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Pulmonary Embolism: A Literature Review

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Abstract: Pulmonary embolism is the blockage of a branch of the pulmonary artery by a substance that has traveled from elsewhere in the body through the bloodstream. The majority of pulmonary embolisms are caused by venous thromboembolism but in some cases it may also come from other sources (fat, injuries, orthopedic surgeries or amniotic fluid during childbirth). The aim of this retrospective study is to highlight some important information about the pulmonary embolism. Extensive review of the recent literature was conducted in electronic database Medline and via the link of the Greek Association of Academic Libraries (HEAL-Link). Pulmonary embolism can be immediately a life threatening situation. Cause of the disease is the blockage of an artery in the lungs usually by a clot (clogging). Pulmonary embolism is divided into (a) small and medium size (b) multiple pulmonary emboli, and (c) massive pulmonary embolism. There are several factors that affect the occurrence of pulmonary embolism such as age over 40 years, major surgery, fractures, particularly those of the basin and prolonged bed stay (more than five days). The treatment of pulmonary embolism and its diagnosis should be direct because many times even today people lose their lives, despite the tremendous development of diagnostic tools.

Keywords: Pulmonary Embolism, Pathophysiology, Clinical Presentation, Diagnostic Tests, Treatment

1. Introduction

Pulmonary embolism is an acute, serious condition that can be directly life threatening. It happens when an artery in the lungs is blocked by a substance that has traveled from elsewhere in the body through the bloodstream. This substance usually results from a blood clot in the legs or pelvis. [1]

The blockage of an artery that supplies the lungs, causes severe damage, interrupts their smooth operation and may, depending on the importance of the blocked artery, directly lead to death.

The most common form of emboli that causes pulmonary embolism is the blood clot described above. But there are also other situations that may occur, such as amniotic emboli during childbirth, scatter tumor emboli from a malignant disease or even traumatic fat emboli originating from the bone or bone marrow in patients with sustained blunt trauma and multiple fractures. [2]

2. Aim

The aim of this retrospective study is to examine the literature sources associated with the pulmonary embolism focusing on the pathophysiology, clinical picture and course of the disease, diagnostic tests and methods of treatment.

3. Methods

Extensive review of the recent literature was conducted in electronic database Medline and via the link of the Greek Association of Academic Libraries (HEAL-Link), using the following key words: pulmonary embolism, pathophysiology, clinical presentation, diagnostic tests, treatment. An

exclusion criterion of studies was the language other than the Greek and English.

4. Pathophysiology

If the venous clot is dislodged from the position in which it was formed, then its embolism to a vessel of pulmonary circulation or arterial systemic circulation follows. (paradoxical embolism, observed in cases of open foramen ovale) [3].

It has been experimentally proved that when 60% of the pulmonary artery's vascular network is blocked, then a severe drop in blood pressure and acute bend of the right ventricular (acute cor pulmonale) is caused. In cases where the blockage reaches 80% of the vascular network, sudden death occurs. Cardiac hemodynamic effects on pulmonary embolism depend on the occlusion rate of the pulmonary vasculature, the distance of this network and the release of vasomotor substances that cause bronchospasm followed byfurther reduction of pulmonary perfusion and an increase of VD (dead space). [4]

Acute pulmonary heart is caused by the sudden increase in the average pressure of the pulmonary artery, in concentrations greater than 40 mmHg due to increased pulmonary vascular resistance. The effects of pulmonary embolism by the cardiovascular system may be acute pulmonary heart, myocardial ischemia, acute circulatory failure, and the left-sided failure and from the lungs, hypoxia and atelectasis. [5] In acute deficiency, the pressure in the right ventricle increases, resulting in septum's displacement to the left, lowering evendototitas of the left ventricular, dysfunction of both ventricles, decrease in cardiac output and a drop of blood pressure. Coronary insufficiency due to pulmonary embolism is attributed to the significant reduction of cardiac output, the drop in aorta's pressure, hypoxia, and possibly to the existence of a reflex through vagal which reduces the width of the coronary vessels. [1, 6]

Pulmonary infarction appears as thickening hemorrhagic lung area when average size pulmonary arteries are unclogged due to left heart failure and drop of pressure in the systemic circulation. Pulmonary infarction occurs only in 10% of cases of pulmonary embolism because the pulmonary parenchyma is satisfactorily oxygenated through the bronchial. [7]

The fate of thromboembolism in the lung will determine the further course of the disease. Several mechanisms restore the circulation of the pulmonary artery, unless the episodes are repeated. When an oversized embolous plugs in the pulmonary circulation, it can cause transient syncope, but then it can be destroyed and dissolved within a few 24-hour, otherwise when it remains, it is organized, shrink and coalesced with the vessel wall. [1, 4]

5. Course of the Disease

Necrosis (infarction) of the lung tissue may follow pulmonary embolism. This development is rare (about 10%), since it requires simultaneous interruption of pulmonary ventilation and pulmonary perfusion via the pulmonary artery and the bronchial arteries. [8]

A few days after the installation of the embolus, the process of re-establishing the damage via the lysis of thrombus by fibrinolytic mechanisms begins and finally the damage is organized in a way that the clot will be converted to small scar adherent to the vascular wall. This process is completed in 10-14 days. [9] In cases where, despite the above mechanisms, the restoration of the circulation, delays, the development of collateral bronchial movement restores flow in capillary network resulting in gradual restoration of surfactant production, atelectasis, hypocapnia and vasoconstriction. [10]

6. Clinical Picture

Pulmonary embolism is not always displayed the same clinical picture. Symptoms and objective findings vary and are depending on the size and number of branches of the pulmonary vascular network that has been occluded. It can occur suddenly with a severe clinical picture and bring even death. Sometimes important dyspnea that is progressively worsening appears, and often it is not attributed to pulmonary embolism. [11]

In massive pulmonary embolism, symptoms are noisy due to apparent disorder of cardiac function and severe hemodynamic disturbances. Sudden chest pain appears, like the pain of acute myocardial infarction. [8]

The pain is accompanied by a large drop in blood pressure (shock) tachycardia, tachypnea, restlessness, pallor, sweating and electrocardiographic changes (deep S in lead I, deep Q in lead III, inverted T in V1, V2, V3, V4 leads, transient right blockade, high pulmonary P etc.) corresponding to «strain of the right heart. "The pain is accompanied by a large drop in blood pressure (shock) tachycardia, tachypnea, restlessness, pallor, sweating and electrocardiographic changes (deep S in lead I, deep Q in lead III, inverted T in V1, V2, V3, V4 leads, transient right blockade, high pulmonary P etc.) corresponding to «strain of the right heart."[12]

In cases of the obstruction of mean size pulmonary artery's branch, symptoms of pulmonary infarctionare observed (in the section of the lung inwhichthe embolism happened, blood flows from the bronchial arteries (hemoptysis), cough, light dyspnea, low-grade fever (40%), tachycardia etc) [9]

Within a small embolism, key and perhaps unique symptom may be the tachycardia. Therefore, postoperative appearance of unexplained tachycardia, should give suspicion of pulmonary embolism. The electrocardiogram is normal (shows only the tachycardia). [3]

Unexplained shortness of breath should make a suspicion of pulmonary embolism. Pulmonary infarction is responsible for the majority of diagnostic errors. As mentioned above, this is caused by inadequate blood supply from the bronchial arteries (the aorta branches).[4]

7. Diagnostic Tests

The clinical diagnosis of pulmonary embolism is not an

easy task and as such, the doctor should think of it, when he/she is facing an acute or of unspecified etiology lung disease. Fever that invades by shivering and marked leukocytosis suggests pneumonia. The expectoration is purulent and on pulmonary infarct is bloody, while the microscopic examination does not indicate the presence of microbes, such as for pneumonia.[1]

The findings on physical examination of patients may be minimal and illusory. Clinical examination of the lung is usually negative. Localized wheezing or atelectatic crackles may occur. Friction sound or pleural effusion findings can be found in case of infarct. The findings from the examination of the circulatory vary. The only consistent finding is tachycardia. [12] In severe situations, abdominal gallop is observed, as well as sharp sound of the occlusal pulmonary artery (as long as good cardiac output exists) and mainstream A-waves tothe cervical vein pulse. Harsh systolic ejection murmur in the hearing focus of pulmonary and continuous or systolic chest murmur is possible to exist. These murmurs are caused by turbulent blood movement due to occlusion and they are disappeared after the clot dissolution. In severe cases, there is a cardiogenic shock image (with jugular distension). [9]

On physical examination of the patient with suspected pulmonary embolism, probable thrombosis of deep veins should always be sought carefully. It is found in only 50% of cases of emboli from clots. The thrombus may be dislodged from the venous system of the limbs with no clinical or laboratory evidence of thrombosis in the deep and remaining venous system.[8]

Lactic deideogenasi and serum bilirubin may be elevated, whereas GOT is usually normal. PO_2 is found to the lower normal limit. The chest X-ray, despite the heavy clinical picture of patient is usually normal. Sometimes, there is a picture of pulmonary hypovolaemic or localized area with a deficit of vascular profiling.[13]

Most of the times, ECG isnormal. However, in cases of mass embolism accompanied by retrosternal pain and shock, it shows tachycardia, a shift of the axis of QRS clockwise, the classical electrocardiogram picture of acute pulmonary heart and reversal of T at the right precordial leads.[1]

The scans of perfusion and ventilation don't facilitate the diagnosis as cold regions in the lung parenchyma are found το many other pathologies, as for pneumonia, for aerial cysts lung, for pleural fluid, asthma, congestive heart failure as well as for diabetic diseases of the lungs. The eminently useful laboratory exam is arteriography of the pulmonary artery and it should be done under special conditions. [14]

8. Treatment

The treatment of pulmonary embolism may be surgical or conservative. Conservative treatment includes administration of anticoagulants, general support circulation, antibiotics, thrombolytics, analgesics. [15]

8.1. Heparin

Heparin linked to antithromvoni III increases its own activity. It prevents the increase in size of the existing thrombus and facilitates the dissolution of the endogenous fibrinolytic system. After administration of heparin for a period of 5-7 days, stabilization of the thrombus occurs in the endothelium of veins or pulmonary artery. Heparin doesn't cause dissolution of the existing clot. [15]

Dosage: The initiation of treatment with heparin becomes after the patient control of the existence of significant bleeding site and after an examination of the rectum, in order microscopic bleeding from the digestive tract to be ruled out. Initially, 5000-10000 units of the drug are administered in a single dose and subsequently 1000-1500 units per hour are administered by continuous intravenous injection. The therapeutic levels of heparin are secured when the time of the patient's partial thromboplastin time is at least twice that of the control. [4]

Complications: The major side effect of heparin is bleeding. If dangerous for life intracranial bleeding exists, the drug is stopped and protamine sulfate is administered. [1]

In patients undergoing long-term treatment with heparin, osteopenia, osteoporosis and pathological fractures of bone may occur. Abnormal liver function tests often occur, but rarely are associated with clinically significant toxicity.[11]

8.2. Warfarin

It is an antagonist of vitamin K and inhibits activation of the coagulation factors II, VII, IX and X, via c-decarboxylation. For the manifestation of its full effect, often, a five days period is required, even when the prothrombin time (through which the action is controlled) is extended in a shorter time. When its administration starts during the active thrombotic condition, levels of proteins C and S are decreased, thus thrombogenesis is favored. The concomitant use of heparin for a period of five days compensates this action of warfarin. [2]

Dose: Initially it is administered at a dose of 7,5-10mg. Usually, however, after a period of some days, the dose should be reduced. In cases of patients with poor nutritional status, or patients who have received long-term treatment with antibiotics and may exhibit some degree of avitaminosis K, the initial dose of warfarin should be substantially less, e.g. 2.5mg. [2, 3]

Complications: As in the case of heparin, the most common complication is bleeding. If threatening for life bleeding appears, freshly frozen plasma (normally two points) is administered in order to achieve hemostasis. [1]

Duration of anticoagulation. After stopping treatment with anticoagulants, the recurrence rate of pulmonary embolism is extremely high. Cancer patients or severe obesity should probably take anticoagulants for all their life. For the other patients, anticoagulation is recommended for a single episode of thrombosis of the leg veins for three months, for an in-the-vein thrombosis of proximal venous leg network for 6 months and for pulmonary embolism for 1 year. [9,16]

8.3. Thrombolysis

Thrombolysis immediately reverses the right heart failure

and thus reduces mortality and the rate of recurrence of pulmonary embolism. By this method is achieved: [3, 17]

1. The dissolution of the larger part of the clot occluding the pulmonary vessels
2. Inhibition of serotonin secretion and other neuro-humoral factors, which could exacerbate pulmonary hypertension and
3. The dissolution of the clot in the veins of the pelvis or in the deep venous system of the legs, thus reducing the rate of recurrence of pulmonary embolism
4. Administration of 100mg recombinant activator of the tissue plasminogen, as a continuous intravenous infusion from a peripheral vein over a period of 2 hours, is preferred. Patients respond to thrombolysis even 14 days after the occurrence of pulmonary embolism. [9]
5. Contraindications method is the existence of intracranial disease, recent surgery or injury. Intracranial hemorrhage occurs in 1%. The best method of preventing bleeding complications is the monitoring of patients for conditions which constitute a contraindication of thrombolysis, prior to the method's application. [12]
6. Thromboendarterectomy Patients who develop pulmonary hypertension due to pulmonary embolism may experience severe dyspnea at rest or with a low degree of fitness. Application of thromboembolectomy, substantially reduces, and in some cases, cures for pulmonary hypertension. [18]
7. Prevention. Prevention of pulmonary embolism is extremely important, because it is difficult to be diagnosed and is costly to treat. There are, fortunately, a large number of preventive methods that show satisfactory success.[2]

The *anticoagulation* with heparin and warfarin is secondary prevention measure of recurrence of pulmonary embolism. The treatment of the disease involves the dissolution of the clot or its removal with embolectomy. It is recommended for high-risk patients due to right heart failure and for those who are in a big risk of the disease recurrence despite the use of anticoagulants. [10]

The echocardiography helps the classification based on the relative risk, so the determination of the right ventricular dysfunction necessitates the application of thrombolysis or embolectomy, even in patients with normal blood pressure. [19]

In cases where it is found by the scan of the lung, attack less than 30%, the function of the right ventricle is not usually affected. The treatment of these patients only with anticoagulant therapy has usually good results. [1, 20]

9. Supporting Measures

These measures include pain relief (mainly administration of nonsteroidal anti-inflammatory agents), supplementary oxygen therapy and psychological support. For the treatment of right heart failure and cardiogenic shock, dobutamine is administered (beta-adrenoceptor agonist with positive inotropic activity and vasodilator activity in the pulmonary circulation). [2, 21] Generally, loading the patient with intravenous fluids, it should be avoided, because the dilatation of the right ventricle is worsened and may lead to further reduction of the left ventricle's supply. [22, 23]

10. Conclusion

Pulmonary embolism is the most common predictable acute fatal disease and the most part of it, remains undiagnosed, which negatively affects the survival, mainly because of relapses.

In suspicion of pulmonary embolism, detailed laboratory tests for diagnosis and the delivery of therapy always in relation to the benefit / risk for the patient, are absolutely necessary.

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