CASE REPORT

Susceptibility-weighted imaging (SWI): A potential non-invasive imaging tool for characterizing ischemic brain injury?

L’imagerie de susceptibilité magnétique (SWI) : un outil non-invasif potentiel pour la caractérisation des lésions ischémiques

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Available online 26 February 2011

KEYWORDS
Susceptibility-weighted imaging; SWI; Diffusion-weighted imaging; DWI; Brain ischemia; Brain infarction; Stroke

Summary
Susceptibility-weighted imaging (SWI) is a new high-resolution magnetic resonance imaging (MRI) tool that uses the paramagnetic susceptibility effects of deoxygenated blood to study the intracranial venous vasculature. We present SWI imaging findings in two children who suffered from acute arterial ischemia. Various patterns of normal/altered venous drainage could be identified. Our case study suggests that SWI assisted mapping of the regional changes of the cerebral venous drainage and correlation with diffusion weighted MRI may identify critically perfused brain at risk for infarct progression. Prospective studies are mandatory to further validate the value of SWI.

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Introduction

Susceptibility-weighted imaging (SWI), originally referred to as high resolution blood oxygen level dependent (BOLD) magnetic resonance venography, is a high-resolution 3-dimensional gradient-echo MR imaging technique that strongly accentuates the paramagnetic properties of intravascular hemoglobin in the brain. SWI is highly sensitive for venous deoxygenated blood, extravascular blood products, such as deoxy-hemoglobin, hemosiderin and calcifications or air [1—3].

The diagnostic value of SWI has been shown in the diagnostic work-up of vascular malformations, cerebral venous thrombosis, hypoxic/anoxic injury, neurodegenerative disorders, multiple sclerosis, traumatic brain injury, and cerebral tumors [4]. Because of the high sensitivity for deoxygenated hemoglobin, SWI seems well suited to study cerebral veins in acute and/or chronic ischemia of the pediatric brain. SWI may give important, easy to collect hemodynamic information about critical brain perfusion by focusing on the venous drainage of ischemic or critically perfused brain tissue. We present the imaging of two children who suffered from acute...
focal ischemic brain injury and correlate the SWI imaging findings with conventional magnetic resonance imaging (MRI) and diffusion weighted imaging.

Patients and methods

In a pilot study, axial SWI was added to the standard departmental stroke protocol in two children who presented with acute focal neurological deficit suspicious for acute ischemia. In addition to the SWI sequence (TR 49 ms; TE 40 ms; field of view 20.1 × 23.0 cm; slab thickness 160 mm; number of slices 73; matrix 256 × 177, minimal intensity projection (MIP) images 80 images 80 × 2 mm), axial T1 and T2-weighted sequences as well as a DTI sequence were measured. The diffusion tensor was sampled by repeating a diffusion-weighted single-shot echo-planar sequence along 21 different geometric directions. Diffusion sensitization was achieved with 2 balanced diffusion gradients centered around the 180° radio-frequency refocusing pulse. An effective b-value of 1000 s/mm² was used for each of the 21 diffusion-encoding directions. An additional measurement without diffusion weighting DTI was performed \( b = 0 \text{ s/mm}^2 \). Scan parameter were TR 8000 ms; TE 91 ms; matrix 128X120; field of view 256 × 240 mm, slice thickness 3 mm. Trace of diffusion and apparent diffusion coefficient (ADC) maps were calculated from the raw data sets. Studies were performed on a 1.5 Tesla MRI scanner (Siemens Avanto, Erlangen, Germany). Images were evaluated by two experienced pediatric neuroradiologists in consensus. The SWI images were evaluated for the signal intensity, presence, size and anatomical distribution of intramedullary and sulcal veins. The SWI findings were correlated with the diffusion weighted findings. Institutional IRB was achieved for the use of the SWI sequence in children.

Patient 1

A 12-year-old boy with known sickle cell disease presented with acute chest pain and fever since 24 hours. On the second day of admission, he became less responsive with expressive aphasia. Computed tomography (CT) demonstrated a hypodense area of acute ischemia in the right frontal lobe (Fig. 1A). Subsequent MRI, done within 13 hours of acute onset of symptoms confirmed an acute right frontal lobe ischemia in the vascular territory of the right anterior cerebral artery (ACA). A matching area of restricted diffusion was seen on DTI indicating cytotoxic edema. On SWI, prominent hypointense intramedullary veins were noted adjacent to and within the area of restricted diffusion (Fig 1B). No additional asymmetry of the venous vasculature was noted. MR angiography (MRA) showed stenosis of multiple intracranial arteries. Collateral vessels were seen suggestive of secondary Moya-Moya disease. The major cerebral veins and dural sinuses were unremarkable. Follow-up imaging showed expected chronic infarction of the right frontal lobe.

Patient 2

A 6-year-old presented with a three days history of gait imbalance, decreased bladder control, bilateral eye lid drooping, abnormal upper and lower extremity movements as well as drooling after a bout of diarrheal illness a week before admission. The patient’s medical history was significant for developmental delay. Electroencephalography showed bilateral slow activity but no seizure-like discharges. No imaging was done during this initial presentation.

One month later the child was re-admitted after becoming less responsive, having difficulty with breathing and not using his left hand and arm. In the emergency department
Susceptibility-weighted imaging (SWI) for characterizing ischemic brain injury

Figure 2  A. acute CT shows a right cortical insular acute ischemia. MRI shows a matching T2-hyperintense, DWI-hyperintense and ADC-hypointense acute ischemia. Moreover, several additional small areas with restricted diffusion are noted within the cortex of the right frontal and left temporal lobe as well as in the left basal ganglia. SWI shows three distinct patterns of veins: a) SWI-hypointense intramedullary veins within the left hemisphere that match the diffusion abnormality, b) wide, SWI hypointense sulcal veins along the left hemisphere and c) an area devoid of intramedullary/sulcal veins within the occipital regions bilaterally. MRA shows high-grade stenosis of both MCA’s and ACA’s. MRV is unremarkable. Follow-up CT shows significant infarct progression especially on the left hemisphere in a distribution matching the SWI-hypointense sulcal veins. The occipital lobes are spared. The right hemisphere showed expected infarct progression. B. fused DWI and SWI image. The SWI-hypointense intramedullary veins match the area of restricted diffusion on DWI. The dilated SWI-hypointense sulcal veins drain a brain area significantly larger than the region of restricted diffusion.

the patient was found to be mildly hypertonic but without overt seizure activity. He was transferred to the pediatric intensive care unit where he was intubated and uploaded with phenytoin. CT showed a right insular/opercular acute ischemic injury (Fig. 2A). MRI done 16 hours after onset of neurologic symptoms confirmed the CT findings of a predominantly cortical, sub-segmental right middle cerebral artery (MCA) infarction. DWI showed a matching area of restricted diffusion as well as several additional, smaller acute ischemic lesions within the right frontal and left insular cortex and in the left basal ganglia. SWI identified three distinct patterns of intracranial veins. Within the area of right MCA infarction, multiple SWI-hypointense intramedullary veins were seen. On the left, multiple dilated, SWI-hypointense sulcal veins were noted without matching veins on the contralateral side. Finally, in the occipital regions no hypointense sulcal or intramedullary veins were seen. The dilated intramedullary veins were seen in a distribution that matched the area of restricted diffusion within the right hemisphere (Fig. 2B). The dilated sulcal veins along the left hemisphere appeared to occur in a distribution significantly larger than the adjacent region of restricted diffusion as seen on DTI. Follow-up CT study confirmed the right-sided infarction, however a growing left hemispheric ischemia was noted. The left-sided ischemia appeared to match the distribution of the dilated sulcal veins as seen on the prior SWI study. The same day, his pupils became fixed and dilated and he was progressively agitated. The child died three days after admission. No further follow-up MRI was performed. Autopsy confirmed bilateral acute infarction.

Discussion

Cerebral perfusion can be assessed directly or indirectly by various MRI techniques, including conventional MRI, MRA, and perfusion weighted imaging (PWI). In the past decade diffusion weighted imaging (DWI) has proven to be highly sensitive in the early detection of acute cerebral ischemia even before conventional T1 or T2-weighted MRI shows pathology [5]. By correlating DWI with PWI, tissue at risk for ischemia may be identified. Areas of restricted diffusion are believed to correspond to the irreversible core of infarction while areas of hypo-perfusion that do not match the area of restricted diffusion are believed to correspond to tissue at risk for future infarction (penumbra) [5]. High-end PWI is however not always available in the acute setting and post-processing may be time consuming. Because “time is brain”, an alternative or additional fast, easy to perform, and reliable imaging technique is desirable. Because SWI is basically a venous BOLD technique, we sought to explore the value of SWI in identifying critical brain perfusion by focusing on the venous side of brain perfusion. SWI is expected to take advantage of the high sensitivity for deoxygenated hemoglobin to identify intra-medullary or sulcal veins that drain brain tissue with critical or diminished arterial perfusion [6]. Recently Sornmachi et al. showed that dilated cortical veins as seen on SWI may reflect areas with increased oxygen extraction fraction (OEF) related to chronic ischemia [7]. In addition, Tamura et al. demonstrated that T2*-sensitive MR imaging can detect local hypointensity in the vascular territory of the occluded cerebral artery in patients with acute ischemic
stroke [7]. They postulated that the SWI-hypointensity might result from increased deoxygenation of hemoglobin in blood, indicating misery perfusion and tissue at risk for infarction. Bearing in mind that increased OEF is associated with an increased risk for stroke [9], SWI may consequently predict future stroke.

In our first child, SWI-hypointense, intramedullary veins were noted within the area of acute cortical/subcortical ischemia. Based upon the high sensitivity of SWI for deoxygenated blood, it seems plausible that these intramedullary veins are draining heavily deoxygenated blood. This finding would match the thesis that the local cerebral hypoperfusion has reached a critically low level and that the autoregulatory mediated compensatory increased oxygen extraction fraction increases the concentration of deoxygenated (SWI-hypointense) blood in the draining veins as discussed by Tamura et al. [8]. Alternatively, the SWI-hypointensity may also be related to the direct visualization of thrombosed intramedullary veins, especially if the arterial perfusion became completely halted. In addition, it should be mentioned that the venous vessels may also appear more prominent than their actual size due to the paramagnetic "blooming" effects of deoxygenated blood and possibly also due to the slow cortical venous outflow/drainage.

In the second child, SWI demonstrated three different "patterns" of venous drainage. Within the area of acute infarction, similar to the first case, mildly prominent, intramedullary SWI-hypointense veins were noted. However, on the contralateral side multiple dilated SWI-hypointense sulcal veins were noted. These vessels were draining an adjacent brain area significantly larger than the area of restricted diffusion as noted on DTI. On follow-up CT, infarct progression was noted matching the area that was drained by the dilated sulcal veins. This constellation of findings and progression of infarction may be explained by the fact that the degree of hypoperfusion of the overlying brain was initially sufficient to keep the involved brain region viable due to an increased oxygen extraction fraction. This also explains the absence of regional DTI abnormalities and a simultaneous abundance of SWI hypointense sulcal veins on the initial study. Infarct progression is however well known to occur if critical perfusion persists for extended time periods. Consequently, the appearance of prominent sulcal veins on SWI may serve as an early, easy to collect data set to identify critically perfused tissue at risk for infarction if hypoperfusion persists. The occipital regions did not show dilated or SWI hypointense veins. Weak venous SWI hypointensity is frequently observed in sedated or intubated, ventilated children and believed to be an indirect sign of a good cerebral perfusion. Under normal physiologic conditions, cerebral venous blood has an oxygenation level of about 50%; venous vessels consequently appear hypointense on SWI sequences. However, with carbogen (95% O₂, 5% CO₂) inhalation, the venous blood oxygenation level increases significantly, which results in a higher SWI signal of the venous blood [10]. The contrast between the veins and surrounding tissue consequently vanishes. This phenomenon was also present in our sedated patient, suggesting adequate occipital perfusion. This was also confirmed by the follow-up CT which was unremarkable for ischemia in the occipital regions.

Our preliminary data show that prospective studies are mandatory to validate our findings in larger controlled patient groups. High quality SWI in combination with DTI studies may provide important non-invasive data about critical brain perfusion by focusing on the venous drainage in acute cerebral ischemia.

**Conflict of interest statement**

Nothing to declare.

**References**