

Morphological Features of Fat Embolism in Fractures of Tubular Bones

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Abstract The article covers a problem of a fatty embolism as a heavy complication of a mechanical trauma that often can be cause of death of the victim or playing a fundamental role in a thanatogenesis. The article briefly clears up the questions of an etiology and pathogenesis, clinical diagnostics and generally touches on an issue of morphological (primary – microscopic) diagnosis of a fatty embolism.

Keywords Fat embolism, Fractures of bones, Morphology of a fatty embolism, Traumatic illness

1. Introduction

Contrary to expectations, the progressive developments of society and scientific-technical progress have not led to a decrease in trauma in the 21st century. Moreover, according to foreign and domestic literature, there has been incessant increase in severe trauma in the structure of diseases leading to long-term disability and disability in patients. According to the World Health Organization, trauma ranks third in terms of frequency and overall mortality in the population. Trauma, as a cause of mortality, is the leading one in the age group from 20 to 60 years, exceeding it by two times the combined number of cardiovascular and oncological diseases [1]. The average age of patients with polytrauma is 38.5 years [2].

A severe complication in the early stages of trauma that increases lethality is fat embolism. However, clear understandings of the pathogenesis, clinical presentation, prevention, and treatment of fat embolism are still lacking. The frequency of fat embolism development varies, according to different authors, from 0.5 to 30% in injuries involving multiple fractures of long tubular bones and fractures of the pelvic bones. Despite intensive modern therapy, the mortality rate ranges from 3 to 67% [3-5].

Fat embolism occurs due to the entry into the blood of droplets of either endogenous or exogenous neutral fat. Causes of this include skeletal trauma (closed fractures or gunshot wounds of long tubular bones, multiple rib fractures, pelvic bone fractures), extensive soft tissue injuries with crushing of subcutaneous adipose tissue, severe burns, intoxication or electrical injuries, hepatic fat dystrophy, closed heart massage, and certain types of anesthesia. Fat embolism can also occur when administering oil-based

therapeutic or diagnostic preparations to patients. Fatty droplets usually enter the lungs and get trapped in small blood vessels and capillaries. Some fat droplets pass through arteriovenous anastomoses into the systemic circulation and are disseminated by blood to the brain, kidneys, and other organs, blocking their capillaries. At the same time, there are no macroscopic changes in the organs. However, targeted examination of histological specimens using fat-revealing dyes allows for the diagnosis of fat embolism in most similar situations.

Due to the complexity of clinical diagnosis and the lack, in most cases, of reliable macroscopic signs indicating its presence, fat embolism is often overlooked in autopsies of individuals who died from mechanical injuries. This, in general, reduces the quality of the conducted examinations.

The aim of the research is to assess the level of fat embolism in blood vessels by staining pieces of internal organs of the corpse with Sudan III dye for histological examination, with the purpose of determining the primary cause of death in fractures of long tubular bones.

2. Materials and Methods

The material for the study consisted of 36 histological specimens prepared from the internal organs of a corpse that was admitted to the hospital with a combined severe trauma during its lifetime. The prepared specimens are stained with hematoxylin-eosin and Sudan III dye for further examination.

To identify lipid inclusions from lung tissue fixed for no more than two days in a 10% formalin solution, thin slices of lung tissue were made using a freezing microtome. These sections were placed on glass slides, stained with Sudan III, then with hematoxylin, covered with glycerin, and a cover slip was applied, the edges of which were secured with a mounting medium. Simultaneously, some pieces of lung

tissue were embedded in paraffin, and the sections were stained with hematoxylin and eosin.

When stained with hematoxylin and eosin, small round-to-oval optical voids resembling fat emboli were found in the lumens of various-sized vessels in all cases. The presence of fat emboli was assessed on Biolam-L-1 and XS 90 microscopes on a section area of 2 square centimeters. When stained with Sudan III, fat emboli were found in vessels of various calibers, appearing as rounded obtrusive forms that filled the entire vessel lumen and sausage-shaped homogeneous droplets of orange-yellow color.

3. Results and Discussion

Internal organs subjected to histological examination included the brain, heart, lungs, and kidneys.

Brain (cortex): The soft meningeal membrane is thin, with venous-venular congestion and brain substance; pronounced perivascular edema, dystrophic changes in neurons throughout the sections. When stained with Sudan III, more than 10 fat emboli were found in vessels in 10 fields of view and with a microscope magnification of 7x8 (Figure 1).

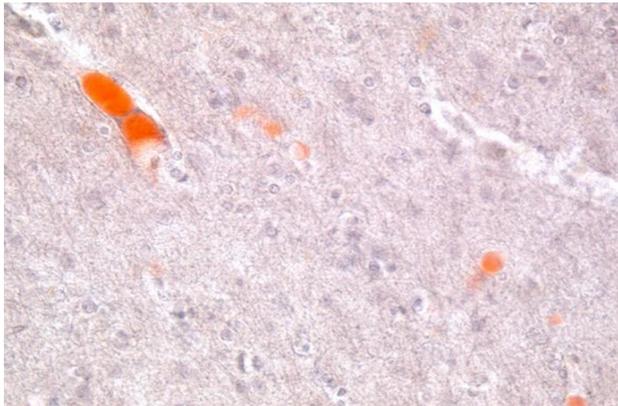


Figure 1. Brain. Orange-colored fat emboli in the lumen of capillaries. Staining with Sudan III, magnification x400

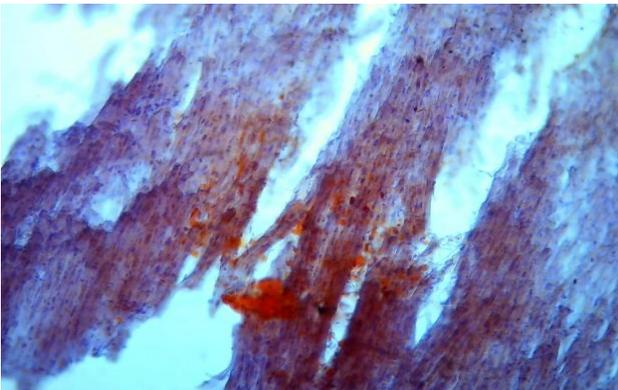


Figure 2. Heart. Orange-colored fat emboli in the lumen of capillaries. Staining with Sudan III, magnification x100

Heart: Focal fatty infiltration of the epicardium, lipoidosis of the epicardial and intramural arteries of the myocardium, spasm of the arteries, uneven blood filling (venous-venular

congestion, stasis of erythrocytes in capillaries alternating with moderate blood filling), hypertrophy of groups of cardiomyocytes, alternation with thinned muscle fibers; intermuscular edema; hyperplasia of stromal elements; fragmentation, undulating deformation, and granular dystrophy of cardiomyocytes. Histologically, emboli are most often visible at the branching points of coronary arteries and in the capillaries of the myocardium (Fig. 2).

Lung: Bronchiolar hypertonus, predominantly preserved epithelial layer on the basal membrane; fibrin threads and groups of leukocytes in the lumens; walls infiltrated with leukocytes, the pathological process extends to the surrounding parenchyma; in the lumens of alveolar ducts and alveoli, in certain fields of view, groups of leukocytes, fibrin threads, homogeneous eosinophilic fluid, hyaline membranes closely adhering to the walls; predominant angiospasm, lumens of some arteries, arterioles, and venules are paralytically dilated, devoid of blood, predominantly venous-venular congestion, stasis, leukostasis; interalveolar septa thickened due to edema, leukocytic infiltration, and congestion; focal and large-focal hemorrhages in the respiratory parenchyma; multiple tuberculosis granulomas at various stages of morphogenesis. In vessels of various calibers (stained with Sudan III), from 110 to 150 disintegrating fat emboli; presence of lipophages in interalveolar septa (Fig. 3).

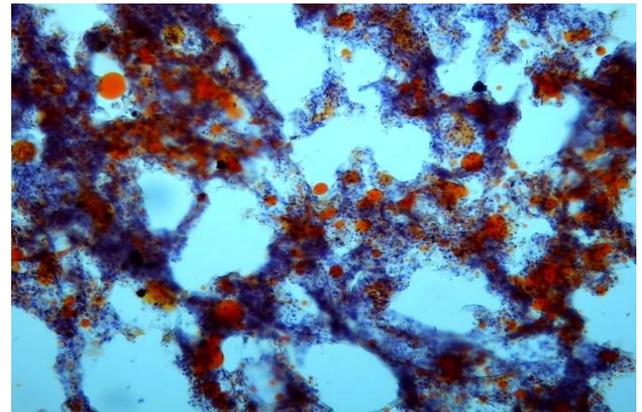


Figure 3. Lungs. Orange-colored fat emboli in the lumen of arterioles. Staining with Sudan III, magnification x100

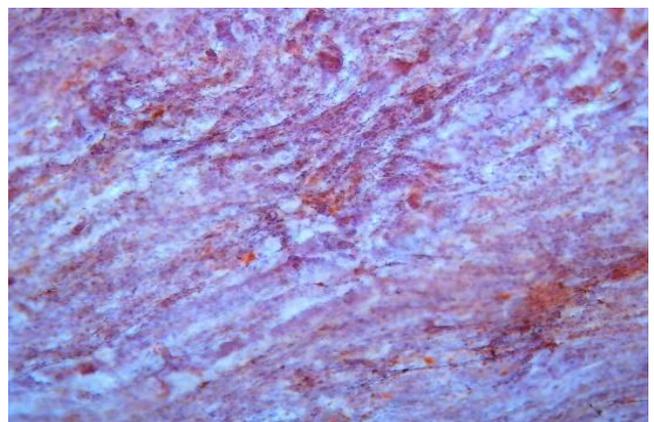


Figure 4. Kidney. Cortical layer. Orange-colored fat emboli in the capillaries of glomeruli. Staining with Sudan III, magnification x200

Kidney: Arterial spasm, uneven blood filling of glomerular capillaries, vessels of the peritubular zone, venous-venular congestion in pyramids and juxtamedullary layer; small-focal hemorrhages in the cortical layer of the kidney, peripheral to hemorrhages venous-venular congestion, stasis, leukostasis, migration of leukocytes beyond the vascular bed (20-80 leukocytes in the field of view with a microscope magnification of 10x40); large-focal necronephrosis, in certain fields of view, homogeneous eosinophilic fluid in the lumens of capsules. In the capillaries of most renal glomeruli, fat inclusions were identified when stained with Sudan III (Fig. 4).

4. Conclusions

The particular feature of this study is the identified fat embolism in the brain, lungs, heart, and kidneys, of a significant degree, during forensic-histological examination. This fat embolism was determined to be the immediate cause of death in the case of a mechanical injury with multiple fractures of long bones. The source of fat embolism in this case was the adipocytes, which are consistently present in the red bone marrow of long tubular bones. It is also significant that blunt force always creates conditions for traumatizing subcutaneous adipose tissue and blood vessels, leading to the formation of fat embolism in the pulmonary vessels of varying degrees (often minor). The fact that primary fat droplets in the bloodstream induce the phenomenon of lipid demulsification and become centers of their aggregation is also noteworthy.

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