CASE REPORT

UDC: 616.136-089.06
https://doi.org/10.2298/VSP171224069M

Iatrogenic pulmonary fat embolism after surgery in a patient with fatty liver

Jatrogena masna embolija pluća posle hirurške intervencije kod bolesnika sa masnom jetrom

Dragan Mitrović*, Svetlana Lazarević†

University of Belgrade, Faculty of Medicine, *Institute of Pathology, Belgrade, Serbia;
†Institute for Orthopaedic Surgery "Banjica", Belgrade, Serbia

Abstract

Introduction. Fat embolism refers to the presence of fat globules in the lung parenchyma and its peripheral circulation. Obstruction of the lung vessels by fat emboli can lead to acute *cor pulmonale* when the compensatory capabilities of the pulmonary vasculature are exceeded. **Case report.** We presented a case of a 78-year old man who suffered a dissection of abdominal aortic aneurysm. Urgent surgical procedure was performed and aneurysm replaced with aorto-bifemoral bypass grafting using a Dacron graft. Despite the procedure the patient died the following day. The autopsy revealed that the cause of death was hypovolemic shock. There were no bone fractures (also no fractures of ribs and sternum from cardiopulmonary resuscitation) or injuries of the subcutaneous fat tissue or other organs (besides those from the surgery). However, additional autopsy findings included fatty liver change, small liver hemorrhages (confirmed microscopically), as well as a presence of fat droplets in the hepatic veins, as well as in the pulmonary vessels, i.e. pulmonary fat embolism [confirmed with hematoxylin/eosin (H/E), and Sudan III staining], which could be the contributing cause of death. **Conclusion.** The presented case indicates that pulmonary (or even systemic) fat embolism should be considered as the possible iatrogenic cause of unexpected and unexplained death in the cases where elective surgical procedures were performed in patients with fatty liver change. Pathologists must be aware of this possibility, since it is not easily recognized on routine H/E staining, and some of the special staining technique should be applied.

Key words:
embolism, fat; aneurysm, ruptured; postoperative complications; pulmonary embolism; fatty liver; diagnosis, differential; death.

Apstrakt

Uvod. Masna embolija je prisustvo masnih kapi u plućnoj i perifernoj cirkulaciji. Kada se prevaziđu kompenzatorne mogućnosti plućne cirkulacije, njena opstrukcija masnim kapima može dovesti do akutnog plućnog srca. **Prikaz bolesnika.** Prikazan je muškarac, star 78 godina kod koga je došlo do disekcije aneurizme trbušne aorte. Učinjena je hitna hirurška intervencija – aorto-bifemoralno premošćenje dakronskim graftom. Uprkos primenjenim merama lečenja smrtni ishod nastupio je sledećeg dana. Obdukcijom je ustanovljeno da je uzrok smrti hipovolemijski šok. Obdukcijom nisu ustanovljeni prelomi kostiju (takođe, ni prelomi grudne kosti, niti rebara, koji su mogli nastati prilikom reanimacije) ili povrede potkožnog masnog tkiva ili drugih organa (izuzimajući organe na kojima je izvršena hirurška intervencija). Međutim, nađena je i bolest masne jetre, sitna supkapsularna krvarenja (potvrđena mikroskopskim pregledom), kao i prisustvo masnih kapi u hepatičkim venama, kao i krvnim sudovima pluća, tj. ustanovljena je masna embolija pluća [potvrđena hematoksilin-eozin (H/E) i Sudan III bojenjem], što je moglo biti doprinoseći uzrok smrti. **Zaključak.** Prikazani slučaj pokazuje da masna embolija pluća ili čak sistemska masna embolija mora biti uzeta u obzir kao mogući jatrogeni uzrok neočekivane smrti bolesnika sa masnom jetrom, potvrgnutih elektivnom hirurškom zahvatu. Patolozi moraju biti svesni ove činjenice, pošto masnu emboliju nije lako prepoznati na rutinskim H/E bojenjima, zbog čega se moraju primeniti neka od specijalnih tehnika bojenja.

Ključne reči:
embolija, masna; aneurizma, ruptura; postoperativne komplikacije; pluća, embolija; jetra, masna infiltracija; dijagnoza, diferencijalna; smrt.

Correspondence to: Dragan Mitrović, Institute of Pathology, Dr Subotića starijeg 1 Str, 11 000 Belgrade, Serbia.
E-mail: dramit@med.bg.ac.rs

Introduction

A presence of fat droplets in the lung circulation presents a pulmonary fat embolism. With the obstruction of the sufficient percent of pulmonary vessels by the fat emboli, with the exceeding of its compensatory capabilities, an acute right heart failure may develop [1–3]. Commonly, the lung fat embolism after sustained trauma is a subclinical event: droplets of fat are sucked into the venous system at a site of fracture and then get stuck in the pulmonary circulation. Once entering the lungs, fat globules might enter the systemic circulation, reaching different organs. The clinical manifestation of systemic fat embolism, primarily with progressive respiratory distress and a deterioration in central nervous system function is called fat embolism syndrome [2]. Although most commonly associated with trauma [1–3], there are some described cases of non-trauma related fat embolism [4–10]. We presented one such case, most probably caused iatrogenically, in a setting of previously existing fatty liver change.

Case report

A 78-year-old man was admitted to the hospital, after being transferred from a regional medical center with the computed tomography (CT) confirmed rupture of abdominal aortic aneurysm. The diameter of aneurysm was 87 × 79 mm and the rupture was localized on the right lateral side of the abdominal aorta, just below the renal arteries. The urgent surgical procedure was performed: open transperitoneal resection of the ruptured part of the aortic aneurysm, which was replaced with aorto-bifemoral bypass grafting using a Dacron graft. During the entire course of the operation, the patient was hypotensive. Several hours after the procedure, the patient died with the clinical signs of hemorrhagic shock.

The autopsy was performed the following day. The macroscopic examination showed signs consistent with hemorrhagic shock, as well as signs of the described surgical procedure. The signs of severe atherosclerosis were most prominent in the aorta, and slightly less in coronary arteries. The lungs were livid and heavy (total lung weight was about 1,600 g), while the liver showed signs of fatty change, as well as small subcapsular bleedings on its surface. There were no bone fractures (and also no fractures of ribs and sternum from cardiopulmonary resuscitation) or injuries of the subcutaneous fat tissue or other organs (besides those from the surgery). Microscopic examination [hematoxylin/eosin (H/E) staining] of the liver showed moderate to severe fatty change in hepatocytes, but also small subcapsular fresh hemorrhages (Figures 1a and 1b). Examination of the lungs, however, revealed the possible presence of fat droplets in the pulmonary vessels, i.e. the pulmonary fat embolism (Figure 2a). Therefore, additional Sudan III-staining was performed on samples taken from the lungs, as well as the brain, kidneys and liver. The histological findings of frozen sections of the lungs confirmed the pulmonary fat embolism: red sausage-shaped or rounded, multiple, disseminated fat emboli were present in every microscopic field (Figures 2b, 2c and 2d). These findings corresponded with the moderate, second grade lung fat embolism [1]. Fat droplets were not found in the brain and kidneys. On the other hand, this special staining confirmed fatty change in the hepatocytes, but also showed the presence of multiple fat globules in middle caliber hepatic veins (Figures 1c and 1d). The main cause of death was attributed to hemorrhagic shock due to rupture of aortic aneurysm, while pulmonary fat embolism was the contributing factor.

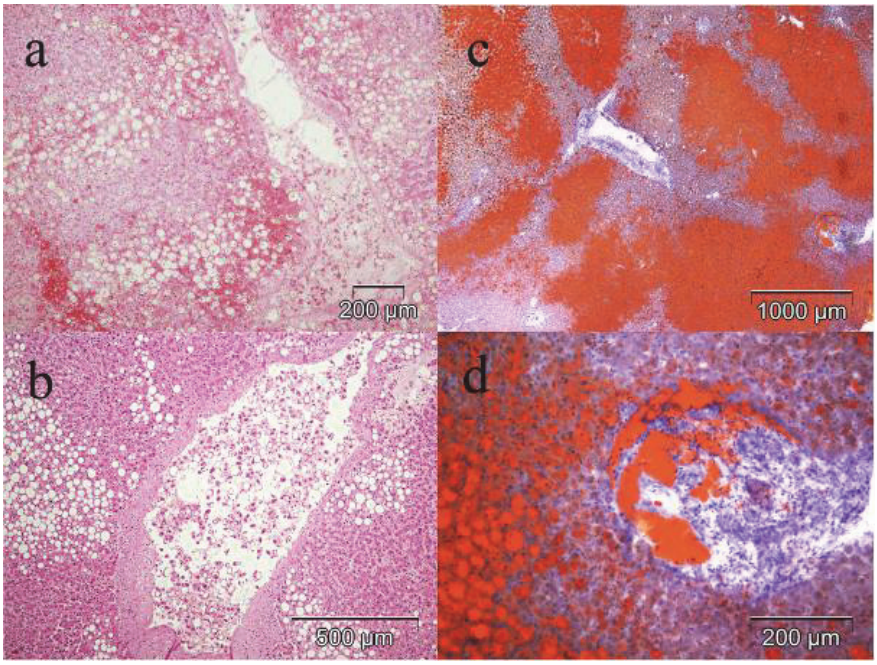

Fig. 1 – Liver: a, b) Fatty changed liver with small tissue hemorrhages due to injury during the surgical procedure [hematoxylin/eosin (H/E) staining]; c, d) Note the presence of fat globules in the hepatic veins (frozen Sudan III-stained sections), visible both in H/E and Sudan III stains.

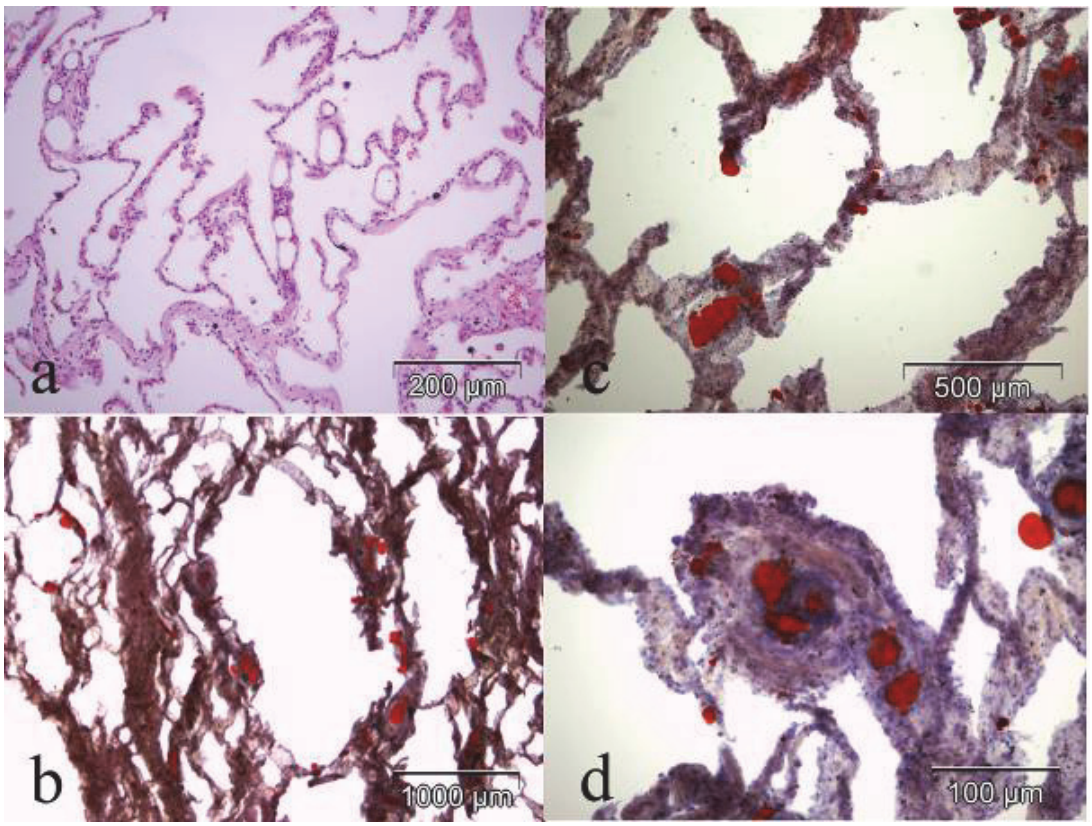

Fig. 2 – a) Hematoxylin/eosin (H/E) staining of the lungs shows the presence of fat globules in small pulmonary vessels; b, c, d) Frozen Sudan III-stained sections of the lungs show red, drop, sausage- and branching-shaped fat emboli in pulmonary vessels.

Discussion

Fat embolism is most commonly associated with trauma (i.e. fractures) [1–3], however there is relatively small number of described cases of fat embolism in the absence of trauma, associated with alcoholic or steroid-induced fatty liver, acute hepatic necrosis or diabetes [4–8], and only a few papers of postoperative fat embolism [9]. One of studies found quite surprising incidence of fat embolism in cases of sudden death – in 34 out of 65 cases [10].

Fat embolism represents the mechanical blockage of blood vessels by circulating fat globules. Lungs are most commonly affected organ, but fat embolism also might affect organs such as the brain, retina, and skin [11]. Systemic fat embolism syndrome is defined as clinical manifestation of systemic fat embolism [2], and it could be potentially fatal complication of trauma or surgical procedure. Typical occurrence is 12h to 72 h after the surgery, presenting with progressive respiratory insufficiency, consciousness disorders and petechiae. Different neurological disorders may be present, ranging from mental confusion to altered level of consciousness, and also transient and reversible disorders manifesting as generalized convulsions and focal deficits [11].

There are several theories that explain the pathophysiological features of fat embolism. So called infloating theory, or the traditional view of fat embolism explains that fat is physically pushed into the veins after trauma, most typically after fracture of long bones [1]. Lipase theory explains that trauma causes an elevation of plasma lipase titer, which then destabilizes circulating fats by de-emulsification, saponification and mobilizing lipid stores. A second biochemical theory invokes the possible histotoxic effects of free fatty acids from bone marrow. Finally, shock and coagulation theory is based on noting that many patients who develop post-traumatic fat embolism are also hypovolemic. Hypovolemia leads to a slowing down of circulation with "slugging" of blood components and forming of micro-aggregates in the lungs. Tissue trauma worsens this by damaging vessels' intimal layer causing platelet activation. Fat from bone marrow then might provide a possible adherence surface for the activated platelets [1].

In the presented case, in the absence of fractures or extensive necrosis of fat tissue, the lung fat embolism could be explained by some of the latter theories. However, both gross and microscopic findings of small subcapsular hemorrhages in the liver, together with the presence of fat globules in hepatic veins and fatty changed hepatocytes indicated a somewhat different mechanism. Most probably, the mechanical pressure on the fatty changed liver during the urgent surgical procedure of abdominal aorta, led to liver injury with small hemorrhages and release of fat from injured hepatocytes. Fat emboli from hepatocytes then entered venous circulation, and finally led to the pulmonary fat embolism which could additionally exacerbate the existing hypovolemic shock. Al-

though undoubtedly the main cause of death in the presented case was hypovolemic shock due to abdominal aortic aneurysm rupture, iatrogenic pulmonary fat embolism could be considered as additional, contributory cause of death.

There is yet no specific treatment for either the pulmonary embolism or fat embolism syndrome [11]. However, it is essential that support measures, especially in treating adult respiratory distress syndrome, such as oxygen therapy or mechanical ventilation, have to be undertaken in more severe cases.

Conclusion

The presented case indicates that iatrogenic pulmonary (or even systemic) fat embolism should be considered as the possible cause of unexpected and unexplained death in the cases where elective surgical procedures were performed in patients with fatty liver change. The pathologists must be aware of this possibility, since it is not easily recognized on routine H/E staining, and some of the special staining technique should be applied.

R E F E R E N C E S

1. *Mellor A, Soni N.* Fat embolism. Anaesthesia 2001; 56(2): 145–54.
2. *Nikolić S, Živković V, Babić D, Djonić D, Djurić M.* Systemic fat embolism and the patent foramen ovale – a prospective autopsy study. Injury 2012; 43(5): 608–12.
3. *Nikolić S, Živković V, Strajina V, Djonić D, Popović V, Djurić M.* Lung fat embolism in a body changed by putrefaction: a hip fracture ante-mortem in origin – case report. Med Sci Law 2012; 52(3): 178–80.
4. *Durlacher SH, Meier JR, Fisher RS, Lovitt WV Jr.* Sudden death due to pulmonary fat embolism in persons with alcoholic fatty liver. Am J Pathol 1954; 30: 633–4.
5. *Kent SP.* Fat embolism in diabetic patients without physical trauma. Am J Pathol. 1955; 31(3): 399–403.
6. *Hill RB Jr.* Fatal fat embolism from steroid-in-duced fatty liver. N Engl J Med 1961; 265: 318–20.
7. *Hulman G.* Fat macroglobule formation from chylomicrons and non-traumatic fat embolism. Clin Chim Acta 1988; 177(2): 173–8.
8. *Schulz F, Trübner K, Hildebrand E.* Fatal fat embolism in acute hepatic necrosis with associated fatty liver. Am J Forensic Med Pathol 1996; 17(3): 264–8.
9. *Schulz F. Tsokos M.* Fatty liver and fat embolism. A case report. Rechtsmedizin 2004; 14(6): 463–6. (Luxembourgish)
10. *Kim PJ, Pollanen MS.* Osmium impregnation detection of pulmonary intravascular fat in sudden death: A study of 65 cases. J Forensic Leg Med 2012; 19(4): 201–6.
11. *Costa AN, Mendes DM, Toufen C, Arrunátegui G, Caruso P, de Carvalho CR.* Adult respiratory distress syndrome due to fat embolism in the postoperative period following liposuction and fat grafting. J Bras Pneumol 2008; 34(8): 622–5. (English, Portuguese)

Received on December 15, 2017.
Revised on September 12, 2018.
Accepted on September 13, 2018.
Online First September, 2018.