



Original Article

Arterial spin-labeled perfusion for vascular anomalies in the pediatric head and neck^{☆,☆☆}



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ABSTRACT

Objective: To study the arterial spin-labeled (ASL) imaging features of vascular anomalies in the head and neck.

Materials and methods: The presence or absence of ASL signal was evaluated for each vascular anomaly, and a comparison with conventional magnetic resonance (MR) sequences was performed.

Results: Twenty-five children were identified with vascular anomalies. All proliferating infantile hemangiomas demonstrated hyperintense ASL signal. There were eight cases that ASL imaging provided additional information when compared with conventional MR sequences, including increased lesion conspicuity and altered perfusion.

Conclusions: ASL imaging of cutaneous vascular anomalies shows specific signal intensity patterns and can provide additional value when compared to conventional MR sequences.

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1. Background and purpose

Conventional MRI, namely fat-suppressed T2-weighted and gadolinium-enhanced T1-weighted sequences, serves as the cornerstone for diagnosing vascular anomalies [1]. Classic imaging appearances, such as phleboliths in venous malformations or large macrocysts in lymphatic malformations, are highly specific features for these lesions but have variable sensitivity given the frequency of atypical imaging features and combined malformations. For example, differentiating between hemangiomas and venous malformations when phleboliths are absent or in extramuscular locations may be challenging, as both lesions exhibit T2 hyperintensity and enhance following gadolinium chelate administration. There can also be further difficulty to interpret these atypical lesions when the clinical profile is not straightforward.

Our institution routinely uses arterial spin-labeled (ASL) imaging, which measures perfusion by magnetically labeling arterial protons, for intracranial disorders such as stroke and brain tumor. Advantages of ASL imaging include its noncontrast technique, that it can be repeated in cases of patient motion, and the short duration of scanning (3–4 min) [2]. Given the perfusion capabilities of ASL, we sought to (a) characterize the ASL features of vascular anomalies in the head and neck;

(b) determine whether ASL imaging might help with diagnosis; (c) investigate flow dynamics of certain lesions, especially as they relate to alterations in cerebral blood flow (CBF) or arteriovenous shunting; and (d) determine if ASL imaging might affect clinical management and treatment in selected cases.

2. Materials and methods

This study was approved by the institutional review board, which waived the requirement for informed consent. Electronic medical records were reviewed for all children (<18 years) with vascular anomalies that underwent ASL MRI over the 5-year period from June 2010 to June 2015. Our investigation focused on lesions within the head and neck; cerebral arteriovenous malformation (AVMs) or dural arteriovenous fistula (AVFs) were excluded. Two neuroradiologists (9 years and 2 years of experience working in our vascular anomalies clinic) reviewed all available magnetic resonance (MR) images, reaching consensus on each finding. MRI examinations were performed with ASL, fast-spin echo T1-weighted, fat-suppressed T2-weighted, and gadolinium-enhanced, fat-suppressed T1 sequences on a 1.5 or 3 T MRI system. Our vendor-supplied ASL technique (GE Healthcare, Milwaukee, Wisconsin, USA) was performed using a pseudocontinuous labeling period of 1500 ms, followed by a 1500-ms postlabel delay. 3D whole-brain images were acquired while suppressing the background with fast-spin echo stacked technique. Other ASL MRI parameters included TR/TE, 4409/10.6 ms, FOV, 20 cm, matrix, 512×8, and NEX, 3. Our ASL images of the head are displayed in two sets of images, with and without cropping of the extracranial soft tissue tissues, of which the latter set

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was analyzed. The arterial labeling plane was set inferior to the cutaneous lesion prior to scanning in the head or neck.

Vascular anomalies were characterized according to the 2014 classification of the International Society for the Study of Vascular Anomalies [3]. Diagnosis was determined from the combination of MRI and with the consensus of our vascular anomalies team of physicians. We made specific mention if infantile hemangiomas were in the proliferating or involuting phase, as the imaging appearances varied. On conventional MRI, proliferating infantile hemangiomas show enhancement and multiple internal flow voids, while involuting infantile hemangiomas show no or minimal enhancement and contain fat. Venous malformations show enhancement and may contain phleboliths. Lymphatic malformations are either macrocystic or microcystic and show T2 hyperintense signal, with occasional minimal peripheral enhancement [1,4].

The presence or absence of ASL signal was evaluated for each vascular anomaly, with comparison to the muscle and skin soft tissues. In patients with PHACE syndrome (posterior fossa malformations, hemangiomas, arterial anomalies, cardiac defects, and abnormalities of the eye) or soft tissue AVM/AVF, CBF was qualitatively investigated with ASL to determine if there was decreased or increased perfusion or vascular shunting compared to normal brain; regions of interest analysis was not performed. In cases of AVM/AVF, evaluation of arteriovenous (AV) shunting within veins was also assessed. Lastly, comparison with conventional MR sequences was done to evaluate if ASL imaging provided any additional information.

3. Results

From the hospital records, 25 patients were identified with cutaneous vascular anomalies (Table 1). There were 8 males and 17 females with ages ranging from 2 days to 14 years (mean, 2.3 ± 3.8 years). Of the 25 patients, 14 had proliferating infantile hemangiomas (3 of

these were diagnosed with PHACE syndrome [5]), 2 had involuting infantile hemangiomas (1 of these was diagnosed with PHACE syndrome), 4 had venous malformations, 3 had lymphatic malformations, and there was 1 case each of soft tissue AVM and facial lipomatosis in the setting of CLOVES (congenital, lipomatous, overgrowth, vascular malformations, epidermal nevi and spinal/skeletal anomalies and/or scoliosis) syndrome.

All proliferating infantile hemangiomas, including the three with PHACE syndrome, demonstrated hyperintense ASL signal when compared to surrounding head and neck muscles and skin soft tissues (Fig. 1). Many of these patients had more than one hemangioma, and all hemangiomas were hyperintense on ASL images. Two of the four venous malformations were isointense on ASL to the surrounding musculature (Fig. 2), while two venous malformations within the scalp showed mildly hyperintense signal. All three lymphatic malformations and both involuting infantile hemangiomas were isointense to the surrounding musculature on ASL images (Fig. 2). In the single case of AVM, there was hyperintense ASL signal within the nidus and shunted veins (Fig. 3). Isointense ASL signal was seen in the sole case of facial lipomatosis.

Two patients with PHACE syndrome (Patients 12 and 13) had significant stenosis of an internal carotid artery and had corresponding decreased CBF in the supplied territory on ASL imaging (Fig. 4). Both of these patients had normal signal intensities of the parenchyma on T2-weighted imaging, and there was no evidence of infarction. One patient with a scalp hemangioma (Patient 11) had markedly decreased ASL signal in the contralateral cerebral hemisphere, mimicking vascular shunting, but was due to artifact from an anesthesia device placed on the contralateral neck (Fig. 5). The patient with facial lipomatosis (Patient 24) also had hemimegalencephaly that showed increased ASL CBF in the enlarged cerebral hemisphere. There was a total of 8 cases in which ASL imaging provided additional information when compared with the conventional MR sequences (Table 1).

Table 1
Patient demographics of vascular anomalies and ASL imaging features

| Patient | Age/Sex | Lesion location | Vascular Anomaly | ASL signal | Additional info from ASL compared with other sequences |
|---------|-----------|--|--|---------------------|--|
| 1 | 2 days/M | R temporal scalp | Hemangioma | Hyperintense | Excluded AVM in setting of multiple vessels |
| 2 | 5 wks/F | R parotid | Hemangioma | Hyperintense | None |
| 3 | 4 wks/F | L parotid | Hemangioma | Hyperintense | None |
| 4 | 9 wks/F | R periorbital/premalar | Hemangioma | Hyperintense | None |
| 5 | 15 mths/F | L frontal scalp | Hemangioma | Hyperintense | None |
| 6 | 5 wks/F | L parotid & L occipital scalp | Hemangioma | Hyperintense | None |
| 7 | 2 mths/M | L parotid & submental | Hemangioma | Hyperintense | None |
| 8 | 2 wks/F | R periorbital & R parotid | Hemangioma | Hyperintense | Lesion was more conspicuous |
| 9 | 6 mths/F | R buccal | Hemangioma | Hyperintense | None |
| 10 | 2 yrs./M | L nasal ala | Hemangioma | Hyperintense | None |
| 11 | 10 mths/F | L frontal scalp | Hemangioma | Hyperintense | None, artifact |
| 12 | 1 yr./F | L orbital & R suboccipital scalp | Hemangioma + PHACE | Hyperintense | Decreased CBF |
| 13 | 5 mths/F | Majority of L scalp, L orbit, subocciput | Hemangioma + PHACE | Hyperintense | Decreased CBF |
| 14 | 5 wks/F | L periorbital & L IAC | Hemangioma + PHACE | Hyperintense | IAC hemangioma more conspicuous, Symmetric CBF |
| 15 | 6 yrs./F | L submandibular/buccal | Venous malformation | Isointense | None |
| 16 | 8 yrs./M | L frontal scalp | Venous malformation | Mildly hyperintense | None |
| 17 | 8 yrs./F | L parietal | Venous malformation + sinus pericranii | Mildly hyperintense | None |
| 18 | 2 yrs./M | L parietal scalp | Venous malformation | Isointense | None |
| 19 | 4 wks/F | Several spaces of neck bilaterally | Lymphatic malformation | Isointense | None |
| 20 | 5 mths/F | R supraclavicular | Lymphatic malformation | Isointense | None |
| 21 | 8 mths/M | Buccal, sublingual, submental | Lymphatic malformation | Isointense | None |
| 22 | 2 yrs./M | Glabella | Involuting hemangioma | Isointense | None |
| 23 | 14 yrs./F | R periorbital | Involuting hemangioma + PHACE | Isointense | Symmetric CBF |
| 24 | 5 mths/M | L cheek | Facial lipomatosis + CLOVES | Isointense | Increased CBF in hemimegalencephaly |
| 25 | 10 yrs./F | L frontal scalp | AVM | Hyperintense | AV shunting |

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