Cardiac Arrest as an Uncommon Presentation of Fat Embolism

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Abstract

Fat Embolism Syndrome has been recognized for over 100 years but considerable controversy remains as to its incidence and clinical significance. We report here the case of a 38 years old female patient with several risk factors of fat embolism including fractured ribs, complex fracture of the humerus, morbid obesity, osteopenia and rough transportation. She presented a cardiac arrest with post mortem examination showing intra-cardiac fixation of fatty hematopoietic tissue. This is the first case report of such a presentation of fat embolism syndrome.

CASE PRESENTATION

A 38 year-old woman was admitted to emergency department in Tunisia following a road traffic accident. She was diagnosed four rib fractures and a distal complex closed fracture of the left humerus (Figure 1A,B). She had a history of morbid obesity (Body Mass Index 42 kg/m²), fibromyalgia and osteopenia since her last pregnancy. Operative treatment was indicated. Whilst intramedullary nailing of the humerus was being performed, she demonstrated sudden severe hypotension requiring vasopressive support. Surgery was immediately stopped. The patient was admitted with unfixed fracture to intensive care unit (ICU). On day 9, the patient was transferred to our ICU in France. She was still under mechanical ventilation but she was off vasopressor support. Completion of surgical repair was delayed because of local septic condition. On day 13, the patient was fully alert and ready to be extubated. She suddenly presented extreme bradycardia immediately followed by asystole. Mechanical cause of hypoxemia (accidental extubation, ventilation circuit, tube obstruction...) and electrolytic disorders were rapidly ruled out. After 25 minutes of unsuccessful cardiac pulmonary resuscitation, pulmonary embolism was suspected and a 50 mg intravenous bolus of alteplase was administrated. Five minutes later, she demonstrated return of spontaneous circulation. Post-cardiac arrest shock required continuous adrenalin infusion. Neither the electrocardiogram nor the Computed Tomography (CT) pulmonary angiogram showed any evidence of primary cardio-pulmonary cause of asystole. Cerebral CT scan ruled out cerebral cause and bacteriological investigations remained negative. Transthoracic echocardiography was difficult because of poor echogenicity but the intensivist who performed the examination did not notice any abnormality. Rapid hemodynamic recovery enabled to discontinue sedation on day 15. After a week, the patient remained comatose (Glasgow Coma Scale 3) and EEG recordings were flat. The dosage of benzodiazepine, barbiturics and morphine were negative. This clinical exam was compatible with brain death (secondary to cardiac arrest). Nevertheless, in the meantime the patient presented a ventilation-acquired pneumonia and a progressive refractory hypoxemia leading to her death on day 24 because of withholding of life sustaining therapies.

Post-mortem examination was performed on day 26. It showed no thrombotic pulmonary embolism but a 4 cm wide yellow engraftment of embolised marrow to the right auricular wall and tricuspid valve (Figure 1C). Microscopic examination showed blood coagulation material mixed with multiple nests of haematopoietic cells from the three lineages (Figure 1D). Microscopic examination did not mention any other evidence for fat or bone marrow deposition in other area.

Fat Embolism Syndrome (FES) has been recognized for over 100 years but considerable controversy remains as to its incidence and clinical significance. It has been reported as a clinical, biological and radiological syndrome due to Fat Embolism (FE) characterised by the release of fat into systemic circulation [1]. FE is found in approximately 90% of post-mortem examination in trauma patients [2], whereas only 5% of trauma patients demonstrate FES [1]. FE is mostly observed after trauma to the fat-containing bony...
derived conglomerate was hypothesised to be secondary to multiple fat and bone marrow embolism from ribs and humerus fractures and resulting coagulation activation [6]. The formed auricular thrombus was potentially blocking the tricuspid valve and leading to cardiac arrest. Our hypothesis is supported by the fact that spontaneous circulation was restored shortly after thrombolytic therapy. We cannot exclude that FE may have been caused by chest compressions of fractured ribs, but in that case, inaugural cardiac arrest would remain unexplained. We also think that early intraoperative severe hypotension observed in the OR operating room might have been the first symptom of FE. We did not find any skin rash or conjunctival signs of FES. Nevertheless, the pulmonary CT scan performed after the cardiac arrest showed bilateral pleural effusion with posterior passive consolidations associated to ground glass opacities predominantly on the right lung. This could be correlated to the clinical course of FES.

So far there are no specific treatments for FES. Heparin and corticosteroids have been proposed as treatments but have not reliably demonstrated improved morbidity or mortality. In case of unexplained cardiac arrest during the management of trauma patients, the hypothesis of FE has to be considered. As the differential diagnosis between FE and fibrino-cruoric embolism is clinically impossible, the treatment would be thrombolysis with the balance of all the underlying hemorrhagic risks.

CONCLUSION

We report here the first intra-cardiac fixation of fatty hematopoietic tissue with fatal outcome. Early fixation of fractures remains probably the best prevention for such life-threatening condition.

REFERENCES