CASE REPORT

Spinal cord infarction as a rare complication of fat embolism syndrome following bilateral intramedullary nailing of femur fractures

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SUMMARY

Fat embolism syndrome (FES) is a rare and potentially fatal complication occurring most often after long bone or pelvic fractures and orthopaedic procedures. It can consist of pulmonary, central nervous system and cutaneous manifestations. The exact pathophysiology of emboli reaching the arterial circulation is poorly understood.¹ It is suggested that this may occur by either 'paradoxical' embolism or microembolism.^{2,3} Its true incidence is unknown but increases in the presence of multiple closed fractures. It can be a diagnostic dilemma for clinicians and if suspected diffusion-weighted MRI is the modality of choice for the investigation of the central nervous system.⁴ We present the case of a 22-year-old man who developed multifocal cerebral infarcts, a right-sided cerebellar infarct and an infarct in the anterior cord bilaterally at the level of C5–C6 as a result of FES.

BACKGROUND

While the presence of cerebral microemboli in the absence of a right to left shunt is documented in the literature, there are no reports of emboli to the spinal cord. It is important to highlight the fact that this may occur as part of fat embolism syndrome (FES), especially in a trauma patient.

CASE PRESENTATION

A 22-year-old male patient presented to hospital following a road traffic accident. He was the restrained driver of a car travelling ~100 km/hour that collided with a stationary car. Airbags deployed at the time of the collision. On arrival to the emergency department he had no signs or symptoms of head injury, and a normal neurological examination and he had a Glasgow Coma Scale (GCS) of 15. Plain radiographs demonstrated multiple fractures including bilateral transverse mid-shaft femoral fractures, a distal fibula fracture and displaced mid-shaft clavicle fracture. A CT scan of his thorax, abdomen and pelvis demonstrated multiple rib fractures with a fracture of the xiphisternum and no evidence of pulmonary contusion. His cervical spine was cleared clinically and radiologically using plain radiographs. He was transferred to the high dependency unit (HDU) prior to surgery.

His medical history was unremarkable apart from his elevated body mass index (BMI) of 34 kg/m². He also smoked 10–15 cigarettes per day.

Prior to surgery, he had a heart rate of 117 bpm, a respiratory rate of 28 breaths/min, his oxygen saturation was 93–95% on 2 L nasal prongs (FiO₂ 28%) and his blood pressure was 159/85 mm Hg and he was non-feverish. Arterial gas at the time showed a pH of 7.35, pCO₂ of 5.29 kPa and pO₂ of 17.9 mm Hg. He was prescribed morphine via a patient-controlled analgesia pump using the standard hospital protocol while awaiting surgery.

In the absence of pulmonary contusions on CT scan of the thorax he was deemed fit for early total care for his femur fractures and underwent bilateral retrograde reamed femoral nailing 16 hours postadmission. Retrograde nailing was performed in view of his body habitus and the presence of bilateral fractures. The canals were reamed to 12.5 mm and an 11 mm locked T2 femoral nail was inserted bilaterally. Reaming nails were inserted to allow a greater diameter nail to be inserted to prevent implant failure. Full weight bearing would be required postoperatively owing to bilateral fractures and there was increased risk of implant failure due to his increased BMI. The procedure was technically straightforward and reduction was achieved by closed means. A below knee slab was applied for his fibula fracture.

He had a general anaesthetic and bilateral femoral nerve blocks (20 mL 0.25% levobupivacaine bilaterally). Intraoperatively he received morphine 20 mg, paracetamol 1 g and diclofenac 50 mg. He did not receive any steroid. In total he received 4 L of crystalloid intravenous fluid. He was haemodynamically stable intraoperatively with heart rate between 100 and 110 bpm and was normotensive throughout. There were no difficulties with ventilation or oxygenation.

Postoperatively he was drowsy in the postanesthetic care unit with a GCS of 12–13. On return to the HDU, he remained drowsy. His clinical state did not change following administration of intravenous naloxone. An urgent CT scan of the brain was arranged to investigate his symptoms. A non-contrast CT scan of the brain demonstrated an evolving right-sided cerebellar infarct and no other intracerebral abnormalities were detected.

On return from the radiology department, he had a further fall in his GCS to 9; he had divergent gaze and was unable to follow commands. Missed cervical spine instability with perioperative cord and/or vertebral artery injury was considered and immobilisation of the cervical spine was reinstated. He was not compliant for a neurological

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examination. He had some spontaneous movement in his right upper limb and responded to painful stimuli to all four limbs. His plantar reflexes were going down bilaterally and there was no evidence of clonus. He was transferred to the intensive care unit (ICU) where he was intubated and ventilated.

An urgent MRI of the brain and the cervical spine and MR angiography (MRA) of cerebral vasculature was performed which demonstrated an acute focal area of infarction in the right cerebellar hemisphere (figure 1) as well as infarctions in both cerebral hemispheres anteriorly (figure 2), in keeping with a shower of emboli.

In the spinal cord at the level of C5–C6, symmetrical areas of high signal were demonstrated in the anterior cord suggestive of infarction of the spinal cord (figure 3). There was no abnormal

Figure 1 Right-sided cerebellar infarct.

Figure 2 Multifocal cerebral infarcts.

Figure 3 Spinal cord infarct at the level of C5–C6.

signal identified in the soft tissue ligaments, as well as no areas of disc protrusion. Both vertebral arteries and the intracranial circulation were intact and patent.

In the ICU, he had a transoesophageal echocardiogram and bubble study for the investigation of the potential source of emboli, which did not demonstrate any communication between the right and left circulation, a normal aortic bifurcation and no evidence of dissection.

There was no evidence of a petechial rash and ophthalmic examination was unremarkable.

On day 2 in the ICU, a bronchoscopy was performed which demonstrated emulsification on bronchiolar-alveolar lavage, and then separation into two distinct layers signifying possible pulmonary fatty infiltrates. His chest X-ray did not demonstrate any abnormalities. Blood work did not demonstrate a thrombocytopenia.

He remained in the ICU for six more days, receiving supportive management. During the course of his stay in the ICU he had elevated temperature. During sedation holidays in the ICU, movement in all four limbs was elicited. He had an uneventful extubation on day 8 and was discharged to the HDU and then to the orthopaedic ward. Examination of his neurological system did not demonstrate any motor or sensory deficits.

He spent another 4 weeks in the hospital where he underwent physiotherapy and rehabilitation.

On review in the outpatient clinic 4 months after the accident he was well with no evidence of neurological deficit.

INVESTIGATIONS

Initially a CT scan of the brain was chosen to investigate the cause of the fall in GCS and drowsiness.

On the basis of the findings of an evolving cerebellar infarct and no other explanation for the deterioration an MRI of the brain and C-spine as well as an MRA of cerebral vasculature were performed. This is the gold standard to investigate for FES. Other possible injuries such as a vertebral artery dissection or a spinal cord injury could also be assessed with this modality of imaging.

On the basis of the findings of multiple focal emboli, a transoesophageal echocardiogram was performed. This was to assess the presence of an abnormal communication between the right

and left sides of the heart. A bubble study was performed which was negative.

DIFFERENTIAL DIAGNOSIS

- ▶ Traumatic intracerebral haemorrhage
- ▶ Dissection of aorta with clot emboli
- ▶ Epidural haematoma
- ▶ Spinal cord injury
- ▶ Vertebral artery dissection
- ▶ Embolic stroke

TREATMENT

The initial decision to perform intramedullary nailing was taken by the operating surgeon. The decision was made for early total care as there was no evidence of pulmonary contusion preoperatively on CT scan. If there was a severe chest injury external fixation of both femurs could have been considered as part of damage control approach. Consideration was also given to the type of intramedullary fixation. Previous studies have suggested that an unreamed nailing and venting can reduce intramedullary pressures.⁵ In our case, however, venting in the subtrochanteric area may have led to a periprosthetic fracture and there would have also been difficulty with access owing to increase muscle bulk proximally. Venting is much easier in antegrade nailing as it is easier to access the distal metaphyseal and there is a lower risk of fracture due to the increased circumference of the femur in that area. Another disadvantage of antegrade nailing is that it increases operative time as the patient has to be set up again on the traction table for the contralateral surgery. Also, in this case his body habitus also makes antegrade nailing more difficult. The use of an unreamed nail would have dramatically decreased the diameter of the nail putting him at risk for implant failure due to his high BMI and the requirement for full weight bearing in the early postoperative period due to the bilaterality of the femoral fractures.

Regarding the treatment of his FES this patient was intubated and ventilated and received supportive cares in the ICU due to the fall in his GCS and the findings on CT scan of the brain. He was ventilated with high positive end-expiratory pressure (PEEP). For his initial fluid management, albumin 20% was used.

There was consideration for the use of high-dose steroids; however, it was felt that the benefit from steroids would have been earlier in the course of his illness. The use of heparin was confined to prophylactic low-molecular-weight heparin.

From other case reports,^{6–9} we know that the most common course of this illness is resolution with conservative and supportive management. In the case of our patient, the cerebral and the spinal infarcts appear to have followed this course.

OUTCOME AND FOLLOW-UP

He has some subjective personality changes as reported by his family and some mild intermittent confusion that resolved over the course of his stay. He also had dysidiadochokinesia on the left in keeping with the right-sided cerebellar infarct. On review in the orthopaedic outpatient clinic 4 months after the accident he was well with no neurological deficits. Owing to the resolution of his symptoms, no follow-up MRI was required.

DISCUSSION

First described in 1873, FES remains a rare but potentially fatal complication of long bone and pelvic fractures, associated with closed rather than open fractures. It is often a diagnostic dilemma for clinicians as it may vary in presentation and

complicate other clinical conditions. The incidence of FES in a single long bone fracture is ~1–3%, increasing with increased number of fractures.¹⁰

Several mechanisms have been proposed relating to the pathogenesis of FES. ‘Mechanical’ fat embolism proposes that when a bone is fractured fluid fat is mobilised and enters into the disrupted venules from the trauma at the site.¹¹ ‘Toxic’ injury to the endothelium occurs following the release of fatty acids from the fat globules.¹² The vascular damage is aggravated by platelet activation and recruitment of granulocytes. This can occur in addition to mechanical embolism. It is postulated that FES depends on the breakdown of embolised fat into toxic intermediaries.

It can be divided into pulmonary and systemic emboli. Movement of the emboli from the venous circulation into the arterial circulation can occur by two mechanisms. Paradoxical embolism happens when there is a connection between the venous and arterial systems such as a patent foramen ovale. In the absence of paradoxical embolism, it is thought that fat emboli enter the arterial circulation via microembolism. This occurs when the emboli are small enough to pass from the venous to arterial circulation through the lungs.

Clinically FES can be a triad of pulmonary, cerebral and cutaneous symptoms, presenting as hypoxia, neurological abnormalities and a petechial rash.

Cerebral FES is a rare complication, and usually occurs after pulmonary manifestations. It can present as confusion, altered level of consciousness, seizures or focal deficits. These usually present within 24–72 hours following the initial injury.¹³ This is best investigated with an MRI of the brain.

There have been no reports of emboli to the spinal cord in the literature, but it is presumed that the fat embolises to the spinal arteries in the same mechanism as the other vessels.

In this case there was only radiological evidence of this embolus; however, the usual course of these emboli is transient and reversible and the patient was intubated and sedated for 8 days.

Learning points

- ▶ Rare cause of neurological deficit in a polytrauma patient.
- ▶ High index of suspicion with a decline in Glasgow Coma Scale following nailing of femoral fractures.
- ▶ Potential for emboli to present in the arterial supply to spinal cord.
- ▶ MRI is the modality of choice for suspected fat embolism syndrome. Investigation of venous–arterial shunt is important.
- ▶ Symptomatic management and supportive measures are mainstays of treatment.

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Contributors RK and CM were responsible for the care and management of the patient in the intensive care unit. RK, JG and DD wrote the manuscript. DD and JG managed the orthopaedic care of the patient. JG and CM revised the manuscript.

Competing interests None declared.

Patient consent Obtained.

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