Detection of Hemosiderin Deposition by T2*-Weighted MRI After Subarachnoid Hemorrhage

Toshio Imaizumi, MD, PhD; Masahiko Chiba, MD, PhD; Toshimi Honma, MD; Jun Niwa, MD, PhD

Background and Purpose—Subarachnoid hemorrhage (SAH) is very difficult to diagnose several months after its onset. We thus investigated subarachnoid hemosiderin deposition well after SAH by T2*-weighted MRI, a sensitive method for hemosiderin detection.

Methods—To investigate how hemosiderin deposition as confirmed by T2*-weighted MRI contributes to the determination of prior SAH and how the extent of hemosiderin deposition is associated with a number of clinical factors, we retrospectively analyzed 58 patients ≥3 months after SAH associated with ruptured aneurysms. We also investigated 209 healthy volunteers as controls.

Results—T2*-weighted MRI demonstrated subarachnoid hemosiderin deposition in 72.4% of the SAH patients, whereas no deposition was seen in the healthy volunteer group. The hemosiderin was preferentially deposited in the subarachnoid space near a ruptured aneurysm. Odds ratios (ORs) were estimated from logistic regression analyses correlating hemosiderin deposition with other factors. Age (≥54 years) (OR, 5.1; 95% CI, 1.03 to 25.0; \(P=0.046\)), Fisher grade 3 on initial CT (OR, 8.0; 95% CI, 1.26 to 50.4; \(P=0.027\)), and Karnofsky Scale score ≥80% 6 months after onset of SAH (OR, 12.8; 95% CI, 1.97 to 83.3; \(P=0.0077\)) were all found to be independently associated with hemosiderin deposition levels.

Conclusions—T2*-weighted MRI is an effective means of diagnosing prior SAH and may also reveal the location of a ruptured aneurysm. The extent of hemosiderin deposition was significantly associated with several factors, including age, CT findings, and poor prognosis. (Stroke. 2003;34:1693-1698.)

Key Words: diagnosis ■ hemosiderin ■ magnetic resonance imaging ■ subarachnoid hemorrhage

A recent international study revealed that unruptured cerebral aneurysms, especially small ones, seldom rupture and that clipping of these unruptured aneurysms is not recommended universally.1 However, if the cerebral aneurysm is known to have ruptured, clipping or embolization is necessary because of the high rate of aneurysm rerupture.2,3 Therefore, determining whether or not an aneurysm has bled has tremendous prognostic significance. In some patients, however, this determination is not a simple one to make. It is easy to diagnose subarachnoid hemorrhage (SAH) by CT soon after onset of symptoms; a diffuse, thick, high-density area in the basal cisterns on the second CT, however, reverts to an isodensity profile as soon as 24 hours after the first CT in some patients.4 The probability of recognizing an aneurysmal hemorrhage on CT is 85% after 5 days, 50% after 1 week, 30% after 2 weeks (mostly patients with hematomas), and almost 0% after 3 weeks.5 If the diagnostic CT findings are obscure, cerebrospinal fluid (CSF) examination may provide more relevant clinical information. Ito and Inaba6 demonstrated that SAH could be diagnosed by searching for iron-positive cells in CSF up to 4 months after the onset of SAH. Despite this finding, it remains difficult to diagnose SAH retrospectively on examination several months after the acute event.

On gradient-echo T2*-weighted MRI, signal loss can represent hemosiderin, calcification, physiological ferritin, melanoma, air, and some paramagnetic contrast agents.7 In particular, T2*-weighted MRI is regarded as a sensitive method for the detection of hemosiderin deposition.8–11 For example, hemosiderin may be detected as an area of signal loss on T2*-weighted images several years after the occurrence of intracerebral hemorrhage (ICH). Detection of an old SAH in this way would prove quite beneficial in assisting with therapy decisions that rely on this prior history. Moreover, we thought it likely that the extent of hemosiderin deposition observed long after acute SAH would be related to other factors, including the CT findings on admission and the prognosis. To this end, we studied the location and level of hemosiderin deposition in 58 patients with previously ruptured cerebral aneurysms. Our results reveal that prior SAH may be diagnosed by T2*-weighted MRI and that the extent of subarachnoid hemosiderin deposition is significantly associated with several factors, including age, initial CT findings, and prognosis.
Subjects and Methods

Subjects
In this study we excluded patients with SAH associated with moyamoya disease, primary ICH, arteriovenous malformation, or head trauma, leaving 97 patients who were admitted to our hospital from November 1999 to February 2002 with SAH associated with ruptured cerebral aneurysm. Of these 97, we excluded 3 patients with pacemakers or other metal objects that put them at risk during MRI, 20 patients who survived <6 months, 7 patients who were moved to other hospitals, and 2 patients with past history of brain surgery. We also excluded 1 patient who experienced massive hemorrhage in the lateral ventricles and third ventricle during a surgical operation for a ruptured aneurysm. With the exclusion of those patients, 58 of the 64 remaining ruptured aneurysms were in the anterior portion of the circle of Willis and were treated by aneurysmal clipping. During neurosurgical operations with craniotomies, we observed in all patients subarachnoid hematomas and ruptured aneurysms that were surrounded by relatively hard hematomas and had rupture points. We did not completely remove subarachnoid hematomas with surgical treatment.

The patients examined in the study consisted of 25 men and 33 women aged 23 to 79 years (mean age, 55.6 ± 14.5 years). The ruptured aneurysms were located in the middle cerebral artery in 27 patients, the anterior communicating artery (AComA) in 14 patients, the internal carotid artery in 12 patients (lesions in the posterior communicating artery in 10 and anterior choroidal artery in 2), and the distal portion of the anterior cerebral artery (ACA) in 5 patients. As a control, we also investigated 209 healthy volunteers in our hospital (106 men and 103 women aged 38 to 79 years; mean age, 56.4 ± 8.3 years) using “Brain Dock,” a formalized screening system for asymptomatic brain diseases that is popular in Japan.12

Radiological Examination
Of the 58 patients, 55 were diagnosed with SAH by plain CT. SAH was diagnosed definitively in 3 patients by CSF examination. Fisher grade was determined by initial CT.13

MRI was performed on a 1.5-T scanner 3 to 18 months after the onset of SAH. We obtained axial T2*-weighted images with the following parameters: repetition time (TR)/echo time (TE)/excitations 450/26/2, flip angle of 20°, section thickness of 10 mm without gaps, and matrix of 256 × 256. Axial spin-echo T1-weighted images (TR/TE/excitations 400/14/2), fast spin-echo T2-weighted images (TR/TE/excitations 4000/133/2; turbo factor, 15) and fluid-attenuated inversion recovery images (FLAIR) (TR/TE/excitations 8000/115/1) were also obtained with the same section thickness and matrix.

Hemosiderin deposits that had developed from hematomas were observed in subarachnoid spaces, cisterns, and the ventricular system as focal areas of signal loss (linear, curvilinear, and spotty low intensity) on T2*-weighted images. We checked the corresponding areas by CT and ruled out cerebral calcifications and calcified cerebral arteries as causes of the T2*-weighted low signal intensity. In addition to T2*-weighted MRI, FLAIR, T1-, and T2-weighted MRI were performed to identify hemosiderin deposits from cerebral arterial and venous flow voids.

Statistical Analysis
We divided the subarachnoid area of each cerebral hemisphere into 5 regions (frontal, temporal, occipital, parietal, and sylvian fissure) and determined the number of regions, from 0 to 10, displaying hemosiderin deposits, excluding the ventricular system and cisternae in which hemosiderin was rarely deposited. The SAH group was then divided into 2 subgroups according to this number (≥4) (described in detail in Results) on the basis of the hypothesis that the extent of hemosiderin deposition would be associated with the severity of SAH.

Age, Glasgow Coma Scale (GCS)14 and Hunt and Hess score15 grading of consciousness level at admission, and performance status (Karnofsky Scale score 6 months after onset of SAH)16 were considered continuous variables (mean ± SD values of specific parameters [as appropriate]). The Student t test was applied to compare ages, and the Mann-Whitney U test was used to compare GCS score, Hunt and Hess score, and Karnofsky Scale score data. Probability values <0.05 were considered statistically significant.

For univariate analyses (χ2 analysis), overall frequencies were compared by dichotomous variable statistics for the following categorical variables (Table 1): age (≥54 years); sex; GCS score on admission (≥14); Hunt and Hess score (≥3); walking SAH (the patient walked to our hospital with some symptoms, including nausea, vomiting, and headache); Fisher grade (grade 3)13; location of aneurysm rupture (distal ACA); ICH on initial CT; intraventricular hematoma on initial CT; interval between onset of SAH and aneurysm clipping (<3 days); acute hydrocephalus requiring ventricular drainage before aneurysm clipping; cisternal drainage; Terson’s syndrome; symptomatic vasospasm including transient neurological deficits; secondary hydrocephalus requiring a ventriculoperitoneal CSF shunt; seizure; Karnofsky Scale score (≥80%) 6 months after onset of SAH; and interval between SAH onset and T2*-weighted MRI (<4 months).

The thresholds for dichotomous variables were set at the point exhibiting the smallest difference between the sensitivity and specificity of the variables. Probability values <0.05 were considered statistically significant.

Multivariate logistic regression analyses were performed (Table 2), initially including all variables in the model and then excluding variables that were not applicable in a stepwise manner. Independent

<table>
<thead>
<tr>
<th>TABLE 1. Univariate Analysis of Factors Associated With Extent of Hemosiderin Deposition as Measured by T2*-Weighted MRI</th>
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<tbody>
<tr>
<td><strong>Hemo Regions</strong></td>
</tr>
<tr>
<td>≤3</td>
</tr>
<tr>
<td>No. of cases</td>
</tr>
<tr>
<td>Age</td>
</tr>
<tr>
<td>≥54 years (≥)</td>
</tr>
<tr>
<td>Sex (F/M)</td>
</tr>
<tr>
<td>GCS</td>
</tr>
<tr>
<td>GCS ≥14 (≥)</td>
</tr>
<tr>
<td>Hunt and Hess score</td>
</tr>
<tr>
<td>Hunt and Hess score ≥3 (≥)</td>
</tr>
<tr>
<td>Walking SAH (≥)</td>
</tr>
<tr>
<td>Fisher grade 3 (≥)</td>
</tr>
<tr>
<td>Distal ACA (≥)</td>
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<tr>
<td>Intraventricular hematoma (≥)</td>
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<tr>
<td>Intraventricular hematoma (≥)</td>
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<tr>
<td>Interval (onset-ope ≥3 d (≥))</td>
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<tr>
<td>Acute hydrocephalus (≥)</td>
</tr>
<tr>
<td>Cisternal drainage (≥)</td>
</tr>
<tr>
<td>Terson’s syndrome (≥)</td>
</tr>
<tr>
<td>Vasospasm (≥)</td>
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<tr>
<td>Secondary hydrocephalus (≥)</td>
</tr>
<tr>
<td>Seizure attack (≥)</td>
</tr>
<tr>
<td>Karnofsky scale, %</td>
</tr>
<tr>
<td>Karnofsky scale ≤80% (≥)</td>
</tr>
<tr>
<td>Interval (onset- MRI) &lt;4 mo</td>
</tr>
</tbody>
</table>

Hemo Regions: number of hemosiderin-positive regions (described in text in detail). GCS indicates Glasgow Coma Scale; SAH, subarachnoid hemorrhage; ACA, anterior cerebral artery; ope, surgical operation.

*Student t test.
†Mann-Whitney U test.
‡P < 0.05.
variables assessed by multivariate analyses were as follows: age (≥54 years); sex; GCS score on admission (≤14); walking SAH; Fisher grade (grade 3); location of aneurysm rupture (distal ACA); ICH; intraventricular hematoma; interval between onset of SAH and aneurysm clipping (≥3 days); acute hydrocephalus; cisternal drainage; Terson’s syndrome; symptomatic vasospasm; secondary hydrocephalus; seizure; Karnofsky Scale score (≥80%); and interval between SAH onset and T2*-weighted MRI (≥4 months). Where applicable, 95% CIs were calculated for the estimated odds ratios (ORs) (Table 2). P<0.05 was also considered statistically significant in these tests. All statistical analyses were performed with the use of Statview 5.0 (SAS Institute Inc).

**Results**

**Hemosiderin Deposits**

The Figure demonstrates that subarachnoid hemosiderin deposition, as measured by T2*-weighted MRI 5 months after onset of SAH in walk-in patients suffering from headache and nausea, was well-correlated with the location of subarachnoid hematoma on initial CT. T2*-weighted MRI, however, revealed little hemosiderin deposition in cisterns, even though there was significant subarachnoid hematoma seen in the cistern system by CT.

T2*-weighted images demonstrated hemosiderin deposition in subarachnoid areas of 42 of the 58 patients (72.4%). In 3 patients, CT could not detect SAH well on admission. In 2 of these patients, however, T2*-weighted MRI detected hemosiderin deposition in the subarachnoid space 4 months after the onset of SAH. The incidences of hemosiderin-positive regions in subarachnoid space on T2*-weighted MRI were 76.0% (19/25 patients), 66.7% (8/12), and 76.2% (16/21) at 3 to 4, 4 to 12, and 12 to 18 months after SAH, respectively; no significant difference existed between any of these 3 groups. Hemosiderin deposition was found to be increasingly widespread in these patients; however, the specific regions affected varied from case to case. Of 58 patients, deposition was found in the frontal (41.3%), temporal (39.7%), parietal (43.1%), and occipital areas (20.7%) and in the sylvian fissure (65.5%). T2*-weighted MRI showed that the hemosiderin was deposited preferentially in the cortical sulcus and sylvian fissure but also in small foci in the ventricular (6.9%) and cisternal systems (1.7%). Hemosiderin deposition was positive in both cerebral hemispheres in 35 patients. In 6 of 7 patients with hemosiderin deposition in only 1 hemisphere, ruptured aneurysms were found on the ipsilateral side.

The laterality of hemosiderin deposition was calculated as the difference in the number of hemosiderin-positive regions in each cerebral hemisphere. By this method, hemispheric laterality of hemosiderin deposition was positive in 9 of 35 patients. Five of 9 ruptured aneurysms were located on the side of greater hemosiderin deposition, while 2 were on the opposite side and the remaining 2 were aneurysms located in the AComA. T2*-weighted images of patients with ruptured CT on admission and T2*-weighted MR image 5 months after onset of SAH. A 60-year-old man walked into our hospital with sudden onset of headache and nausea. A right middle cerebral artery aneurysm was detected and treated with a tuit aneurysmal clip. The marked artifact of the clip, the tuit plates used for a cranioplasty, and air sinuses prevent the T2*-weighted image from demonstrating hemosiderin deposition. Subarachnoid hemosiderin deposition is associated with the hematoma but is barely visible in the cisterns and ventricular system.

### Table 2. Multivariate Analysis of Factors Associated With the Extent of Hemosiderin Deposition as Measured by T2*-Weighted MRI

<table>
<thead>
<tr>
<th>Factor</th>
<th>Odds Ratio</th>
<th>95% CI</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>54≥ years</td>
<td>5.1</td>
<td>1.03–25.0</td>
<td>0.046*</td>
</tr>
<tr>
<td>Sex, F</td>
<td>0.32</td>
<td>0.06–1.72</td>
<td>0.185</td>
</tr>
<tr>
<td>GCS ≤14</td>
<td>0.66</td>
<td>0.09–4.96</td>
<td>0.684</td>
</tr>
<tr>
<td>Walking SAH</td>
<td>0.89</td>
<td>0.10–7.80</td>
<td>0.922</td>
</tr>
<tr>
<td>Fisher grade 3</td>
<td>7.97</td>
<td>1.26–50.4</td>
<td>0.027*</td>
</tr>
<tr>
<td>Acute hydrocephalus</td>
<td>2.99</td>
<td>0.45–19.8</td>
<td>0.256</td>
</tr>
<tr>
<td>Cisternal drainage</td>
<td>2.66</td>
<td>0.33–21.3</td>
<td>0.356</td>
</tr>
<tr>
<td>Karnofsky scale ≤80%</td>
<td>12.8</td>
<td>1.97–83.3</td>
<td>0.008*</td>
</tr>
<tr>
<td>Interval (onset–MRI) &lt; 4 mo</td>
<td>3.38</td>
<td>0.69–16.4</td>
<td>0.130</td>
</tr>
</tbody>
</table>

GCS indicates Glasgow Coma Scale; SAH, subarachnoid hemorrhage.

*P<0.05.
AComA aneurysms showed either hemosiderin deposition in both hemispheres or no deposition. We also analyzed the side of surgical approach (trans-sylvian approach with frontotemporal craniotomy) of ruptured AComA aneurysms and the location of hemosiderin. Fourteen ruptured AComA aneurysms were clipped; 8 were done with a right trans-sylvian approach, and 6 were performed by means of a left trans-sylvian approach. T2*-weighted MRI demonstrated hemosiderin deposition in 9 of these 14 patients. In 2 of these 9, T2*-weighted images demonstrated clear laterality of hemosiderin deposition. In one case the deposits were ipsilateral to the side of approach; in the other they were contralateral, suggesting that hemosiderin deposition cannot be attributed to surgical intervention. T2*-weighted images of every patient with ruptured distal ACA aneurysms showed hemosiderin deposition in both hemispheres, especially in interhemispheric areas. In general, hemosiderin deposition was preferentially located in regions proximal to the ruptured aneurysms.

It is also noteworthy that T2*-weighted MRI revealed hemosiderin deposition in 7 of 10 patients with walking SAH. In contrast, hemosiderin deposition was never detected in the healthy volunteer group. The hemosiderin deposition seen in the subarachnoid areas on T2*-weighted images was very specific for SAH.

**Univariate Analyses**

Univariate analyses are shown in (Table 1). An important goal of this study was to detect old SAH. We assumed that patients with old SAH detected by T2*-weighted MRI several months after the acute event presented with severe symptoms at first. A decrease in Karnofsky Scale score was found in this study to correlate strongly with an increase from 3 to 4 hemosiderin-positive regions in these post-SAH MRI images (data not shown). Vasospasms and secondary hydrocephalus were observed in patients with ≥4 hemosiderin-positive regions, described below. Therefore, we decided for this study to use ≥4 hemosiderin-positive regions as a cutoff for statistical analyses relating prognosis and other associated factors. The SAH group was divided into 2 subgroups according to the number (≥4) of hemosiderin-positive regions. The mean age of the group with ≥4 hemosiderin-positive regions was 59.3±13.8 years, which was not significantly different from that of the group with <4 positive regions (aged 52.5±14.6 years; P=0.076). The Karnofsky Scale score of the latter group was 92±15%, which was significantly higher than that of the former group (73±26%; P=0.0004). There was no significant difference in GCS score on admission (Table 1).

We demonstrated on \( \chi^2 \) analysis that distal ACA (P=0.0095), Fisher grade 3 (P=0.0095), symptomatic vasospasm (P=0.0041), secondary hydrocephalus (CSF shunt system) (P=0.0041), and Karnofsky Scale score (≥80%) (P=0.0004) were significantly associated with the extent of hemosiderin deposition (Table 1).

Univariate logistic regression analysis demonstrated that T2*-weighted MRI at 4 months is 59.4% sensitive but 60.0% specific for prior SAH.

**Multivariate Analyses**

Multivariate analyses are shown in (Table 2). ORs were estimated by logistic regression analysis with the use of the data relating hemosiderin-positive regions to other factors. Walking SAH, distal ACA, intracerebral hematoma, intraventricular hematoma, interval between onset of SAH and aneurysmal clamping, Terson’s syndrome, symptomatic vasospasm, secondary hydrocephalus, and seizure were not applicable for multivariate logistic regression analyses because of the small number of patients with these factors. On the other hand, age (≥54 years) (OR, 5.1; 95% CI, 1.03 to 25.0; P=0.046), Fisher grade 3 (OR, 8.0; 95% CI, 1.26 to 50.4; P=0.027), and Karnofsky Scale score ≥80% (OR, 12.8; 95% CI, 1.97 to 83.3; P=0.0077) were independently associated with the extent of hemosiderin deposition (Table 2). Multivariate analysis demonstrates a sensitivity value of 83.9%, a specificity value of 80.8%, a positive predictive value of 83.9%, a negative predictive value of 80.8%, and predictive accuracy value of 82.5%.

**Discussion**

The clinical significance of subarachnoid hemosiderin deposition after SAH resulting from a ruptured cerebral aneurysm has not been reported. In this study we show that several factors are associated with the extent of hemosiderin deposition.

**Mechanisms of Hemosiderin Deposition**

Arachnoid granulation is observed after SAH, and the granulation core contains channels through which CSF flows. These channels are partially filled with red blood cells. Clusters of iron-laden macrophages can be seen in the basal meninges and are associated with even minor leakage preceding SAH. This suggests that granulation and channel formation may be associated with hemosiderin deposition. Shunt-dependent hydrocephalus, as a late consequence of blockage of the basal cistern and subarachnoid space related to arachnoid granulation, might be associated with more severe hemosiderin deposition in this study, as shown by univariate analysis.

The washout of subarachnoid hematoma fluid during a surgical operation may have a prominent effect on the extent of hemosiderin deposition. We also expected that postoperative CSF drainage (cisternal and ventricular drainages) would accelerate the clearance of blood from the subarachnoid space. Indeed, CSF drainage is typically indicated if an initial CT shows substantial subarachnoid hematoma and/or hydrocephalus. Our results demonstrate, however, that CSF drainage does not reduce the resulting number of hemosiderin-positive areas. It is noteworthy that the incidence of deposition is high in the parietal areas even though this area is distant from the ruptured aneurysms. Most deposition was found to accumulate preferentially in areas near the ruptured aneurysm; among patients with distal ACA aneurysms, for example, 100% showed deposition in the frontal area. On the other hand, ventricles and cisterns close to the site of rupture only very rarely displayed hemosiderin accumulation. These results suggest that abundant CSF flow may disturb deposition, whereas trapped hematoma fluid may promote it.
other words, hemosiderin deposition may be accelerated by complicated or narrow adjacent structures that trap blood and thus result in poor washout or by injuries to the subarachnoid space or cortex caused by the aneurysm rupture. Furthermore, long-lasting blood clots and thick layers of blood (Fisher grade 3) are associated with severe vasospasm,19,20 which might be correlated with hemosiderin deposition in this study, as shown by univariate analysis. Independently, more severe hemosiderin deposition was found in SAH patients with Fisher grade 3.

Coil embolization therapy was not performed for ruptured aneurysms of anterior circulation in our hospital. Some aneurysms of posterior circulation were embolized by a platinum coil. Although there is a difference in the location of ruptured aneurysms, T2*-weighted MRI demonstrated more hemosiderin deposition in the subarachnoid spaces and cisterns with aneurysms treated by coil embolization than aneurysms treated with clipping and craniotomy; craniotomy and aneurysmal clipping with or without cisternal and ventricular drainage may decrease hemosiderin deposition.

From our univariate data analysis, we found that both symptomatic vasospasm and secondary hydrocephalus might be associated with the extent of hemosiderin deposition, but these effects may not be independent. Further study is necessary to determine how symptomatic vasospasm and secondary hydrocephalus, which are relatively rare in this study, act as independent factors in their correlation with increased hemosiderin deposition. If the hemosiderin deposition was detected in the early stage after SAH, the symptomatic vasospasm and secondary hydrocephalus might be predicted by the extent of the deposition.

Age (≥54 years) is also independently associated with the extent of hemosiderin deposition. We guess that the enlargement of the subarachnoid space by cortical atrophy associated with aging may be related to the extent of subarachnoid hematoma followed by the extent of hemosiderin deposition and that biophysiological activity associated with aging may be related to washout of the hematoma.

**Diagnosis of Old SAH**

Areas of low intensity on T2*-weighted MRI may represent deoxyhemoglobin in the acute phase of ICH and hemosiderin in the chronic phase19,20; low intensity on T2*-weighted MRI, however, is not specific for hemorrhage. Other causes include calcification, physiological ferritin, melanoma, air, and some paramagnetic contrast agents.7 Ferritin in glial cells and macrophages was found in a histopathologic biochemical study of hematoma in animals to have a wider distribution than hemosiderin in macrophages around an intracerebral hematoma.21 The existence of hemosiderin was pathologically confirmed.17,18 It is possible that iron is stored in the subarachnoid area in the form of ferritin; however, previous pathological examinations did not mention ferritin deposition after SAH. Further pathology studies are needed to demonstrate conclusively that the low intensity seen in the subarachnoid space is associated with ferritin deposition.

A possible source of bias in our study is the selective enrollment in Brain Dock, a medical examination of the brain or screening for asymptomatic brain disease by MRI, ECG, laboratory tests, ophthalmological examination, and other methods, conducted in Japan. Some employers recommend that employees submit to these examinations, but the decision is ultimately voluntary; individuals tend to vary in their concern, leading to inhomogeneities and self-selection in this screening process. Therefore, we cannot completely exclude bias from this differential enrollment in Brain Dock.

We included walking SAH as a clinical variable. Patients with severe symptoms usually are admitted to a hospital soon after the onset of SAH. One of the goals of this study was to detect old SAH; many patients that come to the hospital months after the onset of SAH may have only minor symptoms. Walking SAH may be analogous to old SAH detected by T2*-weighted MRI >3 months after the acute event. The incidence of hemosiderin deposition in patients with ruptured aneurysms was found to be 72.4% and did not depend on GCS on admission. T2*-weighted MRI was also able to demonstrate hemosiderin deposition in 70% of walking SAH cases, a remarkable percentage when one considers the minor symptoms experienced by these patients.

Except for ruptured AComA and distal ACA aneurysms, the resulting hemosiderin deposition largely tended to share the same laterality as the ruptured aneurysm. As a result, hemosiderin deposition patterns as measured by T2*-weighted MRI may be used in a manner similar to that of SAH distributions measured by CT to help determine the location of ruptured aneurysms22 (Figure).

Because metal objects, such as aneurysmal clips, leave marked artifacts on T2*-weighted images, it may not be possible to scan for hemosiderin deposition around such implants. Surgical operations (eg, opening of the sylvian fissure) and CSF drainage involve early removal of subarachnoid hematoma fluid and thus may affect hemosiderin deposition around a ruptured aneurysm. Because hemosiderin may deposit preferentially in the area surrounding a ruptured aneurysm, T2*-weighted MRI may be able to detect SAH and the location of the aneurysm that ruptured >3 months before examination. Thus, if a patient has a past history of severe headache, nausea, and vomiting, T2*-weighted MRI may be of value for diagnosis. In our limited experience, significant artifact surrounding aneurysms previously treated with coils was less than that seen with aneurysmal clips,23 even on T2*-weighted MRI. T2*-weighted MRI is also suitable for the follow-up of minor bleeding of aneurysms treated with coils.

**Conclusion**

Even with our limited patient sample, we have strong evidence to support the utility of T2*-weighted MRI for diagnosis of old SAH and localization of aneurysm rupture. In addition, we found a strong association between the extent of subarachnoid hemosiderin deposition and various clinical factors, including age, CT findings on admission, and SAH prognosis.

**References**


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