CASE REPORT DOI: 10.15713/ins.bhj.155

Rare Presentation of a Patient with Cardiac Arrest due to Cerebral Fat Embolization Following Polytrauma

ABSTRACT

Cerebral fat embolism syndrome is a known complication following polytrauma, especially fracture of long bones, but the cardiac arrest is a rare presentation following cerebral fat embolization. Our patient met with a road traffic accident, sustaining multiple long bones injury with hypovolemic shock. After 10 h of admission and achieving hemodynamic stability, the patient developed cerebral fat embolization. He developed sudden cardiac arrest and he was resuscitated. Instituted ventilator support, inotropic infusion, antibiotics, and IV fluids. Our patient regained consciousness without neurological deficit for 10 days and underwent surgery for all three major fractures with due precautions. The patient was discharged after 3 weeks of treatment from the hospital.

Key words: Cardiac arrest, Cerebral fat embolization, Long bone fracture, Road traffic accident

INTRODUCTION

The incidence of cerebral fat embolism (CFE) ranges from 0.9% to 11%, with a mean mortality rate of around 10%. Fat embolization syndrome (FES) is characterized by multisystem dysfunction, typically presenting 10–72 h after the initial insult. The classic triad of FES includes hypoxemia, neurological abnormalities, and petechiae. The clinical manifestations of respiratory failure, petechiae, and a diffuse or focal cerebral disturbance, are characteristic but not pathognomonic of the syndrome. Petechial rash is considered pathognomonic of FES and is reportedly present in up to 60% of patients, usually on the conjunctiva, oral mucous membranes, skin folds of the neck and axillae.

CASE REPORT

A 51-year-old male, with no comorbidity and no addiction, presented to the emergency department with an alleged history of road traffic accident, while traveling in an Auto rickshaw which collided with the car, sustaining trauma to his face, bilateral lower limbs, and left arm.

On admission to the emergency department, he was fully conscious, and oriented. On arrival, his pulse rate was 130 beats/min, with a blood pressure of 90/60 mm Hg measured in his right arm. Blood oxygen saturation (SpO_2) was 90% on room air, with a respiratory rate of 28/min and patent airways. Clinical examination revealed multiple abrasions over the right arm and right shoulder, CLW over the right side of the chin measuring 1 cm × 0.5 cm × 0.5 cm. X-ray AP and lateral view of bilateral lower extremity showed (1) Right Femur shaft segmental fracture [Figure 1]. (2) Left femur shaft segmental fracture [Figure 2]. (3) Left patellar fracture [Figure 3]. X-ray of the left upper limb and shoulder revealed a displaced fracture of the shaft of the humerus [Figure 4]. X-ray of the Head revealed left zygomatic bone fracture and left maxillary sinus fracture. After initial resuscitation with IV fluid, inotropes, and analgesics, POP slab was applied on the left arm. Temporary stabilization of the bilateral femur fracture was done using Thomas splint. Routine blood investigations [Table 1] were carried out, which indicated a picture suggestive of acute blood loss with the initial rise in hemoglobin, hematocrit levels, and total leukocyte counts due to hemoconcentration followed by an eventual drop in (Hb, Haematocrit, and Total leucocyte count) parameters.

Patient was immediately shifted to intensive care unit (ICU) in view of hemodynamic instability associated with multiple long bone fracture. Inotrope and vasopressor support was continued. Multiple (four) units of packed red blood cells were transfused. The patient was having right conjunctival bleed and small petechial hemorrhage over the right axilla and chest wall. After 8 h of stabilization, patient developed convulsion, which was followed by cardiac arrest. He was

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Table 1: Routine investigations

	16/11/22	17/11/22	19/11/22	25/11/22	1/12/22	19/12/22
HB(g/dL)	14.1	8.8	9.7	8.8	10.4	10.2
HCT (%)	43.9	26.6	29.7	27.1	32.3	31.4
TLC(/microL)	14860	6680	11760	15770	7760	11050
PLATELETS(/mi	419000	138000	126000	255000	578000	570000
PT(sec)	10.8	14.5	19.5			
INR	1.04	1.39	1.87			
NA(mEq/L)	137	136	144	134	133	134
K(mEq/L)	3.69	4.13	4.26	3.78	4.16	4.5
CL(mEq/L)	96	102.6	110.7	100.4	94	95.5
ESR(mm/hr)						
CREAT(mg/dL)	0.98	0.75	0.95		0.45	
CPK(U/L)		2689	4424			
BIL(mg/dL)			1.005			
SGOT(U/L)			25.53			
SGPT(U/L)			21.06			
PROTEIN(g/I)			3.98	4.5		
ALBUMIN(g/I)			2.3	2.3		
pH		7.38	7.36	7.43		
$PaCo_2$(mmHg)		30.4	40.5	37.1		
$Pa0_2$(mmHg)		526	73	163		
$HC0_3$(mmol/L)		17.5	22.3	23.9		
LACTATE (mmo	1.6	5	2	1		
UREA(mg/dL)			37.5	39.4		
BUN(mg/dL)			17.52	18.41		
CRP(mg/L)				163.72	156.63	30.07
ESR(mm/hr)	10		1.79			
Mg(mg/dL)			6.82			
Ca(mg/dL)						
PCT(ng/mL)						
URINE ROUTINE						
SPUTUM CULTURE				NO GROWTH	KLEBSIELLA PNEUMONIA PSEUDOMONAS AERUGI NOSA	
URINE CULTURE					NO GROWTH	
					PSEUDOMONAS AERUGINOSA STAPHYLOCOCCUS AUREUS	
BLOOD CULTURE						

immediately resuscitated, intubated, and put on ventilator. Post resuscitation and stabilization CT scan of the head, chest, and abdomen were performed. CT brain did not show any evidence of acute infarct or bleed. CT scan chest did not reveal evidence of ground-glass opacity or pulmonary embolism. The next day, in view of the deteriorating neurological status and reduced power in the limbs, MRI with MR Angio (brain) was done, which revealed [Figures 5a-7a] multiple small lesions in the whole brain consistent with a "star field" pattern. It showed numerous tiny foci of restricted diffusion in the

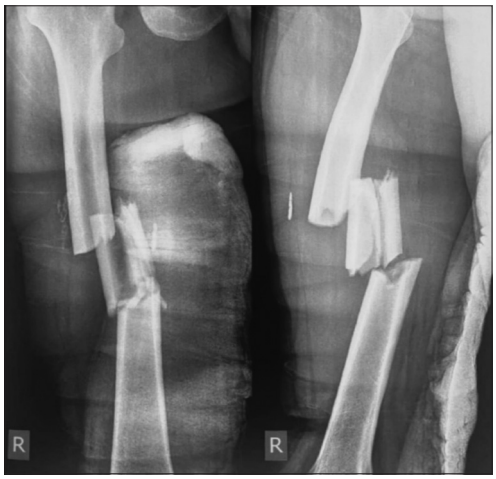

Figure 1: Right femur fracture

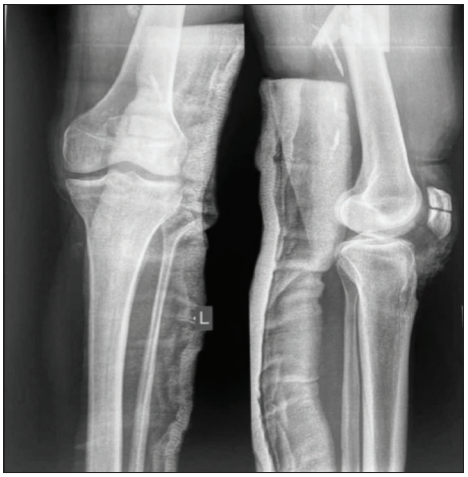

Figure 3: Left patellar fracture

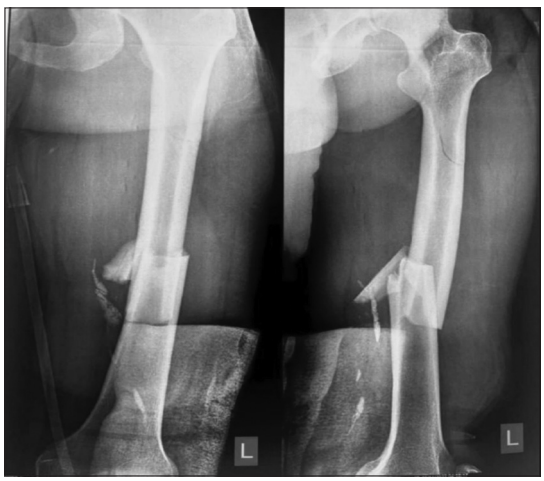

Figure 2: Left femur fracture

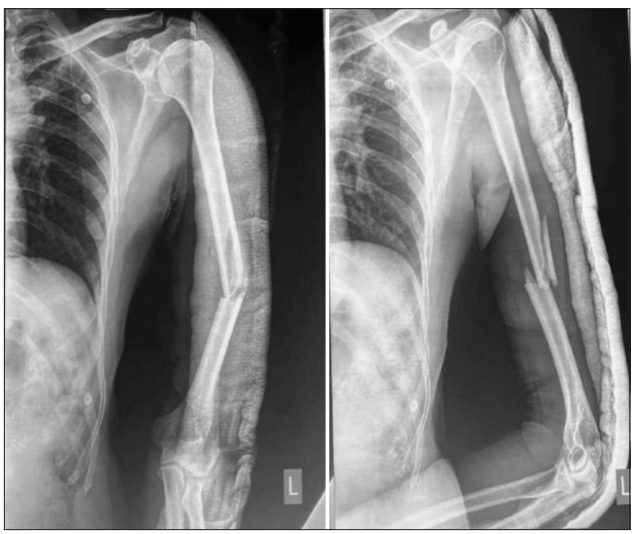

Figure 4: Left humerus fracture

bilateral frontal, parietal, occipital and temporal gray-white matter junction and basal ganglia, thalami, selenium of corpus callous, and both cerebellar hemisphere. These findings represent the sequel of Fat embolism. On 3D arterial spin labeling (non-contrast perfusion), there was symmetric and maintained cerebral perfusion. A 2D-echo was done, which revealed good left ventricular function with left ventricular ejection fraction = 55%, no regional wall motion abnormality, no clot or vegetation in the left ventricle. There was no intra-atrial septal or intra-ventricular septal defect or shunt. There was neither right ventricular enlargement or hypertrophy, nor evidence of pulmonary artery hypertension, thus ruling out pulmonary embolization. A bilateral lower limb Doppler was done, which showed evidence of echogenic thrombus seen from the left distal SFV- Popliteal junction extending into the left popliteal vein. He was started on DVT prophylaxis with LMWH. Anti-epileptic was started. Ventilator and inotrope support was continued along with IV antibiotics. Ryle's tube was inserted, and the patient was given tube feeds. Hemodynamic stability was achieved in 4 days and an MRI brain [Figures 5b-7b] was repeated, which revealed partial resolution of the previously seen areas of restricted diffusion and blooming in both cerebral hemispheres. No new areas of altered signal intensity were seen in brain parenchyma on repeated scans.

As soon as hemodynamic stability was achieved, the patient was planned and taken up for surgical repair (closed reduction and internal fixation with nailing) of right mid-shaft femoral fracture. LMWH was withheld 12 h before surgery. The surgery was uneventful. The patient was vitally stable in the post-operative period. LMWH was restarted after 12 h of surgery. He was then tracheotomized for further oxygen delivery, and subsequently patient was weaned off the ventilator with tracheostomy *in situ*. He maintained blood SpO_2 of 97% on 2 L O_2/min through T-tube (Tracheostomy tube). Arterial blood gas was repeated, which showed improvement in all the

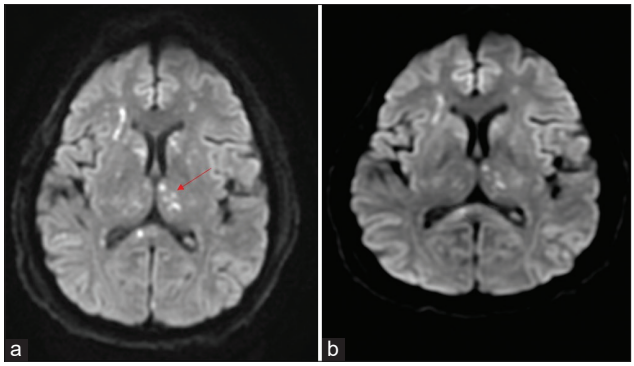

Figure 5: (a) Diffusion weighted images show multiple areas of restricted diffusion. (b) These show temporal evolution

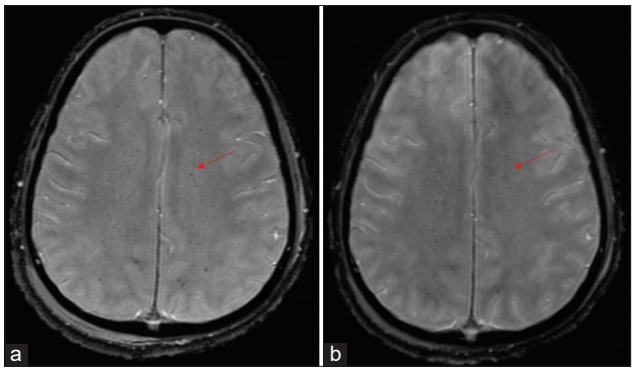

Figure 6: (a) Tiny subtle areas of blooming are seen throughout bilateral cerebral parenchyma (b) These show reduction

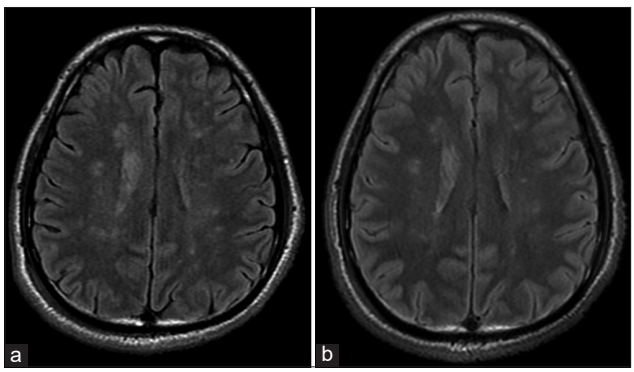

Figure 7: (a) Flair images show hyperintensity in affected areas (b) These show reduction

parameters (pH = 7.43, pCO2 = 37.1, HCO3 = 23.9 mmol/L, Lactate = 1 mmol/L). Physiotherapy was initiated with careful monitoring of all vital parameters.

Subsequently, patient underwent two more surgeries for (I) Left femur shaft fracture closed reduction and internal fixation, open reduction and internal fixation with tension band wiring for left patellar fracture. (II) The left shaft of humerus fracture open reduction and internal fixation with plating. Patient's neurological status started improving. He was fully conscious, obeying verbal commands. The patient continued to receive RT feeds. Slowly and gradually oral clear liquids were initiated and were well tolerated by him.

With symptomatic improvement and hemodynamic stability, patient was shifted to Ward from the ICU. Physiotherapy sessions were continued in the form of active therapeutic movements, active release technique, static quadriceps exercise, and sitting over the edge of the bed with support and bed-to-chair mobilization, breathing exercises.

Patient's condition improved symptomatically with well-maintained hemodynamic stability, with a novel oral anticoagulant and discharged from the hospital.

DISCUSSION

After a "bone burst," according to the accepted pathophysiology, "fat droplets" are shot into the systemic circulation, giving rise to emboli. There is no univocal explanation existing to describe how the syndrome develops. The mechanical theory postulates that fat micro emboli enter venous sinusoids, collect in the pulmonary microvasculature, and migrate into the systemic circulation via the pulmonary capillary bed. The fat embolism syndrome is believed to be caused by the toxic effects of free fatty acids liberated at the endothelial layer, which cause capillary disruption, perivascular hemorrhage, and edema.

Our patient developed fat embolization after 10–12 h of injury (long bone fractures). The patient also developed cardiac arrest following cerebral fat embolization. After adequate resuscitation, the patient improved without any neurological deficit.

CONCLUSION

When patients have long bone and pelvic fractures or multiple bone fractures and deteriorated neurological status, CFE should be considered. CFE may occur without the presence of a patent foramen ovale. Diffusion-weighted imaging often demonstrates the characteristic "star field pattern;" diffuse microhemorrhages on susceptibility-weighted imaging may be the only characteristic finding.

Clinical significance

The early diagnosis and appropriate management of FES are important, and patients should be monitored comprehensively in the ICU. With appropriate treatment, CFE patients may achieve good results.

REFERENCES

1. Sung AJ, Lee SH, Shin H, Lim D. Cardiac arrest occurred by cerebral fat embolism. J Korean Soc Emerg Med 2021;32:712-5.
2. Vetrugno L, Bignami E, Deana C, Bassi F, Vargas M, Orsaria M, *et al.* Cerebral fat embolism after traumatic bone fractures: A structured literature review and analysis of published case

reports. Scand J Trauma Resusc Emerg Med 2021;29:47-52.
3. Mellor A, Soni N. Fat embolism. Anaesthesia 2001;56:145-54.
4. Godoy DA, Di Napoli M, Rabinstein AA. Cerebral fat embolism: Recognition, complications, and prognosis. Neurocrit Care 2018;29:358-65.
5. Aman J, van Koppenhagen L, Snoek AM, van der Hoeven JG, van der Lely AJ. Cerebral fat embolism after bone fractures. Lancet 2015;386:e16.

How to cite this article: Rathod V, Rathod A, Parikh B, Rathod N. Rare Presentation of a Patient with Cardiac Arrest due to Cerebral Fat Embolization Following Polytrauma. Bombay Hosp J 2023;65(3):21-25.

Source of support: Nil, **Conflicts of interest:** None