



Orthopaedics

A CASE REPORT ON SURVIVAL OF A POLYTRAUMA PATIENT WITH FAT EMBOLISM SYNDROME TREATED BY PRINCIPLES OF DAMAGE CONTROL ORTHOPAEDICS AND INTENSIVE CARE

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ABSTRACT In a patient with Polytrauma, the initial trauma (the first hit) and subsequent impact (a second hit) like a major open surgery, sepsis or an adverse event can lead to high risk of death. The principle of avoiding a second hit by minimum emergent surgical intervention to ensure return of stable physiological state in polytrauma is called "Damage Control Orthopaedics"(DCO). Severely injured patients are at risk of hypovolemic shock, Systemic Inflammatory Response Syndrome(SIRS) and Multiple Organ failure(MOF). This is a report of a patient with polytrauma and an impending fat embolism syndrome which was recognised very early and management based on the principles of DCO. It is essential to recognise early the ominous clinical signs which indicate such poor physiological and biological status. In such cases, the risk for further deterioration can be reduced by a carefully staged management of major fractures with initial external fixation and secondary conversion to definitive procedures which prevents the burden of the second hit which is life threatening to such patients.

KEYWORDS : Polytrauma, Fat Embolism Syndrome, Damage Control Orthopaedics, Injury Severity Score, External Fixator

Introduction

In 1861 Zenker was the first to describe Fat embolism in a patient with thoracolumbar injury.¹ In 1873, Ernst von Bergmann was first to make a clinical diagnosis of fat embolism in a patient who fell off a roof and sustained a comminuted fracture of the distal femur.² Fat Embolism Syndrome(FES) is most commonly associated with orthopedic trauma especially in multisystem injury or polytrauma patients, with highest incidence among closed, long bone fractures of the lower extremities, particularly the femur and pelvis.³ The term damage control was originally coined by the US Navy, in reference to keeping afloat a badly damaged ship by procedures to limit flooding, stabilize the vessel, isolate fires and explosions, and avoid their spreading. The same principle, named damage control orthopedics (DCO), was applied to the management of multi-injured patients with long bone and pelvic fractures.^{4,5}

DCO seeks to avoid provoking a severe inflammatory response and confines itself to more modest goals: sufficient stabilization of fractures to prevent further tissue damage and the potential compartment syndrome, while allowing the patient to be mobilized for tests and improved pulmonary care. Regarding the timing of definitive osteosynthesis, the period defined "window of opportunity" has been set between the 5th and the 10th days. The post trauma days 2 to 4 have been reported to be unsuitable for performing definitive osteosynthesis.⁶

Case Report

A 40 year old otherwise healthy man was brought to the emergency after being referred from another hospital following a Road Traffic Accident 7 hours prior with chest injury and closed injury to his left lower limb. On presentation the patient had altered mental status and respiratory distress. He had persistent drowsiness, tachycardia, tachypnoea and low SPO₂ in room air inspite of sufficient fluid resuscitation. The Xrays from outside hospital showed fractures of the left femur, tibia and fibula as well as chest injury (signs of left lung contusion and left 4th rib fracture) (Fig.1). Both FAST scan and CT brain did not show any signs of internal bleeding. His Injury Severity Score (ISS) was 12. He required 6 Litre of oxygen on flow via nasal cannula to maintain adequate oxygen saturation. There were conjunctival petechiae (Fig.2). The clinical picture was consistent with an impending Fat Embolism Syndrome. Principle of "DCO" was followed with a plan for two staged procedure. First stage Emergency stabilisation of both fracture femur and tibia were done using external

fixator (Fig.3). Patient was shifted to ICU for monitoring. Postoperatively IV antibiotics, IV fluids, blood transfusions and DVT prophylaxis were continued. By 5th postoperative day, patient was no longer drowsy and was well oriented to time, place and person. All his clinical parameters were within physiological limits. Patient was planned for Stage two definitive surgery in which External fixator of the femur was removed followed by Open Reduction and Internal Fixation of the femur bone with a Locked Titanium Plate (Fig.4). The external fixator of the tibia was retained as definitive treatment for 3 more weeks since it was an undisplaced tibia fracture. Postoperatively patient was stable and physiotherapy was started. Patient had tremendous overall clinical improvement and discharged after 5 days of further stay. We had achieved in saving the life of the patient by preventing complications of an impending fat embolism as well as treatment of the fractures.

Discussion

Fat embolism is most commonly associated with skeletal injury especially in patients with polytrauma. There are numerous studies which report the factors that increase the risk of FES development: young age, closed fractures, multiple fractures, and conservative therapy of long-bone fractures.⁷ The rate of mortality in patients with FES is about 5% - 15%.⁸ There are two theories which explain the process of development of FES in patients, a mechanical theory and a biochemical theory. According to the mechanical theory, FES occur when large fat globules enter the venous circulation resulting in the obstruction of the pulmonary vascular system. However, this theory cannot substantiate the delay in the development of symptoms.⁹ The biochemical theory suggests that hormonal changes after extensive trauma induce hydrolysis of triglycerides and release of free fatty acids, causing toxic endothelium damage in pulmonary capillary beds, as well as ARDS in animal models. In this theory, the time required to produce these toxic intermediaries explains the delay in development of symptoms. Despite the large number of studies supporting the involvement of these mechanisms in the development of FES, evidence is considered circumstantial.⁹

Among the reasons for difficulty in diagnosis of FES is the complication of widely different clinical conditions that may vary in severity. In our case, the diagnosis of FES was prompted on the basis of neurological symptoms, hypoxemia, tachycardia, petechial rash with no evidence of brain injury, sepsis, cardiogenic pulmonary edema or any cause of ARDS. The patient had three major and one minor criteria

of Gurd and Wilson classification to establish the diagnosis of FES.¹⁰

The treatment of fat embolism is only supportive and includes maintenance of adequate oxygenation, stable hemodynamic, normal blood levels, hydration, prevention of deep venous thrombosis and gastrointestinal bleeding and nutrition.¹¹ The timing and type of surgery for fractures constitute modifiable factors for the development of FES. Early stabilisation of femoral shaft fractures provides benefits for patients with multi-system-trauma. However, there is evidence in the literature suggesting that subsequent surgery after severe trauma acts as a "second hit" to the patient's system, effectively serving to worsen his or her situation. Therefore, the timing of definitive surgery in patients with multisystem trauma must be discussed – and carefully considered. Based in part upon the concept of avoiding this "second hit", "Damage Control Orthopaedics" (DCO) has become a popular procedure in severely injured patients – as it serves to provide early stabilisation, while also taking advantage of the benefits of definitive surgery for fracture union. Within this concept, the initial surgical trauma can be reduced by using external fixation. After recovery of the patient, most likely on an intensive care unit, external fixation is removed and plating/nailing is implemented to be in favour of stable osteosynthesis.

One of the most important issues in DCO is the timing of the secondary surgical procedures (definitive osteosynthesis). Many studies have shown that days 2, 3 and 4 are not safe for performing definitive surgery. During this period, marked immune reactions are ongoing and increased generalized edema is observed. A study demonstrated that multiply injured patients subjected to secondary definitive surgery between days 2 and 4 had a significantly ($P < 0.0001$) increased inflammatory response compared with that in patients operated on between days 6 and 8 after the primary diagnostic work-up.¹² In our case patient was diagnosed early and adequate symptomatic treatment was given followed by early stabilisation of the fractures and a final definitive surgery.

CONCLUSION

In summary, there is no specific therapy for FES; prevention, early diagnosis, and adequate symptomatic treatment are very important. Most of the studies in the last 20 years have shown that the incidence of FES is reduced by early stabilization of the fracture and prompt intensive care which in turn have saved the lives of many patients who presented as polytrauma or multiple injury patients in the emergency department.

Figures

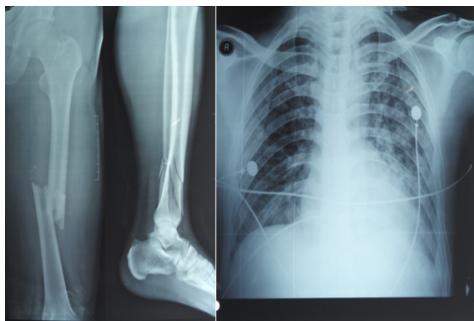


Fig1: Femur, Tibia and fibula fracture (Left) with Chest Injury



Fig.2: Conjunctival Petechial rash



Fig.3: External fixator Femur and Tibia



Fig.4: Definitive surgery (Plating Femur)

REFERENCES

1. Zhenker FA. Beitrage zur Anatomie und Physiologie de Lung. J Braunsdorf 1871. Dresden, Germany.
2. Fulde GW, Harrison P. Fat embolism - a review. Arch Emerg Med. 1991;8(4):233-239.
3. Gurd AR. Fat embolism: An aid to diagnosis. J Bone Joint Surg Br. 1970; 52:732–7.
4. Rotondo MF, Schwab CW, McGonigal MD, et al. 'Damage control': an approach for improved survival in exsanguinating penetrating abdominal injury. Journal of Trauma. 1993;35(3):375–383.
5. Shapiro MB, Jenkins DH, Schwab CW, Rotondo MF. Damage control: collective review. Journal of Trauma-Injury, Infection and Critical Care. 2000;49(5):969–978.
6. Nicola R. Early Total Care versus Damage Control: Current Concepts in the Orthopedic Care of Polytrauma Patients. ISRN Orthop. 2013 Mar 21; 2013:329452.
7. Dillerud E. Abdominoplasty combined with suction lipoplasty: A study of complications, revision and risk factors in 487 cases. Ann Plast Surg. 1990; 25:333–8.
8. Mellor A, Soni N. Fat embolism. Anaesthesia 2001;56:145-54.
9. Parker FB, Wax SD, Kusajima K, Webb WR. Hemodynamic and pathological findings in experimental fat embolism. Arch Surg. 1974; 108:70–4.
10. Gurd AR, Wilson RI. The fat embolism syndrome. J Bone Joint Surg Br. 1974; 56B:408–16.
11. Porpodis K, Karanikas M, Zarogoulidis P, Konoglou M, Domvri K, Mitrakas A, Boglou P, Bakali S, Iordanidis A, Zervas V, Courcousakis N, Katsikogiannis N, Zarogoulidis K. Fat embolism due to bilateral femoral fracture: a case report. Int J Gen Med. 2012;5:59-63.
12. Agrawal Alok Chandra, Kalia Roop Bhushan. Damage control in orthopaedic patients. J of Orth, Traum and Rehab. 2013; 6; 1: 23-27.