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CASE SERIES

Cerebral Fat Embolism Syndrome Case Series

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ABSTRACT

Fat embolism is the presence of fat particles within the microcirculation, while FES is the systemic manifestation of fat emboli within the microcirculation. FES commonly presents with ards, altered mental status, and dermatological manifestations which occur commonly after traumatic long-bone fractures, though the incidence reported by varies studies range from 1%-33%. Here we discuss the two young patients presented with cerebral fat embolism syndrome. Cerebral FES may manifest from minimal changes in mentation to deep coma. Early suspicion in patients with no traumatic neuro parenchymal injury and MRI of brain will be helpful for the early diagnosis.

KEYWORDS

MRI of brain; Early suspicion; Fat embolism syndrome

CASE 1

A 21-year-old youngster after met with an RTA presented to tertiary orthopedic centre with fracture right leg. On admission he was alert and well oriented with GCS 15 and no neurological deficit noted [1]. After 7 hrs of presentation his GCS dropped to 4. He was intubated and ventilated for airway protection and respiratory failure and referred to our centre for further management. On admission to our centre his bp 120/70, PR - 130/min, spo2 97% with 60% fio2 and 8 peep, temperature 38 Deg, GCS 6 et/15, his chest X-ray showed bilateral infiltration. He had both bone compound fracture right lower limb for which Thomas splint was applied outside. He was oliguric and had high coloured urine [2]. Our initial suspicion was traumatic brain injury. As his CT brain was normal which doesn't explain his low GCS, he was done MRI brain after stabilization which showed Multiple scattered punctate t2/flair hyperintensities in cerebral and cerebellar hemispheres. His Cpk was 1050, CR was 1, his cardiac status was normal. He was resuscitated with iv fluids, IV Augmentin, anti-convulsant. He was done wound debridement after initial stabilisation on the day of admission. Next day he had worsening hypoxemia requiring fio2 60% and peep of 10. His Cxr showed worsening infiltrations. His procalcitonin was low. ET c/s not grown organisms. His platelets decreased to 1.2 lakhs, He had petechial rashes over the trunk and left conjunctiva.

On day 3 he had further drop in platelets to 1 lakh. On 4th day had drop in CPK, Platelets started improving. His chest infiltrations were started clearing and able to wean ventilator support though his neurological status was showing non improvement. At 7th day of hospitalisation, he underwent Orif tibia and surgical tracheostomy for ventilatory weaning. He was weaned from ventilator and shifted to step down unit in another couple of days. His neurological condition showing mild improvement of E2 m5 after 2nd week. During further follow up his GCS improved to E3M6, started obeying at the end of 6th week and he was weaned from tracheostomy [3,4].

CASE 2

A 17-year-old studying 12th grade was sustained with femur fracture after met with an road traffic accident in the middle of the night. He had no loc/vomiting/ altered mental status. Initially he was admitted in tertiary ortho centre where he was initially treated. He had right femur mid shaft closed fracture [5]. His CT abdomen showed grade 4 liver laceration, perinephric hematoma without hemoperitoneum. He was resuscitated with I.V fluids, Thomas splint and planned for surgical intervention for femur in the following day. When the patient was shifted to theatre after 8 hrs of presentation, found to have patient was in confused state and irritable. So, the procedure was abandoned, and CT brain was done which showed no neuroparenchymal abnormality. After that patient was referred to our tertiary care trauma centre for further management. On admission patient GCS was E2V2M5. Bp - 110/70, Spo_2 95% with 15 l O_2, chest on auscultation showed bilateral crackles, he was tachyonic with respiratory rate of 32/minutes blood gas analysis showed Dpo_2 of 65 mm hg, chest X-ray showed bilateral infiltration. He was intubated and ventilated. His echo cardiogram showed mild dilatation of right ventricle and normal lv function, he had oliguria and mild haematuria which improved after hydration. on admission his plat - 1.5 lakhs hb-10.8 gms%, sgot - 182 u/l, sgpt-227 u/l, cr-0.7 ptt-32 sec, inr-1.46. Next day his Hb dropped to 7.9 gms%, haematocrit dropped to 24.3% and Cpk - 2675 u/l, plat- 1,34000. cell. Third day further drop in Hb -7.5 gms%, plat-113000. Pt/inr-1, ptt-30 seconds sgot-57 u/l, sgpt87 u/l. t. bil1.18 mgs%. He was requiring ventilatory support of 40%/6 peep. His po2 was improving. He was given 2 units of packed cells. On 4th day patient has mild improvement in GCS and he was obeying simple commands. He was undergone ORIF FEMUR on 6th day. Patient was given trial extubation as his GCS was E3V3M6 and he was protecting airway and oxygenation was improved. He was observed in stepdown unit before shifting to ward. On 13th day patient was well oriented and GCS was 15/15, wound was healing well. Patient was discharged on 15th day with good neurological status [6-8].

MRI IMAGING

Multiple punctate haemorrhages in the brain due to FES (Figure 1 and Figure 2).

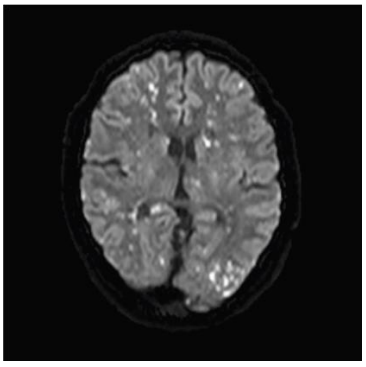

Figure 1: MRI Imaging. Multiple punctate haemorrhages in the brain due to FES.

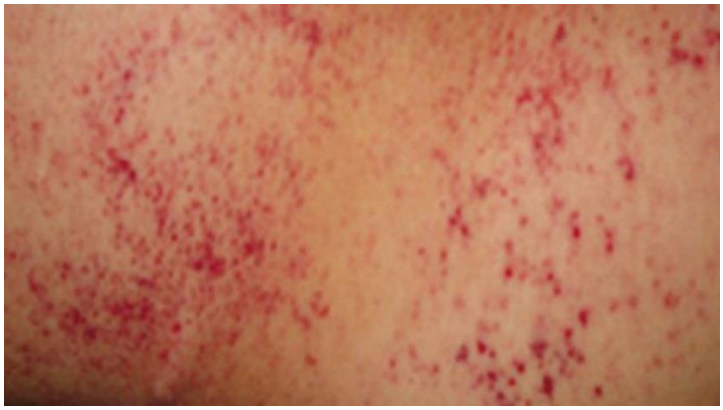

Figure 2: Skin petechiae image.

DISCUSSION

FES is known clinical entity since 1862 when it was reported after autopsy in which fat globules are identified in pulmonary vessels. Though FES is less identified with non-traumatic causes, incidence reported by varies studies range from 1%-35% due to traumatic long bone injuries. Varies factors which will increase the incidence of FES include closed long bone fractures, pelvic injury, rib fractures and multiple bone injuries, 1%-3% incidence reported with single femoral fracture and nearly 33% with bilateral femoral fractures. In our case first patient had open both bone fracture leg and second one had femoral mid shaft closed fracture. Clinical presentation usually starts between 12 hours to 72 hours of initial insult predominantly characterised by sudden onset of respiratory insufficiency and altered mental status. Our both patients had altered mental status within 12 hours and respiratory insufficiency requiring mechanical ventilation in the initial 24 hours. Mechanical and biochemical theory proposed are fat globules entering into the circulation via sinusoids, producing pro inflammatory response, Sequestration of platelets, fibrin causing emboli formation and obstruction of the capillaries of pulmonary and systemic circulation. Lungs and brain are most commonly involved. According to Gurd and Wilson criteria our first patient had all three major criteria Hypoxemia, CNS involvement, Skin petechiae and most of the minor criteria tachycardia, Hyperthermia, Low platelets though most common presentation is sudden onset respiratory insufficiency in 96% of the cases which makes the clinician to suspect fat embolism, our patients had profound alteration in mental status along with breathing difficulty within 12 hours of insult and MR imaging features are consistent with FES. In both patients CT showed no neuroparenchymal abnormality. Management of FES mostly of supportive therapy though corticosteroids, albumin infusion have the favourable outcome benefit, our patients were not given either of these. Our patients are resuscitated with crystalloids, ventilator support, and LMWH. Mortality of FES was 5%-15% and various neurological outcome has been reported in the previous studies. In our patient’s good neurological outcome happened in both patients though first patient took longer to recover.

CONCLUSION

Early suspicion of FES in traumatic bone injuries and MRI imaging modality for the early diagnosis of fat embolism syndrome, adequate hydration, ventilator support and other supportive measures favour the good neurological and clinical outcome. As varies studies noted Early stabilisation with surgical intervention as early as possible within 6 hours - 8 hours will be helpful to prevent FES and to reduce the morbidity and mortality in the case of long bone fractures of lower limbs.

PATIENTS CONSENT

Our patient's consent has been obtained for publication.

COMPETING INTERESTS

There were no competing interests for the article.

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