doi: http://dx.doi.org/10.11606/issn.1679-9836.v99i2p197-201 Rev Med (São Paulo). 2020 March-April;99(2):197-201.

Cerebral fat embolism in polytraumatized: case report

Embolia gordurosa cerebral em politraumatizado: relato de caso

Isadora Dias Lacerda[1], Josué da Silva Brito[2], Luísa Lobo Sousa[3], Talitha Araújo Velôso Faria[4], Wesley Lobo Costa Júnior[5]

Lacerda ID, Brito JS, Souza LL, Faria TAV, Costa Júnior WL. Cerebral fat embolism in polytraumatized: case report / *Embolia gordurosa cerebral em politraumatizado: relato de caso*. Rev Med (São Paulo). 2020 March-April;99(2):197-201.

ABSTRACT: Cerebral fat embolism (CFE) results from the release of fat globules into the bloodstream, mainly from long bone fractures. The presence of significant fat emboli in the blood can cause vascular occlusion and clinical manifestations depend on the location of the obstruction. In this work, we report a case of a young patient, victim of an automobile accident with acetabulum fracture and right femoral diaphysis, who showed a decrease in the level of consciousness. In view of the clinical picture, magnetic resonance imaging was requested, which showed results compatible with the diagnosis of CFE. After proper care for embolism management, the patient had a favorable evolution.

Keywords: Intracranial embolism; Embolism, fat; Multiple trauma; Case reports.

RESUMO: A embolia cerebral gordurosa é uma patologia decorrente da liberação de glóbulos de gordura na circulação sanguínea provenientes, principalmente, de fraturas de ossos longos. A presença de êmbolos significativos de gordura no sangue resulta em oclusão vascular e manifestações clínicas dependentes da localização da obstrução. O caso relatado apresenta um paciente jovem, vítima de acidente automobilístico com fratura de acetábulo e diáfise femoral direita, que evoluiu com rebaixamento do sensório. Diante do quadro clínico, foi solicitada ressonância magnética, que apresentou resultado compatível com o diagnóstico de embolia cerebral gordurosa. Realizado os devidos cuidados para tratamento da embolia, o paciente apresentou evolução favorável.

Descritores: Embolia intracraniana; Embolia gordurosa; Traumatismo múltiplo; Relatos de casos.

INTRODUCTION

Fat embolism (FE) consists of the presence of fat globules in the bloodstream, usually resulting from the exposure of yellow bone marrow in fractures of long bones, such as femur, tibia and pelvis bones, polytrauma and orthopedic surgery, such as knee and hip arthroplasty, which can cause occlusion of small vessels[1,2,3].

Zenker made the first description of FE in 1862, when he found fat droplets in the pulmonary capillaries of a railway worker who suffered fatal thoracoabdominal injuries. The description of clinical manifestations, however, was only made by von Bergmann, in 1873, who reported dyspnea, cyanosis and coma in a patient 60 hours after a femoral fracture. Two years later, Czerny associated

1. Atenas University Center. Orcid: https://orcid.org/0000-0003-1228-4621. Email: isadoradias_lacerda@hotmail.com.
2. Atenas University Center. Orcid: https://orcid.org/0000-0003-2142-3689. Email: josuedasilvabrito1998@gmail.com.
3. Atenas University Center. Orcid: https://orcid.org/0000-0003-4956-8089. Email: luisalobo10@outlook.com.
4. Atenas University Center. Orcid: https://orcid.org/0000-0002-4252-6794. Email: talithabio@yahoo.com.br.
5. Atenas University Center. Orcid: https://orcid.org/0000-0001-9485-5824. Email: Wesley.jr@hotmail.com

Correspondence: Isadora Dias Lacerda. Rua Alumínio, 1326, B. Esplanada. Paracatu, MG, BR. Email: isadoradias_lacerda@hotmail.com

fat embolism with brain manifestations[2,4-6].

The presence of fat emboli is common, being observed in 67% to 95% of patients who have suffered long bone fractures or major trauma. There are, however, few patients with emboli capable of causing clinical manifestations and complications [4,6]. The clinical incidence is less than 1%, but this is not a real incidence. Many cases are overlooked and undetected. In post-mortem examination, the incidence can reach 20%. The real incidence value is estimated to be between 0.25% and 33%; however, there has been an observed drop since 1970, when the first studies reported a clinical incidence of 20%. The reduction is attributed to early immobilization and fracture fixation. Lethality, in severe cases, is between 10% and 20%. This depends on the size of the embolus, the amount of droplets and the presence or absence of systemic involvement[1,5].

FE is more commonly found in patients with an average age of 30, young adults, since they are better able to survive high-energy trauma, a risk factor for the pathology. Patients younger than 12 years old hardly ever present the condition, since they have a greater preponderance of hematopoietic tissue in relation to yellow marrow and, also, a higher concentration of triolein in medullary fat[2,7].

It is conceived that they are risk factors for fat embolism: closed fractures, polytrauma — the main risk factor —, conservative therapy and delayed immobilization. Patients with higher glycemic status, reduced alpha-lipoprotein to beta-lipoprotein ratio, abnormal capillary fragility test, reduced cortisol levels or thrombocytosis are also in the increased risk group [2,4]. As for the initial treatment instituted for fracture and the development of FE, there may be no relation, according to a retrospective study with 272 patients with diaphyseal lesion of the femur, submitted to rest until the surgical procedure, external fixation, immediate definitive treatment or skeletal traction until definitive treatment, carried out by Silva et al.[7].

FE, according to Sevitt's clinical classification, can present systemic manifestations, which include tachycardia, neurological and respiratory disorders, fever and petechiae, when it comes to the classic syndrome, or, more commonly, only some of these symptoms, in the so-called partial or incomplete syndrome. There are three categories of clinical pictures in the incomplete syndrome: 1) without respiratory symptoms; 2) without brain symptoms; and 3) without significant brain and respiratory symptoms[8]. Fulminant manifestation is the most serious presentation, occurring mainly in cases with multiple and severe fractures[9].

CASE REPORT

Male patient, 19 years old, admitted to the emergency department after car trauma with excoriations on trunk and lower limbs with complaints of pain in the pelvic region and in the right lower limb. He was lucid, oriented, with a heart rate (HR) of 120 bpm, without neurological, abdominal or respiratory changes. Laboratory tests performed on the patient's admission revealed: hemoglobin (Hb) 16 g/dL; hematocrit (Ht) 46%; leukocytes 25,000/mm^3; platelets 293,000/mm^3.

Three hours after the first ones, new laboratory tests were requested: Hb 14.2 g/dL; Ht 45%; leukocytes 25,700/mm^3; platelets 256,000/mm^3. During orthopedic evaluation, a radiography of the pelvis and lower limbs was also requested, which showed fracture of the acetabulum and right femoral shaft. Splint immobilization was performed and he was referred for orthopedic admission after general surgery evaluation, which found that the patient did not present changes in the thoracic and abdominal exam, but was progressively less responsive.

Within 24 hours, the patient remained drowsy and confused, with no neck stiffness and no focal neurological signs. New laboratory tests were performed showing a decrease in the number of Hb (11 g/dL), Ht (34%) and platelets (187,000/mm^3), however, with normalization of the amount of leukocytes (12,000/mm^3). Skull and thorax computed tomography (CT) was performed. There were no changes.

In 48 hours, the patient evolved with significant psychomotor agitation and subsequent lowering of consciousness, in addition to increased HR, fever spikes and the appearance of petechiae on the trunk and neck, being accompanied by neurology. He was referred to the intensive care unit, where prophylactic antibiotic therapy (intravenous clindamycin 600 mg — IV — 6/6 h + ceftriaxone 1 g IV 12/12 h) was started and foci of abdominal hemorrhage were investigated by CT and ultrasonography (USG) of the abdomen, due to the progressive drop in the values of Hb (9 g/dL), Ht (27%) and platelets (140,000/mm^3).

Due to the drop in Hb to 8 g/dL, Ht to 24% and platelets to 130,000/mm^3, the patient underwent blood transfusion (600 ml of red blood cell concentrate), 72 hours after admission, and had significant improvement, except from drowsiness.

Awaiting the schedule of orthopedic surgery, the patient continued to be evaluated by neurology, who suspected, from the clinical picture and laboratory values, of cerebral fat embolism. He also had easy crying, mood swings, and repetitive movements. Magnetic resonance imaging (MRI) was performed, which proved to be compatible with the picture of cerebral fat embolism, revealing: extensive areas of signal change located in the periventricular and bilateral frontoparietal subcortical white matter and diffusion restriction, in addition to foci of marked hyposignal on the right side of the corpus callosum, which had a slight enhancement to the contrast medium.

As a form of treatment attempt, it was decided to use methylprednisolone, in addition to ventilatory support and an urgent request to correct the fracture, which is the focus of FE.

Surgery to fix the acetabulum and femoral diaphysis was performed on the same day as the diagnosis of cerebral fat embolism. The patient evolved significantly well, with complete improvement to the state of consciousness.

After 30 days of the preview external fixation procedure, with the fracture already aligned, osteosynthesis with reverse nail was performed. The patient evolved without clinical complications, being discharged from neurology and orthopedics, and referred to outpatient physiotherapy.

DISCUSSION

The term fat embolism is used interchangeably to refer to two entities that, although related, expose different clinical and pathological concepts: fat embolism, that is, the presence of fat emboli in the bloodstream, and fat embolism syndrome (FES), defined as the occurrence of injury and dysfunction of one or more organs, suggesting a complication or atypical evolution of FE[1,9-11].

Factors described as triggering go from arthroplasty and osteotomy of the femur, to extensive burns, soft tissue trauma and necrotizing pancreatitis. The most frequent factor, however, is trauma to the musculoskeletal system, particularly fractures of the femur and tibia[11].

In a long bone fracture, there is fragmentation of the parenchyma, damage to the vascular wall and increased tissue pressure — elements responsible for the appearance of fatty acids or free fat in the bloodstream or in organs such as the lungs, kidneys and brain, mainly characterizing FES[1,11-13].

Some mechanisms have been described by which fat emboli can injure other organs, in addition to the lung, through arterial circulation: 1st) through anatomical pulmonary arteriovenous microfistulas; 2nd) by the deformation of the fat droplet itself, which, assuming a more elongated shape, manages to cross the pulmonary capillaries; 3rd) by passage through the foramen ovale. Recent studies using transesophageal echocardiography have shown that, in about 20 to 34% of adult individuals, this foramen is still patent. In patients who present with foramen ovale, the fat microemboli cross from the right to the left heart, reaching the arterial circulation and noble organs such as the brain, triggering all the symptoms of FES[1,9,11]. Therefore, it was investigated the existence of a patent foramen ovale in the patient, which was excluded by echocardiography.

The pathogenesis of FE is not yet fully understood. For its development, two distinct but interconnected theories are postulated: the biochemical theory and the mechanical theory[1,10,11]. The biochemical theory alludes that changes in lipid metabolism, even without a previous fracture, lead to the emulsion of fatty acids and chylomicrons, which directly reach the pneumocytes, generating injuries and, therefore, ineffective gas exchanges[5,10]. The mechanical theory, the most widespread, suggests that drops of fat, coming from the intramedullary content of the bone after a fracture, occupy the blood microcirculation, reaching the lung, heart and, later, the brain. The number of emboli varies a lot, depending on the energy and extent of the trauma, the involved bone, the type of fracture, since the exposed fractures cause less FE and, consequently, FES[1,9,11,13].

FES does not install itself immediately after the trauma. There is a lucid, or latent, period of 12 to 24 hours. The time until the onset of symptoms depends on the type of trauma and severity. Clinical manifestations involve the acute fulminant, subacute and subclinical forms. Fulminant FES, more common in polytrauma patients and submitted to artoplasties — exposed to numerous fatty emboli —, is manifested by acute cor pulmonale, multiple organ failure, hydroelectrolytic disorders. It has the worst prognosis, causing death within a few hours after the trauma. Subacute FES, the most frequently reported form, presents the triad of symptoms represented by progressive respiratory difficulty, altered consciousness and/or behavior and cutaneous petechiae, which is a pathognomonic finding [1,5,10]. The subclinical form shows minimal changes, with no respiratory failure[5].

This case report is compatible with the subacute form of FES, starting with a nonspecific picture of tachycardia, hyperthermia and malaise, evolving with mental confusion and petechial rash in the trunk and neck. The symptoms resulting from brain lesions can simulate the existence of intracranial hematoma, however, magnetic resonance imaging showed cerebral fat embolism in the patient, enabling the best treatment approach[1,11].

FES has an eminently clinical diagnosis, with no laboratory and imaging exams that alone provide the diagnosis. Therefore, it is extremely important to investigate the main differential diagnoses (traumatic brain injury — TBI —, pulmonary thromboembolism — PTE —, and sepsis), which were ruled out by the clinical history and initial physical examination presented by the patient. The hypothesis of TBI was ruled out due to the absence of suggestive brain injuries. The suspicion of PTE could be ruled out by the absence of signs of acute cor pulmonale and the septic syndrome was disregarded by the rapid clinical and hematological improvement after blood transfusion and the absence of infectious foci[11].

FE, as an isolated phenomenon, does not necessarily produce symptoms when compared to FES, which is a pathophysiological consequence. Therefore, there are established criteria for the diagnosis of FES[11]. Gurd and Wilson's criteria classify the findings as major and minor. The major criteria are respiratory failure ($pO_2 < 60$ mmHg and $FiO_2 > 40\%$), central nervous system depression and petechial rash (petechiae in mucous membranes, anterior chest and neck). Minor criteria are tachycardia (HR > 120 bpm), fever (axillary temperature > 38 °C),

thrombocytopenia (platelets < 150 x 10^9/L), lipiduria, retinal embolism and unexplained anemia. At least one major and three minor or two major and two minor criteria are necessary to characterize the clinical picture[10,11,12]. In the present case, the patient had two major criteria (decreased level of consciousness and petechial rash) and three minor ones (tachycardia, fever peaks and thrombocytopenia), allowing the diagnosis of FES, corroborated by MRI.

Treatment is often empirical, with no specific measures proven in the case of FES[1,5,10-13]. Prevention and early diagnosis are the best interventions during the development of the syndrome. Prevention is based on the immobilization of initial fractures within the first 24 hours[10,12]. In this case, the patient underwent surgery for fixation of the acetabulum and femoral diaphysis after the critical time of 24 hours, allowing the presenting of FES.

Most of the treatment is supportive and consists primarily of oxygen supplementation, with a face mask, which was provided to the patient, or mechanical ventilation, associated with measures aimed at hemodynamic stabilization such as: correction of acid-base disorders, volumetric replacement and adequate nutritional support. Continuous monitoring of O_2 saturation is recommended, trying to keep it above 95%. Oxygen therapy has proven efficacy, but it does not act on the cause of the problem and cannot be seen as specific treatment[1,510,11].

There is also evidence that high-dose corticosteroids stabilize the membranes and reduce the patient's inflammatory response, reducing the risk of lung injury. It is not proven, though, that they are beneficial after the manifestation of FES. There is evidence, however, that they are beneficial in preventing the development of the syndrome, especially when FES is triggered after a fracture. The drug of choice is methylprednisolone up to 10 mg/kg, IV, in pulse therapy, which the patient underwent, resulting in remission of FES[1,5,10-14].

CONCLUSION

We presented a case of cerebral fat embolism resulting from a fracture of the acetabulum and right femoral diaphysis, characterized as a long bone fracture, which confirms the typical appearance of the condition.

FE is a condition of difficult diagnosis, which must be performed clinically, using, complementarily, imaging and laboratory tests in order to exclude differential diagnoses. When facing a long bone fracture, first, prevention is necessary, however, once the symptoms are installed, an accurate and rapid diagnosis together with the hemodynamic stabilization determine the patient's prognosis. Therefore, the physician must be able to recognize predisposing conditions and the different symptomatic spectra of fat embolism, thereby reducing complications.

Authors' Contributions: *Lacerda ID, Brito JS, Souza LL, Faria TAV, Costa Júnior WL* - conception study; *Lacerda ID, Souza LL, Costa Júnior WL* - data obtenting; *Lacerda ID, Brito JS, Souza LL, Faria TAV, Costa Júnior WL* - data analysis and interpretation; *Lacerda ID, Brito JS, Souza L* - study writing; *Faria TAV, Costa Júnior WL* - critical review of the study.

REFERÊNCIAS

1. Filomeno LT, Carelli CR, Silva NC, Filho TE, Amatuzzi MM Embolia gordurosa: uma revisão para a prática ortopédica atual. Acta Ortop Bras. 2005;13(4):196-208. doi: 10.1590/S1413-78522005000400010.

2. George J, George R, Dixit R, Gupta R C, Gupta N. Fat embolism syndrome. Lung India. 2013;30:47-53. doi: 10.4103/0970-2113.106133.

3. Zhou Y, Yuan Y, Huang C, Cheng X. Pathogenesis, diagnosis and treatment of cerebral fat embolism. Chin J Traumatol. 2015;18(2):120-3. doi: 10.1016 / j.cjtee.2015.03.001.

4. Akoh, CC, Schick C, Otero J, Karam M. Fat embolism syndrome after femur fracture fixation: a case report. Iowa Orthop J. 2014;34:55-62. Available from: https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4127739/.

5. Alves JD, Arantes LD, Magalhães EMS. Síndrome da embolia gordurosa: relato de caso. Rev Med Minas Gerais. 2009;19(1):63-6. Disponível em: http://www.rmmg.org/artigo/detalhes/491.

6. Godoy DA, Di Napoli M, Rabinstein AA. Cerebral fat embolism: recognition, complications, and prognosis. Neurocrit Care. 2017. doi: 10.1007/s12028-017-0463-y.

7. Silva JJAB, Diana DA, Salas VER, Zamboni C, Hungria Neto JS, Christian RW. Síndrome da embolia gordurosa na fratura diafisária de fêmur: o tratamento provisório faz diferença? Rev Bras Ortop. 2017;52(5):535-7. doi: 10.1016/j.rbo.2016.08.011.

8. Sevitt S. The significance and classification of fat-embolism. Lancet. 1960;276:825-8. doi: 10.1016/S0140-6736(60)91901-2.

9. Zhou Y, Yuan Y, Huang C, Cheng X. Pathogenesis, diagnosis and treatment of cerebral fat embolism. Chin J Traumatol. 2015;18(2):120-3. doi: 10.1016 / j.cjtee.2015.03.001.

10. Menéndez DFS, Sousa Júnior LM, Neville IS, Paiva WS, Andrade AF, Teixeira MJ. Embolia gordurosa encefálica. Arq Bras Neurocir. 2014;33(2):132-8. doi: 10.1055/s-0038-1626261.

11. Rangel Rivera DA, Fuentes Abreu S, Guerreiro Caballero Ju, Rodriguez Duran JC. Sindrome de embolismo graso con predominio de sintomas neurologicos. Rev Asoc Argent Ortop Traumatol. 2016;81(4):310-4. doi: 10.15417/595.

12. Ballesteros-Flores CG, Hernández HJ, Sánchez AHE,

Ávila-Romero AS. Embolismo graso y síndrome de embolismo graso. Rev Med Hosp Gen Mex. 2008;71(1):49-56. Disponível em: https://www.medigraphic.com/pdfs/h-gral/hg-2008/hg081i.pdf.

13. DeFroda SF, Klinge SA. Fat embolism syndrome with cerebral fat embolism associated with long-bone fracture. Am J Orthop (Belle Mead NJ). 2016;45(7):E515-E521. Available from: https://mdedge-files-live.s3.us-east-2.amazonaws.com/files/s3fs-public/ajo04511515e.PDF.

14. Silva DF, Carmona CV, Calderan TR, Fraga GP, Nascimento B, Rizoli S. The use of corticosteroid for the prophylaxis of fat embolism syndrome in patients with long bone fracture. Rev Col Bras Cir. 2013;40(5):423-6. doi: 10.1590/S0100-69912013000500013.

Received: August 26, 2018
Accepted: March 06, 2020