when the desired level is reached (46-70 s, 1.5-2.5 times higher than control), this indicator is monitored daily.

APTT, Change in dosage

<35 Increase by 4 IU/kg/h

35—45 Increase by 2 IU/kg/h

46—70 Do not change

71—90 Reduce by 2 IU/kg/h

>90 Stop administration for 1 hour, then reduce by 3 IU/kg/hour

In most cases, low molecular weight heparins are used, as they are more effective, safer and more convenient to use than unfractionated heparins [20]. However, they should be prescribed with ca ution in cases of renal dysfunction (decreased creatinine clearance <30 ml/min) and with a high risk of hemorrhagic complications (the duration of action of unfractionated heparin is shorter and therefore can be discontinued more quickly in the event of bleeding). Of the low molecular weight heparins, the following are recommended: enoxaparin (1 mg/kg every 12 hours or 1.5 mg/kg once a day), tinzaparin [English] (175 IU/kg once a day). In patients with cancer, dalteparin (200 IU/kg once daily) can be used [9].

For anticoagulation, fondaparinux, a selective factor Xa inhibitor, can be used. It is prescribed subcutaneously once a day at 5 mg for body weight <50 kg, 7.5 mg for 50-100 kg and 10 mg for >100 kg. It is highly effective for pulmonary embolism and does not cause thrombocytopenia, unlike heparin. However, fondaparinux is contraindicated in severe renal failure (creatinine clearance <20 ml/min) [10].

It is also advisable to prescribe warfarin on the first day of introducing direct-acting anticoagulants (heparins or fondaparinux). When an international normalized ratio (INR) level of 2-3 is reached and maintained at this level for at least 2 days, direct anticoagulants are discontinued (but not earlier than 5 days from the start of their use). The initial dose of warfarin is 5 or 7.5 mg 1 time per day. For patients under 60 years of age and without serious concomitant pathology, an initial dose of 10 mg is acceptable. Warfarin is continued for at least 3 months [11].

Reperfusion therapy

The goal of reperfusion therapy is to remove the clot and restore normal pulmonary blood flow. Most often, thrombolytic therapy is used for this. It is recommended for high-risk patients. Some authors allow the use of thrombolysis at moderate risk, but the benefit of such therapy in this group of patients is currently unclear [12]. The following drugs are used:

• Streptokinase - loading dose of 250,000 IU over 30 minutes, then 100,000 IU per hour for 12-24 hours or accelerated regimen 1.5 million IU over 2 hours

• Urokinase - loading dose of 4400 IU/kg body weight for 10 minutes, then 4400 IU/kg body weight per hour for 12-24 hours or an accelerated regimen of 3 million IU for 2 hours

• Alteplase - 100 mg over 2 hours or accelerated regimen 0.6 mg/kg body weight over 15 minutes (maximum dose 50 mg)

Bleeding is the main problem of thrombolytic therapy. Massive bleeding develops in 13% of cases, and intracerebral hemorrhage occurs in 1.8% of cases.

Surgical methods

Surgical removal of the blood clot (thrombectomy) is considered an alternative treatment for high-risk pulmonary embolism when thrombolytic therapy is contraindicated. In patients with a high risk of relapse and with absolute contraindications to anticoagulant therapy, vena cava filters can be installed [13]. They are mesh filters that catch blood clots coming off the wall and prevent them from entering the pulmonary artery. The vena cava filter is inserted percutaneously, usually through the internal jugular or femoral vein, and placed below the renal veins (higher in the presence of blood clots in the renal veins) [14].

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