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CASE REPORT

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FAT EMBOLISM SYNDROME CASE IN A WOMAN PRESENTING WITH MULTIPLE INJURIES FOLLOWING A TRAFFIC ACCIDENT SUCCESSFULLY TREATED USING INTERDISCIPLINARY APPROACH – CASE REPORT

Szymon Bialka[1], Adam Ubych[2], Jacek Smereka[3], Tomasz Byrczek[4], Krzysztof Szaniewski[5], Lukasz Szarpak[6]

[1]Department of Anaesthesiology, Intensive Care and Emergency Medicine, School of Medicine with Division of Dentistry in Zabrze, Medical University of Silesia, Zabrze, Poland
[2]Centre of Didactics and Medical Simulation, Medical University of Silesia, Katowice, Poland
[3]Department of Emergency Medical Service and Laboratory of Experimental Medicine and Innovative Technology, Wroclaw Medical University, Poland
[4]Department of Emergency Medicine, Medical University of Silesia, Poland
[5]Department of Vascular Surgery Trauma Centre Hospital No5 District of Silesia, Poland
[6]Medical Faculty of Lazarski University, Warsaw, Poland

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BACKGROUND

Trauma is among the main causes of death among people aged under 30 years. A potential complication of extensive injuries is an episode of fat embolism [1], also referred to as fat embolism syndrome (FES) in literature [2]. The main causes of fat embolism include long bone and pelvic fractures. Depending on the severity of injuries, their character, and the number of fractures, the frequency of this complication ranges from 0.25% to 8.0% [3]. Symptoms of fat embolism may also develop after elective orthopaedic surgery, removal of subcutaneous fat tissue, multiple rib fractures, acute pancreatitis, or parenteral nutrition with fat-containing products [4].

Two mechanisms are considered to lead to the development of clinical signs. At the first stage, lung arterioles are mechanically closed by fat globules released into veins. At the later stage, mediators are released as a result of hydrolysis of the fatty embolism material and the release of a large amount of free fatty acids. Massive free fatty acids release under the influence of increased adrenergic stimulation explains FES in patients without injury [3, 5].

CASE REPORT

Chief complaints

A 44-year-old woman presenting with multiple injuries following a traffic accident (hit by a car) was transported by a medical emergency team to a hospital emergency department.

History of present illness

Patient's symptoms started after a traffic accident.

History of past illness

The patient had a free previous medical history.

Physical examination

At the time of hospital admission, the patient was unconscious, unresponsive; Glasgow Coma Scale: 4 points; respiratory failure. The patient was intubated and mechanically ventilated with FiO2 1.0; oxygen saturation 90%, presented circulatory insufficiency; heart rate: 110/min, blood pressure: 90/50 mm Hg, pulse on radial artery undetectable. The trauma examination showed a wound in the parietal region and chest bruising.

ADDRESS FOR CORRESPONDENCE:
Szymon Bialka, MD, PhD, Department of Anaesthesiology, Intensive Care and Emergency Medicine, School of Medicine with Division of Dentistry in Zabrze, Medical University of Silesia, 41–800 Zabrze, 3 Maja Str 13–15, Poland; e-mail: szymon.bialka@gmail.com, tel.: +48 32 37 04 593

Imaging examinations

The focused assessment with sonography in trauma found no fluid in the peritoneal or pleural cavity. Computed tomography revealed a paracerebral haematoma up to 6 mm wide along the right frontal lobe and a rupture fissure in the left frontal bone passing through the upper wall of the left orbit. A small amount of fluid was visible in the left maxillary sinus, as well as haematoma and emphysema of soft tissues of the head in the right parietal region. Additionally, numerous fractures of ribs, transverse processes of thoracic vertebrae, and right scapula were confirmed.

Final diagnosis

The final diagnosis of the presented case is fat embolism syndrome.

Treatment

After orthopaedic surgical treatment, the patient was transferred to an intensive care unit owing to her severe condition. Mechanical ventilation was continued with FiO2 1.0, SpO2 92%. In the following hours, a decreased ratio of PaO2/FiO2 $<$ 250 mm Hg was observed in the arterial blood gas test. Circulatory insufficiency was present with noradrenaline infusion in the dose of 0.25 μg/kg/min. Fever up to 38.5°C. Follow-up laboratory tests confirmed anaemia (HGB: 8.9 g/dl, HCT: 26.6%) and thrombocytopenia (PLT 117 $\times$ 103/μl). Symptoms of oliguria with renal failure were found; creatinine: 1.2 mg/dl, GFR: 70 ml/min/1.73 m2.

On the second day of hospital treatment, abdominal computed tomography was performed which raised suspicion of spleen injury and revealed left iliac vein thrombosis with suspicion of fat embolism (Fig. 1).

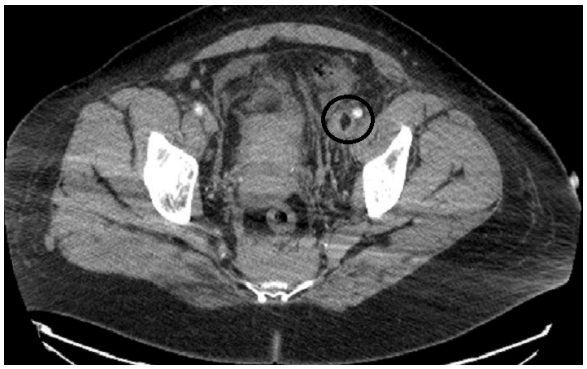

FIGURE 1. Abdominal computed tomography with contrast. Left common iliac vein widened to 16 mm, without visible contrasting. Left external iliac vein widened to 22 mm, without flow maintained, with a 12 $\times$ 10 mm area of fat density visible in its light. The picture probably corresponds to fat embolism

On surgical consultation, the patient was disqualified from laparotomy owing to her severe condition and ambiguous diagnostic tests results. On the basis of the clinical picture confirmed by the results of radiological studies, the diagnosis of fat embolism was made. In order to stabilize the clinical condition, symptomatic treatment was implemented which included filling the vascular bed with human albumin solution (200 mg/ml) with a simultaneous norepinephrine infusion (under control of haemodynamic parameters measured by the Pulse Contour Cardiac Output technology), mechanical ventilation, supply of unfractionated heparin and methylprednisolone. Metamizole, paracetamol, and physical cooling were applied to prevent body temperature rise above 38°C. The implementation of the above treatment resulted in an improvement of the patient's general condition.

The patient was surgically consulted again for the procedure of filter insertion into the inferior vena cava. On stabilization of the ventilation and circulatory parameters, 14 days after the accident, the patient was transferred to a vascular surgery ward for preventive insertion of a vascular filter (Fig. 2).

Before leaving the intensive care unit, the patient informed about the rules of publication and consented to the description of her case in the scientific journal.

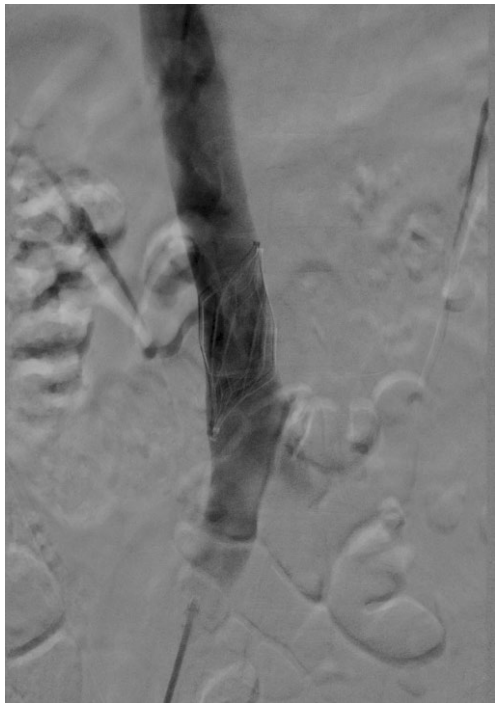

FIGURE 2. X-ray with contrast. Endovascular filter inserted to the inferior vena cava

Table 1. Diagnostic criteria for fat embolism according to Gurd

Large clinical criteria	Hemorrhagic skin bleeding Hypoxia: PaO2 < 60 mm Hg Central nervous system disorders Pulmonary edema
Small clinical criteria	Tachycardia > 120/min Fever > 38°C Changes in the retina: ecchymosis, droplets of fat in vessels Icterus Renal symptoms: anuria or oliguria
Laboratory criteria	Thrombocytopenia Anemia High erythrocyte sedimentation rate Fatty macroglobulinemia

DISCUSSION

Most of the possible clinical signs are non-specific. Initially, an asymptomatic phase (12–72 hours) occurs [6, 7], followed by a progression of symptoms. FES causes central nervous system symptoms (headache, confusion, unconsciousness, convulsions, stupor, or brain oedema) [8], skin symptoms (reddish-brown petechiae visible on the upper body skin and conjunctiva), and a number of non-specific symptoms, such as tachycardia, fever, retinal haemorrhages, exudative oedema, jaundice, or lipiduria [1]. The triad of clinical signs including gas exchange disorders in the lungs, central nervous system disorders, and bloody petechiae on the skin makes FES diagnosis much easier. However, the final diagnosis is based on meeting one large criterion, four small criteria, and one laboratory criterion in accordance with Gurd's assumptions (Tab. 1) [9].

CONCLUSION

Proper diagnosis of FES in patients with severe trauma can be significantly impeded in everyday clinical practice. This is due to the fact that the symptoms typical of FES (such as gas exchange disorders, central nervous system disorders, bloody petechiae on the skin, laboratory abnormalities) commonly occur in injured patients, masking FES presentation.

Precise knowledge of the pathomechanism and clinical presentation of FES seems indispensable in the approach of an interdisciplinary therapeutic team. A quick correct diagnosis is crucial to improve treatment results. Access to laboratory and imaging tests considerably facilitates the diagnostic process, which should be followed by optimal therapeutic management. The lack of a golden standard of treatment necessitates a flexible and interdisciplinary therapeutic approach.

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