

Middle East Journal of Medical Case Reports ISSN: 2789-8660 (Print) & ISSN: 2958-2121 (Online) Frequency: Bi-Monthly DOI: 10.36348/merjmcr.2024.v04i04.001

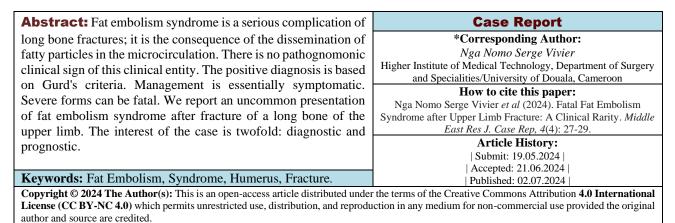


Fatal Fat Embolism Syndrome after Upper Limb Fracture: A Clinical Rarity

Nga Nomo Serge Vivier^{1*}, René Essomba¹, Kuitchet Aristide², Amengle Ludovic³, Binam Bikoi Charles³, Iroume Cristella³, Ngouatna Serge³, Djomo Tamchom Dominique⁴, Metogo Mbengono Junette⁵

¹Higher Institute of Medical Technology, Department of Surgery and Specialities/University of Douala, Cameroon
²Faculty of Medicine and Biomedical Sciences, Department of Surgery and Specialities, University of Garoua, Cameroon
³Faculty of Medicine and Biomedical Sciences, Department of Surgery and Specialities, University of Yaounde1, Cameroon
⁴Faculty of Health Science, Department of Surgery and Specialities, University of Buea, Cameroon

⁵Faculty of Medicine and Pharmaceutical Sciences, Department of Surgery and Specialties, University of Douala, Cameroon



1. INTRODUCTION

The fat embolism syndrome is a set of clinical, biological and radiological manifestations resulting from the obstruction of the microcirculation by fine droplets of insoluble fat [1, 2]. Zenker was the first to find the presence of fat in the lungs of a patient who died of polytrauma [3], in 1862, but it was Von Bergmann who in 1873 had the first clinical description of the disease a fat embolism syndrome [4]. This is a complication generally observed in the context of polytrauma including several long bone fractures. Its frequency is difficult to estimate, because there are no means to establish the diagnosis with certainty. We report here an unusual and fatal case of fat embolism syndrome occurring 24 hours postoperatively after a humerus fracture in an adult. The interest lies in the prevention of this clinical entity whose expression of severe forms is correlated with high mortality.

2. CASE DESCRIPTION

This was a 34-year-old patient admitted to a critical care unit for the management of acute respiratory distress with sudden onset in the early postoperative period. The first symptoms appeared a few hours before his admission to intensive care at the Essos hospital center, marked by the onset of acute respiratory distress, associated with signs of shock, 24 hours after

osteosynthesis, indicated for closed fracture of the left humerus. The mechanism of this fracture was a road traffic accident with a fall and landing on the left hemibody, 2 days previously, in an urban area. The patient was struck by a light motor vehicle traveling at high speed. After first aid at the district hospital in the area where the accident took place, the patient was taken to the Essos hospital center (reference health facility located 300 km from the accident site) via a non-medical transport. For this first aid, it mainly involved a brachioanterior-palmar brachio splint and the administration of non-morphine analgesics. The clinical examination at the emergency reception at the Essos hospital center (Yaounde-Cameroon) found abnormal mobility of the left arm and dermabrasions in the lower back region. Consciousness was clear, pleuropulmonary examination normal, vital parameters stable. The abdomen was supple, without defense or contracture. Standard radiography of the left arm showed images consistent with a multifragmentary fracture of the left humerus. The anesthesia consultation did not find any particular defects. The preoperative laboratory assessment was normal. The surgeon performed osteosynthesis using a screwed plate of the humeral fracture. The surgical procedure took place under general anesthesia and was well tolerated by the patient. Anesthetic recovery took place in the operating room and the patient was transferred to surgical hospitalization for further care.

Peer Review Process: The Journal "Middle East Journal of Medical Case Reports" abides by a double-blind peer review process such that the journal does not disclose the identity of the reviewer(s) to the author(s) and does not disclose the identity of the author(s) to the reviewer(s).

The occurrence of acute respiratory distress with desaturation in ambient air at 70%, associated with signs of shock (arterial hypotension, tachycardia, coldness of the extremities, mottling), on the first postoperative day, justified his transfer to the intensive care unit. The intensive care clinical examination revealed an altered general condition and poorly colored conjunctiva. Consciousness was obtunded, the pupils isochoric and isoreactive to light. There were no localization signs on neurological examination, nor petechial rash. Cardiac auscultation revealed irregular tachycardia at 180 beats per minute. On pleuropulmonary examination, breathing was superficial, presence of crackling rales at both pulmonary bases. The abdomen was still supple, the surgical wound dressing clean and dry. The calves were supple and painless when the feet were dorsiflexed. The rest of the somatic examination was unremarkable. The abdominal CT scan with contrast injection, performed urgently, was normal. Thoracic CT angiography showed bilateral alveolar syndrome. The electrocardiogram suggested a complete tachyarrhythmia due to atrial fibrillation (heart rate 176/minute). On transthoracic echocardiography, the ejection fraction of the left ventricle was preserved, the left ventricle hyperkinetic, the right cardiac chambers moderately dilated with minimal pulmonary hypertension. Furthermore, the inferior vena cava was slightly dilated on ultrasound exploration. Laboratory tests showed a d-dimer level of 7798 ng/ml, an ultra-sensitive troponin I of 1760 ng/l, a procalcitonin of 0.5 ng/ml and anemia of 8.5 g/dl. the level of D-dimer the clinical context of fracture of a long bone, the delay linked to surgery, and the association of radiological alveolar infiltrates, hypoxemia, tachycardia at 176/minute and polypnea at 33 cycles per minute allowed the diagnosis of post-traumatic fat embolism to be retained (Schonfeld score = 9).

Resuscitation treatment was essentially symptomatic, integrating oxygen therapy with a high

concentration facial mask at 12 liters/minute, rehydration with isotonic saline (2000 milliliters/24 hours), administration of catecholamines (Dobutamine 12 μ g/kg per minute and norepinephrine 0.3 μ g/kg/minute) by right subclavian central venous route, anticoagulation based on enoxaparin at a curative dose 6000 International Units/12 hours by subcutaneous route, multimodal analgesia (paracetamol, nefopam, tramadol, ketoprofen), the treatment of heart rhythm disorders using amiodarone and the prevention of stress ulcers by administering omeprazole IV in a single dose. A postoperative blood transfusion was necessary in order to optimize the hemoglobin level to 10g/dl. The outcome was fatal after 48 hours of intensive care in a situation of cardio-respiratory arrest, resuscitated without success.

3. DISCUSSION

Fat embolism syndrome is a rare and serious complication, mainly linked to trauma, involving one or more long bone fractures or revealed in the perioperative context of liposuction [1]. It brings together all the clinical (respiratory, neurological), biological and radiological signs secondary to the obstruction of microcirculation by fatty particles [5]. In 90% of cases, respiratory manifestations appear first [6, 7]. In the classic type of description, the clinical diagnosis is evoked in front of the triad of acute respiratory failure. neurological disorders and petechial syndrome [8]. These signs are used to describe diagnostic scores such as the Gurd (table1) or Schonfeld criteria [7-9]. The Gurd criteria, the most used to make the diagnosis of fat embolism (table 1), include major and minor criteria. The diagnosis is made based on the association of a major criterion and 3 or 4 minor criteria [10]. The prognosis for serious forms is reserved. The related mortality varies between 14 and 87% [11].

Table 1. Guru and Wilson's criteria	
Minor features	
Tachycardia >110/min	
Pyrexia > 38.5	
Retinal fat or petechiae	
Urinary fat globules or oligoanuria	
Sudden thrombocytopenia > 50%	
High Erythrocytose Sedimentation Rate > 71 mm/hour	

Table 1: Gurd and Wilson's criteria

In the case of our patient, the patient presented several risk factors for fat embolism. The diagnosis was considered on the basis of the major criteria (Hypoxemia PaO2 < 60 mmHg, FiO2 = 0.4) and 3 minor criteria (Tachycardia >110/min, oligoanuria, thrombocitopenia > 50%). There was no mucocutaneous involvement. The diagnosis is very difficult because the manifestations are variable and not very specific. Some tests can be useful, but cannot confirm the diagnosis with certainty. It is essentially based on the particular observation of clinical

signs by the practitioner. The literature on fat embolism shows that it is a diagnosis of exclusion. But it must be mentioned in the context of long bone fracture and look for other clinical signs. Our patient's symptoms were dominated by respiratory distress. Respiratory distress is the determining factor in mortality linked to fat embolism [1-13]. The neurological damage was characterized by clouding of consciousness. Respiratory manifestations (tachypnea, dyspnea, hypoxemia) are often the first signs to appear and they usually make the disease serious [13]. In more than half of cases, respiratory compromise leads to severe hypoxemia associated with respiratory failure which may require artificial ventilation. The notion of free interval is important to note. For our case study, the suggestive clinical signs appear on the 4th day after the trauma. The interval is characteristic of the condition. Its duration varies, from a few hours to a few days, with a median around the 24th hour [5-11]. The particularities of our clinical case are the occurrence of this complication after trauma to a long bone of the upper limb, and the association with a serious heart rhythm disorder that is poorly tolerated. The majority of cases described in the literature concern fractures of the long bones of the lower limbs [14]. Postoperative atrial fibrillation is common in the postoperative period of non-cardiac surgery [15]. It is responsible for an increase in postoperative morbidity and mortality. Its management consists of slowing the ventricular rate and anticoagulation [15]. Beta-blockers with a short half-life are recommended to slow the ventricular rate in order to improve myocardial energy balance when hemodynamic tolerance is good [16]. The healthcare team chose amiodarone to slow the heart rate and anticoagulation based on enoxaparin.

4. CONCLUSION

Fat embolism is a clinical entity that frequently occurs in the context of fractures of the long bones of the lower limbs, and rarely reported in fractures of the long bones of the upper limbs. The diagnosis of fat embolism remains above all a clinical diagnosis based on the history and the association of suggestive clinical manifestations. Its prevention is based on early and complete immobilization of the fracture site from the site of the accident until early and definitive surgical fixation. The prognosis for serious forms remains reserved

Declaration of Interest: No interests to be declared

REFERENCES

- Kawakami, D., Yoshino, S., Kawakami, S., & Yamakawa, R. (2022). Fat embolism syndrome. *Intensive care medicine*, 48(6), 748–749. https://doi.org/10.1007/s00134-022-06664-7
- Fukumoto, L. E., & Fukumoto, K. D. (2018). Fat Embolism Syndrome. *The Nursing clinics of North America*, 53(3), 335–347. https://doi.org/10.1016/j.cnur.2018.04.003
- Luff, D., & Hewson, D. W. (2021). Fat embolism syndrome. *BJA education*, 21(9), 322–328. https://doi.org/10.1016/j.bjae.2021.04.003
- 4. Von Bergmann, E. (1873). Ein fall von töldlicher fettembolie. Berl Klin Wochenschr, 10, 385-8.
- 5. Rothberg, D. L., & Makarewich, C. A. (2019). Fat Embolism and Fat Embolism Syndrome. *The Journal of the American Academy of Orthopaedic*

Surgeons, 27(8), e346 https://doi.org/10.5435/JAAOS-D-17-00571.

 Fabian, T. C., Hoots, A. V., Stanford, D. S., Patterson, C. R., & Mangiante, E. C. (1990). Fat embolism syndrome: prospective evaluation in 92 fracture patients. *Critical care medicine*, 18(1), 42–46.

e346-e355.

- Adeyinka, A., Pierre, L., & Fat, E. (2022). In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2023 Jan–. PMID: 29763060.
- Timon, C., Keady, C., & Murphy, C. G. (2021). Fat Embolism Syndrome - A Qualitative Review of its Incidence, Presentation, Pathogenesis and Management. *Malays Orthop J*, 15(1), 1-11. doi: 10.5704/MOJ.2103.001. PMID: 33880141; PMCID: PMC8043637.
- Bailey, K., Wesley, J., Adeyinka, A., & Pierre, L. (2019). Integrating Fat Embolism Syndrome Scoring Indices in Sickle Cell Disease: A Practice Management Review. *Journal of intensive care medicine*, 34(10), 797–804. https://doi.org/10.1177/0885066617712676
- 10. Gurd, A. R., & Wilson, R. I. (1974). The fat embolism syndrome. The Journal of bone and joint surgery. *British volume*, 56B(3), 408–416.
- He, Z., Shi, Z., Li, C., Ni, L., Sun, Y., Arioli, F., Wang, Y., Ammirati, E., & Wang, D. W. (2021). Single-case metanalysis of fat embolism syndrome. *International journal of cardiology*, 345, 111–117.

https://doi.org/10.1016/j.ijcard.2021.10.151

- Samaee, S., Samaee, S., Mihalca, D., Fitzgerald, L., Ahmed, A., Hall, J., & Tsitsikas, D. A. (2023). Mortality Rates and autopsy findings in fat embolism syndrome complicating sickle cell disease. *Journal of clinical pathology*, *76*(7), 497– 500. https://doi.org/10.1136/jcp-2023-208763
- Hilmo, J., Kayser, O., & Nielsen, E. W. (2017). Fettembolisyndrom [Fat embolism syndrome]. *Tidsskrift for den Norske laegeforening: tidsskrift for praktisk medicin, ny raekke, 137*(12-13), 896. https://doi.org/10.4045/tidsskr.17.0009
- Berdai, A. M., Shimi, A., & Khatouf, M. (2014). Le syndrome d'embolie graisseuse post traumatique [Post-traumatic fat embolism syndrome]. *Pan Afr Med J, 17, 83.* French. doi: 10.11604/pamj.2014.17.83.2062.
- Bessissow, A., Khan, J., Devereaux, P. J., Alvarez-Garcia, J., & Alonso-Coello, P. (2015). Postoperative atrial fibrillation in non-cardiac and cardiac surgery: an overview. *Journal of thrombosis* and haemostasis: JTH, 13 Suppl 1, S304–S312. https://doi.org/10.1111/jth.12974
- Royster, R. L., Deng, H., & Whalen, S. P. (2017). Postoperative Atrial Fibrillation. *Anesthesia and analgesia*, 125(1), 10–12. https://doi.org/10.1213/ANE.000000000002070.