Case report

Journal of the Intensive Care Society
0(0) 1–4

DOI: 10.1177/1751143717718664
journals.sagepub.com/home/jics

Neurological effects of fat embolism syndrome: A case report

Emma Shacklock[1], Andrew Gemmell[2] and Nigel Hollister[1]

Abstract

Fat embolism syndrome is a serious multi-system pathology which classically affects the respiratory system, neurological system and causes a petechial rash. We present the case of a 20-year-old farmer who developed fat embolism syndrome following a traumatic femoral fracture. Features developed within 24 h of injury and necessitated a prolonged stay in Intensive Care. He exhibited significant signs of cerebral fat embolism syndrome including coma and seizures but went on to make full functional recovery. Magnetic resonance imaging is the recommended imaging modality for patients with suspected cerebral fat embolism. In this case, computerised tomography was inconclusive, but magnetic resonance imaging demonstrated the "starfield pattern" of multiple high signal foci on a dark background. Supportive treatment of fat embolism syndrome is required in an appropriate setting, such as High Dependency or Intensive Care, for patients at risk of hypoxia or neurological deterioration. Despite major neurological involvement of fat embolism syndrome, full recovery is described by several cases including ours.

Keywords

Fat embolism syndrome, cerebral fat embolism, neurological fat embolism syndrome, starfield pattern

Introduction

Fat embolism is a virtually unavoidable consequence of long bone fractures. Between 0.9%–2.2% of these cases result in the multi-system pathology of fat embolism syndrome (FES).[1–4] The classical triad of signs described in FES are hypoxaemia, neurological impairment and petechial rash, which typically present 12–36 h after injury.[1,2]

Case presentation

A previously healthy 20-year-old male farmer fractured his right mid-shaft femur, after falling under the wheel of a tractor. He was admitted to hospital with an isolated injury to this limb and had no neurological impairment on presentation.

Twenty-four hours later, the patient presented hypoxic on arrival into the anaesthetic room for intramedullary nailing. His oxygen saturations were 83% on air, though he was fully orientated and in no respiratory distress. His low oxygen saturations were thought to be secondary to opiates and quickly corrected to 100% on pre-oxygenation.

Induction of anaesthesia was uneventful and a laryngeal mask was placed. Initially, oxygenation remained satisfactory, but during the final steps of nailing, there was an acute drop in saturations. The patient was intubated but continued to have a significant oxygen requirement, and the decision was made to transfer him ventilated to the Intensive Care Unit (ICU) post-operatively.

Oxygenation improved within 24 h; however, extubation was delayed due to failure to wake off sedation. Two sedation holds were abandoned due to biting on the endo-tracheal tube and no purposeful response to stimulation.

Five days post-operatively, a tracheostomy was performed to facilitate weaning from sedation. The patient was spontaneously opening his eyes but still making no motor response to pain. Reflexes were brisk, and bilateral clonus was elicited. Cerebral computerised tomography (CT) demonstrated subtle

[1]Department of Anaesthetics and Intensive Care, North Devon District Hospital, Barnstaple, UK
[2]Department of Radiology, North Devon District Hospital, Barnstaple, UK

Corresponding author:
Emma Shacklock, North Devon District Hospital, Raleigh Park, Barnstaple EX31 4JB, UK.
Email: emma.shacklock@nhs.net

bilateral focal areas of low attenuation within the basal ganglia, these were considered non-specific and the grey–white matter differentiation was normal. The patient was re-sedated, neuro-protective measures instated and methylprednisolone 120 mg administered three times daily for two days. Echocardiography showed no evidence of a patent foramen ovale.

The patient was able to localise to pain by day 10 following injury, but these promising steps were soon complicated by generalised seizures. Leviteracetam 500 mg was commenced twice daily for seizure prevention and given for five days. Electroencephalogram was not immediately accessible.

Magnetic resonance imaging (MRI) was able to give radiological confirmation that our patient's symptoms were the result of a neurological component of FES, see Figures 1 to 3.

Steady neurological recovery was made from this point onwards. On day 17, the patient was able to be decannulated and subsequently discharged from the ICU. Care continued on the local stroke unit and then a regional neurological rehabilitation unit.

One month after injury, the patient was discharged from hospital. He had made a good functional recovery and was continuing to improve. There remained subtle deficits in concentration, short-term memory and processing.

Discussion

No symptom or investigation is completely specific in this multi-system disorder. Hypoxemia is most commonly a feature of FES with one study reporting a $pO_2 < 9.3$ kPa in 90% cases. Petechial rash is seen in up to 60% cases. Neurological symptoms are also frequently seen but vary widely from minor alterations in mental state to hemiparesis, seizures and unconsciousness.[1]

Thrombocytopaenia and anaemia are commonly present. Our patient did require a transfusion of red blood cells but also developed a considerable haematoma, making this by no means diagnostic. Chest X-ray can exhibit patchy consolidation, typically in the middle and upper zones.[1]

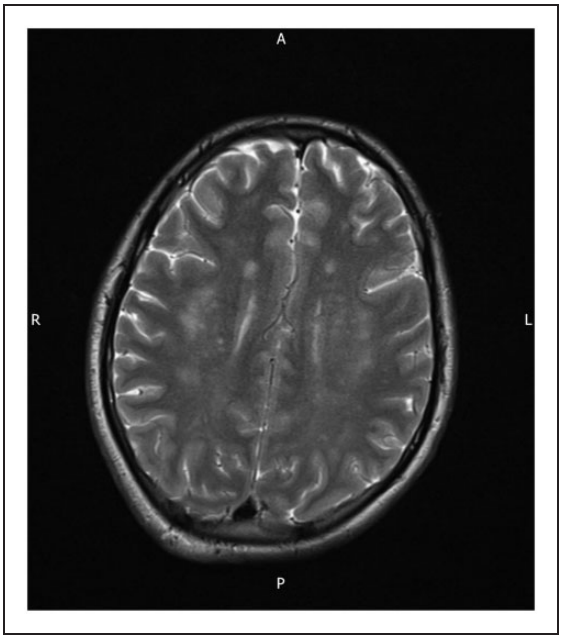

Figure 1. Axial T2 image demonstrates focal areas of high T2 signal change within the centrum semiovale of both cerebral hemispheres.

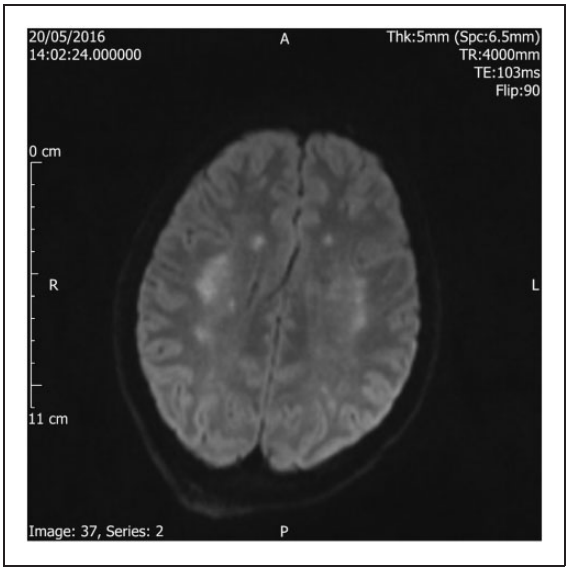

Figure 2. Axial diffusion weighted image demonstrates the "starfield pattern" of multiple high signal foci on a dark background. The lesions are more conspicuous than on T2 weighted imaging.

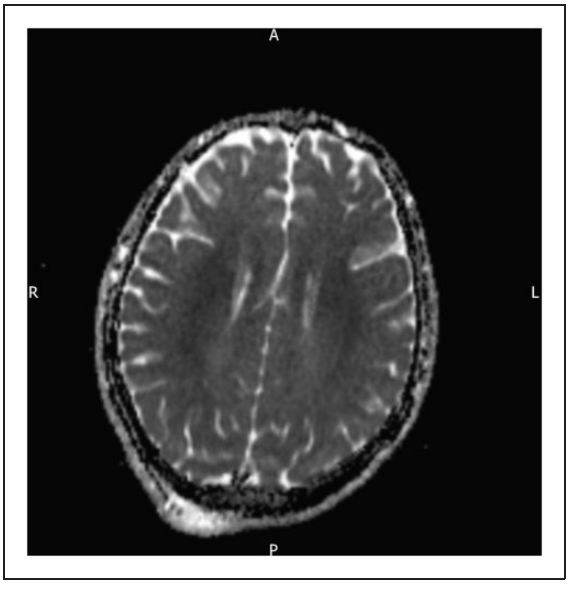

Figure 3. The corresponding ADC map demonstrates low ADC value lesions consistent with diffusion restriction.

Cerebral CT is often the first imaging performed in acute neurological dysfunction, being fast and easily accessible. CT in the acute phase may occasionally identify low attenuation areas of focal or generalised oedema and high attenuation focal areas of haemorrhage have also been reported.[5,6] It is, however, frequently unremarkable in cerebral fat embolism.[5]

MRI is more sensitive and is recommended as the initial imaging modality in patients with clinically suspected fat embolism.[5] Multiple focal areas of high T2 or fluid-attenuated inversion recovery signal within the white matter are frequently present as demonstrated in this case; lesions may also be present within grey matter.[6] Such lesions are relatively non-specific and may also be observed in demyelination, diffuse axonal injury and gliosis.[5] T1 weighted images have been reported as normal[7] but may also demonstrate focal low signal lesions and occasionally high T1 signal lesions consistent with haemorrhagic infarct.[8]

It is important that diffusion weighted sequences are included in all cases of suspected fat embolism. The "starfield pattern" of high signal foci on a dark background, as demonstrated in this case, has previously been reported to enhance the sensitivity and specificity of fat embolism.[2,7] The pathology in this case exhibited abnormal low value on the corresponding apparent diffusion coefficient (ADC) map consistent with restricted diffusion, reflecting acute ischaemia and cytotoxic oedema. Similar findings of diffusion restriction have previously been reported in cases of cerebral fat embolism imaged in the acute phase.[2] Cases imaged at a later stage may fail to exhibit diffusion restriction as a result of T2 shine through resulting from vasogenic oedema.[7]

The pathophysiology of FES remains unclear. Fat emboli may be forced into the venous system, which is supported by ultra-sonography showing most emboli occurring at times of high intramedullary canal pressures such as reaming.[1,2] Surgical strategies could potentially be employed to reduce these chances or load of emboli. External fixation or plate fixation has been shown to produce less pulmonary insult, as has the use of unreamed nails or intramedullary lavage. Reducing length of time to surgery to within 24 h also seems to be beneficial, reducing pulmonary complications and hospital stay.[1]

A patent foramen ovale may account for the more unusual presentation of FES, appearing to cause systemic emboli without pulmonary involvement.[1] Some emboli may be small enough to reach the systemic circulation without being caught by the pulmonary vasculature.[1,3]

Other theories of FES mechanism include the histotoxic effects of free fatty acids produced during inflammation, altering capillary permeability and causing cytotoxic oedema and ischaemic changes visible on MR imaging.[1,9] These changes on imaging seem to be closely linked to clinical severity and subsequent studies have been able to demonstrate their resolution by several weeks post insult.[2–4]

Treatment of FES remains supportive. Up to 44% patients require mechanical ventilation but the effects on respiratory function appear to be self-limiting and resolve within 3–7 days.[1]

Corticosteroids have been shown to have beneficial effects for the prevention of FES and hypoxia by a 2009 meta-analysis but evidence is inconclusive as to whether they are indicated for treatment.[10]

Fortunately, even significant cerebral FES appears to be reversible and full recovery possible, though one case with multiple lower limb fractures took a period of six months to regain full functional status.[2–4,9,11,12] The mortality of FES is less than 10% in the context of supportive care.[4]

Conclusion

Cerebral involvement of FES can be severe. These patients require recognition and supportive management in an appropriate setting, such as High Dependency or Intensive Care. If available, MRI is key for the diagnosis of cerebral FES and correlates with clinical presentation. Outcome can be favourable even in the context of a poor Glasgow coma scale and seizure activity many days post insult.

Consent

Consent for publication has been obtained from the patient.

Declaration of conflicting interests

The author(s) declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

Funding

The author(s) received no financial support for the research, authorship, and/or publication of this article.

References

1. Mellor A, and Soni N. Fat embolism. *Anaesthesia* 2001; 56: 145–154.
2. Parizel PM, Demey HE, Veeckmans G, et al. Early diagnosis of cerebral fat embolism syndrome by diffusion-weighted MRI (Starfield Pattern). *Stroke* 2001; 32: 2942–2944.
3. Chen JJS, Ha JC, and Mirvis SE. MR imaging of the brain in fat embolism syndrome. *Emerg Radiol* 2008; 15: 187–192.
4. Aravapalli A, Fox J, and Lazaridis C. Cerebral fat embolism and the "starfield" pattern: a case report. *Cases J* 2009; 2: 212.
5. Stoeger A, Daniaux M, Felber S, et al. MRI findings in cerebral fat embolism. *Eur Radiol* 1998; 8: 1590–1593.
6. Chrysikopoulos H, Maniatis V, Pappas J, et al. Case report: post-traumatic cerebral fat embolism: CT and MR findings. Report of two cases and review of the literature. *Clin Radiol* 1996; 51: 728–732.

7. Eguia P, Medina A, Garcia-Monco JC, et al. The value of diffusion-weighted MRI in the diagnosis of cerebral fat embolism. *J Neuroimaging* 2007; 17: 78–80.
8. Finlay ME and Benson MD. Case report: magnetic resonance imaging in cerebral fat embolism. *Clin Radiol* 1996; 51: 445–446.
9. Lin KY, Wang KC, Chen YL, et al. Favourable outcome of cerebral fat embolism syndrome with a Glasgow Coma Scale of 3: a case report and review of the literature. *Indian J Surg* 2015; 77: 46–48.
10. Bederman SS, Bhandari M, McKee MD, et al. Do corticosteroids reduce the risk of fat embolism syndrome in patients with long-bone fractures? A meta-analysis. *Can J Surgery* 2009; 52: 386–393.
11. Sethi D, Kajal S, and Saxena A. Neuroimaging findings in a case of cerebral fat embolism with delayed recovery. *Indian J Crit Care Med* 2015; 19: 674–677.
12. Scarpino, et al. Cerebral fat embolism after video-assisted thoracic surgery. *Ann Thorac Surg* 2016; 102: e409–e411.