CASE REPORT

Role of susceptibility-weighted imaging in demonstration of cerebral fat embolism

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SUMMARY

Cerebral fat embolism (CFE) is a rare but potentially lethal complication of long bone fractures. Many cases of CFE occur as subclinical events and remain undiagnosed. We report a case of a 22-year-old man, with multiple long bone fractures from a road traffic accident, who subsequently developed hypoxia, neurological abnormality and petechial rash. CT of the head was normal. MRI of the head confirmed the diagnosis with lesions markedly conspicuous and most widespread on susceptibility-weighted imaging as compared to all other sequences including diffusion-weighted imaging.

BACKGROUND

The imaging diagnosis of cerebral fat embolism (CFE) remains a challenge because of its various presentations and the limited number of reported cases. MRI has been reported to be the most sensitive means of diagnosing CFE with T2-weighted (T2WI), T2*-weighted (T2*WI) gradient-echo, fluid-attenuated inversion recovery (FLAIR) and diffusion-weighted imaging (DWI) being the useful conventional techniques commonly described in the literature. Susceptibility-weighted imaging (SWI) is a newer MRI technique that increases lesion conspicuity and is a useful adjunct to conventional MR for evaluation of CFE. Owing to its high sensitivity for detecting blood products, it is potentially the most sensitive sequence for detecting imaging abnormalities associated with CFE.

CASE PRESENTATION

A 22-year-old man who was involved in a road traffic accident was hospitalised with complex long

bone fractures. His initial neurological status was unremarkable. Soon after his internal fixation of left femoral fracture on day 2, his neurological status deteriorated significantly. He subsequently developed a petechial rash.

INVESTIGATIONS

Admission chest radiograph and skeletal survey performed in the accident and emergency department showed a comminuted fracture of the left proximal femur, Monteggia fracture and right scapular blade fracture.

CT of the head within an hour after admission was normal. CT of the chest, abdomen and pelvis showed pulmonary contusion and compression fracture of T12 vertebral body, and confirmed the fractures mentioned above.

After internal fixation (day 2) of the patient's left femur, CT of the head performed after neurological deterioration was normal ([figure 1](#)). CT of the chest showed changes in keeping with acute respiratory distress syndrome. Subsequent MRI of the brain demonstrated extensive, rather symmetric abnormality in the corpus callosum and bilateral frontoparietal lobes extending into posterior limbs of bilateral internal capsules ([figure 2](#)). These areas showed restricted diffusion on DWI ([figure 3](#)). On SWI, there were extensive numerous scattered minute foci of low signal involving these areas, but more widespread, including the cerebellum ([figure 4](#)). Overall extent of changes on SWI was more widespread than all other sequences including DWI. Given the clinical presentations and other abnormalities, findings confirmed the diagnosis of CFE.

Figure 1 Non-contrast CT of the head (A–C) on day 2 after onset of neurological symptoms. No abnormality could be seen.

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Figure 2 MRI. T2-weighted axial images (A–D), fluid-attenuated inversion recovery sagittal (E), axial (F and G) and coronal (H) showing abnormalities in genu (short white arrows), splenium of corpus callosum (long white arrows), internal capsule (black arrows) and deep white matter (dotted black arrows).

Funduscopy examination revealed evidence of fat emboli, which was in keeping with Purtscher's retinopathy. Echocardiography performed on day 9 excluded the presence of intracardiac shunt.

DIFFERENTIAL DIAGNOSIS

Diffuse axonal injury (DAI) is an imaging differential, although not considered clinically due to the presence of lucid interval. Other potential differentials include vitamin B₁ or B₁₂ deficiency, cerebritis complicated with microbleeds or incidental

cavernomas. These were excluded and diagnosis of CFE was considered due to overall clinical picture.

TREATMENT

Supportive treatment.

OUTCOME AND FOLLOW-UP

The patient had an uneventful recovery (including neurological status) and was discharged from hospital about 4 weeks after admission. He would receive follow-up by his general

Figure 3 MRI. Diffusion-weighted axial images (A–D) and ADC maps (E–H) showing restricted diffusion in genu (short white arrows), splenium of corpus callosum (long white arrows), internal capsule (black arrow) and deep white matter (dotted black arrows).

Figure 4 MRI. Susceptibility-weighted images (A–D) showing extensive punctate low signal areas throughout cerebral white matter, brainstem and cerebellum, the extent of which is significantly more than extent of abnormalities in [figures 2 and 3](#).

practitioner and ophthalmologist in outpatient clinic. No follow-up neuroimaging has currently been planned.

DISCUSSION

CFE is a serious complication of long bone fractures with an incidence of 0.9–2.2%.¹ Fat emboli from the bone marrow may reach the brain through a right-to-left cardiac shunt or through an intact pulmonary circulation in patients without a shunt. The exact pathogenesis of CFE is debated; there are broadly two hypotheses, namely mechanical and biochemical theories. The latter relates to the production of free fatty acids and other products that can incite local inflammatory response, platelet aggregation, endothelial damage and haemorrhages.² Fat emboli cause microinfarcts, vasogenic oedema and petechiae in the brain. Neurological symptoms may vary considerably, ranging from a subclinical presentation to confusion to coma and seizures and, in rare cases, death may result.³

The imaging diagnosis of CFE remains a challenge due to variable presentations and relatively small number of reported cases. Most often, CT of the head is normal despite encephalopathy or focal neurological deficits, as in this patient. A CT scan may show diffuse oedema with scattered low-attenuating areas and haemorrhage in some cases.

MRI of the brain is the most sensitive means of diagnosing CFE. The typical findings described so far are multiple small, scattered hyperintense lesions on T2WI or FLAIR images likely secondary to vasogenic or cytotoxic oedema, predominantly in the white matter, corpus callosum, basal ganglia, thalamus, brainstem and cerebellum.⁴ On DWI sequences, there may be restricted diffusion, particularly if there are microinfarcts with associated cytotoxic oedema. Microhaemorrhages could be detected on conventional T2*WI as hypointense lesions.

In this case, SWI revealed extensive punctate foci of low signal intensity in the cerebral and cerebellar white matter and corpus callosum. Their overall extent and distribution were far more extensive than other sequences including DWI, and involved more areas in the cerebral hemispheres and also cerebellum that were normal on other sequences. These were considered as petechiae, their distribution being consistent with the reported autopsy cases.⁵ It is well known that SWI is much more sensitive than T2*WI for detecting petechial haemorrhages.⁶ Possibility of calcifications was excluded by normal CT scan. It has recently been reported that fat and fat-water interface in lipomas can also have low signal on SWI due to chemical shift artefacts and these appearances can mimic blood products.^{7–9} We did wonder if some of these changes represented

Learning points

- ▶ Susceptibility-weighted imaging (SWI) is a recently introduced MRI technique that is exquisitely sensitive to blood products, iron and calcification.
- ▶ SWI can be very valuable in MRI protocols where presence, extent and distribution of blood products can affect imaging interpretation and diagnosis.
- ▶ Extensive minute or punctate hypointense foci in the cerebral and cerebellar white matter, and splenium of the corpus callosum on SWI, having a greater extent than diffusion-weighted imaging changes, in appropriate clinical circumstances, can be pathognomonic of cerebral fat embolism.

fat emboli in microcirculation, however, this possibility is less likely as the tiny size of fat emboli would be unlikely to be detected by SWI. In addition, the fat emboli are more likely to be lodged at grey-white matter junctions rather than current distribution. The high sensitivity and more widespread distribution of white matter abnormalities on SWI in comparison with other sequences could make SWI the most useful sequence for CFE, and this pattern of more widespread extent of SWI changes a pathognomonic feature of CFE.

The other traumatic injury accompanied by multiple microhaemorrhages is DAI, which is an imaging differential, however, it does not have a lucid interval. It was not considered in this case, since symptoms developed after a while. The distinctive radiological feature of DAI is that the changes are at cerebral grey-white matter junction, splenium of the corpus callosum and dorsolateral brainstem, whereas in CFE these are predominantly in the cerebral and cerebellar white matter and the splenium of the corpus callosum.

However, SWI has limited availability, is performed in selected cases in most centres and is not routinely included in the brain MRI scan protocols. In our case, SWI was carried out since it is part of our vascular protocols. We suggest that SWI

should be considered in routine use for patients requiring MRI of the head post-trauma or orthopaedic surgery and in vascular protocols. We are, however, uncertain if there is a relationship between the number and size of hypointense foci on SWI, and the degree of neurological disability or prognostic importance, as despite the extensive changes, the patient had a subsequent uneventful recovery.

In conclusion, SWI is useful in diagnosing CFE that shows multiple punctate hypointense foci in the cerebral and cerebellar white matter, and splenium of the corpus callosum, with distribution more extensive than DWI and other sequences. In appropriate clinical circumstances, this pattern can be pathognomonic of CFE.

Competing interests None.

Patient consent Obtained.

Provenance and peer review Not commissioned; externally peer reviewed.

REFERENCES

- 1 Suh SI, Seol HY, Seo WK, *et al.* Cerebral fat embolism—susceptibility-weighted magnetic resonance imaging. *Arch Neurol* 2009;66:1170.
- 2 Dash SK, Bansal A, Wankhade BS, *et al.* Alveolar hemorrhage in a case of fat embolism syndrome: a case report with short systemic review. *Lung India* 2013;30:151–4.
- 3 Simon AD, Ulmer JL, Strottmann JM. Contrast-enhanced MR imaging of cerebral fat embolism: case report and review of the literature. *AJNR Am J Neuroradiol* 2003;24:97–101.
- 4 Kuo K-H, Pan Y-J, Lao Y-J, *et al.* Dynamic MR imaging patterns of cerebral fat embolism: a systemic review with illustrative cases. *AJNR Am J Neuroradiol* 2014;35:1052–7.
- 5 Kamenar E, Burger PC. Cerebral fat embolism: a neuropathological study of a microembolic state. *Stroke* 1980;11:477–84.
- 6 Zaitse Y, Terae S, Kudo K, *et al.* Susceptibility-weighted imaging of cerebral fat embolism. *J Comput Assist Tomogr* 2010;34:107–12.
- 7 Lingegowda D, Rajashekar C, Belaval VV, *et al.* Susceptibility artifacts in lipomas. *Neurol India* 2013;61:56–9.
- 8 Schembri N, Kanodia AK, Nicholas RS, *et al.* Sylvian fissure lipoma associated with middle cerebral artery aneurysm—report of a rare case highlighting imaging pitfalls. *Int Neuropsychiatr Dis J* 2013;1:16–23.
- 9 Mehmed TM, Yamamoto A, Okada T, *et al.* Fat-water interface on susceptibility-weighted imaging and gradient-echo imaging: comparison of phantoms to intracranial lipomas. *AJR Am J Roentgenol* 2013;201:902–7.

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