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Susceptibility-weighted imaging in post-treatment evaluation in the early stage in patients with acute ischemic stroke

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Abstract

Objective: This study aimed to investigate the association between abnormal signs on susceptibility-weighted imaging (SWI) and post-treatment outcome in the early stage in patients with acute ischemic stroke.

Methods: Thirty-seven patients with middle cerebral artery territory infarction were recruited. Baseline and 24-hour follow-up magnetic resonance imaging was performed. Pre- and 24-hour post-treatment clinical conditions were assessed with the National Institutes of Health Stroke Scale (NIHSS) score. Prominent vessel sign (PVS) on SWI and infarcted areas on diffusion-weighted imaging (DWI) were assessed using the Alberta Stroke Program Early CT (ASPECT) score system. Susceptibility vessel sign (SVS) was evaluated and recorded. The associations between image abnormalities and clinical scores were analyzed.

Results: PVS was found in 35 patients and SVS in seven patients. The extent of PVS was significantly correlated with the post-treatment DWI ASPECT score ($r = 0.79$), but not with the post-treatment NIHSS score or the post−pre NIHSS difference score. The presence of SVS was significantly correlated with the post-treatment NIHSS score ($r = 0.41$).

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Conclusion: PVS might be a useful predictor of early imaging prognosis and infarct growth in patients with acute ischemic stroke. SVS is related to a poor early outcome and could be useful for assessing stroke.

Keywords
Acute ischemic stroke, magnetic resonance imaging, susceptibility-weighted imaging, clinical outcome, middle cerebral artery, infarction, susceptibility vessel sign

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Introduction

Acute ischemic stroke (AIS) is a serious disease that can lead to severe clinical consequences, and is a major cause of morbidity and mortality worldwide. Rapid and accurate assessment of AIS is essential for improving clinical outcome. Assessment of imaging plays a crucial role in evaluating AIS. Many imaging techniques are used in patients with AIS to provide important pathophysiological information, such as the extent of the infarct core, the hypoperfusion area, and stenosis and occlusion of the involved artery. Associations between various imaging features and clinical outcome have been shown.[1,2]

Susceptibility-weighted imaging (SWI) is a relatively new imaging technique with high sensitivity to hemorrhage components and ability to depict small vein drainage. SWI is mainly based on the susceptibility effects within veins. SWI provides additional magnetization rate information compared with conventional magnetic resonance imaging (MRI). SWI is a potential alternative method that has been applied for assessing AIS in recent years.[3] Prominent vessel sign (PVS) on SWI reflects an increased oxygen extraction fraction (OEF) and correlates well with venous and capillary deoxyhemoglobin levels. PVS shows increased numbers and/or diameters of small vessels, and displays multiple linear low-signal intensity at the involved area of hypoperfusion. Therefore, SWI could be useful for assessing the ischemic penumbra and the target area for acute therapy. PVS and perfusion-weighted imaging are well correlated.[4,5] However, there have been conflicting results on the association between SWI–diffusion-weighted imaging (DWI) mismatch and the clinical outcome.[4,6] Susceptibility vessel sign (SVS) on susceptibility-weighted imaging (SWI) appears as low-signal intensity in the artery. SVS is considered to result from the paramagnetic and susceptibility effect of the component in thrombi. SVS has a high sensitivity and specificity in detecting intra-arterial thrombus in acute stroke.[7] The presence of SVS may predict a poor outcome for patients with AIS who are untreated with thrombolysis.[8] For patients who receive tissue plasminogen activator therapy, proximal SVS in the middle cerebral artery (MCA) pre-cortex area (M1) appears to be a predictor for a poor outcome.[9] Moreover, early hemorrhagic transformation and cerebral microbleeds can be clearly visualized on SWI, which is also important for assessing patients with AIS. SWI is recommended to substitute for the T2* gradient echo sequence in patients with AIS.

However, the value of SWI for predicting clinical outcome is controversial. Early clinical neurological improvement or deterioration of patients with AIS might be related to the long-term prognosis. A previous study showed that early infarct growth on DWI could predict the long-term clinical outcome.[10] Therefore, evaluating imaging and clinical outcome at the early stage of AIS is important. Because SWI can display signs of pathological changes of AIS, it might be useful in evaluating early clinical and imaging outcomes of these patients. This study aimed to investigate whether abnormal signs on SWI are associated with the clinical outcome in the early stage in patients with AIS who receive tissue plasminogen activator therapy.

Materials and methods

Subjects

From the July 2015 to February 2018, consecutive patients with acute brain infarcts in the MCA territory who presented to our emergency care unit were reviewed. Patients were recruited in this study if they had the following inclusion criteria: 1) aged 18 to 80 years old; 2) baseline magnetic resonance imaging (MRI) was performed within 9 hours from onset of AIS; 3) image quality was sufficient for evaluation; and 4) baseline magnetic resonance angiography (MRA) showed large artery occlusion or stenosis responsible for cerebral infarction with thrombolysis in cerebral infarction. The exclusion criteria included patients with one or more of the following conditions: 1) coma or severe stroke (National Institutes of Health Stroke Scale [NIHSS] > 25); 2) seizures; 3) a history of stroke in 3 months; 4) significant sequelae of previous stroke with a modified Rankin score (mRS) > 2; 5) known to have a history of intracranial hemorrhage or suspected intracranial hemorrhage (including subarachnoid hemorrhage); 6) severe central nervous system damage (e.g., tumor, aneurysm, intracranial, or spinal cord surgery); and 7) declined to join the study or provide informed consent. Finally, 37 patients were included in the study. All of the patients received a routine neurological examination. Pre- and post-clinical assessment was performed for all patients by two neurologists. The NIHSS was used when patients arrived for initial neurological evaluation and at 24 hours after treatment. The Institutional Review Board of Beijing Tiantan Hospital approved this retrospective study and all participants provided signed informed consent.

MRI

MRI was carried out at our institution on a 3.0 T scanner (Discovery 750; GE Healthcare, Milwaukee, WI, USA) with a 32-channel head coil. After routine T1-weighted imaging, T2-weighted and T2-weighted fluid attenuation inversion recovery, DWI, and SWI sequences were performed. A single-shot echo-planar imaging DWI sequence was used with the following parameters: repetition time/echo time (TR/TE) = 2300/63.60 ms, b = 1000 s/mm^2, slice thickness = 5 mm, slice number = 24, field of view = 240 mm, and matrix = 128 × 128. Related apparent diffusion coefficient and exponential apparent diffusion coefficient maps were generated. The parameters for three-dimensional (3D) SWI sequences were as follows: TR/TE = 38.90/23.77 ms, flip angle = 15°, slice thickness = 4 mm with a 2-mm slice gap, 60 sections per slab, matrix = 256 × 256, and field of view= 240 mm. Minimal intensity projection images were reconstructed with a thickness of 5 mm. The phase, magnitude, minimal intensity projection, and SWI images were generated automatically. We performed 3D time-of-flight magnetic resonance angiography to display stenosis or occlusion of the

cerebral arteries using the following settings: TR/TE = 19/1.8 ms, flip angle = 25°, field of view = 220 mm, matrix = 256 × 256, three slabs, and slice thickness = 1.2 mm. Follow-up MRI including DWI was performed with the same parameters for 34 patients at 24 hours (±3 hours) after intravenous thrombolysis treatment.

Imaging assessment

All MRI images were reviewed and interpreted by consensus of two radiologists who were experienced in neuroimaging. All initial MRI images were interpreted blind to the clinical history and evaluation. The acute infarct area was defined by high-signal intensity on DWI with dark signal intensity on an apparent diffusion coefficient map. PVS was defined as local prominence of hypointense vessels with either an increased vessel number or diameter in the MCA territory of the index side on SWI (Figure 1a). Susceptibility-weighted sign on SWI was defined as a focal hypointensity within the intracranial arteries, with the diameter of hypointensity exceeding the parent vessel diameter (Figure 1b). Arterial stenosis and occlusion were assessed on minimal intensity projection images of 3D time-of-flight magnetic resonance angiography.

Pre- and post-treatment DWI infarcts and the extent of PVS were scored using the Alberta Stroke Program Early CT (ASPECT) score system.[11] The total ASPECT score is 10. The MCA blood supply area was divided into 10 regions, including the caudate nucleus (C), lentiform nucleus (L), internal capsule (IC), insula cortex (I), M1, lateral cortex (M2), MCA post-cortex (M3) and MCA territory cortex above M1 (M4), MCA territory cortex above M2 (M5), and the MCA territory cortex above M3 (M6). The three regions of C, L, and IC were defined as the basal ganglia area, and the seven regions of M1, M2, M3, M4, M5, M6, and I were assessed separately.

Statistical analysis

Statistical analysis was performed using IBM SPSS Statistics for Windows version

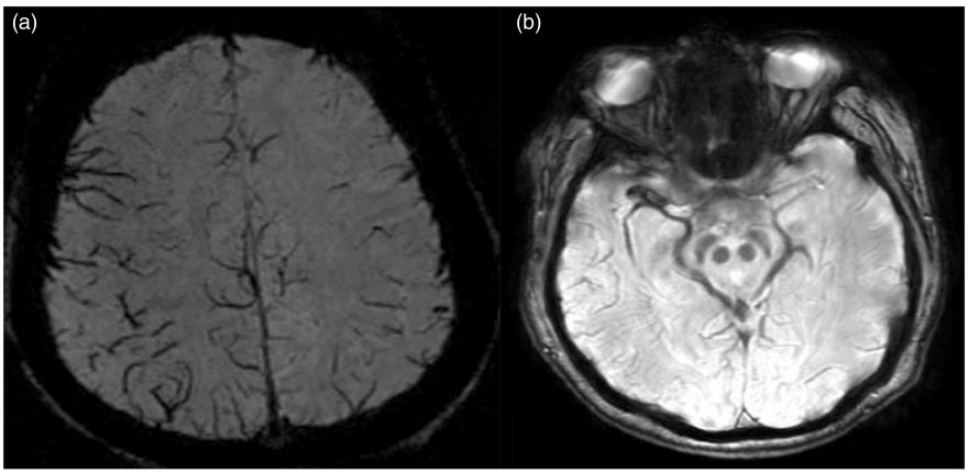

Figure 1. (a) Prominent vessel sign can be seen at the right frontal and parietal lobe, with an increased number and diameter of veins compared with the contralateral side. (b) Susceptibility vessel sign can be seen at the M1 segment of the right middle cerebral artery, displaying intense low-signal intensity in the artery with enlargement of the diameter of the right MCA

23.0 (IBM Corp., Armonk, NY, USA). Continuous variables are expressed as mean ± standard deviation or median/range (non-parametric variables). For correlation analysis, the Pearson correlation test was used for parametric variables and Spearman's rank correlation test for non-parametric variables. A P value < 0.05 was considered statistically significant.

Results

This study included 23 men and 14 women, aged 38 to 87 years (mean age: 59.9 ± 9.7 years). Demographic clinical and imaging data of the subjects are shown in Tables 1 and Table 2, respectively. A total of 18 patients had infarction in the left side and 19 had infarction in the right side. Follow-up MRI showed no infarct growth in 19 patients and infarct growth in 15 patients. Arterial occlusion was detected in five patients without any distal branches displayed. SVS was found in seven patients at the M1 segment of the MCA. Among these seven patients, one patient did not receive follow-up MRI, while the other six patients showed enlargement of infarction on DWI (Figure 2). All five patients with arterial occlusion displayed SVS. Cerebral microbleeds (CMBs) were found in 17 patients.

A significant correlation was found between the pre-treatment DWI ASPECT score and the post-treatment DWI ASPECT score ($P < 0.001$, $r = 0.86$). A significant correlation was found between the PVS ASPECT score and the post-treatment DWI ASPECT score ($P < 0.001$, $r = 0.79$), PVS in the cerebral cortex, ($P < 0.001$, $r = 0.82$), and PVS in the basal ganglia ($P < 0.001$, $r = 0.64$). Among 37 patients, 16 showed an identical PVS ASPECT score and post-treatment DWI ASPECT score. We also found a significant correlation between the PVS ASPECT score and the pre-treatment DWI ASPECT score ($P < 0.001$, $r = 0.59$) and the pre−post DWI difference ASPECT score ($P < 0.001$, $r = 0.61$). There was no significant correlation between the PVS ASPECT score and the post-treatment NIHSS score or the post−pre difference NIHSS score. The presence of PVS was significantly negatively correlated with arterial occlusion ($P = 0.011$, $r = 0.42$).

Table 1. Clinical characteristics of the patients.

	n = 37	%/range
Age (years)	59.9 ± 9.7	
Sex (male)	23	62.2%
Hypertension	26	70.3%
Hyperlipidemia	4	10.8%
Diabetes	10	27.0%
Heart disease	5	13.5%
NIHSS score (baseline)	Median: 5.5	Range: 0–15
NIHSS score (follow-up)	Median: 4	Range: 0–18

Values are mean± standard deviation, number, median, or percent. NIHSS: National Institutes of Health Stroke Scale.

Table 2. Imaging findings of the patients.

	Number/ score	%/range
DWI ASPECT score (baseline)	Median: 8	Range: 5–9
DWI ASPECT score (follow-up)	Median: 7	Range: 3–9
PVS	35	94.6%
PVS ASPECT score	Median: 7	Range: 4–9
SVS	7	18.9%
Arterial occlusion	5	13.5%
Cerebral microbleeds	17	45.9%
Grade I	13	35.1%
Grade II	1	2.7%
Grade III	3	8.1%

DWI: diffusion-weighted imaging; ASPECT: Alberta Stroke Program Early CT; PVS: prominent vessel sign; SVS: susceptibility vessel sign.

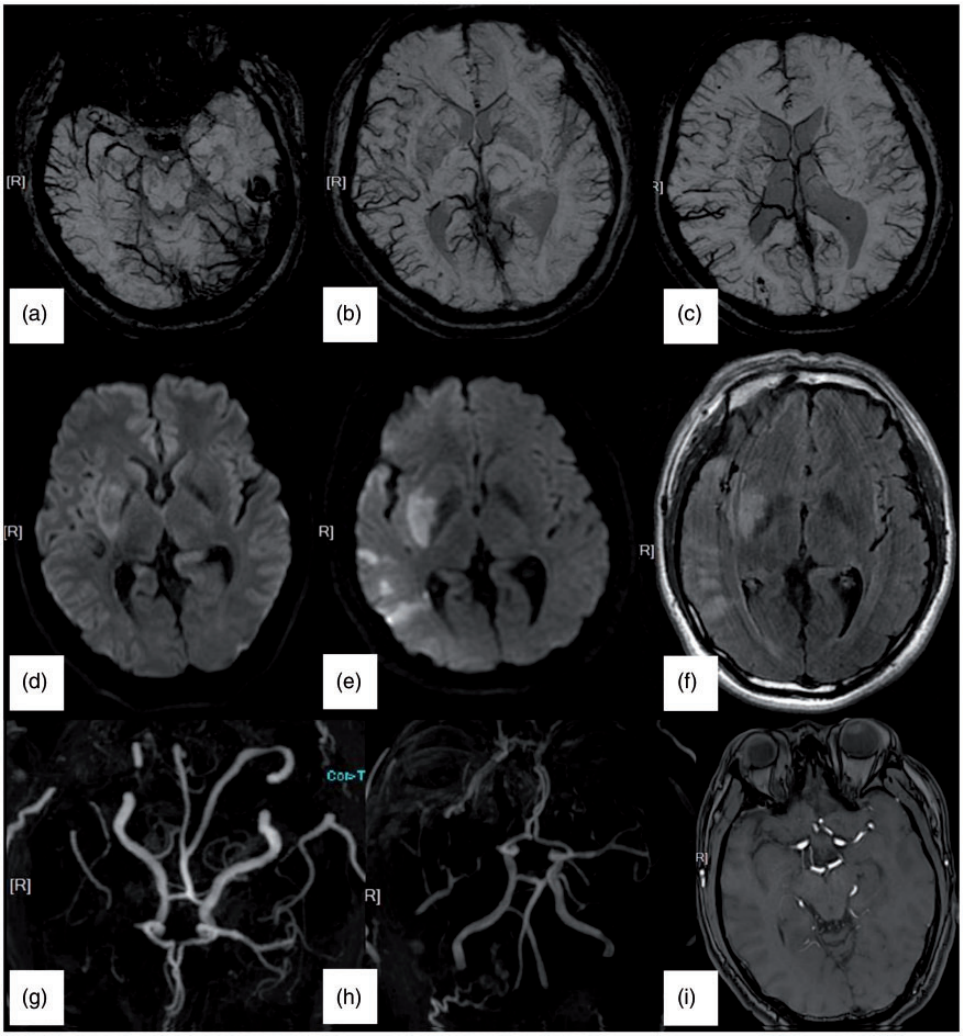

Figure 2. Magnetic resonance images of a 45-year-old man. The baseline and follow-up National Institutes of Health Stroke Scale score was 10 and 6, respectively. (a–c) Baseline susceptibility-weighted imaging minimal intensity projection images show susceptibility vessel sign at the right middle cerebral artery. Prominent vessel sign can be seen at the right middle cerebral artery territory, with an Alberta Stroke Program Early CT score of 4. (d) Baseline diffusion-weighted imaging shows infarction in right basal ganglia, with an Alberta Stroke Program Early CT score of 6. (e) Follow-up magnetic resonance imaging was carried out at 27 hours after baseline imaging. Diffusion-weighted imaging shows enlargement of the infarction, with an Alberta Stroke Program Early CT score of 4. The abnormal area corresponds with the susceptibility-weighted imaging–prominent vessel sign area. (f) A follow-up fluid attenuation inversion recovery image shows a high-signal intensity area at the right temporal lobe, and right insular and right basal ganglia area, which also corresponds with the abnormal susceptibility-weighted imaging–prominent vessel sign area. (g) Baseline minimal intensity projection image of three-dimensional time-of-flight magnetic resonance angiography shows arterial occlusion without distal branches of the right middle cerebral artery. (h–i) Follow-up three-dimensional time-of-flight magnetic resonance angiography shows that the middle cerebral artery is not recanalized after thrombolysis treatment.

The presence of SVS was significantly correlated with the post-treatment NIHSS score ($P = 0.011$, $r = 0.41$). A negative correlation was found between PVS and SVS ($P < 0.001$, $r = 0.54$).

No significant correlation was found between cerebral microbleeds and the post-treatment NIHSS score.

Discussion

Some studies have reported correlations between SWI manifestations and clinical outcomes.[12,13] There are controversial results for PVS prediction values for clinical outcomes of patients with stroke.[14,15] In this study, early outcomes of patients with AIS after intravenous thrombolysis treatment, including imaging and clinical criteria, were studied. We found that PVS was associated with post-treatment areas of diffusion restriction and the pre−post DWI difference ASPECT score, but not with the NIHSS score. Our results indicate that PVS on SWI, but not clinical assessment, might be useful for predicting early post-treatment imaging outcomes for patients with AIS. The different findings between the mean post-DWI ASPECT score and the NIHSS score also suggested that there was some inconsistency between imaging and clinical presentation at the early stage after stroke. Our results are not consistent with some previous studies,[13,16] which might be due to the difference in recruitment of patients and evaluation time points. Zhao et al's study showed that low-intensity venous signals in SWI could be used to evaluate post-thrombolytic prognosis.[16] Luo et al showed the predictive value of SWI for early infarct growth and early clinical outcome, but clinical assessment was performed in 7 days.[17] Long-term prognosis of ischemic patients is often evaluated by the 90-day mRS. Previous studies have shown a correlation between PVS sign with the 90-day mRS score.[18]

When blood flow is significantly decreased in acute stroke, the OEF of the involved brain tissue is elevated. This leads to an increase in deoxyhemoglobin in veins and capillaries, displaying low-signal intensity on SWI. The presence of PVS sign on SWI might be explained by hemodynamics of ischemic brain tissue and the related change in deoxyhemoglobin in veins. Because the OEF is a well-accepted hallmark of penumbra, PVS on SWI might be an indicator of salvageable ischemic tissue that is correlated with changes in hypoperfusion. Multiple hypointense vessels on SWI is considered as a possible surrogate marker for predicting an increased OEF and diffusion–perfusion mismatch in an acute ischemic hemisphere.[19] A previous study showed a positive predictive rate of 87% and a negative predictive rate of 100% of PVS sign.[20]

In our study, the correlation between PVS and the post-treatment DWI ASPECT score was relatively good. Although this correlation was slightly lower than that with the pre–post treatment DWI difference, it could offer information that is not available by assessing baseline DWI. Nearly half (16/37) of the patients showed identical PVS ASPECT and post-treatment DWI ASPECT scores, which suggested that PVS could be useful in prediction of the final infarction area.

However, PVS was not displayed in all patients with AIS. A meta-study reported that the presence of PVS ranged from 34% to 100% on SWI in patients with AIS.[21] This inconsistent finding might be due to different recruitment of patients among different studies. PVS is a sign of hemodynamic changes in the involved brain tissue. If there are no related cerebral hemodynamic and OEF changes, PVS sign is negative. Most of our patients had PVS. All of the patients received thrombolysis

treatment, which indicated that hypoperfusion areas were present. Identification of PVS might be more difficult than visualization of changes in perfusion. In this study, comparison of the contralateral cerebral cortex was used to identify PVS. Previous reports have shown that SWI might not be good for assessment of deep areas, such as the basal ganglia and thalami.[20] In our study, ASPECT scores were assessed for the cerebral hemisphere and deep areas separately. PVS and the post-treatment ASPECT score for deep areas were significantly positively correlated, but the r value was lower than that of the cerebral hemisphere. In 2017, a systemic review on SWI in AIS concluded that there is still controversy about the relationship between signs on SWI and thrombolytic therapy.[22]

Chen et al found that PVS was correlated with a worse early clinical outcome when only conservative treatment was provided.[20] In our study, all of the patients received intravenous thrombolysis treatment, and PVS was correlated with a good early outcome as shown by imaging. Potentially vulnerable tissue might persist up to 24 to 48 hours after the onset of ischemic stroke.[23,24] A previous study reported that PVS on T2*gradient-echo was a marker of a risk for hemorrhagic transformation in patients who received intravenous tissue plasminogen activator treatment.[2] However, in our study, no hemorrhagic transformation was found in follow-up MRI. This difference between studies might be due to the relatively small baseline infarction volume and better clinical baseline condition of patients in our study.

An increased SVS diameter on SWI may predict cardioembolism.[26,27] Cardiogenic stroke has a poor outcome after thrombolysis treatment. In our study, seven patients displayed SVS at the M1 segment of the MCA. In six of these patients, the follow-up infarcted area on DWI was enlarged. The presence of SVS was significantly correlated with the post-treatment NIHSS score. Our results are consistent with previous studies.[26,27]

Good collaterals and recanalization are correlated with a better clinical outcome. A previous study showed that hypointense cortical vessels with a change in signal on follow-up SWI might indicate the condition of metabolism.[28] Additionally, PVS might reflect, not only veins, but also small arteries with deoxyhemoglobin in the penumbra area. However, in our study, 24-hour follow-up MRI did not include SWI. A further study including follow-up SWI might provide more information about collateral circulations.

This study has some limitations, as follows. The sample size of the patients was small. The main limitation of this study was that the long-term clinical outcome was not analyzed. Although this study mainly focused on the early outcome of patients, the correlations between imaging findings and long-term outcome should be studied in the future. Another limitation is that SWI was not performed using a follow-up MRI protocol. Follow-up SWI might provide information of the clinical outcome by displaying apparent normalization of PVS in veins after successful recanalization or the hyperperfusion status with iso- or hyperintensity of draining veins on SWI.[28,29] Further studies including baseline and follow-up SWI might provide more pathophysiological information on evolution of AIS. Semi-quantification using the ASPECT score for assessing PVS is debatable. Further study using the quantitative susceptibility mapping technique would allow quantification of the OEF and provide more information for predicting the outcome of patients.

In conclusion, our study shows that PVS on SWI is correlated with the early imaging outcome, but not clinical outcome. SVS at the large MCA territory might be an

indicator of a poor early clinical outcome. SWI should be able to replace the T2*gradient-echo sequence in the stroke imaging protocol for assessing patients with AIS.

Declaration of conflicting interest

The authors declare that there is no conflict of interest.

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