Case Report

Increase in optic nerve sheath diameter predicts early cerebral involvement in fat embolism syndrome

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A B S T R A C T
Early diagnosis of cerebral fat embolism in a patient with contradiction to MRI is challenging. Here we report an interesting case, where the raised optic nerve sheath diameter helped us to predict the early cerebral involvement with fat emboli in a left femoral shaft fracture patient. MRI scan could not be performed due to the presence of a metallic implant in the patient from a previous surgery. He was later diagnosed as an atypical presentation of fat embolism syndrome. Optic nerve sheath monitoring also helped us to guide further management of the patient.

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Introduction

Fat emboli released into the blood after traumatic fracture of long bones sometimes leads to intensive care unit (ICU) admission and critical care management. Early diagnosis and management can be challenging in cases of atypical presentations of fat embolism syndrome (FES). In this case report, we discussed the importance of raised optic nerve sheath diameter (ONSD) as a tool to accurately detect and guide the management of cerebral fat embolism in FES.

Case report

A 21 years old male presented to the emergency department with left femoral shaft fracture after a motor vehicle crash. On presentation, he was conscious, oriented with the Glasgow coma scale (GCS) of 15. Initial management was done by the emergency team which revealed no associated comorbidity, stable vital parameters and normal blood investigation reports. After 4 h of admission, the patient developed altered sensorium, restlessness and confusion, and the GCS decreased to 12 (E3V4M5). Patient was then admitted to the ICU.

A working diagnosis of embolism was made. However, further investigations including Doppler ultrasound of the lower limbs, D-dimer values, CT pulmonary angiography, electrocardiogram, echocardiogram, fundoscopy and CT scan of the brain provided no evidence of the same. Urine fat globules were also negative.

ONSD was measured using bedside ultrasonography, which was recorded as 0.56 cm. After 12 h of ICU admission, the GCS of the patient further decreased to 8 (E2V2M4) and the ONSD increased to 0.67 cm (20% increase).

Mechanical ventilation was then decided as per the ICU protocols. Bedside repeat brain CT scan revealed normal result. Brain MRI was contraindicated because of a metallic implant placed to fix the traumatic right femoral shaft fracture in the past. On day 3, the GCS was 7 (E1V2M4) with a raised ONSD of 0.78 cm (Fig. 1). On day 4, the typical signs of FES started to reveal themselves in the form of bilateral subconjunctival petechiae (Fig. 2), bilateral cotton wool spots with retinal haemorrhages on fundoscopy and fat globules in the urine test. Blood investigations reported decreasing blood platelets.

To prevent further release of fat emboli, operative fixation of the femoral fracture was planned. Before surgical fixation, bedside brain CT was repeated after neurosurgical consultation in view of decreased platelet counts, which showed a normal finding and ruled out any fresh intracranial haemorrhage.

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Open reduction and internal fixation of the left sided femoral fracture was done under general anaesthesia with minimal intra-operative blood loss of 300 mL. There was no intraoperative complication. On day 5 of ICU admission, the GCS remained 8 with ONSD of 0.67 cm. The patient was gradually weaned from the ventilator. On day 7, GCS was 10 with a decrease of ONSD to 0.52 cm (22% decrease). Till day 10, the patient had normal GCS with an ONSD of 0.42 cm (19% further decrease in ONSD) (Fig. 3). After 15 days of total hospital stay, the patient was discharged.

Discussion

Cerebral involvement of the fat emboli in cases absent of typical clinical manifestations and investigational findings of FES remains a diagnostic dilemma for the clinicians. It is reported that 70%–86% of FES cases have neurological manifestations, like acute confusion, altered level of sensorium, dementia, focal deficits, seizures and coma, mostly appearing within 10 h but may sometimes present after 5 days. In our case the time of cerebral manifestation from injury was 8 h. It was the only manifestation in our case and did not fulfill the Gurd’s criteria to diagnose FES. Petechial rashes are seen in only 50%–60% of cases and appear within the first 36 h of injury. In our case report, however, multiple bilateral subconjunctival petechial haemorrhages was detected 84 h after injury.

We were able to rule out venous thrombus emboli after multiple investigations in the form of Doppler ultrasound of the lower limbs, D-dimer values, CT pulmonary angiography, electrocardiogram, echocardiogram, fundoscopy and brain CT. Although brain MRI remains the most sensitive imaging modality to diagnose cerebral fat embolism, it was contraindicated in our case as the patient had a metallic implant in-situ.

Bedside measurement of ultra-sonographic ONSD has proved its efficacy as early surrogate marker of raised intracranial pressure and helps the clinicians in diagnosing conditions with high suspicion of cerebral involvement.

After admission to the ICU, we measured the ONSD of the patient, which was 0.56 cm and increased in the subsequent days by 33% to 0.67 cm. During the same time the GCS dropped from 12 to 8. After diagnosing FES as per Gurd’s criteria on day 4, open reduction and internal fixation of the femur fracture was done to halt further release of fat emboli. The GCS remained low till the 6th day of ICU stay, along with raised ONSD. On the third post-operative day (7 days after ICU admission), GCS of the patient improved to 10 with 22% decrease in ONSD to 0.54 cm. In our case the raised ONSD indicated manifestation of cerebral fat embolism as other causes of this had been successfully ruled out by thorough investigations. An MRI scan could not be performed due to the presence of a metallic implant in patient from a previous surgery. Clinical improvement may be due to washout of the fat emboli from the cerebral tissue and reduction in the local tissue inflammation together with absence of further release of fat emboli from fracture site after operative fixation.

In conclusion, early diagnosis of raised intracranial pressure due to cerebral fat emboli in absence of typical presentation of FES is always challenging. A raised ONSD in such situation does not indicate a new cerebral insult but can help us in establishing the cause, guide our management approach and predict prognosis of the patient.

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Ethical statement

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient has given his consent for his images and other clinical information to be reported in the
journal. The patient understand that his name and initials will not be published and due efforts will be made to conceal his identity, but anonymity cannot be guaranteed.

Declaration of competing interest

The authors declare no competing interest.

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