A Lethal Case of Fat Embolism Syndrome in a Nine-Year-Old Child: Options for Prevention

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Abstract

We report a lethal case of fat embolism syndrome in a nine-year-old child after a direct blunt trauma leading to a pelvic fracture. On the second day, signs of bowel perforation and septic shock led to an acute aggravation of the pulmonary symptoms, cardiac arrest and death. Fat embolism is seldom thought to occur in pediatric trauma patients; however, this case illustrates it can lead to disastrous sequela. Since there is no specific treatment for it, prevention by early fracture stabilization is the only option.

Key Words

Abdominal trauma · Fracture care · General trauma · IC treatment · Management of skeletal injuries in trauma · Pediatric trauma · Pelvic fractures · Trauma management and education

Eur J Trauma Emerg Surg 2009;35:311–3

DOI 10.1007/s00068-008-8141-6

Introduction

Fat embolism syndrome (FES) is a potential lethal condition threatening severely injured patients. Younger patients are especially at risk in the first 48 h after admission [1, 2]. Most children recover; however, complications from fat embolism can influence physiological compensation mechanisms that operate in response to shock, sepsis or pulmonary trauma. We describe a pediatric case in which trauma from a large impact led to a pelvic fracture with a lethal cascade of complications.

Case Report

A nine-year-old boy was referred to our tertiary trauma center after blunt injury to his abdomen and pelvis due to a falling tree. He presented with a patent airway and adequate breathing. Chest radiography was normal. Circulation improved after a fluid challenge. Abdominal ultrasound showed a retroperitoneal hematoma. His pelvis was tender, with a compression injury observed on plain X-ray (Figure 1). The dorsal ligaments of the sacroiliac joints were assumed to be intact, reflecting a type B lesion. Instability of the pelvis was limited. A T-Pod was applied to provide temporary stabilization. Head-to-toe examination revealed a bruise on the head, although an intact neurological outcome was obtained. He was awake and had a maximal Glasgow coma scale. Computed tomography scans of the pelvis and abdomen showed a dislocated pelvic fracture consisting of a left-sided ilium fracture and bilateral fractures of the superior and inferior frami of the pubic bone, retroperitoneal hematoma, intact retroperitoneal vessels, and no intraperitoneal free fluid or air. The pediatric trauma score was 11 and the injury severity score (ISS) was 29. The patient was admitted to the pediatric intensive care unit. Pelvic reconstruction was planned for the third day; however, the patient's condition deteriorated on the second day. He was in mild shock and complained of abdominal pain. Physical examination showed signs of peritonitis and the patient was transferred to the operating theater for laparotomy with the suspicion of bowel perforation. There was a rise in CO_2 level from 4.8 to 6.3 kPa. At laparotomy, a perforation of the terminal ileum was found, with only minor abdominal contamination. A wedge resection and

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Received: August 25, 2008; revision accepted: October 15, 2008; Published Online: November 26, 2008

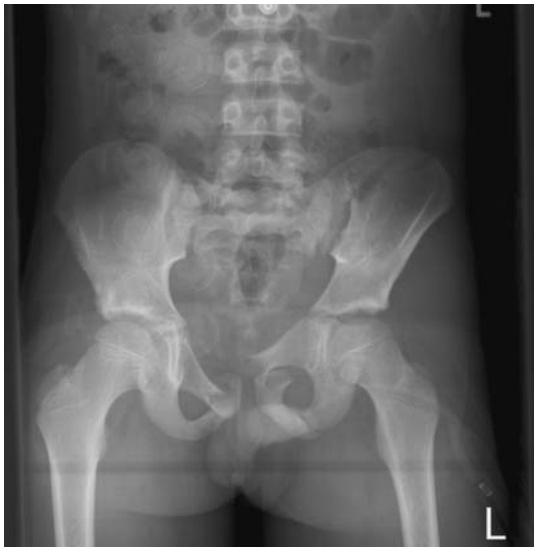

Figure 1. Plain X-ray of the pelvis, showing a dislocated pelvic ring fracture.

anastomosis was performed. After the wound had been closed and the boy transferred into bed and to the ICU, his oxygen saturation dropped and severe bradycardia occurred, necessitating cardiopulmonary resuscitation. Unfortunately, the CPR was unsuccessful and the boy expired. Autopsy showed massive bilateral pleural effusion and, on microscopic examination of the lung biopsy, signs of fat drops in the vessels (Figure 2). The diagnosis of FES was confirmed post mortem.

Discussion

The association between fracture of a long bone and respiratory insufficiency in the form of FES, fat globules in the lung with arterial hypoxemia, mental changes and skin or retinal petechiae was classically described by Peltier [3]. Clinical FES typically involves multiple organ systems; however, pulmonary, neurological, hematological and dermatological systems involvement is most common. Diagnostic criteria are proposed that consist of at least one major (respiratory insufficiency plus bilateral signs with positive radiological changes, cerebral signs and petechial rash) and at least four minor (tachycardia, pyrexia, retinal fat or petechiae, urinary fat globules or oligoanuria, sudden drop in Hg level, sudden thrombocytopenia, high erythrocyte sedimentation rate, fat globules in sputum) criteria as well as fat macroglobulinemia (Gurd's criteria) [4, 5]. In pediatric patients, the diagnosis of FES can be made when one of three signs are present: retinal embolism, positive skin, lung or kidney biopsy, or

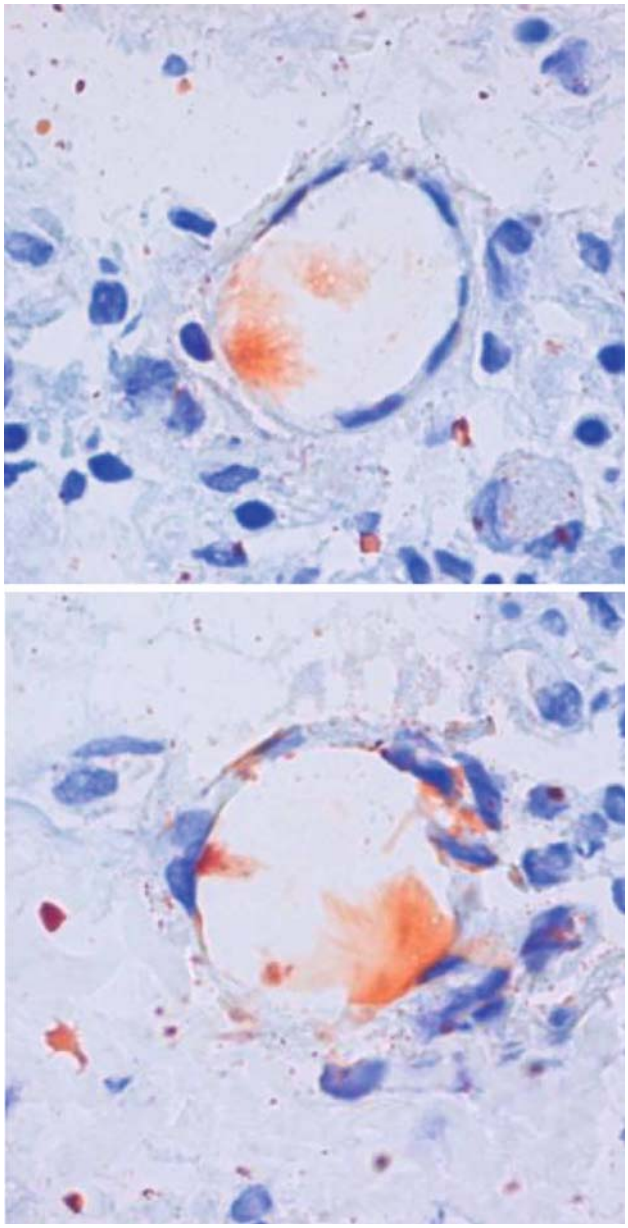

Figure 2. Microscopy (400× magnification) of the lung biopsy, showing fat droplets in the vessels (ORO fat stain).

histological findings at autopsy. The incidence of FES in children is said to be up to 100 times lower than in adults. It has been postulated that this is due to different compositions of bone marrow fat; children's bone marrow contains less olein and more palmitin and stearin, and more hematopoietic tissue than fat. However, the incidence could be higher if signs and symptoms were to be carefully sought [2].

The signs of fat embolism syndrome arise from fat globules liberated from the long bones, embolizing

primarily to the lungs. Smaller globules may pass through the lungs (and a patent oval foramen) and reach the systemic circulation. Movement of the ends of the fractures has been shown to result in showers of fat emboli in the circulation, and early fixation of the fractures is recommended. Rigid fixation soon after injury has been indirectly supported by three observations: (1) development of hypoxemia in patients with long-bone fractures not treated surgically; (2) occurrence of two separate episodes of FES in the same patient with multiple fractures; (3) reappearance of skin petechiae in ''crops,'' supporting repeated episodes of embolization [6]. Fixation can be achieved by damage control (external fixators) or definitive care (nailing or plating), depending on the clinical situation. Fat embolism can occur through the disruption, formation and release of thrombi from an injured, unstabilized limb. Delayed stabilization results in prolonged activation of the components of the systemic physiological stress response. Furthermore, the immobile supine patient is prone to atelectasis, pneumonia and reduced functional residual capacity, which causes shunting and impairs oxygenation. In the multitrauma patient, a delay of more than 24 h in pelvic or long-bone fracture stabilization after the injury increases the overall incidence of ARDS from 7 to 39% [7]. In patients with severe thoracic trauma, delaying stabilization of femur fractures led to an increase in pulmonary complications from 16 to 56% [8]. Bone et al. [9] showed in a randomized trial that early stabilization of femur fractures decreased the incidence of pulmonary complications. In our case, rigid stabilization of the pelvic fracture was postponed for three days. The fracture fragments in this unstable fracture probably moved when the patient was transferred from bed to operating table or when he was turned during washing procedures in bed, although an external stabilization device (T-POD) had been applied. Movement can result in fat embolism and even cardiac arrest [10]. In our case, the signs of FES were aggravated by the septic shock and pleural effusion and led to sudden cardiac arrest. Perhaps the instability of the fracture was underestimated, and bilateral traction on the abdominal wall during the operation increased the risk of FES.

A specific treatment for FES does not currently exist. FES is mostly self-limiting after fracture stabilization and the treatment is largely supportive. Adequate fluid resuscitation is necessary, and albumin is recommended for its fatty acid binding capacity. Treatment with high-dose corticosteroids may reduce the incidence of FES, but evidence is poor. The use of activated protein C or vena cava filters is still experimental. Therefore, prevention by early stabilization is the still the best option for FES [11–16].

References

1. Moore P, James O, Saltos N. Fat embolism syndrome: incidence, significance and early features. Austr N Z J Surg 1981;51:546–51.
2. Wiesz GM, Rang M, Salter RB. Posttraumatic fat embolism in children: review of the literature and of experience in the hospital of sick children. J Trauma 1973;13:529–34.
3. Peltier LF. An appraisal of the problem of fat embolism. Int Abstr Surg 1957;104:313–24.
4. Gurd AR. Fat embolism: an aid to diagnosis. J Bone Joint Surg (Br) 1970;52:732–7.
5. Talbot M, Schemitsch EH. Fat embolism syndrome: history, definition, epidemiology. Injury 2006;37:S3–7.
6. Tachacra SS, Sevitt S. Hypoxaemia after fractures. J Bone Joint Surg Br 1975;57:197–203.
7. Johnson KD, Cadambi A, Seibert GB. Incidence of adult respiratory distress in patients with multiple musculoskeletal injuries: effect of early operative stabilisation of fractures. J Trauma 1985;25:375–84.
8. Charash WE, Fabian TC, Croce MA. Delayed surgical fixation of femur fractures is a risk factor for pulmonary failure independent of thoracic trauma. J Trauma 1994;37:667–72.
9. Bone LB, Johnson KD, Weigelt J, Scheinberg R. Early versus delayed stabilization of femoral fractures. A prospective randomized study. J Bone Joint Surg Am 1989;71:336–40.
10. Van Miert M, Thornington RE, Van Velzen D. Cardiac arrest after massive fat embolism. BMJ 1991;303:396–7.
11. Taviloglu K, Yanar H. Fat embolism syndrome. Surg Today 2007;37:5–8.
12. Habashi NM, Andrews PL, Scalea TM. Therapeutic aspects of fat embolism syndrome. Injury 2006;37:S68–73.
13. White T, Petrisor BA, Bhandari M. Prevention of fat embolism syndrome. Injury 2006;37:S59–67.
14. Glover P, Worthley LI. Fat embolism. Crit Care Resusc 1999;1:276–84.
15. Parisi DM, Koval K, Egol K. Fat embolism syndrome. Am J Orthop 2002;31:507–12.
16. Bulger EM, Smith DG, Maier RV, Jurkovich GJ. Fat embolism syndrome: a 10-year review. Arch Surg 1997;132:435–9.

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