

Fat Embolism Syndrome Treated with Methylprednisolone: A Different Perception or a Misconception?

Mohd Shaffid Md Shariff¹, Hanizah Ngadiron², Firdaus Hayati³, Affiril Chairil Ariffin²

¹Orthopaedic Surgery, Universiti Sains Islam Malaysia, Negeri Sembilan, Malaysia

²Faculty of Medicine and Health Sciences, Universiti Sains Islam Malaysia, Negeri Sembilan, Malaysia

³Department of Surgery, Faculty of Medicine and Health Sciences, Universiti Malaysia Sabah, Sabah, Malaysia

*Corresponding author's email: firdaushayati@gmail.com

(Received: 20 June 2017; Accepted: 18 September 2017)

ABSTRACT

Fat embolism syndrome is manifested by the fat globule presence in the pulmonary and systemic circulation. A 34-year-old man was involved in a motor vehicle accident with a fracture of the left femur and avulsion fracture of the left posterior cruciate ligaments. He developed signs and symptoms that suggested an early diagnosis of fat embolism syndrome. Intravenous methylprednisolone administration was administered as part of the treatment. The role of methylprednisolone in a patient with fat embolism syndrome is controversial due to unproven effectiveness. In this case, fat embolism syndrome after a femur fracture was treated successfully with methylprednisolone.

Keywords: fat embolism syndrome, methylprednisolone, femur fracture.

INTRODUCTION

Fat embolism syndrome (FES) is often seen in association with long bone or pelvic fractures. It remains a diagnostic challenge for clinicians and surgeons. It is hard to diagnose, and the severity

of its consequences may vary. The incidence of FES varies and often underestimated by the physicians. Its clinical manifestation includes respiratory, cerebral dysfunction and petechial rash. Corticosteroid treatment in fat embolism is a topic of interest since 40 years ago.¹ Few studies had postulated regarding corticosteroid treatment before, but its outcome is arguable. Herein, a case of fat embolism syndrome after a long bone fracture treated successfully with methylprednisolone.

CASE PRESENTATION

A 34-year-old technician had skidded while riding a motorbike during a torrential downpour rain. He sustained closed fracture mid-shaft of left femur (Figure 1) with left posterior cruciate ligament avulsion fracture (Figure 2). On admission, his Glasgow coma scale was full. His vital sign was stable. There was no other injury noted. His left thigh was swollen but soft. Sensation of the left lower limb was present and distal pulses were palpable. Haemoglobin level on admission was 12.2 g/dl. Skin traction was applied.

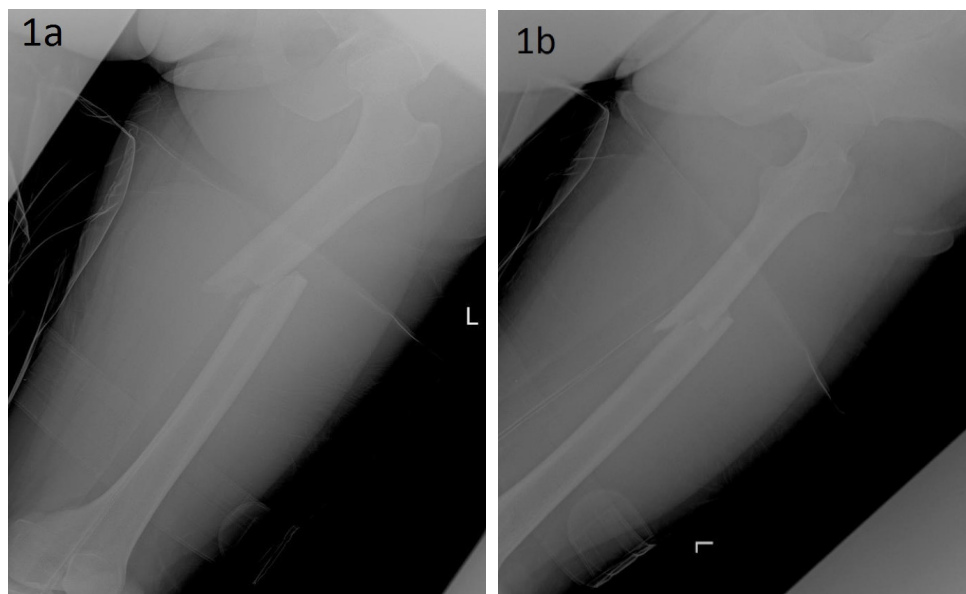


Figure 1 Radiograph AP (1a) and lateral view (1b) of left femur showing fracture of mid-shaft femur

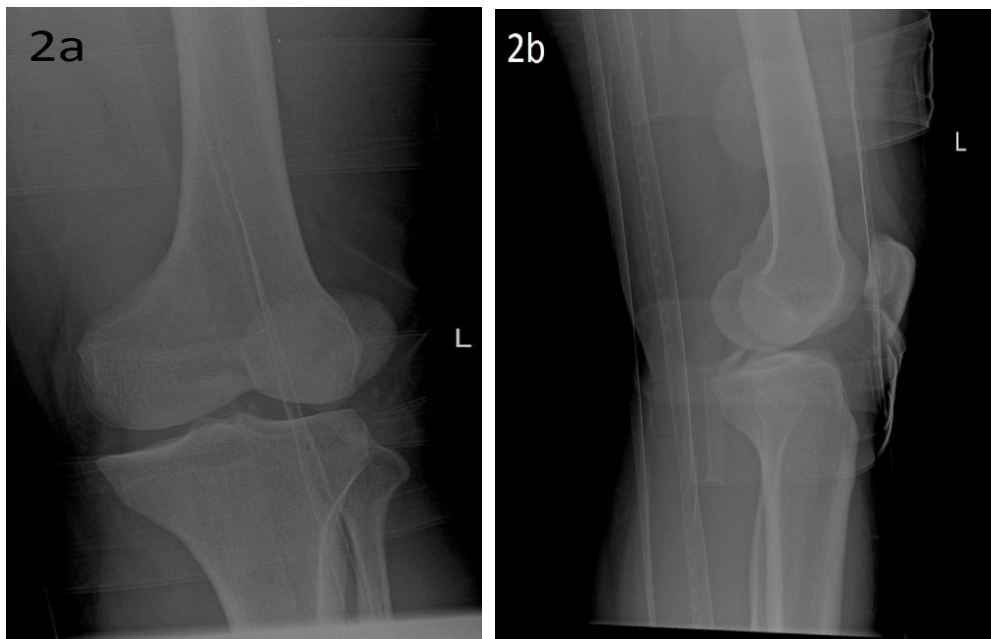


Figure 2 Radiograph AP (2a) and lateral (2b) of left knee showing avulsion fracture of the posterior cruciate ligament

A calcaneal pin was inserted after one day of trauma with a skeletal traction of 10% body weight. He was scheduled for an interlocking nail for left femur fracture and screw fixation for his posterior cruciate ligament avulsion fracture. However, at day two post trauma, he developed a spiking temperature of 38.5°C, tachycardia with

a heart rate of 105 beats/minute, tachypnoea with 20 breaths/minute and low SpO₂ under room air of 94%. Arterial blood gases (ABG) air showed respiratory alkalosis with renal compensation. ECG showed pulmonary embolism type of changes (Figure 3). Chest X-ray was clear. Repeated haemoglobin was 10.3 g/dL.

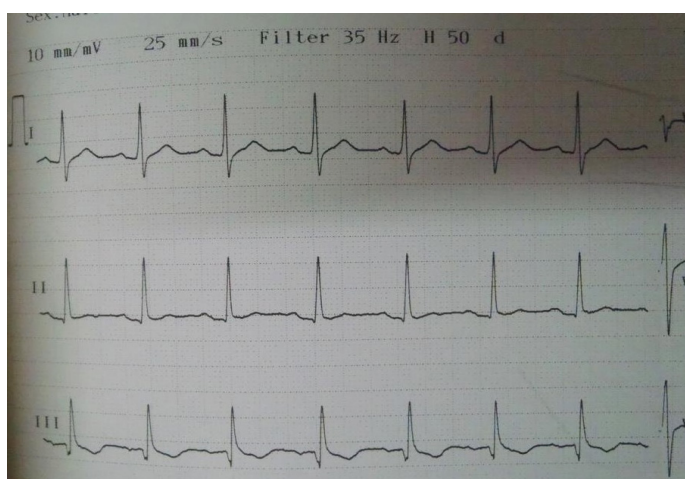


Figure 3 ECG showing S1Q3T3 changes suggestive of pulmonary embolism

Differential diagnoses at that period were fat embolism syndrome and pulmonary embolism secondary to either deep vein thrombosis or from fat globules. The patient was given 6-pint normal saline infusion, 2 pints

of packed cell transfusion and oxygen 3 litres via nasal prong. Computed tomography of pulmonary angiogram (CTPA) was performed with no evidence of pulmonary embolism.

Intravenous methylprednisolone was started with a dosage of 7.5 mg/kg every 8 hours for three days. Supportive management with adequate hydration and oxygenation was continued. The symptoms resolved after completed methylprednisolone dosage three days later. The patient underwent interlocking nail of the femur and posterior cruciate ligament repair. The surgery was successful and uneventful postoperatively. He was fit for discharged three days later.

DISCUSSION

The diagnosis of FES is very challenging. Gurd's Criteria (Table 1) is the best way to the diagnosis of FES.² The combination of two major or one

major with four minor criteria may point us to the diagnosis. FES actual incidence is undetermined due to the rarity of diagnosis.³ Compared to the upper limb, the association between lower limb long bone fracture and FES is more significant. This feature is possible due to the larger size and easy access to the vasculature.⁴ The patient presented with shortness of breath associated with fever. Few differential diagnoses may manifest with the similar signs and symptoms but given his age, injuries and symptoms, differential diagnoses were narrowed down into fat embolism syndrome and pulmonary embolism secondary to deep vein thrombosis and fat globules. Even though the ECG showed high suspicious of pulmonary embolism, however, the CTPA showed negative results.

Table 1 Criteria for the diagnosis of fat embolism syndrome according to Gurd²

| Major Criteria | Minor Criteria |
|---|---|
| <ul style="list-style-type: none"> • Petechial rash: axillary or subconjunctival petechiae • Respiratory symptoms: Positive radiographic changes; hypoxaemia (PaO₂ < 60 mmHg; FiO₂ < 0.4) • Cerebral depression disproportionate to hypoxaemia • Pulmonary oedema | <ul style="list-style-type: none"> • Tachycardia (>110 beats/min) • Pyrexia (>38.5) • Retinal fat or petechiae • Presence of urinary fat globules • Sudden drop in haemoglobin level or platelet values • High erythrocyte sedimentation rate • Fat globules in the sputum • Urinary incontinence |

The diagnosis of FES is relatively challenging and confusing. Its presentation is non-specific and posed a challenge in diagnosis. Gurd first describes this condition in 70's and was refined together with Wilson in 1974.²⁻⁵ Even though few authors argued regarding Gurd's criteria, however, it has become the tool of determining the diagnosis of FES in our setting. Our patient presented with respiratory symptoms, tachycardia, pyrexia, and sudden drop in haemoglobin. There were not enough criteria to fit into the diagnosis of FES. However initial presentation of FES needs to be considered.

The treatment of FES requires a close monitoring and respiratory support if indicated. Although the presence of Gurd's criteria may aid in the diagnosis, its universal usage is somewhat lacking. Despite the supportive treatment given, the patient's condition remains the same. He showed significant recovery after the administration of 12 dosages of methylprednisolone. The role of methylprednisolone in FES treatment has been debatable for the last decade. Although various studies describe the role of methylprednisolone in FES treatment, it is not used universally due

to the unverified effectiveness.¹ Its role has been investigated extensively in both animals and humans. Ashbaugh and Petty (1996) recorded a successful treatment of a massive respiratory failure secondary to fat embolism syndrome using corticosteroid.⁶ They postulated that corticosteroid causes stabilization of pulmonary capillary membrane and halts inflammatory response. The treatment also suggested a stabilization of complement system and platelet aggregation.⁷ One article showed a successful use of methylprednisolone as a prophylaxis against FES. The study revealed a protective effect in reducing the incidence of FES and hypoxaemia related complications.⁸ However, given the unwanted side effects of corticosteroid, the practice is not widespread in some locations. Among the effects include, it can progress to peptic ulcer disease, hyperglycaemia and poor wound healing. There was a case report whereby patients with long bones fracture developed atrial fibrillation after receiving intravenous methylprednisolone.⁹ Blood glucose should be monitored closely in these patients. Proton pump inhibitor would be a wise option to prevent any gastrointestinal side effects.

The recommended dosage in most literature is between 9 mg/kg and 90 mg/kg in divided dose. This dosage range is safe and postulated to provide maximum benefit. One series showed a 10-fold reduction of fat embolism syndrome episodes with methylprednisolone use.¹⁰ These findings should convince the medical world of its beneficial role. A meta-analysis of 389 patients using corticosteroids concluded a reduction of FES in traumatic patients up to 43%. However, the sample size is not adequate to show significant differences in mortality or infections with the administration of corticosteroids.

CONCLUSION

Methylprednisolone is an enticing treatment option in fat embolism syndrome. Although the role is debatable, its usage has been shown to be safe and efficient. A randomized trial would be ideal to evaluate its true advantage.

CONFLICT OF INTEREST

The authors declare that they have no conflict of interest in publishing this case.

CONSENTS

Written informed consent was obtained from the patient to publish the case. A copy of the consent is available with the Chief Editor.

REFERENCES

1. Bederman SS, Bhandari M, McKee MD, Schemitsch EH. (2009). Do corticosteroids reduce the risk of fat embolism syndrome in patients with long-bone fractures? A meta-analysis. *Can J Surg* 52 (5): 386 – 393.
2. Gurd AR. (1970). Fat embolism: An aid to diagnosis. *J Bone Joint Surg* 52 (4): 732 – 737.
3. Robert JH, Hoffmeyer P, Broquet PE, Cerutti P, Vasey H. (1993). Fat embolism syndrome. *Orthop Rev* 22: 567 – 571.
4. Shaikh N. (2009). Emergency management of fat embolism syndrome. *J Emerg Trauma Shock* 2 (1): 29 – 33.
5. Gurd AR, Wilson RI. (1974). The fat embolism syndrome. *J Bone Joint Surg* 56 (3): 408 – 416.
6. Ashbaugh DG, Petty TL. (1996). The use of corticosteroids in the treatment of respiratory failure associated with massive fat embolism. *Surg Gynecol Obstet* 123 (3): 493 – 500.
7. Gossling HR, Pellegrini VD. (1982). Fat embolism syndrome: A review of the pathophysiology and physiological basis of treatment. *Clin Orthop Relat Res* 165: 68 – 82.
8. Stoltenberg JJ, Gustilo RB. (1979). The use of methylprednisolone and hypertonic glucose in the prophylaxis of fat embolism syndrome. *Clin Orthop Relat Res* 143: 211 – 221.
9. Afsaneh-Vazin GR, Firoozifar M, Zand F. (2010). Intravenous Methylprednisolone, a Possible Cause of the Atrial Fibrillation. *Iranian Journal of Pharmaceutical Sciences* 6 (1): 13 – 18.
10. Kubota T, Ebina T, Tonosaki M, Ishihara H, Matsuki A. (2003). Rapid improvement of respiratory symptoms associated with fat embolism by high-dose methylprednisolone: A case report. *J Anesth* 17: 186 – 189.