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Clinical Article

Lesions identified on T2*-weighted gradient echo images in two patients with suspected diffuse axonal injury that resolved in less than ten days

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Summary

T2*-weighted gradient echo (GE) imaging is useful for detection of intracranial hemorrhage in the patients with diffusion axonal injury (DAI). However, the temporal changes in the DAI-related lesions on T2*-weighted GE images are not clear. We report two very rare cases with DAI in which lesions identified on T2*-weighted GE images resolved in less than ten days.

Introduction

Diffuse axonal injury (DAI) is a specific type of primary traumatic brain injury. Since evidence exists in the neuropathological literature that DAI is typically accompanied by small hemorrhages, or so-called tissue tear hemorrhages, we utilized the technique of magnetic resonance (MR) imaging to detect petechial hemorrhages in patients with DAI. T2*-weighted gradient echo (GE) imaging is useful for detecting intracranial hemorrhages. Magnetic susceptibility differences resulting from the presence of paramagnetic blood breakdown products create local magnetic field inhomogeneities, which manifest themselves as areas of marked hypo-intensity on T2*-weighted GE images. However, the temporal changes in the DAI-related lesions on T2*-weighted GE images are not clear. We herein report two cases with DAI in whom the lesions identified on T2*-weighted GE images resolved in less than ten days.

Case report

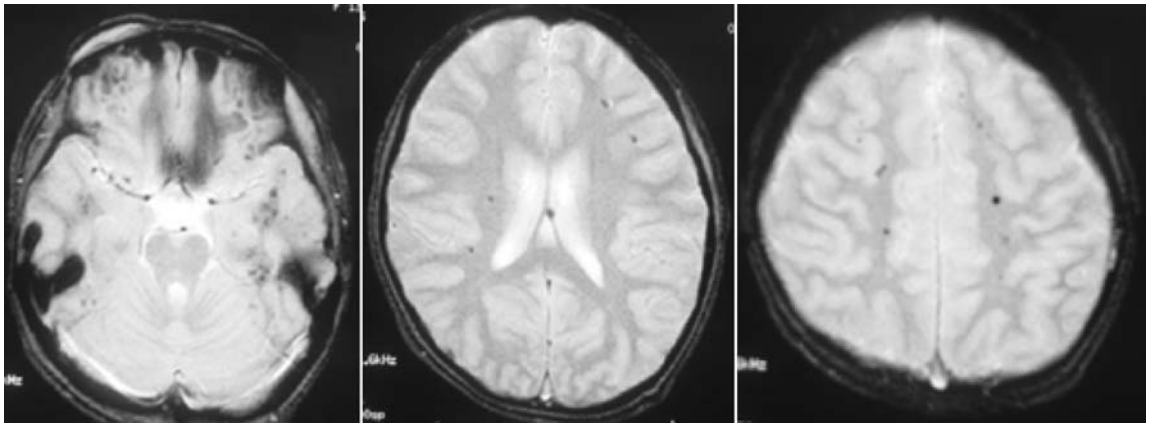
Case 1

A 13-year-old female sustained a severe closed head injury in a traffic accident. On admission, she was unconscious and demonstrated a score of 6 on the Glasgow Coma Scale (GCS). Computed tomography (CT) showed a slight subarachnoid hemorrhage in the interhemispheric fissure. Since the clinical status was worse than the CT findings, MR imaging was performed immediately. Multiple signal hypo-intensities in the gray-white matter junctions of the left temporal lobe and both fronto-parietal lobes were observed on T2*-weighted GE images (Fig. 1a). No lesions were found in the other sequences including T1, T2, fluid-attenuated inversion-recovery imaging, and diffusion-weighted imaging. Given her clinical status and the findings revealed by CT and MR images, the patient was suspected to have DAI and she was therefore initially treated conservatively.

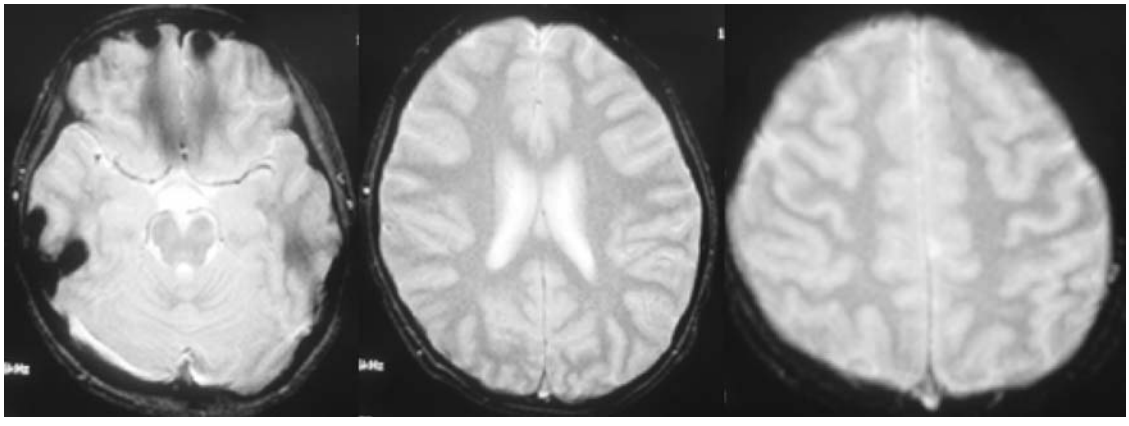
A week later, subsequent MR imaging was performed, and the patient was still unconscious at that time (GCS 6 = E2V1M3). Almost all lesions that had been identified on admission had either resolved or had become fainter on follow-up T2*-weighted GE images (Fig. 1b). In addition, no new lesions were detected in any other imaging sequences. Subsequently, the patient's status improved daily, and she was discharged 31 days after injury with only mild deficits in her cognition and memory.

Case 2

The second patient with DAI was a 22-year-old male who suffered a head injury in a car accident and was immediately transferred to our hospital. On admission the patient was unconsciousness and had a GCS score of 9 (E2V2M5). CT showed no apparent intracranial mass lesion due to unconsciousness. First MR imaging was obtained within 24 h after injury. Multiple signal hypo-intensities in the gray-white matter junctions and the left basal ganglia were observed on T2*-weighted GE



a



b

Fig. 1. The MR images of a 13-year-old female who was injured in a motor vehicle accident. (a) Multiple small foci showing signal hypo-intensity are shown at the corpus callosum, the white matter and the gray matter-white matter border on the T2*-weighted gradient-echo images on admission. The signal loss of the both frontal lobes seemed to be an artifact caused by the frontal bone. (b) Additional T2*-weighted GE images, taken one week after the injury, demonstrated the hypo-intense foci to have become substantially fainter

images (Fig. 2a). No abnormal lesions were found in the other sequences. The patient was diagnosed to have moderate DAI and was thus managed conservatively.

On the fourth day, he opened his eyes spontaneously. His neurological examination revealed almost normal findings except for mild somnolence. The serial MR imaging performed 10 days postinjury demonstrated partial resolution of the multiple lesions detected on admission on T2*-weighted GE images, especially those in the left frontal lobe (Fig. 2b). The other sequences showed no signal changes in the left frontal lobe and basal ganglia, in which hypo-intensities were demonstrated on the T2*-weighted GE images at onset. The patient was discharged 2 weeks after injury. At that time, he was alert and oriented with no cranial, motor or sensory nerve deficits.

Discussion

DAI is identified as one of the most important causes of morbidity and mortality in patients with traumatic brain injury [9]. In our cases, few lesions of the corpus callosum, brainstem and basal ganglia were observed, which would be the key hallmarks of a radiological definition of DAI. According to pathological study results [1], about 50% of DAI lesions are located in the deep white matter or the corticomedullary junction of the frontal and temporal lobes, although the corpus callosum, basal ganglia and brain stem are also commonly affected. Therefore, I diagnosed these two patients to have DAI according to the following criteria established by Gennarelli [6]; A) loss of consciousness from the time of injury that persisted beyond six hours. B) no cause of unconsciousness found other than the primary brain injury. C) no apparent intracranial mass lesion on CT.

CT and conventional MR imaging are known to underestimate the extent of DAI and correlate poorly with the final outcome [7, 8]. T2*-weighted GE images are useful for detecting microbleeding associated with DAI, which causes a marked signal loss due to magnetic susceptibility [5]. However, the correlation between the

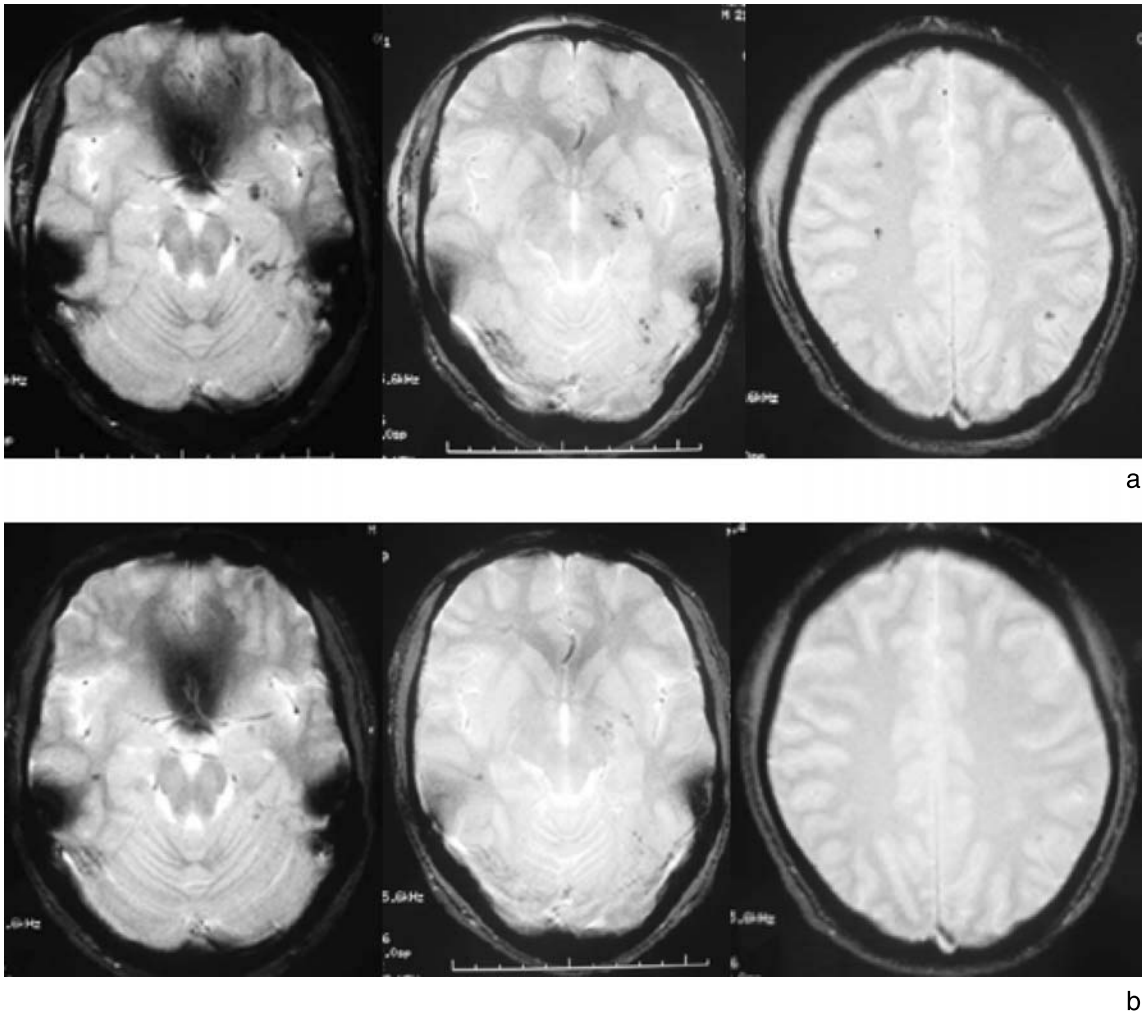


Fig. 2. The MR images of a 21-year-old male who was injured in a motor vehicle accident. (a) Multiple signal hypo-intensities in the gray-white matter junctions and the left basal ganglia were observed on T2*-weighted GE images within 24 h after injury. (b) T2*-weighted GE images showed partial resolution of the multiple lesions detected on admission, especially those of the left frontal lobe

hypo-intensities on T2*-weighted GE images and histopathological microbleeding is not absolute. Fazekas *et al*. reported the histopathological findings of microbleeding to be observed only in 21 of 34 areas with a signal loss on T2*-weighted GE images in 11 patients who had died of an intracranial hemorrhage [5].

To our knowledge, this is the first report of patients demonstrating a dynamic change in the T2*-weighted GE image findings suffering from DAI in the acute period. Because patients with an extremely severe degree of head injury are not stable enough to undergo emergency MR imaging, they are usually only evaluated by CT during the acute period, and MR imaging is normally performed in the subacute period or even later. Therefore, the pathological correlation with the temporal variation in the DAI-related lesions on T2*-weighted GE images is not clear.

There are numerous studies documenting the change in appearance on MR imaging of hemorrhage through acute and subacute to chronic stages [3], but none of these state whether any hemorrhages disappeared and none were systematic studies of a defined population, since all were imaged late after the event. Time-dependent changes of MR appearance of hemorrhage depend on several factors; the structure of hemoglobin and its various oxidation products, red blood cell membrane integrity and dehydration, hematocrit [3]. Hemoglobin from a traumatic hematoma is found in four main forms (oxyhemoglobin, deoxyhemoglobin, methemoglobin, and hemosiderin). Hyperacute hematomas are primarily composed of the oxygenated form of hemoglobin. Though oxyhemoglobin has little magnetic susceptibility effects, deoxyhemoglobin, methemoglobin, when intracellular, and hemosiderin have a susceptibility effect

resulting in a low signal on T2-weighted GE images. In our cases, follow-up MR images were performed in the early subacute stage (7–10 days after injury), at which time the oxidation of deoxyhemoglobin to methemoglobin takes place. Since methemoglobin may have less of a susceptibility effect than deoxyhemoglobin, the lesions detected on admission by T2-weighted GE images had either disappeared or became fainter. With the formation of methemoglobin, T1 is markedly shortened due to a dipole–dipole interaction, thus resulting in an increased intensity on the T1-weighted images. However, our follow-up MR imaging demonstrated no signal changes on the T1-weighted images.

Since the lesions detected on T2*-weighted GE images were very tiny in our cases, the partial volume effects with such tiny lesions obviously became lost in the slice thickness and slice gap. MR imaging was performed on 1.5-T scanners. We obtained T2*-weighted GE images in the axial plane with the following parameters; 700/25/20/2 [TR/TE/flip angle/excitations], field of view 22 cm, acquisition matrix 256×224, 6-mm slice thickness with 2-mm gap. We carefully made radiographs in the same manner using the same 1.5 T scanner and the same developer. Although exactly identical T2*-weighted MR imaging slices could not be made, we simultaneously investigated the hypo-intense foci from the upper and lower slices to compare the condition and number of the hypo-intense foci.

Since it is difficult to assume that the paramagnetic blood degradation products caused by DAI could have disappeared within only a week, the MR images may thus have detected some other type of morphological changes in our cases. It must be noted that the signal intensity loss on T2*-weighted GE images is not specific for hemorrhages and it can also be caused by calcification, ferritin, air, and paramagnetic contrast agents [2, 4]. Considering that shear injuries are typically not situated on the cortical surface or at contusion sites close to the skull base, this nonspecificity is not a main limitation for the evaluation of DAI. Other causes of the signal intensity loss on T2*-weighted GE images could be a fat embolism from a skull fracture or some other systemic injury, air bubbles from a compound fracture, and multiple cerebral hemorrhage from medical complications after brain injury, such as hematological disorders or sepsis or side effects of medical treatment [11]. Although no such complications were observed in our patients, their relevance remains unclear.

As a result, even though several reports have shown a good correlation between the MR sequence findings, such as T2*-weighted GE and DAI, and the clinical prognosis of DAI [10], one limitation of these studies is the times that MR imaging could be performed tended to vary greatly. Serial changes in the signals on MR imaging might occur over time, and such changes need to be confirmed by longitudinal studies.

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Comment

Lesions identified on L2*-weighted gradient echo images in two patients with suspected diffuse axonal injury that resolved in less than 10 days.

The authors report on a 13 year-old girl in a coma after head injury. gradient-echo T2*-weighted images showed some hypointense lesions, that had disappeared one week later. As there are only very few reports on early MRI after head injury, any report especially on repeat MRI is important.

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