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Case Report

Subarachnoid hemosiderin deposition after subarachnoid hemorrhage on T2*-weighted MRI correlates with the location of disturbed cerebrospinal fluid flow on computed tomography cisternography

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A 72-year-old male was admitted with subarachnoid hemorrhage associated with a ruptured cerebral aneurysm. The aneurysm was treated with clipping soon after radiological examination. Eight weeks after the treatment, the patient suffered from secondary hydrocephalus resulting from blockage of the subarachnoid space due to subarachnoid granulation. Previous pathological examination revealed the granulation was associated with hemosiderin deposition. We investigated subarachnoid hemosiderin deposition in this patient using T2*-weighted (T2*-w) magnetic resonance image (MRI), a sensitive method for hemosiderin detection. computed tomography (CT) cisternography demonstrated that cerebrospinal fluid (CSF) flow was disturbed adjacent to sites of subarachnoid hemosiderin deposition on T2*-w MRI. Placement of a ventriculo-peritoneal shunt contributed to neurological improvement. In this case, T2*-w MRI was an effective means of diagnosing the location of disturbed CSF flow associated with subarachnoid hemosiderin deposition.

Key words: Computed tomography cisternography, hemosiderin, hydrocephalus, magnetic resonance image, subarachnoid hemorrhage

Secondary hydrocephalus can arise from a disturbance in cerebrospinal fluid (CSF) flow after cerebral aneurysm rupture and subarachnoid hemorrhage (SAH).[1-3] Secondary hydrocephalus might be associated with residual subarachnoid hematoma and blockage of the subarachnoid space due to granulation.[4-6] We reported previously that secondary hydrocephalus was observed only in patients with extended hemosiderin-positive subarachnoid spaces after SAH on gradient echo T2*-weighted (T2*-w) magnetic resonance image (MRI),[4] which is a sensitive method for the detection of hemosiderin.[7] In this case report, the findings suggested that subarachnoid hemosiderin deposition after SAH might be associated with disturbances in CSF flow and secondary hydrocephalus.

Case Report

A 72-year-old male was admitted with sudden onset of headache and altered consciousness. Glasgow coma scale was 10 (E2, V2, M6) on admission and a ruptured anterior communicating artery aneurysm was visualized by radiography. Soon after admission, an aneurysmal clip was placed via an interhemispheric approach with frontal craniotomy and a ventricular drainage and a cisternal drainage around an aneurysm were placed, followed by lumbar CSF drainage until three weeks after the onset. Eight weeks after symptom onset, the patient experienced secondary hydrocephalus with consciousness disturbance and urinary incontinence. The figure demonstrates that subarachnoid hemosiderin deposition [Figures 1D,E,F] eight weeks after the onset of SAH was well-correlated with the location of subarachnoid hematoma on initial computed tomography (CT), in particular the left Sylvian fissure and sulci of the left temporo-parietal area [Figures 1A,B,C]. Additionally, nine weeks after the onset of SAH, CT cisternography performed 22 h after the intrathecal injection of 10 ml 51.3% iotrolan (Isovist, SCHERING) demonstrated restriction of iotrolan to the ventricular system [Figures 1G, H, I] without diffusion into the hemosiderin-rich subarachnoid space [Figure 1D, E, F]. Except ventricle, Sylvian fissure and cistern, there were significant relationships among hematoma-rich subarachnoid

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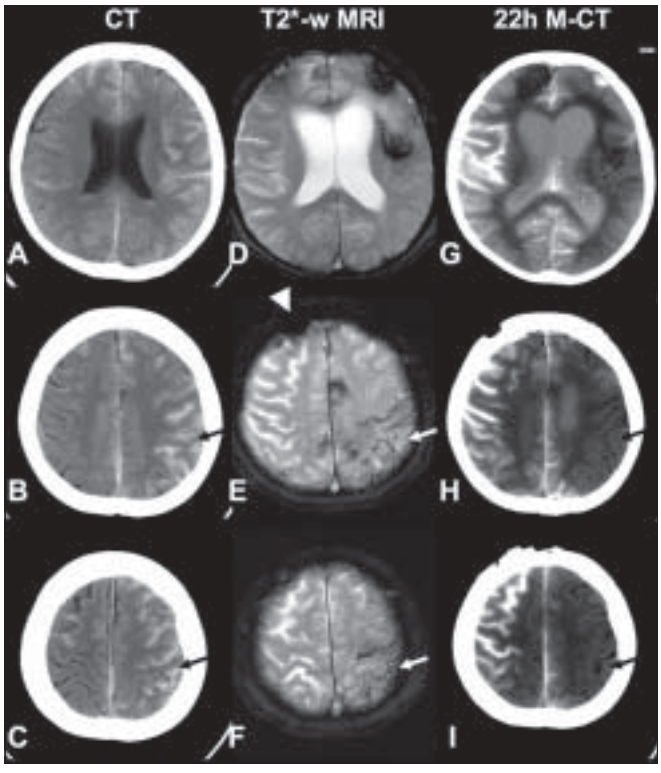

Figure 1: The relationship between subarachnoid hemosiderin deposition and disturbed cerebrospinal fluid flow. Arrows demonstrate hematoma-rich subarachnoid spaces on admission

space, hemosiderin-rich space and iotrolan-free space (arrows). Conversely, the ventricles, Sylvian fissure and cisterns rarely displayed hemosiderin accumulation on T2*-w MRI, despite the subarachnoid hematoma demonstrated on admission CT. A ventriculo-peritoneal shunt was placed and marked neurological improvement occurred.

The MRI was performed on a 1.5-T scanner. We obtained axial T2*-w MRI with the following parameters: 450/26/2 (TR/TE/excitations), flip angle of 20º, section thickness of 8 mm without gaps and matrix of 256×205. We checked the corresponding areas with hemosiderin deposits by CT, fluid-attenuated inversion recovery images, T1- and T2-weighted MRI and ruled out cerebral calcifications, calcified cerebral arteries and cerebral arterial and venous flow voids as causes of the low signal intensity on T2*-w MRI.

Discussion

We previously demonstrated that the extent of subarachnoid hemosiderin deposition after SAH was associated with secondary hydrocephalus.[4] In this case report, CT cisternography suggested that subarachnoid hemosiderin deposition on T2*-w MRI might be associated with disturbed flow of CSF.

Low intensity on T2-w MR image*

Areas of low intensity on T2*-w MRI might represent deoxyhemoglobin in the acute phase of intracerebral hemorrhages and hemosiderin in the chronic phase.[8] In this case, the subarachnoid low intensity signal on T2*-w MRI eight weeks after the onset of SAH was identified as hemosiderin rather than deoxyhemoglobin and this was confirmed by pathological examination.[7] A low intensity signal on T2*-w MRI was not specific for hematoma, however. Other causes might include calcification, ferritin, melanoma, air and some paramagnetic contrast agents.[8] With the exception of ferritin, these causes could be excluded as the source of the low intensity signal in this case. Ferritin in glial cells and macrophages was found to have a wider distribution than hemosiderin in macrophages in a histopathologic biochemical study of intracerebral hematomas in animals.[9] However, previous pathological examinations did not observe ferritin deposition after SAH.

Subarachnoid granulation and CSF flow

Subarachnoid granulation was observed following SAH and the granulation core contained channels through which CSF flows.[6] These channels were partially filled with red blood cells.[6] Clusters of hemosiderin-positive macrophages, which were related to the inflammation and the formation of granulation tissue, could be seen in the meninges four weeks after SAH onset.[5] Iron-positive cells existed in the CSF up to four months after SAH.[10] T2*-w MRI demonstrated that the subarachnoid hemosiderin deposition remained more than one year after the onset of SAH.[4] The granulation with hemosiderin deposition might remain long after SAH resolution[4-6] while continuing to disturb CSF flow.

In the present case, the ventricles and basal cisterns did not display significant hemosiderin accumulation, suggesting that greater CSF flow might disturb accumulation.[4] Hemosiderin deposition may have been accelerated by complicated or narrow adjacent structures, which trap blood and thus result in poor washout.[4]

Low intensities on T2*-w MRI were associated both with the amount of subarachnoid blood and the blood in peripheral cortical sulcal location on admission CT. The CSF flow was not seen surrounding the low intensity lesions on T2*-w MRI and it remains possible that the locations of hemosiderin deposition are correlated with areas of CSF channel narrowing secondary to subarachnoid granulation.[4-6] The poor CSF flow induced by subcortical granulation, which located in same place of hemosiderin deposition, was associated with hydrocephalus. While we present only one patient, this study strongly supports the use of T2*-

w MRI for the diagnosis of subarachnoid granulation with disturbed CSF flow.

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Accepted on 21-07-2007
Source of Support: Nil, **Conflict of Interest:** None declared.