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CASE REPORT

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Maternal Death Due to Non-Traumatic Fat Embolism

ABSTRACT: The aim of this report is to document a case of non-traumatic fat embolism (NTFE) and to address the need for considerition of fat embolism in suspicious deaths resulting from respiratory distress in the postpartum period. A 28-years-old woman autopsied at the Morgue Department of the Council of Forensic Medicine is included to the study. This female became unconscious and developed respiratory distress 4 h after delivery, and this was followed by respiratory arrest. External examination revealed resuscitation marks and normal postmortem changes. Light microscopy revealed massive fat embolization involving most of the alveolar capillaries on several sections. Only in one particular area was a bone marrow embolus. Pathological diagnosis of the lung was diffuse pulmonary fat embolism. There was no evidence of other organ involvement with emboli. Other visceral organs showed no striking findings other than mild congestion. The cause of death was considered to be respiratory insufficiency resulting from severe fat embolism of the lungs.

KEYWORDS: forensic science, forensic pathology, pregnancy, lung, non-traumatic fat embolism

Fat embolism (FE) develops due to lipid globules being released from bone marrow and soft tissue following severe trauma, surgery and/or soft tissue damage (1–3). It was first described by Von Bergmann in 1873 (4) and it can also develop as a result of acute pancreatitis, hepatic steatosis, osteomyelitis, diabetes mellitus, severe burns, and conditions causing bone infarcts such as sickle cell anemia, and prolonged steroid therapy without trauma (3,5).

Nontraumatic fat embolism (NTFE) may clinically mimic traumatic fat embolism. Extensive pulmonary oedema caused by massive fat embolism, thrombocytopenia, dyspnea, mental status changes, and progressive hypoxemia are the most significant clinical findings (1–3). Brain, kidney and heart are the most vulnerable target organs (6).

Although cases with traumatic fat embolism are seen often, there are no published cases documented following normal vaginal delivery. This report documents this uncommon situation and emphasizes the need for considering fat embolism in suspicious deaths resulting from respiratory distress during the postpartum period.

History

The deceased was a 28-year-old female who had given birth vaginally to her fourth child. Four hours after the delivery, she suddenly became unconcious and developed respiratory distress, which was followed by respiratory arrest. Two hours after succesful resuscitation, she again developed respiratory arrest and died. The medical history detailed no chronic disease or family history and she had no medical problems in her previous deliveries. An autopsy was was performed to determine the cause of death.

Autopsy Findings

External examination revealed resuscitation marks and normal postmortem changes. There was no evidence of injury, apart from slight mucosal abrasions due to the vaginal delivery.

Examination of the visceral organs, sternum, and bilateral rib fractures (3,4,5) revelaed hemorrhages, which were most likely caused by resucitation. The heart weighed 400 g, with normal gross findings. The weight of the lungs were 640 g, right; 480 g, left. They were oedematous and displayed extensive subpleural petechiae. There was an area of hemorrhage with a diameter of 2 cm on the pleural surface of the left lower lung. Hemorrhagic changes in the uterus were compatible with the postpartum period. Examination of the other organs did not reveal any remarkable pathology.

Histological Findings

Light microscopy revealed massive fat embolizm involving most of the alveolar capillaries on several sections. There was a bone marrow embolus in only one area (Fig. 1) The severity of fat embolism of the lung was considered as Grade III according to the scoring system used by Mudd et al. (2). In addition, polymorphonuclear leucocytes in interstitial capillary vessels and oedematous fluid in alveoli were present in the lung. Pathological diagnosis was diffuse pulmonary fat embolism. Fat and bone marrow embolism of the lung was confirmed by Oil Red-O lipid stains on fresh tissues, applied with a conventional histochemical method (Fig. 2).There was no evidence of emboli in other organs.

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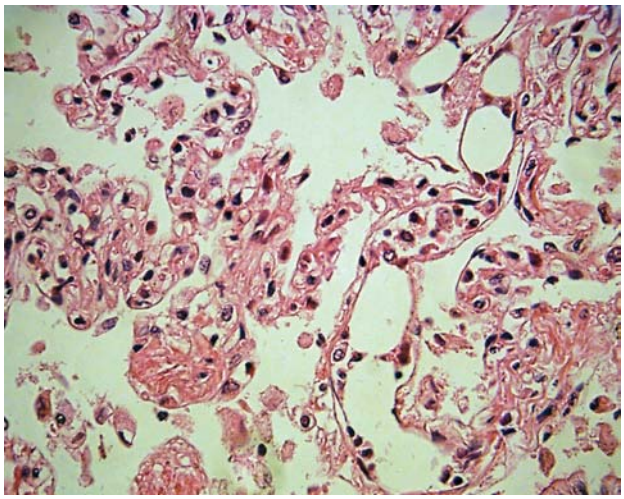

FIG. 1—*Pulmonary microvasculature distended with fat emboli (Hematoxilen-Eosin staining), X200.*

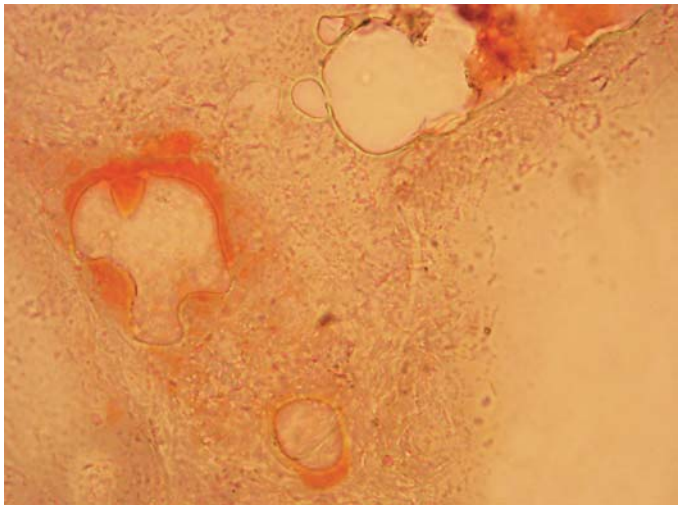

FIG. 2—*Fat embolization of pulmonary microvessels with Oil-red-O stain, X400.*

Mild hypertrophic changes and perivascular fibrosis were determined in myocardium. The liver showed centrilobular congestion without any fatty change. Other visceral organs showed no striking findings other than mild congestion.

Toxicological Examination

Tissues submitted for toxicologic analysis, showed no evidence of any kind of agents. The cause of death was considered to be respiratory insufficiency resulting from severe fat embolism of the lungs.

Discussion

Although the mechanism of fat embolism has not yet been clearly defined, several mechanical and biochemical theories are proposed (7). According to mechanical theory, the relation of fat containing cavities with teared vessels under the effect of pressure enables the passage of fat or bone marrow into the circulation. According to the first biochemical theory, fat droplets within the circulation have local toxic effects on tissues due to their destruction by free fatty acids. On the other hand, the second biochemical theory, proposes fatty acids mobilized by cathecolamines form fat droplets leading to embolism (1). Embolism may occur following long bone and pelvis fractures. However, it may not only occur unassociated with previous simple traumas (8) or secodary to soft tissue injuries (9) but also in non traumatic cases such as pancreatitis, burns, osteomyelitis, epilepsy, sickle-cell disease and fatty liver (7).

Although NTFE is as rare entity, it may follow a fatal course mimicking clinical findings of fat embolism (3). Its pathogenesis is intended to be explained by biochemical theories (1).

In this case, no major trauma other than pregnancy and resuscitation was evident. Her medical past history indicated that there was no major problem during the pregnancy, which was followed by a vaginal delivery. Moreover, no evidence of bleeding, atonia or other findings were seen clinically or on autopsy. The most striking features were fat embolism and sternal and rib fractures due to resuscitation.

Clinical signs of fat embolism frequently include respiratory distress syndrome characterized by tachypnea and dyspnea, cerebral manifestations and petecchial rash on the mucous membranes and skin (5,7). In this case, the two major findings were observed, however the rash was not. Because of the medical history there was no likelihood of the predisposing factors. This specific medical history argues for the importance of histologically proven grade III disseminated fat embolism as the major cause of the rapidly progressive dsypnea and loss of conciousness. However, the effects of resuscitation giving rise to bone fractures as a possible causative factor should also be considered. Pregnancy and puerperal period are the major risk factors in pathogenesis of pulmonary embolism (10). It has also been reported that pregnancy might be a triggering factor for fat embolism (11). Godeau et al. reported that massive fat embolism can develop particularly in pregnant women using prostoglandin E2 (12). And also, different studies indicated that fat embolism resulting from vasoocclusive crisis in pregnant women with sickle cell anemia can lead to maternal deaths (13). Non-traumatic fat embolism has been reported in a variety of conditions including prolonged corticosteroid administration for lymphoma and autoimmune disorders (3). Various studies indicate that fatty change due to longstanding treatment with steroids might be a triggening factor in embolisation However, neither clinical nor laboratory findings related with sickle cell anemia and cortisone treatment could be determined in our case.

Chest compression is not a risk-free intervention. Approximately 30% of victims are found at autopsy to have rib fractures and close to 20% have sternal fractures (14). In some cases, bone marrow embolism are present even when an autopsy does not reveal any trauma (15–17). The possible sources of histologically determined bone marrow and fat embolism in this case could have resulted from three factors: 1. Elevated catecholamine levels and mobilisation of free fatty acids after delivery and sudden changes occuring during delivery, 2. Fat embolism secondary to CPR as an result of iatrogenic sternal and rib fractures, and 3. An underlying disease or disorder giving rise to clinical signs and symptoms without any clinical or autopsy findings.

In cases such as this, it is imperative that the clinically history be thoruughly evalauted and that an autopsy be performed to determine the cause of death.

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