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CASE REPORT

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Fat embolism syndrome and massive middle cerebral artery occlusion: a case report

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Fat embolism syndrome (FES) is common with bones fractures. It often involves several symptoms like respiratory distress, neurological impairment, and cutaneous rash. The symptoms often start after several hours or days after the trauma or the bone reduction. Neurological damages can be seen better with MRI and the pattern is most of the time a diffuse one but changes according to timing. In our case, the symptoms started in the recovery ward and a complete flow interruption by adipose material was seen in the left middle cerebral artery.

Keywords: embolism, Fat, fractures, bone, middle cerebral artery, embolectomy.

INTRODUCTION

Fat embolism is a relatively common complication associated with bone fractures. But most of the time, it has no clinical impact on patients. The fat embolism syndrome (FES), however, is known as a frequent combination of different symptoms involving the respiratory, neurological, cutaneous, and haematological systems. It has an incidence of approximatively 3-4% of patients with bone fractures and has a mortality rate estimated at 10 %[1,2]. The symptoms often start 24-72 hours after the trauma or the surgical correction[3] and frequently begin with a respiratory impairment. Cognitive impairment can rapidly follow or appear at the same time. The neurological symptoms are often diffuse but massive cerebral embolism can lead, in certain cases, to focal deficiency[2].

CASE REPORT

An 85 years old lady was admitted to the emergencies following a mechanical fall at home. At the admission, a femoral fracture, Garden IV, was shown at the left leg. A cerebral scanner was also done due to confusion, but no acute injury was reported. Her medical history was hypertension and a transient ischemic attack 6 months earlier. She had fully recovered from it and had left the hospital before all the investigations could be done. As medicine, she was only taking aspirin and a Beta-blocker for her blood pressure.

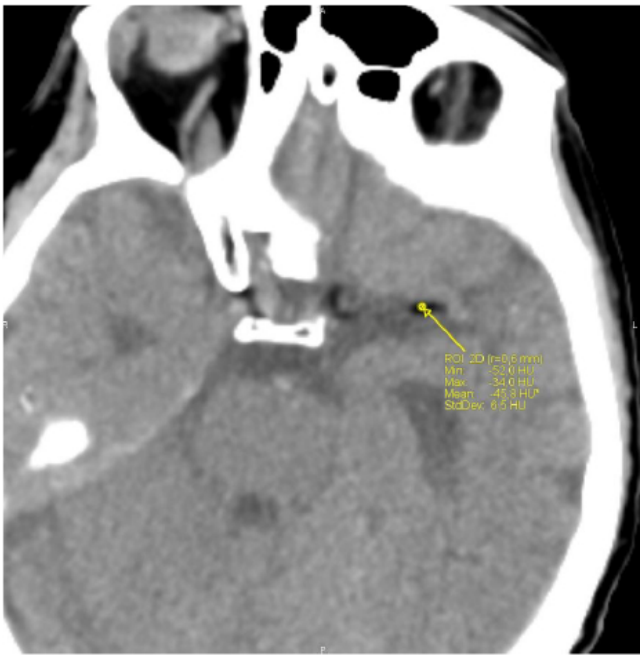

Figure 1.

The next day, a cemented total hip arthroplasty was performed, there was no major incident during the procedure. In the recovery room, she rapidly developed a right hemiplegia with drowsiness. A cerebral angio-CT showed a complete left middle cerebral artery occlusion at M1 segment by fatty material (Figure 1

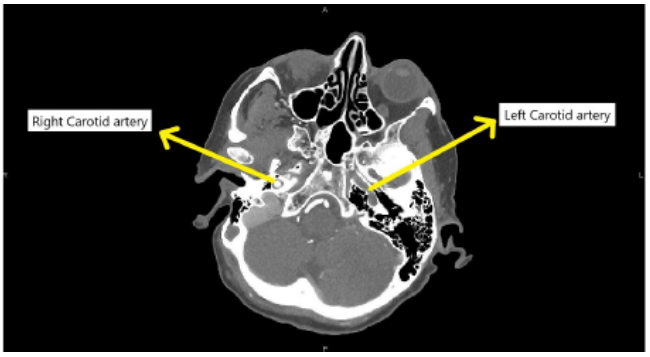

Figure 2.

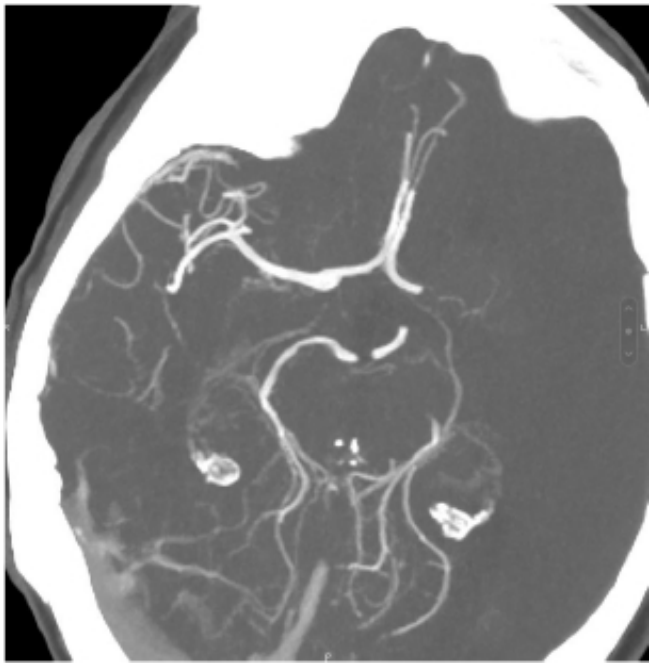

Figure 3.

and 2) and a consecutive thrombus extending to the left internal carotid (Figure 2).

She was then transferred to a comprehensive stroke center where an endovascular clot retrieval (ECR) was successfully performed (mTICI grade 3) (Figure 4 & 5). Unfortunately, her condition deteriorated the day after with a Glasgow Coma Scale of 3/15. A control brain scanner showed a malignant medial infarction with major oedema and a local haemorrhagic conversion (Figure 6). Due to the poor prognosis of the situation, a palliative treatment was decided, and she died the same day.

DISCUSSION

Fat embolism syndrome was first clinically described in 1861 by Zenker[4] who found fat droplets on lung capillaries of a man who died from a crush injury. Then, Von Bergmann described the symptoms in 1873[4].

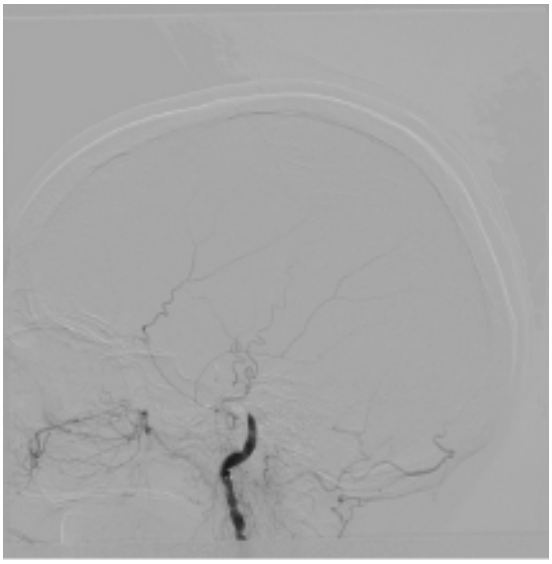

Figure 4.

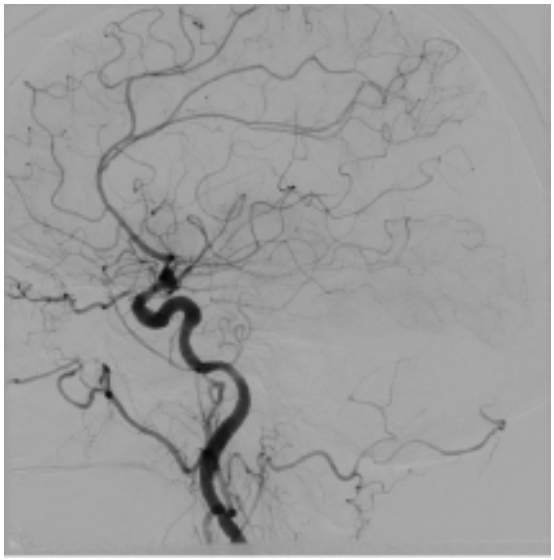

Figure 5.

These symptoms involve a respiratory impairment, emerging in 75 % of patients, going from a transient respiratory distress to 10-20% of patients entering the ARDS criteria[2]. Neurological impairment happens in 59% of patients, including confusion or disorientation to cerebral death. The cerebral symptoms are mostly diffuse but 20% of them are focalised[2]. Petechial rash will happen in approximately 33% of patients and will be prominent on areas like the head, the neck, the upper part of the thorax and the axillae regions[3]. Finally, haematological signs can also appear, with an unexplained anemia occurring in 67 % of the cases, but thrombocytopenia can also occur, coagulopathy or fat droplets can be seen in histology[5]. The symptoms usually appear progressively between 24-72 hours after

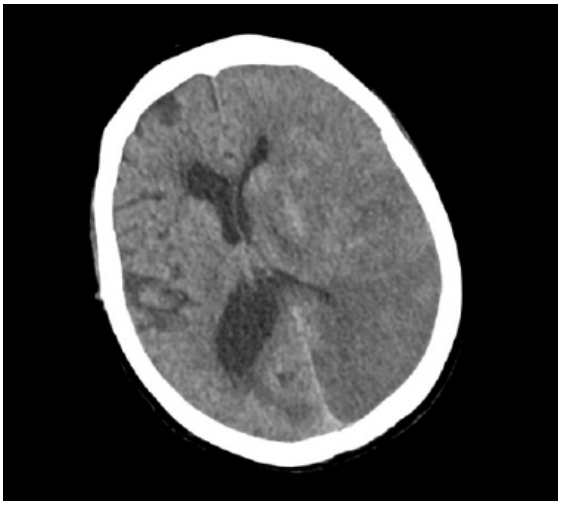

Figure 6.

the trauma or the surgical procedure and the respiratory distress is often the first to take place and might be quickly followed or even preceded by neurological signs[6]. Many of these situations are, in fact, subclinical, but some conditions like a reduced cardiorespiratory reserve, age, hypovolemia or pulmonary impairment will make them more noticeable[7].

Two mechanisms have been proposed to explain this syndrome. The first one was established by Gauss in 1924 and has been named the "Mechanical mechanism". Three years later, Lehman developed another one called "Biochemical mechanism"[2]. The mechanical theory involves the path of fat emboli to the veins due to an increase in the intra-medullary pression. These emboli will create a mechanical obstruction, local ischemia and will lead to endothelial and parenchymal damages. The biochemical theory states that it is the inflammatory response, leading to breakdown of fat elements into free fatty acids and production of cytokine like IL-6, Il-1, TNF-alpha or serum lipase, that will damage the endothelium and the parenchyma[3]. But at this day, the exact scenario is still not fully understood.

As an example, the systemic affect is not fully explained either. A patent foramen ovale (PFO) is known to be effective in 20-25% of the population[8] and has been shown to let fat materials pass into the systemic circulation on multiple TEE, favoured by an increase in pressure on the right atrium and a right to left shunt[9]. Therefore, fat can enter arterial system through cardiopulmonary shunts or via change of shape[10]. It is thought that fragments lower than 7-10 micrometers can pass through pulmonary capillaries due to high pulmonary artery pressure, and only 80% of them remain stuck in the lung[11].

Regarding of cardiopulmonary shunts, a case series suggested clinical differences in large vessel occlusion caused by fat embolisms. For example, the symptom onset was mostly within the first 6 hours after operation, respiratory or dermatological manifestations were usually absent, and the prognosis was worst with a high mortality or permanent disability. In this series, 4 out of the 5 patients had a PFO at ETT, implying that it could mean a higher risk of substantial systemic emboli and large vessel occlusion[12].

Concerning our patient, no cardiac ultrasound had been made in the past. Unfortunately, the evaluation of the transient ischemic attack could not have been done since she had left the hospital. But the restrained localisation and the size of the cerebral emboli could be in favour of a PFO.

The majority of FES are linked to an injury involving a bone fracture. But numerous other situations can be seen; for example: soft tissue trauma, burns, liposuction, cardiopulmonary bypass surgery or even sickle cell disease[13]. FES in relation to bones fractures is more prevalent with long bone fractures, multiple fractures and with male patients. The peak of age is thought to be between 10 and 40 years old in the male population and is thought to be favoured by high velocity injury[6]. It has appeared to decrease during the years due to several changes like response time of handling the fracture, the use of washing vacuum or the choice of the reamer[7]. It went from an incidence of 8% historically to 2% nowadays[14]. However, the exact incidence changes a lot across studies, according to the fact that it is a retrospective or prospective one and because a lot of fat embolisms remain subclinical[15]. A risk factor is the use of cement during the operation, due to the increase in the medullary that pushes fat emboli to the metaphyseal veins. But previous studies showed that it diminished the risk of mechanical complications like periprosthetic fracture from 1.5% to 0.2%, especially in older people[16].

In our case, the only risk factor that were present was the fact that it was a long bone fracture and the use of cement to limit the risk of mechanical complications. The timing remained short; the patient was managed the next day after her fall as recommended by the last guidelines[17].

The diagnostic of FES is mainly clinical, several algorithms have been made during the years but none of them was clinically approved. The main algorithm that is still used is the one from Gurd and Wilson, that was made in 1974[5]. It is composed of major and minor criteria, having two major criteria or one with four minor criteria will suggest a diagnosis of FES (Table I). Since then, the developments in imagery like scanner and MRI gained in importance.

Table I.

Original Gurd and Wilson criteria 2 major criteria or 1 with 4 minor criteria suggest a diagnosis of FES	
Major criteria	Minor criteria
Hypoxemia	Heart rate > 110/minute
Altered mentation	Pyrexia > 38.5°C
Petechiae	Retinal fat embolism
	Oligoanuria or urinary fat globules
	jaundice
	Sudden Thrombocytopenia > 50%
	Unexplained sudden anemia
	High ESR

Table II.

Modified Gurd and Wilson criteria 1 major criteria with 3 minor or 2 major with 2 minor suggest a diagnosis of FES	
PaO2 < 60 mmHg with an FiO2 of 21% with or without pulmonary infiltrate at imagery	Heart rate > 110/minute
Altered mentation with multiple cerebral white matter lesions on brain MRI	Pyrexia > 38.5°C
Petechiae on conjonctiva and upper trunk	Thrombocytopenia < 100 x 103/microL
	Anemia with coagulopathy or DIC without definite ongoing bleeding site
	Retinal Embolism

Pulmonary imagery can show nothing or nonspecific focal of diffuse areas of consolidation, ground-glasses opacities, and small nodules of various sizes. These signs appear often after several hours/days from the start of the symptoms. For cerebral imagery, MRI is considered better than CT as the scanner sometimes does not show any abnormalities in the first hours/days. MRI in T2-weighted images often shows a "starfield" pattern, with fat emboli predominantly seen in the deep white matter area and basal ganglia[15]. It is thought that MRI pattern has a dynamic change through time, involving different phenomena like cytotoxic or vasogenic oedema[18]. These advances lead to propositions of modified Gurd and Wilson criteria (Table II) which included imagery[19].

These criteria are mostly there to guide the practitioner and are not always fulfilled. In our report, the patient had a clear cerebral damage and brain angioscanner helped us to make the diagnosis, even if it does not often show such a localised image usually. The patient was effectively needing 5L/minute of oxygen through nasal cannula, but it is difficult to assess if it was due to emboli or immediate post-operative conditions. No pulmonary imaging was performed in the context of the cerebral damage.

Finally, the care of this kind of syndrome is mainly a supportive therapy. A majority of people will survive with no or little symptoms and 58% of them will recover during the next year even in severe brain damage[2,20]. The mortality rate of cerebral embolism remained at 10% in previous studies[6]. No pharmacological prevention has been proven to be effective. It was thought that corticoids could help but a meta-analysis showed in 2009 that even if it was reducing FES and the respiratory symptoms, it had no effect on mortality[5].

For us, after the embolectomy that was successfully performed four hours after the onset of the symptoms, the patient still developed oedema and haemorrhagic conversion leading to her death. This futile recanalization could be multifactorial, firstly due to ischemia-reperfusion altering the blood brain barrier, as middle cerebral artery is responsible for a great part of the cerebral cortex, basal ganglia, and internal capsule. But also due to the proper cytotoxic oedema following local toxicity of free fatty acids and residuals micro-emboli[21].

CONCLUSION

Fat embolism syndrome is a widespread complication of surgical operations and various trauma. The precise rate is not known as a great part remains subclinical, but the recent studies suggest that its incidence is decreasing. Advances like change in timing of handling or new methods and materials can explain this regress. The symptoms are diverse and often involve respiratory, dermatologic, and cerebral systems, going from mild clinical presentation to severe, needing intensive care. Fat materials are most of the time of small size and have a diffuse spread but can be greater and lead to a large vessel occlusion. In this scenario, a cardiopulmonary shunt like PFO can be involved, but the percentage of this kind of embolism remain unknown. The prognosis seems worst when a large vessel occlusion is included, even if endovascular clot retrieval is performed. Usually, there is no specific treatment, and patients care is mostly supportive. Algorithms exist but none of them has been clinically approved and the syndrome is largely clinical. Progress in imagery can be helpful to

determine the exact location and extent of *the* damages. Unfortunately, no prevention treatment has been found either. Attentiveness and responsiveness remain the cornerstone of the handling of this syndrome and every clinician must be aware of its existence.

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Informed consent statement: Not applicable.

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