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Cerebral Fat Embolism or Another Cause? A Diagnostic Dilemma After Polytrauma: A Case Report

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Abstract

Background: Fat embolism syndrome (FES) is an uncommon complication of long-bone fractures. Its clinical criteria are primarily designed for a syndrome that evolves over time and may be difficult to apply during the first hours after trauma. Hyperacute focal neurological deterioration therefore requires consideration of several competing mechanisms.

Case Report: A 70-year-old man was brought to the emergency department after being struck by a vehicle as a pedestrian, with bilateral forearm fractures and a proximal fibular fracture. He was initially stable with a GCS of 15, SpO2 97% on room air, no petechial rash, and sinus rhythm. Approximately 15 minutes after splint application, his GCS decreased to 12 with confusion, aphasia, and focal seizure-like movements. Cranial CT showed no acute pathology. Diffusion-weighted MRI approximately 2 hours after trauma demonstrated restricted diffusion in the left temporal region. Chest CT obtained early after arrival showed no diffuse parenchymal abnormality. The patient was transferred to a tertiary center before echocardiography and neurovascular imaging; the final diagnosis and outcome were unavailable.

Conclusion: The temporal relationship with fracture splinting raised suspicion for cerebral fat embolism, but the focal DWI lesion was not specific and the available findings were insufficient to establish FES. Cerebral fat embolism, cardioembolic or paradoxical embolism, blunt cerebrovascular injury, traumatic brain injury, and peri-ictal change should be considered in hyperacute neurological deterioration after trauma.

Keywords: Cerebral fat embolism; Fat embolism syndrome; Diffusion-weighted MRI; Polytrauma; Case report

Introduction

Fat embolism syndrome (FES) is an uncommon, potentially life-threatening complication of long bone fractures, classically presenting with respiratory distress, neurological impairment, and petechial rash [1]. Diagnosis is clinical. Gurd's criteria require two major criteria, or one major and four minor criteria [1-6]. Alternative scoring systems, such as those of Schonfeld and Lindeque, place more weight on respiratory findings [7,8]. FES typically becomes clinically apparent 24–72 hours after injury, so several diagnostic findings, including thrombocytopenia, anemia, fever, and pulmonary infiltrates, are not expected in the first hours [9]. On diffusion-weighted MRI, cerebral fat embolism classically produces multiple small punctate foci of restricted diffusion, often described as the “starfield” pattern [2,10,13]. MRI findings are supportive rather than pathognomonic, and current reviews emphasize that no single clinical criterion has been prospectively validated for FES or CFE [12,13]. Fulminant or paradoxical fat embolism through a patent foramen ovale (PFO) can cause very early cerebral involvement [3,4]. Elderly polytrauma patients who deteriorate early also require evaluation for other causes, including cardioembolic stroke, blunt cerebrovascular injury [11], and traumatic brain injury. We report a patient with hyperacute neurological deterioration soon after fracture manipulation and splinting and a restricted-diffusion lesion on early MRI, whose diagnosis remained unresolved at our center.

Case Report

A 70-year-old man was brought to the emergency department after being struck by a vehicle as a pedestrian. His past medical history and medication use were unavailable at presentation. Radiographs showed displaced fractures of both forearms and a proximal fibular fracture (Figures 1-3).



Figure 1: *AP radiograph demonstrating displaced comminuted fractures of the forearm long bones.*

Figure 2: *AP radiograph demonstrating a proximal fibular fracture.*

Figure 3: *Contralateral forearm radiograph showing displaced shaft fractures.*

On arrival the patient was hemodynamically stable, with a blood pressure of 110/70 mmHg, heart rate of 95/min, respiratory rate of 16/min, SpO2 of 97% on room air, and temperature of 36.5 °C. ECG showed normal sinus rhythm. The GCS was 15 and the neurological examination was unremarkable. No petechial rash was seen on the trunk, axillae, or conjunctivae. Long leg and long arm splints were applied 30 minutes after arrival, with NSAID analgesia and no sedation.

Approximately 45 minutes after arrival, about 15 minutes after splint application, the patient's GCS fell to 12. He was confused and had aphasia and focal seizure-like movements, and an antiepileptic drug was administered. Non-contrast cranial CT showed no acute intracranial pathology. Because of persistent altered consciousness, diffusion-weighted MRI was obtained approximately 2 hours after the trauma. According to the official radiology report, it showed restricted diffusion in the left temporal region (Figures 4,5). Non-contrast chest CT, obtained within the first 30 minutes after arrival as part of the trauma evaluation and therefore before the neurological deterioration, showed no diffuse ground-glass opacities or other parenchymal abnormality (Figures 6,7).

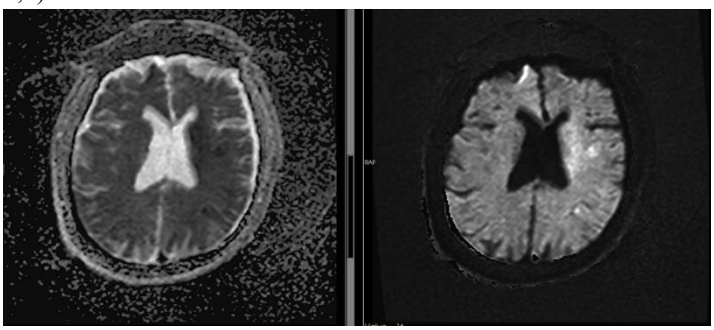


Figure 4: *Upper axial ADC (left) and DWI (right) images demonstrating restricted diffusion in the left temporal region.*

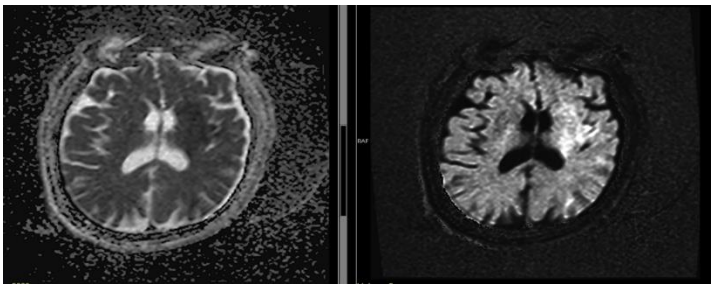


Figure 5: *Lower axial ADC (left) and DWI (right) images demonstrating persistent left-sided restricted diffusion.*



Figure 6: *Non-contrast chest CT obtained within 30 minutes of arrival.*

Laboratory results were: WBC 8.49 × 10^3/μL, hemoglobin 13.6 g/dL, hematocrit 41.7%, platelets 275 × 10^3/μL, ALT 14 U/L, AST 25 U/L, ethanol 0 mg/dL, INR 1.09, creatine kinase 183 U/L, and blood glucose 175 mg/dL.

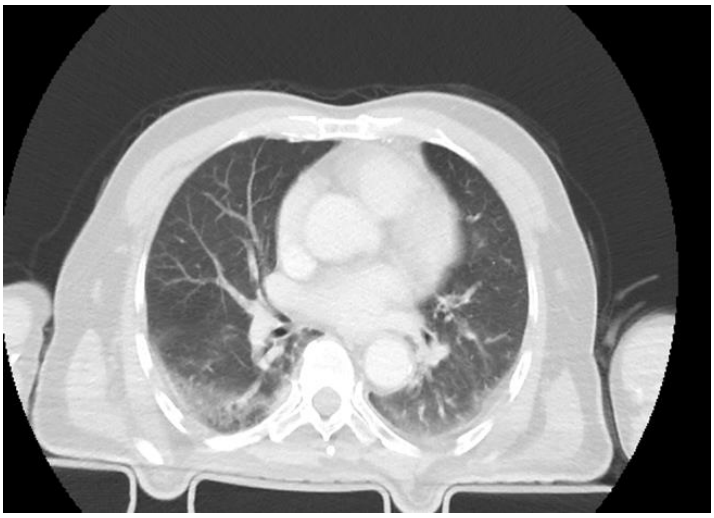


Figure 7: *Non-contrast chest CT at a different level, showing no diffuse ground-glass opacities or parenchymal abnormality.*

Echocardiography and vascular imaging could not be performed at our institution, and the patient was transferred urgently to a tertiary center for multidisciplinary evaluation. Attempts to obtain follow-up information from the receiving center were unsuccessful, and the final diagnosis and outcome are unknown (Table 1).

Table 1: *Timeline of events (T0 = collision).*

Time	Event
T0	Collision
Arrival	GCS 15; BP 110/70, HR 95, RR 16, SpO2 97% (room air), 36.5 °C; sinus rhythm; normal neurological examination; no petechiae
Arrival + ≤30 min	Chest CT: no ground-glass opacities
Arrival + 30 min	Long leg and long arm splints applied (NSAID analgesia, no sedation)
Arrival + ~45 min	GCS 12; confusion, aphasia, focal seizure-like movements; antiepileptic drug given
After deterioration	Cranial CT: no acute pathology
T0 + ~2 h	DWI MRI: restricted diffusion, left temporal region
After imaging	Emergency transfer to tertiary center; outcome unknown

Discussion

This case illustrates the diagnostic difficulty of attributing hyperacute neurological deterioration in a polytrauma patient to fat embolism when evaluation is limited to the first hours after injury and the final diagnosis is unavailable.

Limits of clinical criteria in the first hours

By Gurd's criteria the patient had a neurological finding but no petechial rash and no respiratory compromise (SpO2 97% on room air, respiratory rate 16/min). Thrombocytopenia, anemia, fever, tachycardia, retinal changes, and pulmonary imaging abnormalities may appear later, so their absence about 2 hours after injury cannot be taken as evidence against FES [9,12]. This applies in particular to the chest CT, which was obtained within 30 minutes of arrival, before the neurological deterioration; a normal study at that time says little about later pulmonary involvement. Cerebral involvement can also occur without prominent pulmonary findings [13]. Creatine kinase is not a Gurd criterion, and 183 U/L within hours of trauma is uninformative. The Gurd cerebral criterion applies to neurological signs not explained by another cause, so it cannot be counted in favor of FES if the lesion is attributed to a different mechanism. Using the Schonfeld index, the patient scored 1 point for confusion and 0 points for petechiae, diffuse pulmonary infiltrates, hypoxemia, fever, tachycardia, and tachypnea, for a total score of 1; this is well below the commonly used diagnostic threshold of >5 points [7,12]. These clinical criteria therefore neither supported nor excluded FES.

Timing and fracture manipulation

Symptom onset 45 minutes after arrival is earlier than the usual clinical presentation of FES [1,9,12,13]. However, the deterioration began roughly 15 minutes after splint application. Handling and manipulation of fractured limbs can release marrow fat into the circulation, and fat embolism is described after closed fractures without surgery, although it is more often discussed in association with orthopedic procedures. Very early cerebral presentations, particularly through a right-to-left shunt such as a PFO, have been reported [3,4]. Because no sedation was given and analgesia was limited to an NSAID, the fall in GCS is not readily explained by drug effect. This sequence is temporally compatible with an embolic event, but the association with splinting is non-specific and does not establish causation.

Table 2: *The principal mechanisms considered in this case and the investigations that could have helped distinguish them are summarized.*

Mechanism	Supporting / against in this case	Discriminating test
Cerebral fat embolism (incl. paradoxical via PFO)	Long bone fractures; deterioration about 15 min after splint application; but no rash, SpO2 97%, very early onset, focal pattern	Echocardiography with bubble study, SWI for microhemorrhages, serial MRI
Embolic / cardioembolic stroke	Age 70; sinus rhythm on arrival ECG (paroxysmal arrhythmia not excluded); cardiac history unknown	Telemetry, echocardiography, CT/MR angiography
Blunt cerebrovascular injury (carotid/vertebral dissection)	High-energy mechanism; neck imaging not performed	CT angiography of head and neck [11]
Traumatic brain injury (contusion, DAI)	Temporal lobe is a common contusion site; early CT may be normal	Follow-up CT/MRI, SWI, FLAIR
Seizure / peri-ictal change	Seizure-like movements and temporal lesion; seizure can be a consequence of a cortical lesion or cause transient peri-ictal DWI change; no EEG performed	EEG, follow-up MRI
Hypoperfusion / watershed infarct	BP 110/70 on arrival, no documented hypotension; lesion pattern less typical	Vascular imaging, hemodynamic record
Metabolic or drug-related	Ethanol 0 mg/dL; no sedation; NSAID only; glucose 175 mg/dL (excludes hypoglycemia)	Glucose, electrolytes, medication review

Focal features and imaging pattern

Cerebral fat embolism most often produces diffuse encephalopathy with multiple punctate diffusion-restricting foci, classically described as the starfield pattern [2,10,13]. Our patient had aphasia and a restricted-diffusion lesion in the left temporal region, compatible anatomically with a focal dominant-hemisphere cortical event and potentially explaining part of the recorded confusion. Focal deficits and seizures have been described in CFE, but a single focal lesion is atypical for the classic starfield pattern. Thus, the

MRI finding supports acute cerebral injury but does not by itself establish cerebral fat embolism.

Differential diagnosis

The principal mechanisms considered in this case and the investigations that could have helped distinguish them are summarized in (Table 2).

Seizure-like movements

The focal seizure-like movements pose a diagnostic problem in both directions. They may have been a consequence of a cortical embolic or traumatic lesion, or they may themselves have produced transient peri-ictal diffusion restriction. EEG and follow-up MRI assessing persistence or evolution of the lesion would have helped separate these possibilities, but neither was available before transfer.

Watershed hypothesis

Watershed infarction results from compromised perfusion at arterial border zones, classically in systemic hypotension [5]. This patient had a blood pressure of 110/70 mmHg on arrival, no documented hypotensive episode, and a lesion pattern less typical of watershed infarction, so hypoperfusion was considered less likely. Blunt cerebrovascular injury remained a relevant alternative given the high-energy mechanism and the absence of head-and-neck vascular imaging; current trauma guidance supports CTA-based evaluation when BCVI is suspected [11].

Limitations

This report has several limitations. The patient was transferred to a tertiary center, and neither the final diagnosis nor the outcome could be obtained. Past medical history, including cardiac disease, anticoagulant use, and PFO status, was unknown. Echocardiography with bubble study, head and neck vascular imaging, EEG, and follow-up neuroimaging were not available before transfer, so cerebral fat embolism could not be distinguished from paradoxical or arterial embolism, blunt cerebrovascular injury, peri-ictal diffusion abnormality, or other mechanisms. The temporal relationship with splinting should therefore be regarded as a clinically important association rather than proof of causality. The value of this report lies in documenting the first hours of presentation, when the information available to the emergency physician is limited.

Take-home message

In a trauma patient with hyperacute neurological deterioration after fracture manipulation and splinting, normal oxygenation, platelet count, and early chest imaging do not by themselves exclude cerebral fat embolism. Conversely, a focal DWI lesion is not diagnostic of cerebral fat embolism. Early neurovascular imaging, echocardiography, and EEG when seizure is suspected are important for distinguishing competing mechanisms.

Conclusion

Hyperacute neurological deterioration after polytrauma, particularly when temporally associated with fracture manipulation, should prompt consideration of cerebral fat embolism but should not be attributed to it solely on the basis of an early diffusion-restricting lesion. In this patient, the available findings could not distinguish cerebral fat embolism from paradoxical or cardiac embolism, blunt cerebrovascular injury, traumatic brain injury, or seizure-related change. Early targeted neurovascular and cardiac evaluation, with EEG when seizure is suspected, is important when the presentation is atypical for classic FES.

Declarations

Informed consent: Written informed consent for publication could not be obtained because the patient was transferred emergently to another institution and the receiving center could not be reached despite attempts to obtain follow-up information. All identifying information has been removed, and the presented images contain no identifiable personal information. The manuscript is limited to clinically relevant, anonymized information.

Patient data: All identifying information has been removed. The original clinical documentation is retained by the corresponding author and can be made available to the journal upon reasonable request, subject to applicable institutional and privacy requirements.

Conflict of interest: The author declares no conflict of interest.

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Author contributions: The author was responsible for the conception of the report, clinical data review, literature review, manuscript preparation, revision, and final approval of the submitted version.

Patient perspective: Not available because the patient was transferred emergently and could not subsequently be contacted.

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