Arterial spin labeling magnetic resonance imaging: toward noninvasive diagnosis and follow-up of pediatric brain arteriovenous malformations

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OBJECT Arterial spin labeling (ASL)-MRI is becoming a routinely used sequence for ischemic strokes, as it quantifies cerebral blood flow (CBF) without the need for contrast injection. As brain arteriovenous malformations (AVMs) are high-flow vascular abnormalities, increased CBF can be identified inside the nidus or draining veins. The authors aimed to analyze the relevance of ASL-MRI in the diagnosis and follow-up of children with brain AVM.

METHODS The authors performed a retrospective analysis of 21 patients who had undergone digital subtraction angiography (DSA) and pseudo-continuous ASL-MRI for the diagnosis or follow-up of brain AVM after radiosurgery or embolization. They compared the AVM nidus location between ASL-MRI and 3D contrast-enhanced T1 MRI, as well as the CBF values obtained in the nidus (CBF nidus) and the normal cortex (CBF cortex) before and after treatment.

RESULTS The ASL-MRI correctly demonstrated the nidus location in all cases. Nidal perfusion (mean CBF nidus 137.7 ml/100 mg/min) was significantly higher than perfusion in the contralateral normal cortex (mean CBF cortex 58.6 ml/100 mg/min; p < 0.0001, Mann-Whitney test). Among 3 patients followed up after embolization, a reduction in both AVM size and CBF values was noted. Among 5 patients followed up after radiosurgery, a reduction in the nidus size was observed, whereas CBF nidus remained higher than CBF cortex.

CONCLUSIONS In this study, ASL-MRI revealed nidus location and patency after treatment thanks to its ability to demonstrate focal increased CBF values. Absolute quantification of CBF values could be relevant in the follow-up of pediatric brain AVM after partial treatment, although this must be confirmed in larger prospective trials.

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KEY WORDS arterial spin labeling; arteriovenous malformations; technique

Brain arteriovenous malformations (AVMs) are the main cause of intracerebral hemorrhage in children and represent 30%–50% of pediatric hemorrhagic strokes.12,14 Although successful treatment of small AVMs can be safely and quickly achieved with microsurgery, more complex lesions often require multimodal therapy consisting of endovascular procedures and radiosurgery.3,4,8 Effects of the latter are delayed, and obliteration rarely occurs before 2 or 3 years afterward. Moreover, obliteration is not always achieved, and long-term imaging follow-up is needed to assess the completeness of treatment. The recurrence of pediatric brain AVM has been reported in up to 5% of cases, even as late as 16 years after initial complete treatment.4,8,19 Thus, long-term imaging follow-up is required even after complete occlusion of a brain AVM. Digital subtraction angiography (DSA)
is the reference standard for diagnosis and for tailoring the appropriate treatment. Apart from offering high diagnostic performance in the detection of untreated brain AVM, DSA enables visualization of residual shunt after partial treatment. However, DSA remains an invasive tool, requiring sedation or general anesthesia in children, with the potential risk, albeit very low, for periprocedural complications, and it exposes both patients and medical staff to repeated ionizing radiation, which in children is associated with an increased risk of developing solid cancers or leukemia.

Numerous noninvasive imaging techniques, such as arterial spin labeling (ASL)-MRI, have already shown promise in the management of acute and chronic cerebrovascular disease in adults. Arterial spin labeling is an MRI technique that can noninvasively assess cerebral blood flow (CBF) by magnetically labeling the arterial water spins with a radiofrequency pulse. The general principle of ASL-MRI consists of using the arterial blood as an endogenous contrast agent. By continuous inversion of the arterial blood’s water proton nuclear spins in the cervical arteries, ASL-MRI has allowed accurate and reliable quantification of CBF. A direct application is acute and chronic cerebrovascular disease because quantification of reduced CBF allows one to detect areas of penumbra and vascular collaterals and to objectively follow cerebral reperfusion after therapeutic interventions. Moreover, ASL-MRI gives an overview of cortical perfusion in the brain besides the pathological process. We hypothesized that—as recently shown in small adult cohorts—by enabling focal detection of increased CBF in the nidus of a malformation, ASL-MRI may have interesting applications in the management of pediatric brain AVMs.

Our aim was to study the ability of ASL-MRI to demonstrate nidus patency and localization in untreated and partially treated pediatric brain AVMs.

Methods
Study Design
The study conformed to the scientific principles and research ethics standards of our institution. The paper was prepared in accordance with Standards for the Reporting of Diagnostic Accuracy Studies (STARD) guidelines. Our institutional review board waived the need for written informed consent from participants. We retrospectively analyzed all children (age < 18 years) with a brain AVM, either untreated or partially treated, who had undergone ASL-MRI and DSA during the same week in the last 3 years.

Magnetic Resonance Imaging Procedures
Magnetic resonance imaging was performed during the last 3 years on a 1.5-T GE Signa HDxt system (General Electric Healthcare) using a 12-channel head-neck-spine coil. The brain MRI protocol included at least 3D T1 sequences before and after Gd injection (3D-Gd-T1 MRI), axial FLAIR sequences, axial T2 sequences, and diffusion and noncontrast perfusion sequences with 3D pseudo-continuous ASL-MRI (axial partitions 40, field of view 240 × 240 × 4 mm³, acquisition matrix 8 spiral arms in each 3D partition, TR 1105 msec, flip angle 155°, acquisition time 4 min 17 sec). No anesthesia or sedation was required during MRI acquisition. When consecutive ASL-MRI studies were conducted following radiosurgery or embolization, the last imaging session was performed between 5 days and 2 years after the last procedure.

Catheter Angiography
All endovascular procedures were performed with the patient under general anesthesia, using 3- or 4-Fr catheters introduced via the right femoral route, according to patient age and weight. A 3- or 4-Fr coaxial system (guiding catheter, Glidecath, Terumo) was positioned over a 0.28 or 0.35 guide successively in the proximal internal and external carotid artery and cervical vertebral artery. Face, lateral, and oblique views (IGS Innova 530, GE Healthcare) were obtained after selective injection of 3–5 ml of iodinated contrast (Guerbet).

Imaging Analysis
All imaging data were anonymized and analyzed on a dedicated workstation (Advantage Windows, version 4.2). Three-dimensional T1 sequences and ASL sequences were automatically realigned and fused in a volume viewer. A CBF map was automatically generated, and the color scale was set to rainbow.

Two readers (a neuroradiologist and a neurosurgeon with 10 and 8 years of experience in pediatric imaging, respectively) decided by consensus whether CBF maps and 3D-Gd-T1 sequences showed anatomical colocalization of the AVM nidus. For each patient, we performed, in the axial plane of the ASL-MRI study in which the nidus was the largest, a manual segmentation of the AVM to define the region of interest (ROI) in which the AVM nidus area and nidus CBF (CBF_nidus) mean values were automatically calculated by the software. Average CBF values for normal cortex of the contralateral hemisphere (CBF_cortex) were calculated using the average values of 2 cortical ROIs of 2 cm² that were positioned in the frontal and parietal regions. This allowed us to calculate the relative AVM CBF, defined as the ratio of CBF_nidus/CBF_cortex.

Statistical Analysis
We compared values between CBF_nidus and CBF_cortex using a nonparametric Mann-Whitney test. Significance level was set at p < 0.05. We did not perform statistical analysis for patients who had CBF quantification before and after a therapeutic procedure (embolization or Gamma Knife) because of the small number of patients. Values are expressed as the mean ± standard deviation, unless indicated otherwise.

Results
Patients and AVM Characteristics
Among 21 included patients (14 males), whose mean age was 10.8 ± 3.9 years (range 3.6–17 years), 14 had imaging for a ruptured AVM associated with an intracerebral hematoma (Table 1). Magnetic resonance imaging was performed for headaches in 3 cases, for late follow-up of
### TABLE 1. Summary of patients and imaging characteristics*

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Patient Age (yrs), Sex at Diagnosis</th>
<th>Mode of Revelation</th>
<th>AVM Characteristics</th>
<th>AVM Treatment (date)</th>
<th>ASL1 Characteristics (date of imaging after diagnosis; (\text{CBF}<em>{\text{nidus}}) [ml/100 mg/min]; nidus diameter [cm2]; (\text{CBF}</em>{\text{cortex}}) [ml/100 mg/min])</th>
<th>ASL2 Characteristics (date of imaging; (\text{CBF}<em>{\text{nidus}}) [ml/100 mg/min]; nidus diameter [cm2]; (\text{CBF}</em>{\text{cortex}}) [ml/100 mg/min])</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>13.5, M ICH</td>
<td>Rt temporal lobe, SM G1</td>
<td>Embol ×2, GK (01/12)</td>
<td>06/2012; 148.7; 2.4; 59</td>
<td>06/2013 (18 mos after GK); 156; 1.2; 54</td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>8.6, F Headaches</td>
<td>Lt basal ganglia, SM G1</td>
<td>Embol x1, GK (02/12)</td>
<td>05/2011; 135; 3.5; 65</td>
<td>12/2012 (10 mos after GK); 113; 2.7; 68</td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>13.7, M ICH</td>
<td>Rt basal ganglia, SM G1</td>
<td>Embol x6, GK (05/11)</td>
<td>10/2011; 112; 5; 50</td>
<td>3/2013 (22 mos after GK); 97; 2.7; 48</td>
<td></td>
</tr>
<tr>
<td>4</td>
<td>4.1, F ICH</td>
<td>Lt temporal lobe, SM G11</td>
<td>Embol x1, GK (3/13)</td>
<td>6/2012; 71; 3.4; 51.6</td>
<td>7/2013 (4 mos after GK); 70; 1.7; 60</td>
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<td>5</td>
<td>9.5, M ICH</td>
<td>Rt basal ganglia, SM GIV</td>
<td>Embol ×2, GK (12/11)</td>
<td>5/2012; 244; 3.1; 87</td>
<td>5/2013 (18 mos after GK); 72; 0.9; 66</td>
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<td>6</td>
<td>11, M ICH</td>
<td>Rt frontal, SM G1</td>
<td>Embol ×3</td>
<td>12/2012 (pre-embol); 260; 2.15; 59</td>
<td>07/2013 (incomplete embol); 128; 1.4; 62</td>
<td></td>
</tr>
<tr>
<td>7</td>
<td>13.4, M Epilepsy</td>
<td>Lt central, SM GIV</td>
<td>Embol ×4</td>
<td>At diagnosis; 242; 11.4; 59</td>
<td></td>
<td></td>
</tr>
<tr>
<td>8</td>
<td>13.3, F ICH</td>
<td>Cerebellar culmen, SM GIV</td>
<td>Embol ×3</td>
<td>NA</td>
<td></td>
<td></td>
</tr>
<tr>
<td>9</td>
<td>8.1, M ICH</td>
<td>Lt temporal lobe, SM GII</td>
<td>NT</td>
<td>At diagnosis; 124; 0.5; 68</td>
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<td></td>
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<tr>
<td>10</td>
<td>3.5, F Developmental delay</td>
<td>Brainstem, SM GIV</td>
<td>NT</td>
<td>At diagnosis; 139; 0.6; 57.5</td>
<td></td>
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<tr>
<td>11</td>
<td>16.9, M ICH</td>
<td>Lt parietal, SM G1</td>
<td>Embol ×1, surgery</td>
<td>At diagnosis; 115; 0.7; 69</td>
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<tr>
<td>12</td>
<td>10, F ICH</td>
<td>Lt occipital, SM GII</td>
<td>Embol ×2 GK</td>
<td>At diagnosis; 98; 1.2; 68</td>
<td></td>
<td></td>
</tr>
<tr>
<td>13</td>
<td>8.5, F ICH</td>
<td>Rt occipital, SM GII</td>
<td>Embol ×1</td>
<td>At diagnosis; 128; 81; 137</td>
<td></td>
<td></td>
</tr>
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<td>14</td>
<td>15, M FU for obliterated AVM</td>
<td>Lt frontal, SM GII</td>
<td>Surgery (4 yrs earlier)</td>
<td>At diagnosis; 92; 0.9; 63</td>
<td>Postop; no AVM on ASL MRI</td>
<td></td>
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<tr>
<td>15</td>
<td>5.7, M ICH</td>
<td>Rt central, SM GII</td>
<td>Embol ×1</td>
<td>12/2013 (pre-embol); 92; 1; 57</td>
<td>02/2014 (complete embol); No AVM on ASL-MRI</td>
<td></td>
</tr>
<tr>
<td>16</td>
<td>16.5, F FU for obliterated AVM</td>
<td>Lt frontal, SM GII</td>
<td>GK (5 yrs earlier)</td>
<td>At diagnosis; 117.8; 2; 71</td>
<td></td>
<td></td>
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<tr>
<td>17</td>
<td>8.4, M ICH</td>
<td>Lt frontal, SM GIV</td>
<td>NT</td>
<td>At diagnosis; 105; 1.4; 42</td>
<td></td>
<td></td>
</tr>
<tr>
<td>18</td>
<td>10, M ICH</td>
<td>Lt central, SM GIII</td>
<td>NT</td>
<td>At diagnosis; 123; 3.7; 51</td>
<td></td>
<td></td>
</tr>
<tr>
<td>19</td>
<td>12.9, M ICH</td>
<td>Lt occipital, SM GII</td>
<td>Embol ×1</td>
<td>02/12/2013 (pre-embol); 131; 1.3; 51</td>
<td>07/12/2013 (incomplete embol); 86; 0.7; 45</td>
<td></td>
</tr>
<tr>
<td>20</td>
<td>17, M Headaches</td>
<td>Brainstem, SM GIV</td>
<td>NT</td>
<td>At diagnosis; 145; 5.1; 53</td>
<td></td>
<td></td>
</tr>
<tr>
<td>21</td>
<td>8.2, M Headaches</td>
<td>Lt basal ganglia, SM G11</td>
<td>NT</td>
<td>At diagnosis; 121.6; 1.6; 41</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

* Comparison of 3D-Gd-T1 sequences with ASL-MRI demonstrated colocalization in all patients.

Embol = embolization; FU = follow-up; GK = Gamma Knife; ICH = intracerebral hemorrhage; NA = not available; NT = not treated; SM G(I–V) = Spetzler-Martin grade.
a presumably obliterated AVM in 2 cases (4 and 5 years after treatment), for epilepsy in 1 case, and for developmental delay in 1 case.

The AVM was located on the left side in 12 cases, and lobar localization was as follows (number of patients): basal ganglia (4), central region (3), frontal lobe (4), occipital lobe (3), temporal lobe (3), posterior fossa (3: medulla oblongata, mesencephalon, and cerebellum vermis), and parietal lobe (1). Spetzler-Martin grades were as follows: Grade I, 3; Grade II, 4; Grade III, 6; Grade IV, 6; and Grade V, 2.

**Diagnosis of AVM and ASL-MRI**

Visual analysis of the CBF maps revealed the AVM nidus in all 21 cases. Fusion of 3D-Gd-T1 images and ASL-MRI studies showed anatomical colocalization of the nidus in all 21 cases (Figs. 1–3).

The mean AVM nidus area was 2.7 ± 2.5 cm² (range 0.5–11.4 cm²). The mean CBF_{nidus} (137.7 ± 51 ml/100 mg/min, range 92–260 ml/100 mg/min) was significantly higher than CBF_{cortex} (58.6 ± 9 ml/100 mg/min, range 41–71 ml/100 mg/min; p < 0.0001). The mean relative AVM CBF was 2.25 ± 0.7 (range 1.43–4.10). The mean CBF_{nidus} was higher in larger AVMs, and there was a correlation between mean CBF_{nidus} and mean AVM nidus areas as well (Pearson correlation, r = 0.631, p = 0.003). There was no difference in the mean values for CBF_{nidus} according to venous drainage type (p = 0.739, Mann-Whitney test) or localization (superficial vs deep, p = 0.824, Mann-Whitney test).

**Follow-Up of AVM**

Among the 21 patients, 9 had undergone ASL-MRI both before and after therapeutic procedures: surgery (1), embolization (3), or radiosurgery (5; Table 1). After surgical treatment, no nidus was left, and therefore CBF was normal. After complete embolization in 1 case, ASL-MRI was normal. In 2 cases of incomplete embolization, we measured a reduction in the AVM nidus surface (2.15 and 1.3 cm² before vs 1.4 and 0.7 cm² after treatment) and the mean CBF_{nidus} (260 and 131 ml/100 mg/min before vs 128 and 86 ml/100 mg/min after embolization). As 3D angiography was not performed, we could not correlate the reduction in nidal volume on angiography with the reduction in the surface of the nidus on ASL-MRI. After radiosurgery (Fig. 4), the mean CBF_{nidus} (142.1 vs 102.8 ml/100 mg/min) and AVM nidus areas (3.48 vs 0.82 cm²) decreased before and after treatment, respectively, whereas CBF_{cortex} was stable (62.5 vs 59.2 ml/100 mg/min). However, statistical analysis could not be performed owing to the small number of patients.

**Pseudo-Continuous ASL-MRI in a Case of Seizure**

One patient was admitted for a right-sided occipital hematoma (Case 13), intracranial hypertension, and partial status epilepticus with visual hallucinations and metamorphopsia in the left visual field that disappeared after 2 days of antiepileptic drugs but were present during MRI acquisition. Interestingly, we observed hyperperfusion (CBF: 135 ml/100 mg/min in epileptic cortex vs 81 ml/100 mg/min in normal cortex and 128 ml/100 mg/min in AVM) in the parietooccipital junction, where there was no AVM or draining veins (Fig. 5). We postulate that this area was the symptomatogenic epileptic zone. A control MRI study performed 2 months later, when the patient was without seizure, showed the normalization of CBF in the parietooccipital cortex (104 ml/100 mg/min), whereas the nidus remained patent.
In this preliminary study, we found that 1.5-T ASL-MRI demonstrated nidus location and patency in a cohort of 21 pediatric patients with brain AVM, in accordance with DSA and contrast-enhanced T1 sequences. To our knowledge, this is the first description of clinical interest in ASL-MRI in pediatric brain AVMs. We quantified the hyperperfusion of the nidus as compared with perfusion in remote normal cerebral cortex, and we objectively measured the modifications of CBF after therapeutic interventions.

Different imaging techniques are available to evaluate brain hemodynamics, such as PET, SPECT, xenon-enhanced CT, susceptibility-weighted MRI perfusion, or Doppler ultrasound.2,20 All of these techniques except ultrasound require the injection of a contrast agent.

We demonstrated that visual detection of nidus patency was possible in all cases, and image fusion showed excellent anatomical colocalization between ASL-MRI and 3D-Gd-T1 sequences. These results are in accordance with those in a recent study on 26 adults with 15 cases of DSA-proven vascular malformations (8 dural arteriovenous fistulas and 7 AVMs) and 11 cases of intracerebral hemorrhage without vascular malformations.5 Detection of venous ASL signal hyperintensity enabled the authors to detect vascular lesions with a sensibility and specificity of 78% and 85%, respectively. Moreover, multivariate analysis showed that the detection of ASL-MRI abnormality increased 17 times the chance of detecting an AVM on DSA. Increased CBF was also noted in 7 cases of AVM by using ASL-MRI, and these authors observed a “steal phenomenon,” as decreased CBF was noted in the ipsilateral basal ganglia as compared with that in the contralateral side.2,23

We encountered one false-positive case in a patient...
with status epilepticus and increased CBF values distant from the AVM nidus. Focally increased signal intensity on ASL-MRI may be produced by true hyperperfusion related to neurovascular coupling or by an artifact such as motion or intravascular spin label. Arterial spin labeling is sensitive in revealing hyperperfusion in a number of conditions related to tumor, seizure, or loss of autoregulatory function of blood vessels. The regional hyperperfusion

FIG. 4. Case 5. Conventional DS angiogram obtained before radiosurgery (A); ASL-MRI obtained 6 months after Gamma Knife (B); and ASL-MRI obtained 18 months after Gamma Knife (C). Graph (D) showing the evolution of nidal CBF in Cases 1–5 after radiosurgery. Graph (E) showing evolution of the nidal area, in cm², in Cases 1–5 after radiosurgery. There was a reduction in the nidos area but not in the perfusion values inside the nido. Case 6. Initial angiogram (F) showing a right frontal AVM. Initial ASL-MRI (G). Last angiogram (H) showing partial treatment of the AVM. Last ASL-MRI (I) showing a decrease in the size of the nidus. Figure is available in color online only.

FIG. 5. Case 13. Conventional DS angiogram (A) shows a small compact right occipital AVM with 2 feeders (middle cerebral artery and posterior cerebral artery). An axial 3D-Gd-T1 MR image (B) shows the right occipital hematoma and a small compact AVM nidus. An ASL-MR image (C) shows hyperperfusion of the nidos at the posterior part of the hematoma. An ASL-MR image (D) shows hyperperfusion of the parietooccipital area, compatible with ictal hyperperfusion of the epileptogenic zone. Axial FLAIR MR image (E) reveals the absence of cerebral edema in the parietooccipital junction. An ASL-MR image (F) performed 2 months after intracerebral hemorrhage, when the patient was seizure free. The CBF map is back to normal. Arrows indicate the ROI—that is, the AVM or the hyperperfusion related to the seizure. Figure is available in color online only.
implicated in hypertensive encephalopathy and reversible encephalopathy syndrome can be readily disclosed by ASL-MRI, which on serial follow-up can demonstrate the normalization often observed in these conditions.6,23 Our results showed that ASL-MRI offers reproducible quantitative measurement of CBF. This imaging modality uses radiofrequency-labeled water protons in the neck vessels as an endogenous tracer. Hence, there is no variability in the concentration of the contrast agent. Moreover, in children, the absence of atherosclerotic stenosis of the carotid or vertebral arteries is another advantage in regard to the reproducibility of blood flow analysis. We observed a small standard deviation (6.5 ml/100 mg/min) in the CBF of normal cortical areas, in agreement with published data, and we did not observe significant variations in CBF in the normal cortex on subsequent acquisitions despite an interval of 18 months between MRIs.

We observed mean $\text{CBF}_\text{adu}$ values that were twice those of $\text{CBF}_\text{cortex}$. This result may explain why visual analysis could easily detect the nidus and could be relevant in the early diagnosis of brain AVM in children with intracerebral hematoma, although this must be confirmed in dedicated prospective studies.

One can argue that ASL-MRI performed at 1.5 T has limitations. In adults, 1.5-T ASL-MRI suffers from relatively low signal-to-noise ratio. Interestingly, in children, a consistent pattern of increased signal-to-noise ratio as well as globally elevated absolute CBF has been observed, as compared with that in adults. Possible explanations for this globally increased signal intensity include higher baseline CBF, faster mean transit time, increased baseline magnetization values in gray and white matter, and increased T1 values in blood and tissue (that is, a longer tracer half-life). Decreased susceptibility artifact at the base of the skull from immature parasanal sinus development may also play a role in improved image quality and added signal intensity in the frontal and inferior regions. Finally, the ASL-MRI sequence lasts 5 minutes, is feasible with all MRI set-ups, and does not require intravenous injection. In the adult population, these factors are highly relevant for the etiological diagnosis of an intracerebral hemorrhage or the diagnosis of an AVM recurrence. Another major application of ASL-MRI is the follow-up of AVMs treated with radiosurgery even though DSA is still the gold standard for proving AVM obliteration.15

Numerous noninvasive imaging modalities, such as time-of-flight or dynamic contrast MRA, have also proven useful in monitoring obliteration dynamics, although they only provide volume of the nidus and do not quantify CBF variations.11,16,22 In the present study and as previously reported in an adult cohort with 8 AVMs, 1 dural fistula, and 1 vein of Galen malformation treated with partial embolization, quantification of the nidus diameter and perfusion values allowed us to follow up patients after embolization or radiosurgery. Some authors have noted an average reduction of 36% in the relative amount of signal measured with ASL.16 There has been good correlation between the estimation of nidus obliteration with DSA and the ASL-MRI observations. As obliteration after radiosurgery rarely occurs before 2 or 3 years after treatment, with an obliteration rate of about 70%–80% and a rebleeding rate of 6%, long-term follow-up is mandatory in such patients.9,14 Thus, ASL-MRI could help in the noninvasive monitoring of nidus obliteration before DSA confirms it, as previously suggested.15 Moreover, this imaging modality could decrease the economic cost of the follow-up of brain AVMs. Last but not least, ASL-MRI studies can be used during therapeutic procedures. The fusion of CBF maps with anatomical MRI can be integrated into neuronavigation software during microsurgical or radiosurgical procedures.

Our study suffered several limitations. First, although data were prospectively acquired, analysis was retrospective. Magnetic resonance imaging sequences were acquired at different times after therapeutic procedures, and this could affect their interpretation. Second, the population consisted only of patients referred for brain AVM. Because readers were aware that all patients had been referred for the brain AVMs, sensitivity in detecting the nidus location might have been artificially increased. In addition, we did not analyze the specificity of ASL-MRI. Third, pediatric CBF is prone to large variation from a neonatal age until 8 years old and then gradually decreases to adult levels. This may hamper the possibility of a quantitative long-term follow-up based on CBF values. These limitations might be overcome in the near future with the further refinement of ASL-MRI techniques and the use of 3-T MRI. At our institution, we now use 1.5-T ASL-MRI in all patients harboring a brain AVM. We continue to strongly advocate for the use of DSA in patients with presumed nidus obliteration on ASL-MRI. However, the latter technique might help to reduce the number of DSA examinations performed for brain AVMs, and thus saving children from potential procedural risks. This strategy may change as we gain more experience and with technical improvements in this sequence.

Conclusions

Our preliminary observations support ASL as a sensitive MRI sequence for the diagnosis of nidus patency in brain AVMs. Quantification of nidal CBF may enable objective monitoring of AVM obliteration after radiosurgery or partial embolization. Additional, larger prospective studies are required to confirm our initial results.

References


Author Contributions
Conception and design: Blauwblomme, Sainte-Rose. Acquisition of data: Blauwblomme, Brunelle, Grévent, Di Rocco, Beccaria, Paternoster, Bourgeois, Kossorotoff. Analysis and interpretation of data: Blauwblomme, Naggarra, Brunelle. Drafting the article: Blauwblomme. Critically revising the article: Naggarra, Grévent, Puget, Kossorotoff, Sainte-Rose. Approved the final version of the manuscript on behalf of all authors: Blauwblomme. Statistical analysis: Blauwblomme. Administrative/technical/material support: Zerah, Sainte-Rose. Study supervision: Zerah, Sainte-Rose.

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