Two Different Imaging Patterns In A Case With Cerebral Fat Embolism

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Summary

We present a case of cerebral fat embolism after long-bone fractures with changing imaging findings on follow-up magnetic resonance imaging (MRI). We also discuss the usefulness of diffusion-weighted MRI and susceptibility-weighted MRI in diagnosis of cerebral fat embolism, especially in cases with indistinct findings on conventional MRI.

Key words: Cerebral fat embolism, Diffusion-weighted imaging, Susceptibility-weighted imaging, Long bone fracture, Brain imaging

INTRODUCTION

Fat embolism syndrome refers to a clinical entity that consist of respiratory distress, neurologic dysfunction and petechial skin rash(7). It is a rare, but potentially life-threatening complication of long-bone fractures. Although it is most commonly associated with long bone and pelvic fractures, it may complicate a wide variety of clinical conditions such as severe burns, liver injury, external cardiac massage, bone marrow transplantation, sickle cell crisis and liposuction(7). Cerebral fat embolism (CFE) syndrome is usually self-limiting, however depending on the severity the outcomes range from complete recovery to death, with reported mortality ranges from 10% to 15%(7). CFE typically manifests 24 to 72 hours after the initial insult(7,2). The true incidence is difficult to assess because of undiagnosed and subclinical events. The purpose of this case presentation is to highlight the value of diffusion-weighted imaging (DWI) and susceptibility-weighted imaging (SWI) for early diagnosis of CFE syndrome.

CASE PRESENTATION

A previously healthy, 24-year-old male patient was admitted to the hospital after a motorcycle accident with right femur and
tibia fractures. On admission, he was slightly disoriented and had Glasgow Coma Scale (GCS) score of 14. On the same day, the fractures were treated with open reduction and internal fixation. After several hours from the uneventful surgery, his level of consciousness started to progressively deteriorate reach to GCS of 9. No focal neurologic deficit was observed. He had no respiratory distress but had petechial rashes in the nail beds. Blood gas analysis showed hyperoxia (SO2 98.6% and PaO2 123.8 mm Hg) and slight hypocarbia (PaCO2 34.5 mm Hg). Thrombocytopenia was detected with a platelet count of 100,000/mm3. Chest computed tomography (CT) obtained 3 days after the accident showed peripheral patchy ground-glass opacities in the upper lung predominantly on the left-side, which were consistent with contusion caused by the accident. Cranial CTs obtained 6 hours and 3 days after the accident were unremarkable (Figure 1A). Initial magnetic resonance imaging (MRI) of the brain was performed 3 days following the deterioration in consciousness revealed T2-weighted (W) images showed barely noticeable small hyperintense, nonspecific lesions in the white matter of bilateral cerebral hemispheres although the image quality was significantly degraded by patient motion (not shown). However, DWI trace images showed hyperintense punctate foci on a dark background, most of which were hypointense on apparent diffusion coefficient (ADC) maps (Figure 1B, C). Over the ensuing days, the GCS worsened to 3. On the ninth day of the accident, follow-up MRI of the brain was performed. The distinct lesions had evolved into patchy and confluent lesions and had spread throughout the bilateral centrum semiovale and corpus callosum on T2W images (Figure 2). SWI revealed innumerable, tiny signal voids consistent with microhemorrhages in the involving regions on T2W images (Figure 2I). Basal ganglia, cerebellum, brainstem, and thalami were normal. Transthoracic and transeusophageal echocardiography were performed to exclude cardiac thromboembolism and showed no vegetation or patent foramen ovale.

Based on the clinical and radiological findings, the diagnosis of CFE was proposed. Haemodialysis was performed because of severe rhabdomyolysis, and antibiotherapy was given because of pneumonia. Nonetheless, the clinical picture was complicated with sepsis and the patient passed away on the sixteenth day of the accident.

Figure 1: (A-C). Initial cranial CT scan showed no significant findings (A). Initial MRI of the brain was performed 3 days after the onset of consciousness disturbance. Axial DWI (B) and corresponding axial ADC image (C) shows scattered spot diffusion-restricted lesions.
DISCUSSION

In the present case of CFE, T2W and FLAIR images nearly detected few scattered white matter lesions at onset. However, DWI images revealed multiple pinpoints, scattered bright lesions on a dark background producing a ‘starfield’ appearance at that time. The lesions pattern altered on the follow-up imaging with lesion spreading and becoming more confluent with lack of the ‘starfield’ pattern in the cerebral white matter including the corpus callosum with restricted diffusion on DWI, as well as tiny petechial hemorrhages on SWI.

CFE remains a diagnostic challenge for both clinicians and radiologists. Moreover, the presentations of CFE can be further complicated by the frequent co-occurrence of head trauma caused by traumatic insults. There is no diagnostic test that is sufficiently sensitive or specific to be useful for confirming or excluding CFE. Although petechial rash is one of the most valuable clinical findings, it occurs in fewer than half of cases. Recently, imaging of the brain is considered as one of the instruments of CFE diagnosis, especially in subclinical cases. Head CT is usually negative. However, depending on the clinical severity, head CT may show edema with low-attenuating areas and hemorrhage with high attenuation. MRI is the most sensitive technique for detection of CFE, especially if DWI sequences are used as part of the examination. In experimental CFE, DWI and T2 abnormalities have been demonstrated as early as 30 minutes after the ictus. As shown in the present case, different MRI findings could be identified in the different stages of CFE. Recently, Kuo and et al. described lesion patterns observed in CFE in detail: 1) The most common and well-known lesion pattern of CFE is scattered

Figure 2: (A-I). On the ninth day follow-up MRI. Serial axial DWI (A–C) and axial ADC images (D-F) demonstrate confluent cytotoxic edematous areas in the corpus callosum and the centrum semiovale, bilaterally. Axial T2W (G) and corresponding FLAIR (H) image show mild hyperintensity in the splenium of the corpus callosum. At the same level, axial SWI image (I) demonstrate punctate foci of low signal intensity throughout the splenium of the corpus callosum.
Cytotoxic edema, which was first named to 'starfield pattern' by Parizel et al. in 2001(4). These scattered spot lesions typically show restricted diffusion on DWI and have iso- or hyperintense T2W signal. The signal abnormalities commonly involve both the gray and white matter, but might also be limited to the white matter, due to lack of rich collateral vascular networks present in gray matter. 2) Confluent symmetric cytotoxic edema in periventricular and subcortical cerebral white matter mainly occur at the subacute stage (Figure 2). The cerebellar peduncles, corpus callosum, and posterior internal capsule may also be involved. 3) Vasogenic edematous lesions might also accompany the imaging findings at the subacute stage. These lesions may show contrast enhancement due to breakdown of the blood-brain barrier(1,5). 4) The final type of lesions that may be encountered in CFE are tiny petechial hemorrhages. The petechial hemorrhages may be identified in acute, subacute, or late stages. Takahashi et al demonstrated that T2W lesions appear as early as 4 hours after onset of CFE, and they diminish within 2 weeks to a few months(6). The radiologic differential diagnosis varies according to the lesion pattern. But DWI and especially SWI findings highly suggest the diagnosis of CFE in the appropriate clinical setting with patient's history. The ‘starfield pattern' could be seen in all kinds of embolic showers. Confluent symmetric cytotoxic edema pattern is quite similar to delayed posthypoxic leukoencephalopathy. Differential diagnoses of petechial lesions included diffuse axonal injury (DAI), however, DAI mainly occurs in the gray-white matter interface, splenium of the corpus callosum, and dorsolateral brainstem, while cerebral and cerebellar white matter and splenium of corpus callosum are the areas most vulnerable to CFE. In patients with CFE, consciousness disturbance often occurs few hours to 4 days after the accident, whereas consciousness disturbance in patients with DAI usually occurs at the time of trauma. Both CFE and DAI may be difficult to detect with conventional brain MRI. SWI is sensitive to detect petechial hemorrhages in CFE and DAI. Zaitsu et al. have proposed that the number of hypointense foci on SWI may affect the prognosis in their limited number of case series(8). Additionally, it is proposed that the number and size of the T2W lesions correlates with clinical outcome that is measured by GCS(6).

The pathophysiology of CFE is not clear. Among the many theories on the pathophysiology of CFE, two have gained acceptance: the mechanical theory and the biochemical theory(7). The mechanical theory states that large fat droplets are released into the venous system. These droplets are deposited in the pulmonary capillary beds and travel through intrapulmonary arteriovenous shunts or cardiac right-left shunt to the brain. Microvascular lodging of droplets produces local ischemia and inflammation, with concomitant release of inflammatory mediators, platelet aggregation, and vasoactive amines. The biochemical theory states that hormonal changes caused by trauma or sepsis induce systemic release of free fatty acids as chylomicrons. The biochemical theory helps explain nontraumatic forms of CFE(3), as the small fat vacuoles in CFE could deform, split into smaller globules, and recycle into the pulmonary circulation, the resulting reperfusion within the cerebral circulation might lead to significant tissue salvage; this possibility may explain the transient imaging change and clinically better outcome than other embolic events(8).

The autopsy findings of CFE include multiple petechiae, anemic lesions, and fat globules. The petechiae are usually most numerous and prominent in the cerebral and cerebellar white matter including corpus callosum, but may also be noted in the brainstem and spinal cord, and under the ependymal lining of the ventricles(7),
the gray matter is spared or only minimally involved. The signal voids on SWI were found in the cerebral white matter and corpus callosum in our case, which is in accord with the distribution of the petechiae in the reported autopsy cases. We consider that the increased hypointense foci on SWI represent petechial hemorrhages caused by CFE. Anemic lesions are less conspicuous and usually less frequent than hemorrhagic lesions. The anemic lesions are commonly seen both in the gray and white matter. Fat globules are found in arteriole’s or capillary-sized vessels throughout the brain and spinal cord as well as in the pia arachnoid and choroid plexus. They are most numerous in the cerebral and cerebellar gray matter, but are less frequent in the white matter. Fat globules are present in the vessels of both the altered and normal brain. The amount of the embolic fat in the brain varies depending on the interval between injury and death.

CONCLUSION

MRI is a helpful tool in the early diagnosis of CFE syndrome, especially clinically subtle cases. Particularly DWI and SWI images may be helpful in determining the presence of characteristic imaging findings and tiny hemorrhages and they should be added in case of indistinct conventional MRI findings. More research is needed to determine the relationship between the MRI characteristics and clinic outcome.

REFERENCES


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