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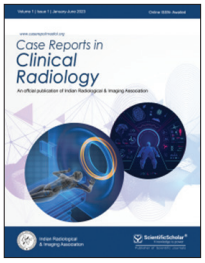

Case Series

Imaging findings of cerebral fat embolism

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ABSTRACT

Cerebral fat embolism (CFE) is a rare complication of long bone or pelvic fractures. The diagnosis is difficult because of non-specific signs and symptoms occurring in conjunction with the features of traumatic injury, where imaging is often deferred in view of unstable vitals. We present two cases of CFE occurring post-internal fixation of femur fracture in young- and middle-aged adult males in early and late subacute stages, respectively; one patient had associated hypoxic-ischemic encephalopathy. It is important for radiologists to know the variable appearance on imaging to make a timely diagnosis.

Keywords: Fat embolism syndrome, Cerebral fat embolism, Magnetic resonance imaging

INTRODUCTION

Fat embolism syndrome was first described by Zenker[1] in 1862 and is known to be associated with the displaced femur and pelvic fractures. The classic triad includes respiratory disability, petechial skin rash, and neurologic symptoms, and reported incidence varies from 0.9% to 2.2%.[2] Although cerebral fat embolism (CFE) is usually self-limiting, it can also be fatal. The presentations of CFE can be further complicated by the frequent cooccurrence of traumatic brain injury or hypoxic-ischemic injury and delayed imaging due to unstable conditions.

The diagnosis of CFE is delayed because the clinical criteria lack specificity, so magnetic resonance imaging (MRI) plays a crucial role in diagnosis, especially diffusion-weighted imaging (DWI) and susceptibility-weighted imaging (SWI).

We describe imaging findings in the subacute stage of CFE in two patients.

CASE 1

A young male in his early twenties with a history of road traffic accident, sustained multiple displaced facial and left femur shaft fractures. Initial imaging of the head [Figure 1a – on admission] showed normal neuroparenchyma. He had suffered multiple facial fractures with right orbital and periorbital emphysema. One day post-intramedullary nailing he developed some visual disturbances along with a few petechial skin rashes. Bedside ophthalmoscopy suggested Berlin's edema. Later, he developed deterioration of sensorium and dyspnea, followed by desaturation for which he was intubated.

Computed tomography (CT) day-5 post internal fixation [Figure 1b] showed confluent areas of hypoattenuation in bilateral cerebral white matter; more so posteriorly with mild effacement

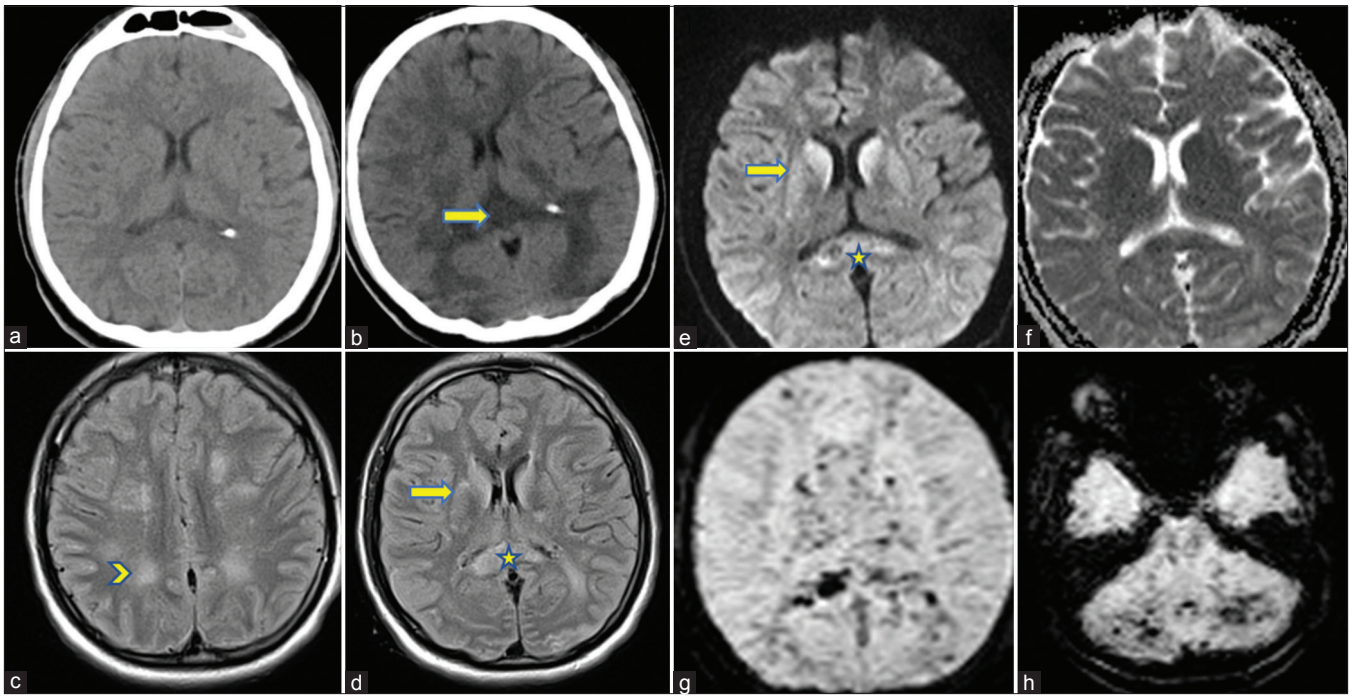

Figure 1: (a) Axial CT Brain at the time of admission and (b) Day 5 post-fixation, there is interval development of bilateral confluent areas of white matter hypoattenuation with mild effacement of adjoining gyri; splenium of corpus callosum (arrow) was bulky and hypodense. (c and d) Corresponding FLAIR MRI images (D12 post fixation) showing confluent high signal in forementioned areas. Also, there is diffusely increased signal of the basal ganglia (arrow). (e) DWI and (f) ADC maps suggesting diffusion restriction only in basal ganglia. The splenium shows altered signal. (g and h) SWI images show bilateral innumerable punctate round foci of hypointense blooming involving the splenium of corpus callosum, bilateral subcortical as well as cerebellar white matter.

of adjoining gyri. Hypoattenuation was also apparent in the splenium of the corpus callosum.

Metabolic encephalopathy was considered the first differential, however, given the clinical scenario there was a suspicion of fat embolism syndrome, and MRI brain was suggested. There was a delay of a week due to unstable patient condition, on day 12 post-fixation MRI brain fluid-attenuated inversion recovery sequence images showed near symmetrical bilateral cerebral confluent white matter hyperintensities, bilateral basal ganglia hyperintensity with mildly bulky and hyperintense splenium of corpus callosum [Figure 1c and d]. True diffusion restriction was seen only in basal ganglia [Figure 1e and f], suggesting the possibility of concurrent hypoxic-ischemic encephalopathy. SWI [Figure 1g and h] showed innumerable small round foci of hypointense blooming in the splenium of the corpus callosum and bilateral subcortical and cerebellar white matter.

Therefore, a diagnosis of CFE syndrome probably in the early subacute phase, as there is no obvious diffusion restriction in most of the lesions with associated hypoxic ischemic encephalopathy was considered.

On follow-up (post 42 days of internal fixation) mild diffuse cerebral and cerebellar atrophy was evident, which is a known chronic sequela. The basal ganglia attenuation was normal. Clinically, the patient was out of the coma and had improving motor functions within a month and regained complete motor function without a focal deficit in 3 months.

CASE 2

Middle aged male in his early fifties sustained fractures of the left femur and patella in a high-velocity collision [Figure 2a]. The sensorium was altered and initially attributed to mild traumatic brain injury. Symptoms worsened after internal fixation. Bedside ophthalmoscopy did not show any significant abnormality.

Initial imaging of the head [Figure 2b – on admission] showed normal neuroparenchyma. One day post-intramedullary nailing his sensorium worsened, the patient underwent intubation and intensive care unit admission. Later MRI brain was performed when the patient was relatively stable (~day 14 post-operative), there were bilateral white matter hyperintensities predominantly in the border zone areas, body, and splenium of corpus callosum [Figure 2c and d]. DWI did not show any evidence of true restriction [Figure 2e and f]. SWI showed punctate round hypointense blooming foci in the corpus callosum and a few in the subcortical region [Figure 2g and h].

Therefore, the diagnosis considered was of CFE syndrome probably in the late subacute phase as there is no obvious

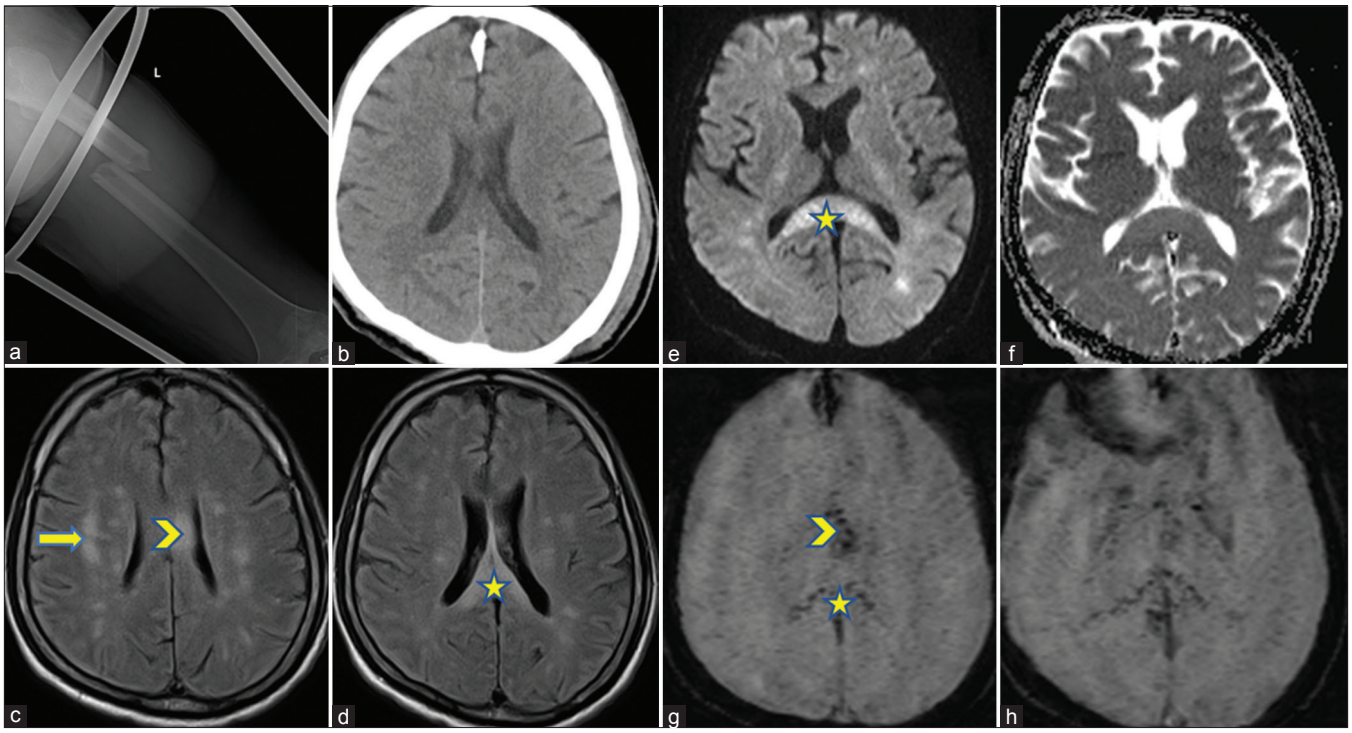

Figure 2: (a) Left femur radiograph showing displaced fracture of shaft with limb in Thomas splint. (b) Admission CT Brain axial image shows essentially normal neuroparenchyma. (c and d) Day-14 post fixation axial FLAIR images show hyperintense areas within bilateral cerebral white matter (arrow), body (arrow head) and in the splenium of corpus callosum. (e) DWI and (f) ADC maps show facilitated diffusion within the corpus callosum. (g and h) mIP SWI images showing obvious punctate hypointense foci in the body (arrowhead) and splenium (star) of corpus callosum, and a few in the subcortical region (h).

diffusion restriction in most of the lesions. The patient regained complete motor function without a focal deficit in 3 months.

DISCUSSION

CFE is a rare entity but has a relatively better prognosis as compared to diffuse axonal injury (DAI) which is one of the main differential diagnosis entities. The clinical history and worsening of sensorium immediately post-trauma without improvement helps to differentiate the two. Furthermore, CT chest can be a supplement at times. It is hypothesized that causation is related to initial mechanical obstruction of arterial circulation by neutral fat and then a delayed toxic injury occurring from free fatty acids. A large amount of disseminated bone marrow fat tissue may flow into the pulmonary circulation and, in the presence of right-to-left shunts, can later pass into the systemic circulation.[2] However, most reported cases in the previous review[3] do not have a shunt; and this is the case in both of our patients. A study on dogs undergoing orthopedic surgery showed that fat vacuoles <5 μm still could directly pass through the pulmonary capillary bed.[4] The size of the impacted intravascular fat vacuole was further identified as approximately 4–42 μm in another animal study.[5]

Parizel *et al*. described showers of microemboli flowing into the brain sporadically resulting in ischemic injury and presenting as a "starfield" (Type 1) pattern seen on DWI at an early stage. It is rarely seen in the subacute and late stages (18% and 0%).[6]

Confluent cytotoxic edema (Type 2A) is a common pattern and is often ignored. Restricted diffusion is still present and this is seen in the early subacute stage. The cerebellar peduncles, corpus callosum, and posterior internal capsule may be involved.

Vasogenic edema lesions (Type 2B) which may enhance another pattern where the lesions are dot or patch shaped without diffusion restriction; however, contrast-enhanced study is not routinely performed in all patients.

Petechial hemorrhages of white matter (Type 2C), this pattern is consistently seen in early, subacute, and late stages.

Chronic sequelae (Type 3) are seen as lacunar areas of high signal on T2 without restriction, with sequelae such as infarction, cavitation, scarring, gliosis, and chronic demyelination. Most commonly there is diffuse parenchymal atrophy.

Both of our patients did not show typical star field patterns or significant diffusion-restricting lesions. The "walnut kernel" pattern was noted in the first patient which was explained in

a recent systematic review by Giyab *et al*.,[7] it is defined as monotonous-sized microbleeds that are diffusely distributed in the subcortical white matter, the internal capsule and the corpus callosum mostly sparing the corona radiata and non-subcortical centrum semiovale.

DAI is a close imaging mimic, it is suggested that CFE shows punctate/round (<3 mm) foci of hypointense blooming (which may be confluent) located in the frontal, parietal, or occipital white matter, the corpus callosum and cerebellum, whereas DAI hemorrhages are ~4–10 mm sized and are relatively linear with mildly scattered abnormalities.[8] Apart from this, the patients have a loss of consciousness immediately after the trauma, unlike CFE which may develop anytime, especially after internal fixation of long bone fractures.

In critically ill patients where there is hypoxemia and respiratory failure the presence of cerebral microbleeds (critical illness-associated microbleeds) has been described[9] which is another differential diagnosis that may be considered, they are typically identified as extensive small hypointense blooming foci on SWI typically located in juxtacortical white matter and corpus callosum but sparing the cortex, deep and periventricular white matter, basal ganglia, and thalami.

Both of our patients regained full motor movement without a focal neurological deficit in the 3 month follow-up.

CONCLUSION

The clinical diagnosis of fat embolism syndrome remains delayed due to non-specific manifestations overlapping with other causes of post-traumatic or post-operative respiratory distress. CFE has to be suspected in a patient with sudden development of altered sensorium after mobilization/orthopedic intervention.

CT chest and MRI brain are helpful to diagnose fat embolism syndrome, and awareness of radiologists is paramount for prompt work-up of these patients.

TEACHING POINTS

- Most often the classical starry-sky appearance may not be present or be missed if imaging is delayed. Another pattern known as the "walnut-kernel pattern" which was seen in our patients has to be remembered. The abnormal white-matter hypoattenuation/signal intensity is seen in border zones due to continued episodic seeding of fat emboli.
- A hawk-eye overview of the entire clinical picture is a must to report imaging of altered sensorium. SWI is by far the best sequence to identify the hypointense blooming artifacts which may be hypodense on CT.
- DAI being a close imaging mimic can often be differentiated using clinical details where the sensorium is worsened immediately post-trauma and the imaging pattern where hypointense blooming foci are slightly linear and ellipsoid.

MCQs

1. Cerebral fat embolism typically occurs between
   a. 0–12 h
   b. 12–24 h
   c. 24–36 h
   d. 12–72 h

Answer key: d

2. Clinical presentation of fat embolism syndrome can be
   a. Loss of consciousness immediately after accident followed by coma and no neurological improvement
   b. Altered sensorium, skin rashes, and dyspnea occurring typically between 12 and 72 h
   c. Subclinical
   d. Fall in GCS score after admission

Answer key: b, c, d

3. Cerebral fat embolism occurs in
   a. Long bone fractures
   b. Pelvic fractures
   c. Pancreatitis
   d. All of the above

Answer key: d

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Declaration of patient consent

Patient's consent is not required as the patients identity is not disclosed or compromised.

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Conflicts of interest

There are no conflicts of interest.

REFERENCES

1. Zenker FA. Beiträge zur Normalen und Pathologischen Anatomie der Lunge. Schönfeld: Royal College of Physicians of Edinburgh; 1862.
2. Bardana D, Rudan J, Cervenko F, Smith R. Fat embolism syndrome in a patient demonstrating only neurologic symptoms. Can J Surg 1998;41:398-402.

3. Kuo KH, Pan YJ, Lai YJ, Cheung WK, Chang FC, Jarosz J. Dynamic MR imaging patterns of cerebral fat embolism: A systematic review with illustrative cases. AJNR Am J Neuroradiol 2014;35:1052-7.
4. Kim HJ, Lee CH, Kim HG, Lee SD, Son SM, Kim YW, *et al*. Reversible MR changes in the cat brain after cerebral fat embolism induced by triolein emulsion. AJNR Am J Neuroradiol 2004;25:958-63.
5. Byrick RJ, Mullen JB, Mazer CD, Guest CB. Transpulmonary systemic fat embolism. Studies in mongrel dogs after cemented arthroplasty. Am J Respir Crit Care Med 1994;150:1416-22.
6. Parizel PM, Demey HE, Veeckmans G, Verstreken F, Cras P, Jorens PG, *et al*. Early diagnosis of cerebral fat embolism syndrome by diffusion-weighted MRI (starfield pattern). Stroke 2001;32:2942-4.
7. Giyab O, Balogh B, Bogner P, Gergely O, Tóth A. Microbleeds show a characteristic distribution in cerebral fat embolism. Insights Imaging 2021;12:42.
8. Rutman AM, Rapp EJ, Hippe DS, Vu B, Mossa-Basha M. T2*-weighted and diffusion magnetic resonance imaging differentiation of cerebral fat embolism from diffuse axonal injury. J Comput Assist Tomogr 2017;41:877-83.
9. Fanou EM, Coutinho JM, Shannon P, Kiehl TR, Levi MM, Wilcox ME, *et al*. Critical illness-associated cerebral microbleeds. Stroke 2017;48:1085-7.

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