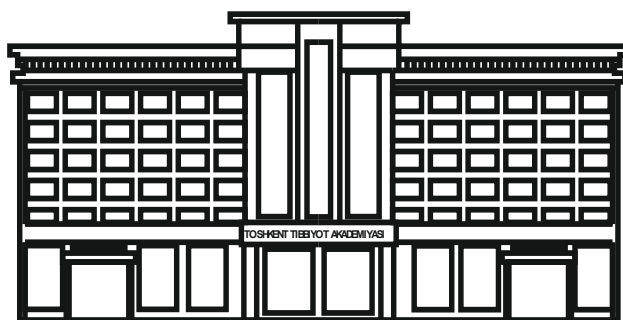


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FAT EMBOLISM IN DEATHS AS A RESULT OF COMBINED INJURIES

Choriev B.A., Tursunov X.Z., Baxriev I.I., Primov X.N., Mirzamuxamedov O.Kh., Sultanov S.B.

ЖИРОВАЯ ЭМБОЛИЯ В СЛУЧАЯХ СМЕРТИ В РЕЗУЛЬТАТЕ СОЧЕТАННЫХ ТРАВМ

Чориев Б.А., Турсунов Х.З., Бахриев И.И., Примов Х.Н., Мирзамухамедов О.Х., Султанов С.Б.

QO'SHMA SHIKASTLANISHLAR NATIJASIDA O'LIM HOLATLARIDA YOG 'EMBOLIYASI

Choriev B.A., Tursunov X.Z., Baxriev I.I., Primov X.N., Mirzamuxamedov O.Kh., Sultonov S.B.

Ташкентская медицинская академия

Цель: оценка уровня жировой эмболии в сосудах с целью определения основной причины смерти при тяжелых травмах с переломами трубчатых костей. **Материал и методы:** материалом для исследования послужили данные 36 гистологических препаратов, изготовленных из внутренних органов трупа, при жизни поступившего в стационар с сочетанной тяжелой травмой. Приготовленные для исследования препараты окрашивают гематоксилином и эозином и красителем Судан III. **Результаты:** как показали исследования, причин смерти пострадавших с тяжелой скелетной травмой много. Как правило, они обусловлены ишемией, пневмонией с отеком легких и головного мозга на фоне тяжелого травматического шока. **Выводы:** одной из основных причин смерти при политравме является пневмония, ДВС-синдром и возникающая в результате черепно-мозговой травмы жировая эмболия.

Ключевые слова: жировая эмболия, комбинированная травма, жировая инфильтрация, тупая травма, осложнение переломов костей.

Maqsad: uzun suyaklarning sinishi bilan og'ir jarohatlarda o'limning asosiy sababini aniqlash uchun tomirlardagi RE darajasini baholash. **Material va usullar:** Tadqiqot uchun material hayot davomida kasalxonaga yotqizilgan murdaning ichki a'zolaridan tayyorlangan 36 ta gistologik preparatning ma'lumotlari edi. Tadqiqot uchun tayyorlangan preparatlar gematoksilin va eozin va Sudan III bo'yoq bilan bo'yalgan. **Natijalar:** tadqiqotlar shuni ko'rsatdiki, og'ir skelet shikastlangan bemorlarda o'limning ko'plab sabablari bor. Qoida tariqasida, ular og'ir travmatik shok fonida ishemiya, o'pka va miya shishi bilan pnevmoniyadan kelib chiqadi. **Xulosa:** politravmada o'limning asosiy sabablari biri – bu pnevmoniya, DIC va miya shikastlanishi natijasida kelib chiqqan yog 'emboliyasi.

Kalit so'zlar: yog 'emboliyasi, kombinatsiyalangan shikastlanish, yog 'infiltratsiyasi, to'mtoq travma, suyak sinishi asorati.

Fat embolism (FE) is a common early complication of trauma in the practice of forensic medicine. The final forensic medical diagnosis of a forensic medical expert in the event of death from a fat embolism is based on the results of an autopsy of the corpse and the study of laboratory tests. Microscopic examination of tissues will be able to determine fat deposits mainly in the lungs, brain, heart, liver and small blood vessels of the kidneys [2,5,12,13].

A fat embolism is a blockage of blood vessels by drops of fat, as a rule, it is the fat of one's own body). As a direct cause of death, fat embolism occurs in 1.9-7.0% of all cases of mechanical damage and in 10.6% of fractures of long tubular bones. These data are based on the data of forensic medical examination and laboratory studies of the corpse, as well as clinical trials [2,7,9,13].

With multiple and combined severe injuries, mortality is higher and is more than 40%. If the causes of death in the first hours after injury are shock and massive acute blood loss, the development of severe changes in the brain and traumatic illness are considered as combined complications [1,6,13].

In a series of studies devoted to the problems of combined injuries, especially in severe combined injuries, it was noted that one of the most characteristic manifestations is shock-traumatic illness. Often, the fat embolism syndrome occurs together with brain edema on the background of pneumonia, pulmonary edema, severe traumatic shock and post-traumatic anemia [11].

According to the literature, in 80-90% of cases in patients with fractures of the bones (lower extremity) of the foot. In the outcome of a fat embolism, fat droplets are saponified and absorbed by phagocytes. 10-36% of the deceased develop clinical fat embolism syndrome [3,13].

The first information about fat embolism is associated with the name R. Lowel. In 1669, it was found that the introduction of milk and foreign fat into the blood vessels of animals lead to their death [13].

This fact has not been explained for a long time. F. Magendie injected olive oil into the neck veins of dogs and studied its latest in experiments. That is, the next day he observed all the signs of pneumonia in the animals. At autopsy of deceased dogs F. Magendie found that the small blood vessels and capillaries of the lungs are clogged with fat droplets. Based on these pathological findings, the scientist gives an explanation of the mechanism of animal death. In the end, he approaches the basic rules of the doctrine of fat embolism [2,10].

F. Zenker and E. Wagner invented the blockage of small blood vessels and capillaries of the lungs with fat droplets in humans. Some scientists attributed the main cause of death to injuries, others to pneumonia. For the first time, targeted experimental studies were conducted by E. Bergman. He injected 6 ml of diluted pork fat heated to 37°C into the superficial veins of the thigh. During the autopsy of the deceased animals, he found fat droplets in the vessels of the lungs, pulmonary edema and expansion of the right half of the heart. The author

found fat in the urine of animals that lived 6-24 hours, and during autopsy - fat embolism in the vessels of the liver and kidneys. F.Buch has established that RE develops mainly after fractures of long tubular bones and that the source of fat entering the bloodstream is the bone marrow. F.Buch sends olive oil to the bone marrow of a rabbit and breaking this bone, he finds fat droplets in the veins of the thigh pulmonary veins of a rabbit. Thus, for a long time, the mathematical theory of the origin of fat embolism, formulated by L.Aschoff and the colloidal-chemical theory proposed by E. Lehrmann in co-authorship in 1927-1928 [13].

Fat embolism often occurs under the guise of pneumonia, adult respiratory distress syndrome, traumatic brain injury and other pathology, which contributes to a significant increase in mortality [4].

Determining the level of fat embolism is important for morphologists to determine the severity of fat embolism syndrome and thanatogenesis. Depending on the number of fat droplets detected and the degree of their distribution in the vessels of the lungs, several degrees of drops are distinguished during histological examination: extremely weak; weak; medium; strong; extremely strong [4,8].

The aim of the study assessment of the level of RE in the vessels by staining pieces of the internal organs of the corpse with Sudan III paint for histological examination in order to determine the main cause of death in cases of severe injuries with fractures of tubular bones.

Material and methods

The material of the study was the data of 36 histological preparations made from the internal organs of a corpse that went to the hospital during its lifetime with a combined severe bodily injury. The preparations prepared for the study are stained with hematoxylin eosin and Sudan III dye.

From the resolution it became known that the citizen B.R. died on October 12, 2021 as a result of an accident. In the reception department of the clinic, the diagnosis was established: HRT.Concussions of the brain? Bruising of the soft tissues of the chin area, the I and V fingers of the right hand, subcutaneous hematoma. Closure fracture of the bones of the V-finger of the right hand? Contusion of the soft tissues of the left pelvic-femoral joint. The doctor recommended stationary treatment. The relatives took the patient home. After about 5-6 hours, the patient suddenly died. After a forensic examination of the corpse, a diagnosis was made: Closed craniocerebral trauma, hemorrhages under the soft meninges, hemorrhages in the brain substance. Closed fracture of both bones of the left shin. Multiple bruises and abrasions of the body. Pulmonary edema. Swelling of the brain. After carrying out additional research methods (forensic chemical, forensic biological and forensic histological), during the commission examination, the final diagnosis was established: Combined trauma: TBI.Brain injury. Subarachnoid hemorrhage. Closed blunt trauma of the chest. Bruised lungs. Post-traumatic pulmonitis. Closure fracture of the bones of the left shin. Multiple subcutaneous hematomas, bruises and abrasions of the body. Complications: Severe fatty embolism of the lungs, brain, heart and kidneys. Background: Alcohol intoxication of severe degree (2.79 ppm).

The brain - in two sections, the soft meninges with focal hemorrhages, there is swelling of the tissue. In other sec-

tions, the tissue of the soft meninges is fragmented, fibrous, focal edema, uneven fullness of blood vessels, some small vessels are empty, and some arteries of medium and large caliber with compressed lumen, dystonia. There is spasm in small arteries and arterioles, some of them are thickened due to plasma impregnation, focal and perivascular diapedetic hemorrhages are also viewed, erythrocyte hemolysis is traced in the lumen of the vessels. In the brain tissue, pronounced perivascular, pericellular edema and focal mesh edema, small arteries, arterioles with compressed lumen, some are spasmodic. Capillaries in many areas are compressed, some are spasmodic, in their lumen are visible exfoliated endothelial cells, in places small veins, capillaries, venules with unevenly weakly expressed fullness, in the lumen of their mesh, homogeneous masses, endothelial cells of the walls of blood vessels with swelling, small focal diapedous hemorrhages. Dystrophic swelling of neurons, in some areas of the "shadow" of neurons in the perivascular and pericellular zones, an uneven accumulation of glial elements. 4-sections of brain tissue are stained with Sudan-III, sometimes fat emboli colored orange are traced in the lumen of the capillaries. In 10 fields of view and magnification of a 7x8 microscope, more than 25 fat emboli were found in the vessels.

Heart - in some sections under the epicardium, large focal zones of adipose tissue proliferation, edema, empty blood vessels, some arteries are spasmed, dystonia, uneven thickening of the intima of a large coronary vessel due to plasma impregnation. There is a loose accumulation of lymphohistiocytic infiltration around some vessels. In the myocardium, blood vessels are anemic, the lumen of large arteries is compressed, dystonia, there is a spasm of small, medium-sized arteries, arterioles. In the lumen of some vessels, loose leukocytes with an admixture of mesh masses are detected in places, pronounced edema in the interstitial tissue. Cardiomyocytes with moderate swelling, with necrobiotic changes, sometimes foci of lipofuscin pigment are detected. Cardiomyocytes are unevenly hypertrophied, sometimes foci of a wave-like nature and acute fragmentation of myocytes. Sometimes small-focal fatty infiltrates, perivascular small-focal sclerosis are visible in the perivascular and interstitial tissue.

Heart - painting by Sudan-III. In stained sections according to the Sudan-III staining technique, interstitial myocardial tissue - in the lumen of small capillaries, fat emboli colored orange are found. Sometimes, in places in the cytoplasm of myocytes, fat emboli colored orange are also visible.

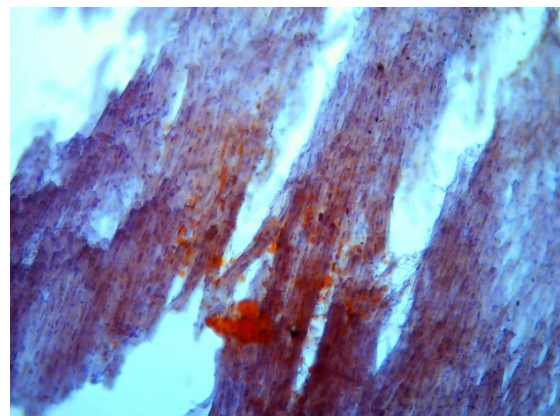


Fig. 1. Fat embolism in the heart.

Lungs – in the parenchyma around the large bronchi and blood vessels, focal hemorrhages with signs of tissue destruction are detected (hemolysis of erythrocytes in hemorrhage foci, loose accumulation of leukocytes with an admixture of lymphocytes and dark brown pigments). In other fields of vision, in interstitial and interalveolar tissue, edema, atelectasis, foci of distelectase, the lumen of some alveoles are emphysematous dilated, their wall is thinned, in some areas with ruptures. There is anemia in the vessels, some small arteries, arterioles and capillaries are optically dilated, empty, sometimes the lumen of some large arteries is compressed, dystonia, there is a spasm of arterioles and small arteries. Focal fullness of blood vessels is revealed, sometimes erythrocytes with an admixture of leukocytes, leukostasis are seen in their lumen. In some areas in the field of vision, the interstitial tissue is thickened due to the accumulation of infiltrate consisting of lymphocytes, histiocytes and leukocytes, which have a focal character. In the lumen of large bronchi, bronchioles, flaked fragments of bronchial epithelium are detected and cellular elements consisting of leukocytes, lymphocytes are visible in small quantities, sometimes spasm of bronchioles and large bronchi are detected in these areas. There is edema in their wall, mild anemia in the lumen of the vessels, loose or thick lymphocytic infiltration is visible in some areas. In the perivascular tissue, lymphoid infiltrates of a focal nature are sometimes detected. When painted with Sudan-III. In the tissue of the interalveolar septum in the lumen of small arteries, arterioles and capillaries, fat emboli colored orange are detected.

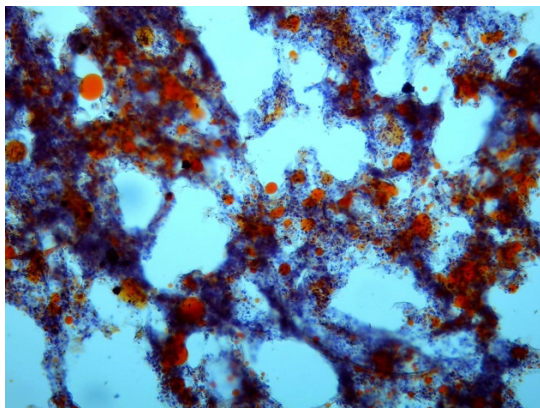


Fig. 2. Fatty embolism in the lungs.

Kidneys – the capsule is thickened due to sclerosis. Anemia is detected in the capillaries of the cortical layer of the parenchyma and in the glomerular capillaries, recalibration is detected in some glomeruli, in other glomeruli the capillaries are optically empty, spasm of small arteries and arterioles, dystonia of large arteries. There is an unevenly pronounced fullness in the cerebral layer, shadows of erythrocytes are detected in the lumen of the vessels, in some areas in the lumen of the vessels there is mesh edema with an admixture of cellular infiltrates consisting of lymphocytes. Interstitial edema, sometimes focal glomerular sclerosis. The epithelium of convoluted tubules with dystrophic changes, in some epithelial cells with necrobiosis. When stained with Sudan

III, orange-colored fat emboli are detected in the lumen of small vessels of the cerebral and cortical layers.

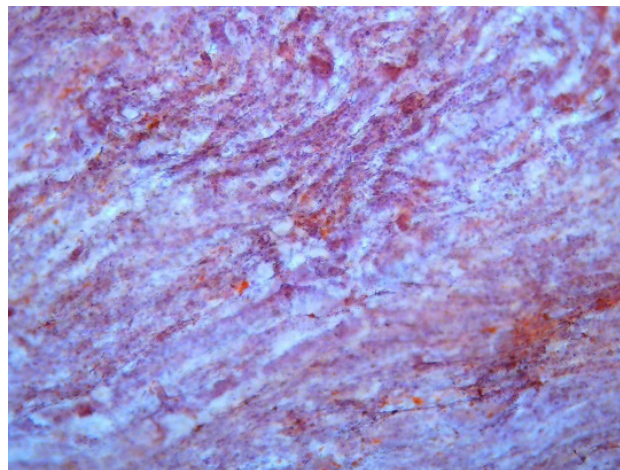


Fig. 3. Fatty embolism in the kidney.

Pancreas – in the interlobular stroma, edema, uneven fullness of blood vessels, spasm of small arteries and arterioles, compression of the arterial lumen in places, vascular dystonia, vascular hemorrhage and diapedesis. In places, focal lipomatosis, sclerosis of the wall of the excretory ducts. In the parenchyma, there is intrauterine edema, uneven fullness, dystrophic changes in the epithelium of acinuses, in places in the lumen of blood vessels, shadows of erythrocytes, with an admixture of loose lymph-leukocyte infiltration. Conclusions: There is a massive fat embolism in the blood vessels of the lungs; there are fat emboli in the capillaries of the brain, heart, and kidney tissue. In the parenchyma of the lungs around the large bronchi and blood vessels, focal hemorrhages with signs of tissue destruction, edema, atelectase, distelectase and emphysema foci; in the soft meninges of the brain, focal hemorrhages, pronounced edema, dystrophic swelling of neurons in the tissue; in organs, acute hemodynamic disturbance in the micro-circulatory bed; in parenchymal organs, dystrophic and necrobiotic changes; in the liver portal (finely nodular) cirrhosis.

A distinctive feature of this observation is a strong degree of ischemia of the brain, lungs and kidneys, revealed during forensic histological examination, which was the direct cause of death in mechanical trauma with multiple fractures of long spongy bones. It is important that the source of fat embolism in this case is the bone marrow, subcutaneous fat or vascular injury and their aggregation centers, which cause the phenomena of demulsification of blood lipids and major fat droplets in the bloodstream.

Thus, embolization of pulmonary capillaries from 2/3 to 3/4 leads to death from pulmonary embolism and only a few embolisms are sufficient for cerebral ischemia with subsequent severe disorders, since the vessels feeding the brain are limited.

Conclusion

In conclusion, it should be noted that there are many causes of death in patients with severe skeletal trauma. As a rule, this is associated with ischemia, pneumonia with edema of the lungs and brain on the background

of severe traumatic shock. According to the study, one of the main causes of death in polytrauma is often pneumonia, DIC syndrome and the resulting fatty embolism of traumatic brain injury.

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FAT EMBOLISM IN CASES OF DEATH AS A RESULT OF COMBINED INJURIES

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Objective: To assess the level of fat embolism in the vessels in order to determine the main cause of death in severe injuries with fractures of long bones. **Material and methods:** The material for the study was the data of 36 histological preparations made from the internal organs of a corpse, admitted to the hospital during life with a concomitant severe injury. Preparations prepared for research are stained with hematoxylin and eosin and Sudan III dye. **Results:** Studies have shown that there are many causes of death in patients with severe skeletal trauma. As a rule, they are caused by ischemia, pneumonia with pulmonary and cerebral edema against the background of severe traumatic shock. **Conclusions:** One of the main causes of death in polytrauma is pneumonia, DIC, and fat embolism resulting from traumatic brain injury.

Key words: fat embolism, combined trauma, fat infiltration, blunt trauma, complication of bone fractures.