Prevention and Modern Treatment of Fatty Embolism in Traumatological Patients

Maxmudov Boburbek Erkinovich,

Teacher, Ferghana Medical Institute of Public Health Uzbekisan, Fergana

ABSTRACT

A fat embolism is a multiple occlusion of blood vessels by lipid globules. In this article highlights of prevention and modern treatment of fatty embolism in traumatological patients

Keywords: modern medicine, embolism, fatty embolism, traumatological patients, treatment.

Fat embolism (FE) is a severe complication that develops mainly with damage to long tubular bones as a result of blockage of vascular pools by lipid complexes that have entered the bloodstream. It manifests itself in the form of respiratory failure, damage to the central nervous system, the retina of the eye. The main symptoms include headache, encephalopathy, floating eyeballs, paralysis, paresis, chest pain, shortness of breath, tachycardia. The diagnosis is made on the basis of the clinical picture, the presence of predisposing factors in the anamnesis and the detection of large lipid particles in the blood. Specific treatment includes ventilators, fat desemulsifiers, anticoagulants, glucocorticosteroids, sodium hypochlorite. In addition, non-specific therapeutic measures are carried out.

The frequency of occurrence ranges from 0.5-30% of the total number of trauma patients. It is usually diagnosed in patients aged 20-60 years. The minimum number of embolisms is registered among people who have been injured while intoxicated. The mortality rate is 30-67%; this indicator directly depends on the severity and type of damage, the speed of medical care.

Reasons

The essence of the pathological process is the obturation of blood vessels by drops of fat. This leads to a violation of blood flow in important structures of the body – the brain and spinal cord, lungs, heart. Among the conditions that can cause are:

Injuries. The main cause of lipid embolism is fractures of the diaphysis of the femurs, tibia, and pelvis. The risk of developing pathology increases with volumetric and multiple injuries accompanied by bone crushing. It is believed that pathology occurs in 90% of people with injuries of the musculoskeletal system. However, its clinical manifestations develop only in a relatively small number of cases. In addition, dyslipidemia, which can provoke vascular obstruction, occurs in patients with burns, damage to a large volume of subcutaneous fat.

Shocks and post-resuscitation illness. Emboli formation occurs with shocks of any origin in 2.6% of cases. The reason is an increase in catabolic processes, a metabolic storm. Symptoms often develop by the end of 2-3 days after the patient is brought out of critical condition.

Intravenous administration of oil solutions. Cases of iatrogenic origin of the disease are

isolated. Fat occlusion occurs due to exogenous fats that have entered the bloodstream due to erroneous actions of a medical professional. In addition, fat embolism is sometimes diagnosed in athletes using synthol to increase muscle mass.

Hypovolemia. With severe hypovolemia, an increase in hematocrit occurs, the level of tissue perfusion decreases, stagnation occurs. All this causes the formation of large fat droplets in the circulatory system. Dehydration develops with prolonged vomiting, diarrhea, insufficient drinking water in hot climates, excessive intake of diuretics.

Pathogenesis.

According to the classical theory, a fat embolism is the result of a direct hit of bone marrow particles into the bloodstream at the time of injury. Further, globules with blood flow spread in the body. When the particle size is >7 microns, they cause blockage of the pulmonary arteries. Small drops of fat bypass the lungs and penetrate into the circulatory network of the brain. There is a cerebral symptomatology. There are other assumptions about the mechanisms of development of the process.

According to the proponents of the biochemical theory, plasma lipase is activated immediately upon injury and after it. This becomes an incentive for the release of fats from the deposit sites, hyperlipidemia develops, coarse fat droplets form. The colloidal-chemical version is that the desemulsification of fine emulsions begins due to the slowing of blood flow in the affected area.

It follows from the hypercoagulation theory that the cause of the formation of fat droplets is a microcirculation disorder, hypovolemia, oxygen starvation. Lipid globules with a diameter of 6-8 microns are formed, which form the basis for disseminated intravascular coagulation. The continuation of the process is systemic capillaropathy, which leads to fluid retention in the lungs and endointoxication by lipid metabolism products.

Classification.

Fat embolism can occur in a pulmonary, cerebral or mixed form. The respiratory form develops with predominant occlusion of the branches of the pulmonary artery and manifests itself in the form of respiratory failure. The cerebral variety is the result of blockage of the arteries and arterioles that provide blood supply to the brain. The mixed form is the most common and includes signs of both pulmonary and cerebral lesions. The period before the appearance of the first symptoms varies widely. According to the time of the latent interval, the following forms of the disease are distinguished:

Lightning fast. Manifests itself immediately after the injury, is characterized by a critically rapid course. The patient's death occurs within a few minutes. Mortality in this type of embolism is close to 100%, because it is impossible to provide specialized care in such a short time. It occurs only with multiple or massive injuries. The frequency of occurrence is no more than 1% of cases of FE.

Spicy. Occurs in less than 12 hours from the moment of injury in 3% of patients. It is a life-threatening condition, but the mortality rate does not exceed 40-50%. Death occurs from pulmonary edema, acute respiratory failure, extensive ischemic stroke.

Subacute. It manifests itself within 12-24 hours in 10% of patients; after 24-48 hours – in 45%; after 48-70 hours – in 33% of victims. There are cases when signs of embolism developed after 10-13 days. The course of subacute forms is relatively easy, the number of deaths does not exceed 20%. The chances of survival increase if the signs of the disease develop while the patient is in the hospital.

Symptoms of fat embolism

Pathology is manifested by a number of nonspecific symptoms that can occur in other conditions. Occlusion of the pulmonary vessels leads to a feeling of tightness in the chest, pain behind the sternum, anxiety. Objectively, the patient has shortness of breath, cough, accompanied by hemoptysis, foaming at the mouth, pallor, sticky cold sweat, anxiety, fear of death, acrocyanosis. There is persistent tachycardia, extrasystole, compressing pains in the heart. Possible development of atrial fibrillation. Changes in the respiratory system occur in 75% of patients and are the first symptoms of pathology.

The consequence of cerebral embolism is neurological symptoms: convulsions, impaired

consciousness up to stupor or coma, disorientation, severe headaches. There may be aphasia, apraxia, anisocoria. The picture resembles that of a traumatic brain injury, which makes diagnosis much more difficult. It is possible to develop paralysis, paresis, there is a local loss of sensitivity, paresthesia, a decrease in muscle tone.

Half of the patients have petechial rash in the armpits, on the shoulders, chest, back. This usually occurs 12-20 hours after the appearance of signs of respiratory failure and indicates overgrowth of the capillary network by emboli. When examining the fundus of the patient, damage to the retina is detected. Hyperthermia develops, in which the body temperature reaches 38-40 ° C. This is due to irritation of the thermoregulatory centers of the brain with fatty acids. Traditional antipyretic drugs are ineffective at the same time.

Complications.

Care for patients with FE should be provided in the first minutes after the development of signs of vascular occlusion. Otherwise, a fat embolism leads to the development of complications. Respiratory failure ends with alveolar edema, in which the pulmonary vesicles are filled with fluid sweating from the bloodstream. At the same time, gas exchange is disrupted, the level of blood oxygenation decreases, metabolic products accumulate, normally moving away with exhaled air.

Obturation of the pulmonary artery by fat globules leads to the development of right ventricular failure. The pressure in the pulmonary vessels increases, the right parts of the heart are overloaded. In such patients, arrhythmia, fluttering and atrial fibrillation are detected. Acute right ventricular failure, as well as pulmonary edema, are life-threatening conditions and in many cases lead to the death of the patient. It is possible to prevent such a development of events only with the fastest possible assistance.

Diagnostics.

An anesthesiologist-resuscitator, as well as consulting doctors: cardiologist, pulmonologist, traumatologist, ophthalmologist, radiologist take part in the diagnosis of embolisms of lipid origin. Laboratory research data play a significant role in making the correct diagnosis. ZHE has no pathognomic signs, so its lifetime detection occurs only in 2.2% of cases. The following methods are used to determine pathology:

Objective examination. The clinical picture corresponding to the disease is revealed, the heart rate is more than 90-100 beats per minute, the respiratory rate is more than 30 times per minute. Breathing is shallow, weakened. Moist, large-bubbly wheezes are heard in the lungs. The SP2 index does not exceed 80-92%. Hyperthermia within febrile values.

Electrocardiography. The ECG shows a deviation of the electrical axis of the heart to the right, non-specific changes in the ST segment. The amplitudes of the P and R teeth increase, in some cases there is a negative T wave. Signs of blockade of the right leg of the Gis beam may be detected: expansion of the S wave, a change in the shape of the QRS complex.

X-ray. Diffuse infiltrates of the lung tissue on both sides, predominant on the periphery, are visible on the radiographs of the lungs. The transparency of the pulmonary background decreases as the swelling increases. There may be a fluid level indicating the presence of pleural effusion.

Laboratory diagnostics. The detection of lipid globules in plasma with a size of 7-6 microns has a certain diagnostic value. It is preferable to take biomaterial from the main artery and central vein. The study of media from both basins is carried out separately. Detection of globules increases the risk of occlusion, but does not guarantee its occurrence.

Differential diagnosis is carried out with other types of embolisms: air, thromboembolism, vascular obstruction by a tumor or a foreign body. A distinctive feature of FE is the presence of microdrops of fat in the blood in combination with the corresponding radiological and clinical picture. In other types of vascular occlusion, lipid globules are absent in the blood.

Treatment of fat embolism

Therapy is carried out by conservative medicinal and non-medicinal methods. To provide medical care, the patient is placed in the

intensive care unit. All therapeutic measures are divided into specific and non-specific:

Specific. They are aimed at deemulsification of fats, correction of the coagulation system, ensuring adequate gas exchange. For the purpose of oxygenation, the patient is intubated and transferred to artificial ventilation. To synchronize with the device, sedatives may be administered in combination with peripheral muscle relaxants. Restoration of the normal consistency of lipid fractions is achieved by using essential phospholipids. To prevent hypercoagulation, heparin is administered.

Non-specific. Non-specific techniques include detoxification by infusion therapy. Prevention of bacterial and fungal infections is carried out by prescribing antibiotics, nystatin. Sodium hypochlorite is used as an antimicrobial and metabolic agent. From the 2nd day, the patient is prescribed parenteral nutrition with subsequent transfer to probe enteral.

The experimental method of treatment is considered to be the use of blood substitutes based on PFOA compounds. The drugs improve hemodynamic parameters, restore normal rheological properties of blood, and help reduce the size of lipid particles.

Prognosis and prevention.

In the subacute course, fat embolism has a favorable prognosis. Timely assistance allows to stop pathological phenomena, provide the necessary perfusion in vital organs, gradually dissolve emboli. In the acute variant of the disease, the prognosis worsens to unfavorable. The lightning-fast course leads to the death of the patient in almost 100% of cases.

Prevention during operations consists in the use of low-traumatic techniques, in particular percutaneous spoke osteosynthesis, performed in a delayed manner. It is recommended to abandon the use of skeletal traction, since this method does not provide a stable position of the fragments and can lead to the development of late embolization. Before hospitalization, it is required to stop bleeding as quickly as possible if it is present, adequate analgesia, and maintaining the blood pressure level at a normal physiological level. A specific method is the introduction of ethyl alcohol in a 5% glucose solution.

Treatment regimen for fat embolism. Many researchers speak in favor of early osteosynthesis, while the role of systemic steroids, heparin and other techniques remains a subject of discussion. A slight decrease in the occurrence of fat embolism is associated with a change in the tactics of clinical management of victims. Firstly, in most developed countries in the late 60s and early 70s of this century, the technique of treating fractures of long tubular bones changed from conservative to operative. Early open reposition and osteosynthesis significantly reduced the risk of severe general complications, such as fat embolism syndrome, adult respiratory distress syndrome, multiple organ failure. Secondly, more attention was paid to anti–shock measures at the scene of the accident, during transportation and especially in the hospital, which revealed the presence of a link between SSE and hypovolemic shock. Immobilization of the fracture at the scene of the accident, gentle transportation to the hospital, exclusion of unnecessary surgical manipulations on the injured limb. With closed fractures, preliminary puncture and suction of the hematoma were performed by injecting 20-40ml of 2-5% novocaine solution. The removal of the patient from shock was considered a preventive measure in comparison with a fat embolism. Primary surgical treatment, plaster immobilization and skeletal traction were used, thorough hemostasis was performed during surgical intervention and fat was removed from the wound. The fallacy of the opinion that fat embolism develops after osteosynthesis was emphasized. Special attention should be paid to the slow introduction of all metal fixators, regardless of their shape and design features, after removing fat from the bone marrow canal. They warned against the use of ether or thiopental anesthesia, referring to data on the increase in fat droplets in plasma and their agglomeration during these types of anesthesia. At the earliest stages of the disease, 25% albumin (200ml) was injected intravenously, and in severe cases repeatedly. Oxygen therapy for severe respiratory disorders - transfer of the patient to a ventilator. In case of fat embolism, targeted and, if possible, early correction of thrombohemorrhagic and microcirculatory

disorders is necessary. It should be mandatory for all patients with moderate and severe forms of traumatic illness throughout the entire stage of adaptation (the first 7 to 10 days), which allows to prevent clinically obvious fat embolism or reduce its severity. The authors recommended the methods of quantum hemotherapy for widespread use – ultraviolet irradiation of autokrovi and intravascular laser irradiation of blood. The next main point is: - adequate anesthesia and analgesia, infusion-transfusion therapy in the mode of moderate hemodilution - heparin in small doses (5 thousand units p / c in the umbilical region 4 times a day) - curantil (100mg per day) + reopoliglyukin (400ml per day) + aspirin (20-40mg per day) - lipostabil or essentiale (40-80ml daily). Prevention of RE is first aid for fractures, a broken limb should be carefully examined and properly splinted: the patient is carefully transported. Open reposition and internal fixation compared to conservative treatment. The frequency of fat embolism syndrome increased if osteosynthesis was postponed. The use of albumin reduced the risk of this complication in patients with multiple damage by binding of free fatty acids. Respiratory support is recognized by the absolute majority of authors as the main condition for the treatment of fat embolism syndrome.

Early and frequent monitoring of respiratory function by pulse oximetry or determination of arterial blood gas contributes to its early diagnosis. And can range from oxygen inhalation to mechanical ventilation. The implementation of the recommendations of Pelter (1988) on the need for immediate application (up to 40%) of oxygen through a facial mask or nasal catheter to all patients with serious fractures can normalize the situation in a number of cases. Prevention and treatment of fat embolism. Surgical methods. Early surgical stabilization of fractures in high-risk patients is the most important aspect in the treatment and prevention of the development of FE. Skeletal traction does not provide proper stability of fragments, therefore, surgical fixation is the optimal treatment for fractures of long tubular bones, especially with combined injuries. Currently, transosseous spoke osteosynthesis is most often used with spoke-rod devices. He is not traumatic, blood loss during surgery is minimal. A number of authors prefer intramedullary osteosynthesis with a pin. However, this type of osteosynthesis, especially performed with the drilling of the bone marrow canal, is accompanied by a significant increase. Fat globule in capillary blood serum. Giant fat globule (1800 microns) in arterial blood serum. Fat globule in venous blood serum. Assistance to a practicing doctor of intramedullary pressure, therefore, it is more often recommended to be carried out in a delayed manner. Medical treatment. Unfortunately, to date, no means of effective drug prevention and treatment of RE have been proposed, therefore, therapeutic measures should be aimed at relieving the main clinical manifestations of injury or disease: blood loss, hypovolemia, shock, coagulopathy, and others. Replenishment and correction of the water-electrolyte balance is carried out depending on the type of dishydria using colloidal and crystalloid solutions. The correct selection of infusion and rheological therapy, eliminating peripheral vascular spasm, helps to reduce the risk of reperfusion complications, which are an important pathogenetic link of RE. In the absence of the phenomenon of "capillary leakage" and normal pulmonary vascular permeability, determined by transthoracic thermodilution using Picco+ technology, it is recommended to use albumin, which is able to bind fatty acids and thereby reduce the degree of globulemia. A number of authors recommend the use of anticoagulants, in particular, heparin, which, along with anticoagulant and disaggregant properties, has the ability to activate plasma lipoproteins and accelerate enzymatic reactions of triglyceride hydrolysis, thus helping to cleanse the lungs of fat globules. Multilevel analgesia, differentiated depending on the type of injury, is of great importance in prevention and treatment. Adequate anesthesia reduces hypercatecholaminemia, and hence the concentration of free fatty acids. The use of narcotic analgesics, prolonged epidural, retropleural blockade after correction of hypovolemia, conduction anesthesia of the extremities, also normalizes microcirculation.

Drug therapy of brain hypoxia, the fight against pathological impulsivity includes antihypoxants (GHB), opiates, barbiturates. There is sufficient data on the use of corticosteroids in FE, based on their ability to stabilize cell membranes, inhibit the neutrophil response to fatty acids, inhibit the release of phospholipase A2, arachidonic acid and platelet aggregation. Currently, the use of corticosteroids for the treatment and prevention of FE is being questioned. In recent years, many publications have appeared on the use of exchange plasmapheresis in the treatment of FE. Exchange plasmapheresis operations normalize hemodynamic parameters, rheological properties of blood, coagulation system, morphological, biochemical and electrolyte composition of blood. The main indications for carrying out exchange plasmapheresis in traumatic fat embolism are: progressive deterioration, violation of vital functions of the body, increased activity of the kinin system, changes in lipid metabolism, immune status and inefficiency of intensive therapy. Contraindications are: agonal condition, severe cardiopulmonary insufficiency, severe concomitant diseases. Due to the fact that the main target organ in RE is the lungs, it is important to conduct respiratory therapy. This is one of the few therapeutic effects that have a high degree of evidence of effectiveness in the treatment of RE. In mild cases of RE, it is possible to carry out inhalations using a mask and a high flow of oxygen supply. With the progression of respiratory failure, mask CPAP is used, and if it is ineffective, tracheal intubation and ventilation are required. The selection of ventilation modes should be carried out individually, in accordance with the indicators of gas exchange and biomechanical properties of the lungs. Lipostabil and essentiale, whose action is aimed at restoring the dissolution of desemulsified fat in the blood, are drugs for the specific prevention and treatment of FE aimed at reducing the concentration of fat globules in the blood. These drugs are recommended for use by almost all domestic authors. However, in studies with the creation of an experimental FE, the use of the Essentiale H solution did not have a preventive and therapeutic effect. It is known that in patients who have been injured while intoxicated, FE occurs much less frequently or occurs in a mild or moderate form, since alcohol is able to inhibit serum lipase, while being a good emulsifier, and also has anti-ketogenic, sedative and analgesic effects. This is the basis for intravenous administration of 5% ethyl alcohol in 5% glucose solution for the prevention and treatment of FE. In experiments on rats, it has been proved that a solution of 5% ethyl alcohol is an effective means of prevention and treatment of experimental RE. In the last decade, there have been publications testifying to the effectiveness of the use of perfluorane in FE.

It has been shown that the early use of perfluorane in the complex treatment of patients with severe trauma and blood loss contributes to the optimization of the parameters of central hemodynamics and oxygen status. In addition, the presence of the emulsifier proxanol in the composition of perfluorane can help reduce the size of fat globules. Its ability to bind lipids, which form the basis of fat globules, has also been proven. These properties of perfluorane make it possible to attribute it to specific means of treating RE. The use of perfluorane for the treatment of FE is accompanied by an increase in the proportion of mild degrees of lung tissue damage by fat emboli and a significant decrease in the number of severe cases of FE. There is evidence of the preventive effect of the drug Gepasol A, which reduces the risk of developing fat globulemia during surgery and in the postoperative period. The drug Gepasol A stabilizes lipid metabolism and reduces the activity of lipid peroxidation. However, there is no convincing evidence of improvement in the results of treatment of RE with its use. Recently, there are data on the preventive effect of sodium hypochlorite, which causes the oxidation of lipid components of the fat globule, the formation of their water-soluble forms, which leads to the destruction of the fat drop itself and a decrease in the severity of fat globulemia. Conclusions. Thus, the complexity and urgency of the problem of fat embolism becomes obvious, and the mortality rate from this complication remains high. There is still no unity of views on

the pathogenesis, prevention and treatment of FE. The early diagnosis of FE is particularly difficult, due to the lack of a clear clinical picture and pathognomonic symptoms, and laboratory diagnostics is not very specific. Fat embolism is much more common than it is diagnosed, and can occur in any critical condition. Prevention and treatment schemes vary from author to author. Based on this, it is necessary to identify the following areas of scientific and practical search:

- development of prognostic criteria for the development of severe forms of FE;
- development of new directions for the prevention of severe forms of FE;
- development of an algorithm for the diagnosis and treatment of FE;
- individualization (personification) of diagnostic and therapeutic criteria of FE.

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