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MORPHOLOGICAL DIAGNOSIS OF FAT EMBOLISM IN COMBINED TRAUMAS WITH FATAL OUTCOMES

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ABSTRACT

The technocratic path of societal development leads to a steady increase in injuries and the escalation of all aspects of traumatic diseases. One of the early severe complications of musculoskeletal injuries is traumatic fat embolism. At the same time, the clinical manifestations of fat embolism in combined injuries are complicated by traumatic shock, traumatic brain injury, and traumatic respiratory failure of various causes. To date, the diagnosis of fat embolism remains a complex and completely unresolved problem, as fat embolism does not have a clear clinical picture and pathognomonic symptoms. Furthermore, forensic diagnosis of fat embolism is considered macroscopically complex, and a definitive conclusion can only be made through microscopic examination of internal organs (lungs, brain, kidneys, and heart).

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

INTRODUCTION

Fat embolism (FE) is generally a complication of trauma, various diseases, and medical procedures. The final forensic medical diagnosis in cases of death due to fat embolism is based on the results of the autopsy and laboratory tests. Microscopic examination of tissues will reveal fat deposits primarily in the lungs, brain, heart, liver, and small blood vessels of the kidneys. [2, 4, 10, 11]

Fat embolism is the obstruction of blood vessels by fat droplets, typically from the body's own fat. As an immediate cause of death, fat embolism occurs in 1.9-7.0% of all cases of mechanical injuries and in 10.6% of fractures of long

tubular bones. These data are based on forensic examinations, laboratory studies of corpses, and clinical research [2, 5, 7, 11].

In cases of multiple and combined severe trauma, mortality is higher, exceeding 40%. If the causes of death in the first hours after trauma are shock and massive acute blood loss, then the development of severe brain damage and traumatic disease are considered as combined complications [1, 6, 11].

In several studies focused on the problems of combined injuries, especially severe combined injuries, it has been noted that one of the most characteristic manifestations is traumatic illness. The fat embolism syndrome often occurs alongside brain edema against the background of pneumonia, lung edema, severe traumatic shock, and post-traumatic anemia [9].

According to the literature, in 80-90% of cases with fractures of long bones in the lower extremities, fat droplets are enveloped and absorbed by phagocytes as a result of fat embolism. Clinical fat embolism syndrome develops in 10-36% of deceased individuals [3, 11].

Fat embolism often presents under the guise of pneumonia, adult respiratory distress syndrome, traumatic brain injury, and other pathologies, which contributes to a significant increase in mortality [12].

Determining the level of fat embolism is important for pathologists to assess the severity of the fat embolism syndrome and its pathogenesis. Based on the number of fat droplets found and their distribution in the vessels of the lungs during histological examination, several degrees of fat droplets are distinguished: extremely weak; weak; moderate; strong; and extremely strong [12, 13].

Research Objective: The aim of the study is to evaluate the level of fat embolism in blood vessels by staining tissue samples from internal organs of the deceased with Sudan III dye for histological examination, with the goal of determining the primary cause of death in cases of severe combined trauma.

Materials and Methods: The study used data from 36 histological specimens prepared from the internal organs of a deceased individual who had been admitted to the hospital with severe combined trauma. The specimens were stained with hematoxylin-eosin and Sudan III dye for examination.

Statistical analysis was based on descriptive statistical methods (determining mean values, standard deviation) and was performed using the program Statistics 5.0 for Windows.

Results and Discussion

Example:

From the decision on appointing the forensic medical examination, it is known that citizen B.R. died on October 12, 2021, as a result of an accident. In the

emergency department of the clinic, the diagnosis was: closed head injury, concussion of the brain, contusion of soft tissues of the chin area, contusion of the 1st and 5th fingers of the right hand, subcutaneous hematoma, closed fracture of the V-shaped finger of the right hand, contusion of the soft tissues of the left hip joint. The doctor recommended hospitalization. The patient's relatives took him home. Approximately 5-6 hours later, the patient suddenly died. After the forensic examination of the body, the diagnosis was: closed head injury, hemorrhages under the soft meninges, hemorrhages into the substance of the brain, closed fracture of both bones of the left shin, multiple bruises and abrasions on the body, pulmonary edema, brain edema. After additional methods of investigation (forensic chemical, forensic biological, and forensic histological examinations), the final diagnosis was: combined trauma, closed head injury, subarachnoid hemorrhage, closed blunt trauma to the chest, contusion of the lungs, post-traumatic pneumonitis, closed fracture of the bones of the left shin, multiple subcutaneous hematomas, bruises, and abrasions on the body. Complications: severe fat embolism of the lungs, brain, heart, and kidneys. Medical history: severe alcohol intoxication (2.79‰).*Complications:* severe fat embolism of the lungs, brain, heart, and kidneys. *Background:* severe alcohol intoxication (2.79 ppm).

Lungs Examination shows focal hemorrhages with signs of tissue destruction in the parenchyma around the large bronchi and vessels, (hemolysis of erythrocytes in the hemorrhagic foci, loose accumulation of leukocytes mixed with lymphocytes and dark brown pigments). In other fields of view, in the interstitial and interalveolar tissue, there is edema, atelectasis, and foci of distelectasis. The lumen of some alveoli is emphysematously expanded, their walls are thinned, with occasional ruptures. The vessels show anemia, some small arteries, arterioles, and capillaries are optically dilated and empty. In some areas, the lumen of some large arteries is compressed, showing dystonia, and spasms of arterioles and small arteries are noted. Focal congestion of vessels is identified, and in some areas, erythrocytes mixed with leukocytes and leukostasis are visible in their lumen. In certain sections, the interstitial tissue is thickened due to the accumulation of infiltrate consisting of lymphocytes, histiocytes, and leukocytes, having a focal character. In the lumen of the large bronchi and bronchioles, scaly fragments of bronchial epithelium and a small number of cellular elements consisting of leukocytes and lymphocytes are found. Occasionally, spasms of the bronchioles and large bronchi are detected in these areas. Their walls exhibit edema, and mild anemia is observed in the lumen of the vessels, with loose or dense lymphocytic infiltration visible in some areas. Focal lymphoid infiltrates are sometimes detected in the perivascular tissue. When stained with Sudan III, fat emboli of orange color

are found in the lumen of small arteries, arterioles, and capillaries in the interalveolar septal tissue (Fig. 1).

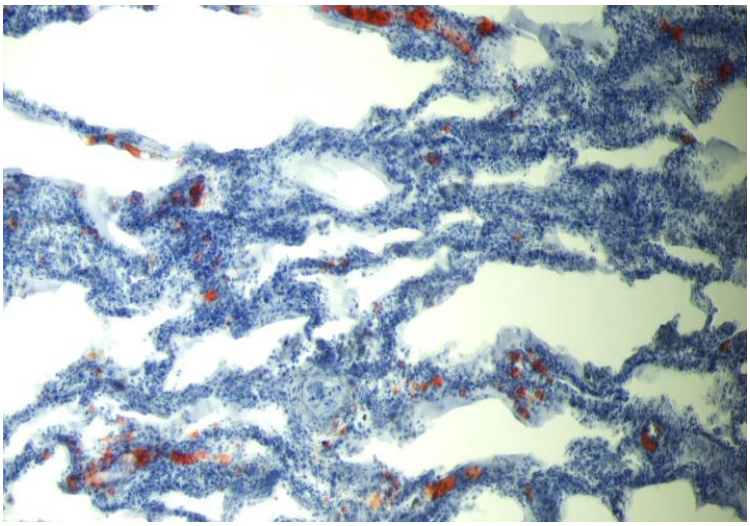

Figure 1. Fat Embolism in the Lungs

Brain Examination shows focal hemorrhages in two areas and edema in the tissues. In other regions, the tissue of the soft meninges is fragmented, fibrous, with focal edema and uneven vessel filling. Some small vessels are empty, while some medium and large caliber arteries have compressed lumens, indicating dystonia. Small arteries and arterioles exhibit spasm; some are thickened due to plasma infiltration. Focal and perivascular diapedeic hemorrhages are also observed, with hemolysis of erythrocytes in the vessels (Fig. 2).

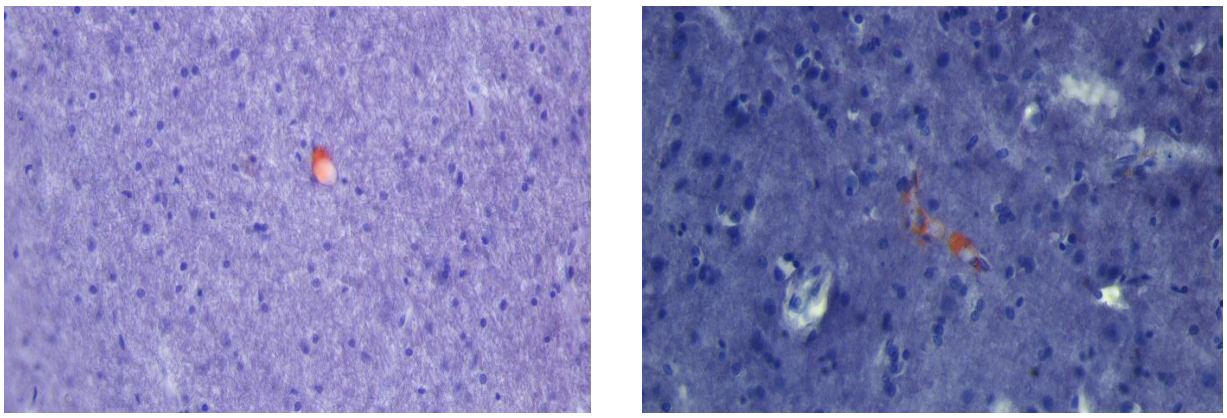

Figure 2. Fat Embolism in the Brain

In the brain tissue, there is pronounced perivascular and pericellular edema, and focal reticular edema. Small arteries and arterioles have compressed lumens, with some showing spasm. Capillaries are compressed in many areas, with some spasm and detachment of endothelial cells visible. Small veins, capillaries, and venules have uneven, weak filling, with a network of homogeneous masses and

swelling of endothelial cells in the vessel walls, accompanied by small focal diapedeic hemorrhages.

Neuronal dystrophic swelling is noted, with "shadows" of neurons in perivascular and pericellular zones and uneven accumulation of glial elements in certain areas. Sudan III staining of brain tissue sections occasionally reveals orange-colored fat emboli in the capillary lumens. In 10 fields of view at 7x8 magnification, more than 25 fat emboli were identified in the vessels.

Heart Examination shows large focal areas of fat tissue growth under the epicardium in certain areas, edema, empty vessels, some arteries are spasmodic, dystonia, and uneven thickening of the intima of large coronary vessels due to plasma impregnation. Around some vessels, there is a loose accumulation of lymphohistiocytic infiltration. In the myocardium, the vessels are bloodless, the lumen of large arteries is compressed, dystonia is noted, and there is a spasm of small and medium arteries, and arterioles. In the lumen of some vessels, loose leukocytes mixed with reticular masses are occasionally found, with marked edema in the interstitial tissue. Cardiomyocytes exhibit moderate edema and necrobiotic changes, and sometimes foci of lipofuscin pigment are detected. Cardiomyocytes are unevenly hypertrophied, with wavy areas and severe fragmentation of the myocytes observed in some places. The heart tissue stained with Sudan III reveals that in the interstitial tissue of the myocardium, orange-colored fat emboli are found in the lumen of small capillaries. Occasionally, fat emboli stained orange are also seen in the cytoplasm of myocytes (Fig. 3).

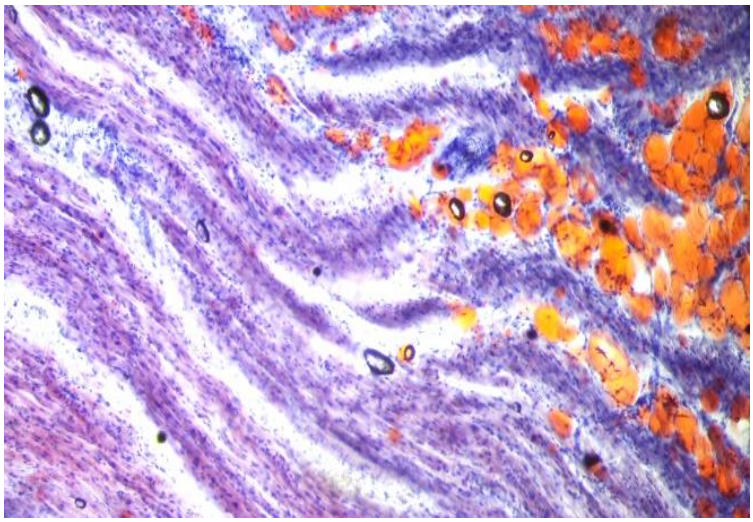

Figure 3. Fat Embolism in the Heart

Examination of kidneys shows that the capsule is thickened due to sclerosis. In the capillaries of the cortical layer of the parenchyma and in the capillaries of the glomeruli, anemia is detected. In some glomeruli, recalibration is observed,

while in others, the capillaries are optically empty, with spasm of small arteries and arterioles and dystonia of large arteries. In the medullary layer, there is uneven hyperemia, with shadows of erythrocytes visible in the lumen of the vessels, and in some places, reticular edema mixed with cellular infiltrates consisting of lymphocytes is found. There is interstitial edema, and occasionally focal glomerular sclerosis. The epithelium of the convoluted tubules shows dystrophic changes, with necrobiosis in some epithelial cells. When stained with Sudan III, fat emboli of orange color are found in the lumen of small vessels in the medullary and cortical layers (Fig. 4).

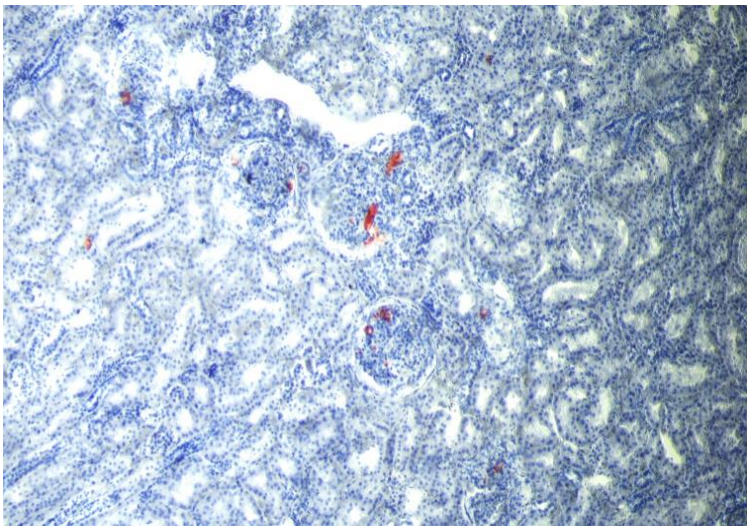

Figure 4. Fat Embolism in the Kidney

There is massive fat embolism in the vessels of the lungs; fat emboli are present in the capillaries of the brain tissue, heart, and kidneys. In the lung parenchyma around the major bronchi and vessels, focal hemorrhages with signs of tissue destruction, edema, foci of atelectasis, distelactasis, and emphysema are found; in the soft membranes of the brain, focal hemorrhages, marked edema, and dystrophic swelling of neurons in the tissue are noted; in the organs, acute hemodynamic disturbances in the microcirculatory bed are observed; in the parenchymatous organs, dystrophic and necrobiotic changes are seen; and with portal (micronodular) liver cirrhosis.

A distinctive feature of this case is the severe degree of ischemia of the brain, lungs, and kidneys identified during forensic histological examination, which was the immediate cause of death in the context of mechanical trauma with multiple fractures of long tubular bones. It is important to note that the source of the fat embolism in this case is damage to the bone marrow, subcutaneous fat tissue, or vessels and their aggregation centers, which cause the phenomena of blood lipid demulsification and large fat droplets in the bloodstream.

Thus, embolization of 2/3 to 3/4 of the pulmonary capillaries leads to death from pulmonary artery thromboembolism, and only a few emboli are sufficient to cause brain ischemia with subsequent severe disturbances, as the vessels supplying the brain become occluded.

Conclusions: In conclusion, it should be noted that the causes of fatal outcomes in patients with severe skeletal trauma are of a multiple nature. Typically, this is a combination of fat embolism, pneumonia with pulmonary edema, and cerebral edema against the background of severe traumatic shock. As shown by the data from the conducted study, one of the main causes of fatal outcomes in victims with combined trauma is fat embolism, which often occurs under the guise of pneumonia, respiratory distress syndrome, or traumatic brain injury.

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