Injury Extra 40 (2009) 147–148

Contents lists available at ScienceDirect

Injury Extra

journal homepage: www.elsevier.com/locate/inext

Case report

A good outcome following cerebral fat embolism

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ARTICLE INFO

Article history:
Accepted 5 March 2009

1. Introduction

Fat embolism syndrome (FES) is a rare but recognised complication of long bone fractures, with a reported incidence of 0.9–2.2% in this patient group.[7] It is often under-diagnosed, since the classical signs of pulmonary, central nervous system, and cutaneous manifestations may not occur.[3]

The outcome of FES is variable, although the prognosis can be improved with early diagnosis and intensive respiratory care.[1,8,12] The diagnosis can be difficult, and while there are published criteria to aid in diagnosis, these are not standardised.[3,5] MRI has been reported to have value in the early diagnosis of FES,[7] with a close relationship between clinical manifestations and MRI appearances.[12]

We present a patient with an acute and profound neurological presentation of FES in whom early supportive treatment led to a complete recovery, with diagnostic confirmation using brain MRI.

2. Patient

A 17-year-old male motorcyclist was admitted following a collision with the side of a car. He sustained a closed multi-fragmentary diaphyseal femoral shaft fracture, and a closed contra-lateral multi-fragmentary diaphyseal tibial fracture. On arrival in the emergency department he was alert and orientated with a Glasgow Coma Score (GCS) of 15. He was haemodynamically stable, eupnoeic, and apyrexial. Secondary survey revealed no other injuries. Five hours after arrival, he underwent general anaesthesia for the application of a bridging AO external fixator (Synthes, 20 Tewin Road, Welwyn Garden City, Hertfordshire, AL7 1LG) to stabilise the tibial fracture before reamed intra-medullary nailing of the contra-lateral femoral fracture (Expert Femoral Nail, also Synthes). After an uncomplicated 2 h procedure, in the presence of continued physiological stability, anaesthesia was reversed and the patient was returned to the orthopaedic ward.

He was reviewed at 11 h post-procedure when he was found to be well, comfortable (using patient controlled intravenous morphine), fully alert, with no signs of compartment syndrome.

During 14 h post-surgery he developed a sinus tachycardia, and became acutely unresponsive, with a GCS of 8 (eyes 3, verbal 2 and motor 3), though the only other physiological abnormality was a temperature of 38 °C. However, further deterioration was rapid to hypoxaemia and hypotension requiring transfer and management on the Intensive Care Unit. He was anaesthetised, his trachea intubated to enable invasive ventilatory support of his rapidly developing Acute Respiratory Distress Syndrome.

A CT of his brain was reported as being normal; the scan of his abdomen revealed a previously undiagnosed stable compression fracture of the body of the second lumbar vertebra. After 24 h in the ICU, an MRI scan of the patient's brain was performed since FES was suspected as the diagnosis. This revealed multiple foci of T2 hyperintensity in the deep white matter and ganglia consistent with small ischaemic foci (Fig. 1). There was no intracardiac shunt demonstrated on a transthoracic echocardiogram.

With clinical and radiological evidence of neurological injury and such significant difficulties with ventilation (high inspiratory pressures and inspired oxygen levels, bilateral pneumothoraces), the chances of his survival appeared extremely poor.

However, 9 days after admission, with physiological improvement, he returned to theatre for a tracheostomy and adjustment of the tibial external fixator. His sedation was stopped after this operation, to demonstrate dramatic neurological improvement, opening his eyes and moving all four limbs to commands. Despite his initial confusion and impaired short-term memory he was able to wean gradually from respiratory support and work with both the dieticians and the physiotherapists. On discharge from ICU 15 days after admission, he had made a full neurological recovery. His L2 fracture was managed with a Jewitt spinal brace and his bridging external fixator was converted to a Taylor-Spatial frame (Smith and Nephew, 15 Adam Street, London, WC2N 6LA).

3. Discussion

Fat embolism syndrome occurs when fat enters the circulation, causing a particular pattern of symptoms and signs. This is separate to fat embolism, where fat enters the circulation causing purely embolic sequelae. The most common cause is trauma, particularly long bone fractures, though other rare causes have been reported.[6]

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1572-3461/$ – see front matter
doi:10.1016/j.injury.2009.03.005

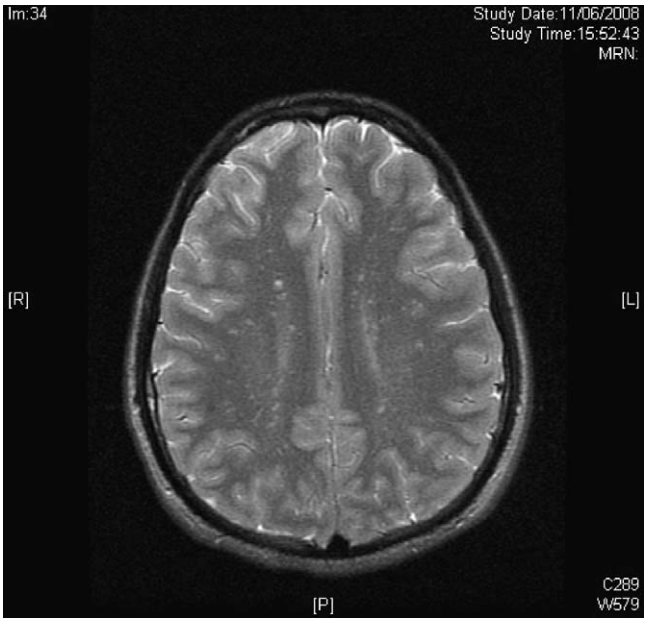

Fig. 1. An MRI of the patient, showing high intensity signal abnormalities in the deep white matter.

Table 1
Gurd and Wilson's criteria.

Major	1. Respiratory insufficiency
	2. Cerebral involvement
	3. Petechial rash
Minor	1. Pyrexial
	2. Tachycardic
	3. Retinal changes
	4. Jaundice
	5. Renal changes
Laboratory	1. Anaemia
	2. Thrombocytopaenia
	3. High erythrocyte sedimentation rate
	4. Fat macroglobulinaemia

There are no standards for the diagnosis of FES, and it can be mimicked by a number of other conditions, making diagnosis difficult.[6] Gurd and Wilson first described a collection of major, minor, and laboratory features in 1974 (Table 1).[3] The diagnosis of FES was suggested in our patient after he became unresponsive, tachycardic, and pyrexial, though the latter two signs were not of a magnitude to be included in Gurd's criteria, and indeed were only two of the required four minor features. However, Lindeque et al. suggested that FES could be diagnosed according to different criteria.[5] Given this patient's femoral and tibial fractures, the diagnosis would not have been made until 24 h after the initial symptoms when the patient's respiratory function deteriorated to give a PaO_2 of less than 8 kPa. Neither of these two criteria takes into account Magnetic Resonance Imaging.

Takahashi et al. have described how FES can be diagnosed on T2 weighted MRI, with diagnostically distinctive findings on T2 weighted images; high intensity signal abnormalities located in the deep white matter, basal ganglia, brain stem, and cerebellum, as found in our patient.[12] However, these haemorrhagic lesions are also consistent with diffuse axonal injuries, though Yanagawa et al. suggested that this type of injury could be ruled out due to the length of time between injury and onset of symptoms.[13] Takahashi et al. also proposed a grading system of the images, correlating with the clinical findings.[12] Fig. 1 depicts Grade 1 changes, which they suggested would be consistent with a good neurological outcome; however, clinically the patient still deteriorated, and thus was not expected to survive.

Treatment of FES is supportive, often requiring intensive care facilities. Habashi et al. described good outcomes after treating hypoxaemia and hypovolaemia, however, suggested that adjuvant therapies are as yet unproven.[4] Prednisolone administration with the aim of reducing the inflammatory response has been published as an effective treatment;[5,10,11] however, the number of patients in these studies were small, and further studies are need to determine the effectiveness of this treatment. Indeed, steroid use provokes alternate problems such as sepsis.

Heparin has also been suggested to increase lipase activity, but contradictory evidence exists to its efficacy.[2,9]

The outcome after developing FES remains variable, but good outcomes are achievable with early diagnosis and the supporting of pulmonary and cardiac functions.[1,6,8]

4. Conclusion

This case is of a patient who did not fit Gurd's triad or Lindeque's criteria of FES, but was managed as this. A later MRI confirmed the diagnosis, and with supportive therapy, the patient made a full recovery. We would recommend that FES is considered as a diagnosis even when all stigmata of the syndrome have not yet fully developed so that early treatment can be initiated.

Early MRI is advocated to confirm the diagnosis and allow appropriate counselling regarding potential neurological outcome.

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