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Reply:

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REPLY:

We thank Dr Nabavizadeh for his interest in our article, “Intracranial Arteriovenous Shunting: Detection with Arterial Spin-Labeling and Susceptibility-Weighted Imaging Combined.”¹

We agree that arterial spin-labeling (ASL) hypersignal in developmental venous anomalies (DVAs) may suggest transitional or mixed malformations with arteriovenous shunting. DVA may also coexist with a true arteriovenous malformation, as reported by Erdem et al,² with selective arterial embolization of the AVM while preserving the DVA. As highlighted by Dr Nabavizadeh, the presence of dilated deep medullary veins in a “spoke wheel” or caput medusae at the venous phase of angiograms is not sufficient for the diagnosis of DVA. Careful attention must be paid to the arterial phase to rule out arteriovenous shunting resulting in early opacification of the draining vein at the arterial phase of the angiogram. In all 8 patients with DVAs included in our study, the main draining vein was never opacified in the early and mid-arterial phases of the angiograms. Only a capillary stain was observed in 2 patients, forming a blush at the arterial phase, without any arteriovenous communication. These findings are in agreement with those reported by Roccatagliata et al,³ suggesting that a large spectrum of lesions exists from typical DVAs to clearly distinct AVMs. These cases probably correspond to DVAs surrounded by dilated arteriolar-capillary channels that may exceptionally lead to ischemic or hemorrhagic complications. This pattern may be due to the lack of adaptability to variations in intracranial venous equilibrium, as suggested by the authors.³

However, the natural history, prognosis, and therapeutic management of such lesions remain unclear and are widely debated in the literature. For a systematic review of digital subtraction angiography images in our study, we chose to classify the vascular malformations as DVAs when angiograms demonstrated a caput medusae appearance without opacification of the main draining vein at the arterial phase of the angiograms. If we had decided to classify these atypical DVAs on DSA as arteriovenous shunting, the specificity of ASL would have been even higher than that reported.

Signal abnormalities on ASL images may be located either in

the brain parenchyma surrounding the DVA or within the draining vein of the DVA. We agree that it may be speculated that a hypersignal of a part of the DVA on ASL images may potentially reflect the presence of an arteriovenous shunting, but little data exist in the literature comparing ASL signal with DSA in patients with DVA. In addition, erroneous interpretation of CBF may be made in patients with prolonged arterial arrival times. Thus, further studies are required to understand the real significance of these findings.

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