International Journal of Surgery and Invasive Procedures
IJSIP, 7(1): 249-251
www.scitcentral.com

ISSN: 2640-0820

Case Report: Open Access

Fat Embolism Syndrome Following Trauma

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Received February 29, 2024; Revised March 04, 2024; Accepted March 07, 2024

ABSTRACT

Background: A young patient, managed as fat embolism syndrome. Proper diagnosis and multi-disciplinary management lead to successful recovery.

Case history: A 22-year-old male, presented following road traffic accident with long bone fracture. Deteriorated requiring invasive ventilation and ICU care, with clinical features of fat embolism syndrome. Fixation of fracture, prolong ventilation, tracheostomy, treating associated infection and supportive care lead to a successful outcome.

Conclusion: Fat embolism syndrome is composed of triad of neurological symptoms, respiratory distress and petechial rash. The management is primarily supportive, which can be prolonged. Proper diagnosis, multidisciplinary supportive care can lead to successful outcome.

INTRODUCTION

Fat embolism syndrome (FES) is a clinical diagnosis, composed of neurological symptoms, respiratory distress and cutaneous manifestations, which is seen after long bone fractures. Both biochemical and mechanical theories suggested to explain pathophysiology. There are several diagnostic criteria including Gurd's criteria. The management of FES is mainly multidisciplinary supportive care and definitive fixation of the fracture. Patients will often need prolonged care. Meticulous supportive care will lead to improved outcomes.

CASE HISTORY

A 22-year-old male patient, presented following a road traffic accident. Diagnosed to have isolated left tibial fracture with no other injuries, and the fracture was immobilized with a splint during initial management.

Six hours after admission to the ward, he desaturated to 90%, requiring oxygen. The GCS dropped to E2V1M5 (8/15). Urgent NCCT brain arranged and patient was transferred to ICU for further management.

As the GCS was low the patient required intubation and ventilation. Careful general examination revealed subcutaneous patches over upper thorax and conjunctiva. With Gurd's criteria, the diagnosis of fat embolism syndrome was made and supportive care continued.

Patient developed one episode of convulsions and required antiepileptics.

He underwent percutaneous tracheostomy on ICU day 7. MRI brain and the EEG were performed, and both findings were suggestive of fat embolism syndrome.

On ICU day 8 the internal fixation of the tibial fracture was done. The weaning of the ventilator was slow and took 15 days, and got complicated with ventilator associated pneumonia. With supportive care GCS slowly improved. When GCS was 10/15 and airway reflexes returned, tracheostomy was decannulated. The patient was safely discharged from ICU to ward on ICU day 20.

DISCUSSION

Fat embolism is presence of fat globules in peripheral and central circulations. When fat globules contribute to the triad of respiratory distress, petechial rash and neurological

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Citation: Vitharana HS & Somaweera S. (2024) Fat Embolism Syndrome Following Trauma. Int J Surg Invasive Procedures, 7(1): 249-251.

symptoms it is referred as fat embolism syndrome (FES) [1]. Diagnosis of fat embolism syndrome is challenging, which requires analyzing clinical symptoms, lab results and imaging. It is an essentially a diagnosis of exclusion [1,2].

Fat in the peripheral circulation is common finding. Around 68-82% of patients who died following trauma found to have fat in their pulmonary circulation. In orthopedic surgeries around 87% of patients' fat will be circulating in the heart, but only 0.9 % will manifest the clinical symptoms of FES [1]. Multiple fracture patients with femur fracture had the highest incidence of FES, which is 2.29% [1,3]. Our patient had a single fracture in the tibia which led to FES.

The first case related to fat embolism was reported in 1861, by Zenker. The patient was a railroad worker died from crush injury with fat droplets in lung tissue. After six decades, in 1920, the physiological theories of fat embolism were first proposed, mechanical theory by Gauss and biochemical explanation by Lehman and Moore. In 1970 Gurd presented first set of diagnostic criteria [2,3].

Two pathophysiological mechanisms were proposed for the development of FES, which are mechanical theory and biochemical theory [1-3]. When there is a long bone fracture, thin-walled veins, remain open. The negative pressure in the venous circulation will draw free fat globules into the circulation. During arthroplasty and intramedullary instrumentation, increased intramedullary pressure will push the fat globules into the circulation. These fat globules will travel through the heart via defect in the heart or via the venous shunts causing the cutaneous manifestations, increased pressure in the pulmonary circulation, and the central nervous system effects.

As fat float in the blood, the globules accumulate in the arch of aorta and distribute via the subclavian veins and carotid vessels. That explains the presence of cutaneous manifestations commonly in upper body, head and neck area, sparing lower body and the back [1-3]. These subcutaneous patches were visible in our patient in conjunctiva and chest. In biochemical theory, the inflammatory reactions are explained, which lead to clinical signs and symptoms, which causes 24-72 h delay in the presentation. The fat globules in the circulation start an inflammatory reaction. The fat embolized in lungs releases lipase, which break down to free fatty acid and glycerol. Free fatty acid causes endothelial cell damage, hemorrhage, and vasogenic edema. This releases proinflammatory cytokines, interleukins (IL-1and 6), which lead to ARDS. These reactions can agglutinate lipids into large molecules, which can occlude the vessels, giving rise to neurological deficits and skin petechiae. The bone marrow fat is prothrombotic, which activate the coagulation cascade, leading to thrombocytopenia and disseminated intravascular coagulation [1,2].

The patients present with classic triad of symptoms, respiratory distress, neurological changes and petechial rash, typically 24-72 h after the injury. Respiratory symptoms are dyspnea, tachypnoea, hypoxemia [2,3]. The neurological symptoms usually occur after the pulmonary symptoms. They are confusion, agitation, delirium, and can progress to hemiplegia, aphasia, seizures and coma. The petechial rash typically distribute over head, neck, thorax, axillae, subconjunctival space and oral mucosal membrane. Other symptoms and signs are tachycardia, hypotension, retinopathy, renal changes, fever, and coagulopathy. Our patient had classical initial presentation within 72 h of the injury with all subcutaneous, respiratory and neurological symptoms.

The diagnosis of FES is a challenge as there is no specific test. Gurd was the first to introduce a diagnosis criterion. This includes three major and nine minor criteria. At least one major and four minor criteria are required for the diagnosis [1-3].

The major criteria are respiratory insufficiency, cerebral involvement and petechial rash. The minor criteria are fever, tachycardia, retinal changes, jaundice, renal changes, anemia, thrombocytopenia, elevated ESR, and fat macroglobulinemia.

The Schonfeld criteria includes, six criteria, out of which five should present for the diagnosis. Those are petechia, chest radiographic changes, hypoxemia, fever, tachycardia and tachypnoea.

The Lindeque criteria have four components and presence of one indicate FES [2]. Those are sustained $PaO2<8kPa$, sustained $PCo2 > 7.3kPa$ or $Ph<7.3$, increased work of breathing, indicated by dyspnea, accessory muscle usage, tachycardia and anxiety.

There are no specific laboratory tests to diagnose FES. These will show evidence of anemia and thrombocytopenia. Elevated CRP, high ESR, high levels of inflammatory cytokines, hypoalbuminemia and fat macroglobulinemia in urine and saliva are some findings. Sometimes bronchoalveolar lavage can show lipid inclusions. Chest x-ray demonstrate bilateral diffuse or patchy opacities. Chest CT can reveal interlobar thickening (Crazy paving). CT brain shows diffuse edema and scattered hemorrhages which are not easily seen. The brain MRI will show multiple small hyperintense lesions known as starfield pattern, which was exhibited in our patient's MRI [1,2].

Treatment is primarily supportive maintaining oxygenation and ventilation, supporting hemodynamics, managing coagulopathy with blood and blood products, and fracture fixation. As there is a lack of direct treatment options, prevention is important.

For the management of the patients with FES, there are no definitive pathways, and it is essentially supportive. The

patients who are suspected to have FES, should be admitted to critical care setting. Continuous detailed monitoring of the physiological parameters and early invasive management in critical situations are essential. Hypoxemia is managed with oxygen supplementation, invasive and non-invasive ventilation when necessary, with lung protective measures [2]. Our patient required ICU admission, invasive ventilation and supportive care.

There are no specific data regarding management of cerebral fat emboli. The same principles as in managing brain trauma patients are applied in order to prevent secondary brain injury [1-3]. Seizure prophylaxis may be considered, which we used in our patient. Conservative management result in full neurological recovery in most of the cases. In some cases, decompression neurosurgery described. It took a longtime course for our patient to get the full neurological recovery. As a supportive measure he had to undergo tracheostomy for weaning from ventilator.

There are various drug regimens studied over past years including corticosteroids, heparin, hypertonic glucose, aspirin NAC and aliskiren. But none of them failed to give evidence for definitive therapy [2,3]. The most commonly used treatments are anticoagulation and corticosteroids. Our patient was on prophylactic anticoagulation and was not started on steroids.

Minimizing movement of fracture site by splinting, and expedited definitive fixation will reduce fat embolism and FES. During definitive management steps should be taken to prevent raised intra-medullary pressure during fixation. Therefore, external and internal plate fixation preferred over intramedullary nailing [1,2]. Our patient underwent fracture fixation as a definitive measure.

CONCLUSION

Fat embolism syndrome is composed of a triad, neurological symptoms, respiratory distress and petechial rash, caused by fat in circulation. The diagnosis is clinical by exclusion with support of diagnosing criteria. The management is primarily supportive, which can be prolonged. Proper diagnosis and multidisciplinary supportive care are key to success.

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