Postcontrast Susceptibility-Weighted Imaging
A Novel Technique for the Detection of Arteriovenous Shunting in Vascular Malformations of the Brain

Bharathi D. Jagadeesan, MD; Josser E. Delgado Almandoz, MD; Tammie L.S. Benzinger, MD, PhD; Christopher J. Moran, MD

Background and Purpose—The purpose of this study was to determine the utility of postcontrast susceptibility-weighted MRI (PCSWI) in the evaluation of vascular malformations of the brain (BVM).

Methods—We retrospectively evaluated PCSWI and digital subtraction angiography data from 16 consecutive patients with known or suspected BVM, which had been entered into a prospectively maintained database during a 1-year period. There had been no intervening treatment or change in patients’ symptoms between the PCSWI and digital subtraction angiography studies. The use of PCSWI in the detection of arteriovenous shunting was compared with that of routine noncontrast susceptibility-weighted imaging, time-of-flight MR angiography, and contrast-enhanced MR angiography using digital subtraction angiography results as the reference standard. The presence of arteriovenous shunting in PCSWI or susceptibility-weighted imaging sequences was defined by the presence of abnormal signal hyperintensity in the venous structures adjacent to the BVM.

Results—A total of 17 BVMs were identified by digital subtraction angiography (9 newly diagnosed arteriovenous malformations, 3 dural arteriovenous fistulas, 4 treated arteriovenous malformations with residual arteriovenous shunting, and 1 complex developmental venous anomaly). PCSWI was 100% sensitive and 100% specific with 100% positive predictive value and 100% negative predictive value for the detection of arteriovenous shunting in these BVMs. The PCSWI/susceptibility-weighted imaging signal intensity ratio in the most prominent early draining venous structure was 1.2 ± 0.32.

Conclusions—PCSWI appears to be superior to susceptibility-weighted imaging, time-of-flight MR angiography, and contrast-enhanced MR angiography in detecting arteriovenous shunting in BVMs and may be useful in the initial diagnosis and follow-up of patients with BVMs. (Stroke. 2011;42:3127-3131.)

Key Words: arteriovenous malformation (AVM) □ arteriovenous shunting □ developmental venous anomalies (DVA) □ susceptibility-weighted imaging (SWI)

Susceptibility-weighted imaging (SWI) is an MRI technique that combines both phase and magnitude signal to produce high-resolution images of the cerebral venous system.1 In SWI images, veins appear hypointense due to the presence of deoxyhemoglobin and the arteries are hyperintense due to time-of-flight effects and lack of T2* effects.1,2 Therefore, using SWI, it is possible to simultaneously and distinctly evaluate both the arterial and venous systems of the brain.

Abnormal hyperintense signal within the veins draining high-flow vascular malformations of the brain (BVM) can be seen on SWI images due to arterIALIZED blood flow from arteriovenous shunting (AVS). We have previously shown that this signal can be used to classify BVMs as those with and without AVS with a high degree of accuracy when the results from SWI are compared with those from digital subtraction angiography (DSA).3 We hypothesize that the accuracy of the SWI sequence in the detection of AVS can be further improved by performing post contrast SWI studies (PCSWI) after the intravenous administration of gadolinium contrast agents. On the postcontrast SWI (PCSWI) studies, the normal arteries become even brighter when compared with noncontrast SWI studies secondary to a time-of-flight effect, and the normal veins become darker when compared with noncontrast SWI studies secondary to a more pronounced T2* effect. Therefore, postcontrast studies are likely to be even more sensitive to the presence of AVS in high-flow BVMs.

Materials and Methods

Patient Selection
The study was approved by our hospital’s Institutional Review Board and conducted in compliance with the Health Insurance Portability and Accountability Act. We retrospectively analyzed the data from

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both MRI studies as well as DSA studies, which had been entered into a prospectively maintained quality assurance database at our institution on a series of 16 consecutive patients with known or suspected BVMs. All 16 patients had undergone both a brain MRI study, which included PCSWI as well as a DSA study for the evaluation of a known or suspected BVM at our institution between January 1, 2010, and December 31, 2010. Patient exclusion criteria were (1) a time interval between the MRI and DSA examinations >365 days; (2) endovascular or surgical treatment for the BVM between the MRI and DSA examinations; (3) contraindications to MRI examinations per hospital protocol including ferromagnetic metallic implants such as pacemakers or severe claustrophobia; (4) decreased renal function with glomerular filtration rate <60 mL/1.73 m² within a period of 6 months prior to the study; (5) allergy to gadolinium contrast agents; or (6) changes in patient symptoms between MRI and DSA studies. Both the MRI with PCSWI and DSA examinations were performed as part of standard of care procedures, and the decision to perform these examinations was at the discretion of the clinical providers.

Image Acquisition
All MRI studies were performed on the 3-T scanners at our institution ( Trio 3-Tesla; Siemens AG, Munich, Germany) and included the standard, Food and Drug Administration-approved SWI sequence. The SWI scanning parameters were: flip angle, 15°; TE, 20 ms; TR, 27 ms; slice thickness, 2.0 mm; and in-plane resolution of 0.9×0.9 mm. Time-of-flight MR angiography (TOFMRA) and contrast-enhanced MR angiography (CEMRA) were performed on all patients using standard vendor-provided sequences (Siemens Inc, Erlangen, Germany). All TOFMRA studies were done using the following imaging parameters: acquisition mode: 3-dimensional multiple overlapping thin slab acquisition, echo time: 4.12 ms, repetition time: 22 ms, slice thickness: 0.8 mm, acquisition matrix: 512×226, flip angle 20°. The CEMRA studies included both an arterial phase and a venous phase acquisition using vendor-provided sequences with bolus timing methods. The PCSWI sequence was performed immediately after the end of the venous phase of the CEMRA sequence and is otherwise identical to the routine precontrast SWI sequence in all respects. The postcontrast sequences in all patients were performed after the intravenous administration of 0.1 mmol/kg Optimark (Gadoversatamide; Mallinckrodt Inc, Hazelwood, MO).

Catheter angiography was performed using a dedicated biplanar neuroangiographic unit (Axiom Artis; Siemens AG, Munich, Germany) with transfemoral arterial access and intravenous contrast injection (Optiray 320; Coviden, Hazelwood, MO) of the vessels of interest.

Image Analysis
Two experienced neuroradiologists, blinded to the results of DSA, TOFMRA, CEMRA, and clinical characteristics, independently reviewed the SWI and PCSWI sequences after multiplanar reformatting of the original transaxial slices. Images were reformatted and reviewed using the Emageon Ultravisual Viewer (Amicas, Inc, Boston, MA) embedded within our hospital’s clinical information system (Clinical Desktop; BJC Healthcare, St Louis, MO), to assess the presence of AVS, as determined by the presence of signal hyperintensity within at least 1 venous structure draining into the BVM when being evaluated or within the nidus of the vascular malformation. When such abnormal hyperintensity was identified, the signal intensity within the most prominent hyperintense draining venous structure was measured on both the SWI and PCSWI studies by drawing a region of interest over this structure. All differences in reader interpretation were resolved by consensus using a panel including an additional board-certified neuroradiologist.

Subsequently, the DSA examinations were reviewed in conjunction with an experienced interventional neuroradiologist to correlate the presence of SWI signal hyperintensity within a given venous structure draining the BVM with the presence of AVS within the same venous structure in the catheter angiogram. On the catheter angiograms, vascular malformations that were identified during the arterial phase of imaging and that had an associated early draining vein were identified as high-flow malformations with AVS, whereas malformations that were visible only during the venous phase of imaging and without an associated early draining vein during the arterial phase of imaging were identified as low-flow vascular malformations without AVS.

The TOFMRA and CEMRA studies were interpreted independently by a board-certified neuroradiologist. Arteriovenous shunting was determined to be present on these studies when a nidus and/or abnormally enlarged draining veins, which were fed by an arterial branch, were clearly identified.

Medical Record Review
Medical records were reviewed for patient age, sex, time interval between the MRI and DSA examinations, hemorrhage on initial head CT images, and prior surgical or endovascular treatments for BVMs.

Statistical Analysis
Statistical analysis was performed using the MedCalc 11.1 software package (MedCalc Software, Mariakerke, Belgium). Interobserver agreement for the presence of AVS on PCSWI was determined with the k statistic. Standard statistical parameters of PCSWI, SWI, TOFMRA, and CEMRA for the prediction of AVS in BVMs were calculated using DSA as the reference standard.

Results
The mean age of the 16 patients (9 male, 7 female) who underwent MRI with PCSWI and DSA for evaluation of a known or suspected BVM was 47±16.65 years. The mean time interval between the MRI and DSA examinations was 29±52.4 days (median, 4 days; range, 0–182 days).

Results of DSA Examinations
A total of 17 BVMs were identified in the 16 patients included in our cohort. Of these, 9 were newly diagnosed arteriovenous malformations (52.9%), 3 were dural arteriovenous fistulas (17.6%), 4 were previously treated arteriovenous malformations with residual AVS (23.5%), and 1 was a complex developmental venous anomaly without AVS (5.9%).

Accuracy of PCSWI for the Detection of AVS in BVMs
PCSWI was 100% sensitive and 100% specific with 100% positive predictive value and 100% negative predictive value for the detection of AVS in these patients with known or suspected BVMs. In contrast, SWI was only 80% sensitive and 100% specific with a 40% negative predictive value and 100% positive predictive value for the detection of AVS in BVMs. TOFMRA was 67% sensitive and 100% specific with a negative predictive value of 29% and a positive predictive value of 100%. CEMRA was 87% sensitive and 50% specific with a negative predictive value of 33% and a positive predictive value of 93% (Table). The PCSWI/SWI signal intensity ratio in the most prominent early draining venous structure in patients with AVS was 1.2±0.32.

Discussion
Currently, the majority of patients with known or suspected BVMs undergo noninvasive evaluation with conventional MRI or MR angiography, which can reliably differentiate
between a typical nontreated arteriovenous malformation (AVM) and a typical developmental venous anomaly with its pathognomonic "medusa-head" appearance from radially oriented small veins converging to a central draining vein. However, the performance of conventional MRI and MR angiographic examinations in the follow-up of patients with treated AVMs and dural arteriovenous fistulas is often suboptimal.4–7 Indeed, conventional MR angiographic studies may continue to show dilated draining veins or enlarged arteries in previously treated AVMs even in the absence of AVS, because the vessel caliber changes often take time to reverse after elimination of AVS.8 Likewise, there is often contrast enhancement in the region of the treated AVM nidus secondary to reactive gliosis.7

Additionally, it is often difficult to differentiate between an atypical developmental venous anomaly with complex vascular anatomy and a high-flow BVM with these techniques.9–12 Likewise, these studies are less reliable in the presence of intracerebral hemorrhage.4–5 The recent development of time-resolved MR angiography offers a valuable new tool in the evaluation of BVMs but diagnostic time-resolved MR angiograms with high temporal resolution are often technically challenging to obtain.8,11 Hence, invasive evaluation with DSA is often required in patients in whom differentiating between atypical developmental venous anomalies and AVMs/arteriovenous fistulas is necessary, and in those patients presenting with intracerebral hemorrhage and a suspected BVM.

We had previously shown that SWI offers unique advantages in the detection of AVS.3 This property of the SWI sequence arises from the intrinsic contrast between hypointense rapidly flowing oxygenated arterial blood and hypointense slowly flowing deoxygenated venous blood on this sequence.14,15 This unique contrast between arteries and veins is further improved when the SWI sequence is performed after intravenous contrast administration (Figure 1). More importantly, this improvement in contrast is achieved without the need for accurate timing of image acquisition or technically demanding dynamic acquisition techniques. Unlike routine rigorously timed CEMRA studies, the PCSWI studies in this study were performed after the venous phase of the CEMRA studies regardless of interindividual differences in timing of the PCSWI study from the administration of the contrast bolus dose. This phenomenon is likely to be particularly advantageous in the pediatric population because cumbersome bolus timing methods are of limited use in children given the limitations on contrast dose that can be used in pediatric patients.16 Our PCSWI studies also retain high spatial resolution unlike many of the dynamic postcontrast MR angiography sequences.

In addition to our recently published study, Fujiyama et al used SWI to perform quantitative oxygenation measurements in the anterior spinal veins to detect changes in blood oxygenation within these veins after the endovascular treatment of spinal dural arteriovenous fistulas.17 Tsui et al described faint SWI signal hyperintensity within a venous varix in a patient with a cerebral dural arteriovenous fistula.18 Saini et al also described other SWI findings in a patient with a dural arteriovenous fistula but did not describe abnormal hyperintensity in a venous structure.19 George et al also recently described the use of SWI in the evaluation of brain AVMS and used the magnitude images from the SWI studies to differentiate between the components of AVMs.20 However, our study is the first to use PCSWI in the evaluation of brain AVMs and to directly compare these results with the results from SWI, TOFMRA, and CEMRA studies. In our current study, we found the sensitivity of SWI to be lower than we had previously reported; this may partly be accounted for by the smaller number of patients included in the current study. It is also likely that the lower sensitivity of the SWI sequence in this study arises from the inclusion of

### Table. The Performance of PCSWI, SWI, TOFMRA, and CEMRA Techniques in the Detection of Arteriovenous Shunting in the 17 BVMs Included in Our Study

<table>
<thead>
<tr>
<th>Technique</th>
<th>Sensitivity</th>
<th>Specificity</th>
<th>PPV</th>
<th>NPV</th>
</tr>
</thead>
<tbody>
<tr>
<td>PCSWI</td>
<td>100</td>
<td>100</td>
<td>100</td>
<td>100</td>
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<tr>
<td>SWI</td>
<td>80</td>
<td>100</td>
<td>100</td>
<td>40</td>
</tr>
<tr>
<td>TOFMRA</td>
<td>67</td>
<td>100</td>
<td>100</td>
<td>29</td>
</tr>
<tr>
<td>CEMRA</td>
<td>87</td>
<td>50</td>
<td>93</td>
<td>33.3</td>
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PCSWI indicates postcontrast susceptibility-weighted MRI; SWI, susceptibility-weighted MRI; TOFMRA, time-of-flight MR angiography; CEMRA, contrast-enhanced MR angiography; BVMs, vascular malformations of the brain; PPV, positive predictive value; NPV, negative predictive value.
smaller AVMs and dural arteriovenous fistulas in the current study. Ultimately, the true sensitivity of precontrast SWI studies is likely to be more accurately evaluated with a prospective study. Moreover, even in those patients with positive SWI or CEMRA studies, the PCSWI studies were more useful in depicting crucial AVS patterns such as retrograde flow in superior ophthalmic veins or additional deep draining veins in cerebral parenchymal AVMs (Figure 2). PCSWI was also superior to CEMRA in demonstrating very small vascular malformations with AVS. This included 2 AVMs that were demonstrated in a patient with a family history of hereditary hemorrhagic telangiectasia (Figure 3) and a small dural arteriovenous fistula (Figure 4) in a patient with headaches that was only demonstrated on the PCSWI and DSA studies.

These preliminary results suggest that PCSWI can reliably detect AVS in both de novo and previously treated high-flow BVMs. However, there are several limitations to our study. First, our study includes only a small number of patients; this makes assessment of the true diagnostic accuracy of this technique difficult. Second, our study does not elucidate the degree of AVS that is required before PCSWI signal hyperintensity can be identified in a venous structure draining a BVM, which may be particularly crucial in the follow-up of patients with treated AVMs/arteriovenous fistulas with small residual AVS. Third, the negative predictive value of PCSWI needs to be studied further given that only 1 lesion without AVS on DSA was included in our study. A prospective multicenter comparative study including a larger number of patients is likely to be useful in addressing these issues.

**Conclusions**

The presence of PCSWI signal hyperintensity within the venous structures draining a BVM is an accurate indicator of AVS from an underlying high-flow vascular malformation. Although a larger prospective study is needed, our results suggest that PCSWI appears to be superior to SWI, TOFMRA, and CEMRA for the detection of AVS in BVMs. Hence, this novel sequence may be useful in the initial diagnosis and follow-up of patients with BVMs.
Figure 4. Axial (A) and coronal (B) maximal intensity projection images from a PC SWI study in a 51-year-old man show a small nidus (white arrow in A) and an abnormally hyperintense draining vein (white arrow in B) situated over the convexity of the right frontal lobe. Three-dimensional volume-rendered PC SWI image (C) again illustrates the abnormal hyperintense vein on the surface of the brain (white arrow). Anteroposterior projection from a right common carotid angiogram performed on the same patient (D) shows a small dural arteriovenous fistula (black arrow) over the right frontal convexity, which is fed by the right middle meningeal artery and drains medially onto a small vein over the surface of the right frontal lobe. PC SWI indicates post-contrast susceptibility-weighted MRI.

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References
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Abstract

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