

Hemodynamic Evaluation of Cerebral Arteriovenous Malformations with Combined 4D Flow and Perfusion MRI

Can Wu¹, Timothy Carroll^{1,2}, Sameer Ansari^{2,3}, Amir Honarmand^{2,3}, Parmede Vakil^{2,3}, Michael Hurley^{2,3}, Bernard Bendok^{2,3}, James Carr², and Michael Markl^{1,2}

¹Biomedical Engineering, Northwestern University, Chicago, IL, United States, ²Radiology, Northwestern University, Chicago, IL, United States, ³Neurological Surgery, Northwestern University, Chicago, IL, United States

Introduction: Cerebral arteriovenous malformations (AVMs) are highly complex and heterogeneous cerebrovascular diseases with direct connection between arteries and veins. Digital subtraction angiography (DSA) is the gold standard for the diagnosis and evaluation of cerebral AVMs. The Spetzler-Martin grading system (SMG) is widely used for surgical risk stratification by evaluating AVM size, eloquence of adjacent tissue and venous drainage [1]. However, SMG does not provide any insight into local and systemic hemodynamics of AVM vasculature and its diagnostic value for prediction of bleeding or rupture is limited. In this study, 4D flow [2,3] and dynamic susceptibility contrast (DSC) perfusion [4] MRI were combined to quantitatively evaluate macro- and micro-vascular hemodynamics in cerebral AVMs, investigate the influence of SMG on AVM hemodynamics, and identify potential relationships between changes in macro- and microvascular flow and perfusion.

Methods: Seventeen pre-treatment cerebral AVM patients (10 Male+7 Female, age=39±15 years, SMG1=3, SMG2=6, SMG3=4, SMG4=4) were included in this study. DSC perfusion (TR/TE=1100/34ms, spatial resolution=1.7×1.7×5.0mm³, TA=2.5min) and 4D flow (TR/TE=5.2/2.8ms, flip angle=15°, VENC=80-100cm/s, spatial resolution=1.1×1.1×1.5mm³, temporal resolution=42ms, TA=15-20min) MR imaging were performed for all AVM patients on clinical 1.5T (Avanto) and 3.0T (Trio) MRI scanners (Siemens, Erlangen, Germany). A single dose (2ml/s, 0.1mmol/kg bodyweight) of GD-DTPA contrast agent (Magnevist, NJ, USA) was administered prior to perfusion imaging. 4D flow data were pre-processed by a customized Matlab program [5] before hemodynamic quantification using commercial software (EnSight, CEI, USA). Peak velocity (m/s) and mean blood flow (ml/s) were quantified at AVM feeding and normal contralateral arteries, draining veins, and the straight sinus. Perfusion images were analyzed using a model including 4 pairs of regions of interest (ROIs) in the AVM nidus (Ni, Nc), hemisphere without the nidus (Hi, Hc), perinidal (Pi, Pc) and remote (Ri, Rc) areas at the ipsilateral and contralateral hemispheres, respectively (Figure 1, middle). Perfusion ratios (CBF: cerebral blood flow, CBV: cerebral blood volume, and MTT: mean transit time) between the affected and non-affected hemispheres were calculated. Figure 1 illustrates the analysis plane locations (Figure 1, left) for a left medial occipital AVM (SMG=3) used for hemodynamic quantification and the corresponding CBF image with ROI labels (Figure 1, right). Macro- and microvascular hemodynamic measures were compared between the low (SMG-A, SMG=1&2) and high (SMG-B, SMG=3&4) risk groups.

Results: 20 AVM feeding arteries (MCA=10, PCA=8, ACA=1, PICA=1) and normal contralateral arteries were identified in the 4D flow data. Figure 2 shows the peak velocities and blood flow in all feeding arteries, normal contralateral arteries, draining veins, and the straight sinus. Both the peak velocity and blood flow in the feeding arteries were significantly higher compared to normal contralateral arteries (p<0.001). High risk group AVMs had significantly higher peak velocity in comparison with the low risk group (p=0.007). Blood flow (p=0.001) in the draining veins and both peak velocity (p=0.003) and blood flow (p=0.012) in the straight sinus were significantly higher in the high risk group compared with the low risk group. The asymmetric ratios of perfusion revealed that the AVM nidus had increased CBF (Ni/Nc=2.13±0.77) and CBV (2.98±0.98) but decreased MTT (0.87±0.14) compared with the normal tissue in the contralateral hemisphere (Figure 3a). In addition, significant relationships (Spearman's correlation) were observed between the SMG and CBF ratios (Ni/Nc) in the AVM nidus (r=0.61, p=0.012), SMG and summation of the blood flow in all feeding arteries (r=0.85, p<0.001), SMG and blood flow in the major draining vein (r=0.80, p<0.001). We also found significant relationships (r=0.59, p=0.033, Pearson's correlation) between the AVM feeding to normal contralateral arteries flow ratio and the CBV ratios (Ni/Nc) in the AVM nidus. Perfusion differences in other regions (Hi/Hc, Pi/Pc, and Ri/Rc) between the affected and nonaffected hemispheres were not found to be significant.

Discussion: 4D flow and DSC perfusion MRI were successfully employed to provide quantitative hemodynamics in the large intracranial vessels and brain tissue in patients with cerebral AVMs. Hemodynamic information in the AVM feeding, draining and sinus systems may provide new insights into the pathophysiology and risk stratification of cerebral AVMs. The results of this study demonstrated the influence of SMG on the AVM hemodynamics in the feeding arteries, draining veins, and the straight sinus. In addition, we could identify significant relationships between large vessel flow and perfusion ratios. These findings demonstrate the potential of our imaging protocol for the comprehensive characterization of the impact of vascular malformation such as AVMs on macro- and microvascular hemodynamics. Further studies with large patient cohorts are needed to determine additional factors (size, location, or venous drainage) responsible for the hemodynamic changes. In addition, fully quantitative perfusion imaging would provide further insight into the controversial AVM steal and hemodynamic disturbance and enable investigating the relationship between macrovascular flow in large AVM vessels and microvascular perfusion within corresponding vascular territories.

Acknowledgements: Grant support by NIH/NIBIB T32 EB005170.

References: [1] Spetzler RF, et al. J Neurosurg 1986; 65:476-483. [2] Markl M, et al. J Magn Reson Imaging 2012; 36:1015-1036. [3] Markl M, et al. J Cardiovasc Magn Reson 2011; 13:7. [4] Srouf JM et al. J Cereb Blood Flow Metab 2011; 31:1272-1282. [5] Bock J, et al. ISMRM 2008; 16:3050.

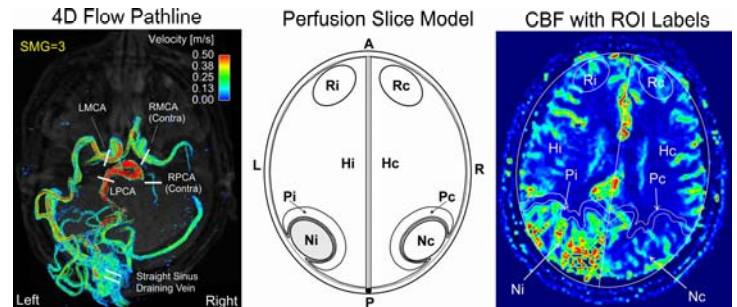


Figure 1: 4D flow visualization (pathlines) and analysis plane positions for flow quantification in a left medial occipital AVM (left); Perfusion model with four ROIs at the ipsilateral (affected) and contralateral (non-affected) hemispheres for perfusion ratio analysis (middle); CBF image with the ROI labels (right).

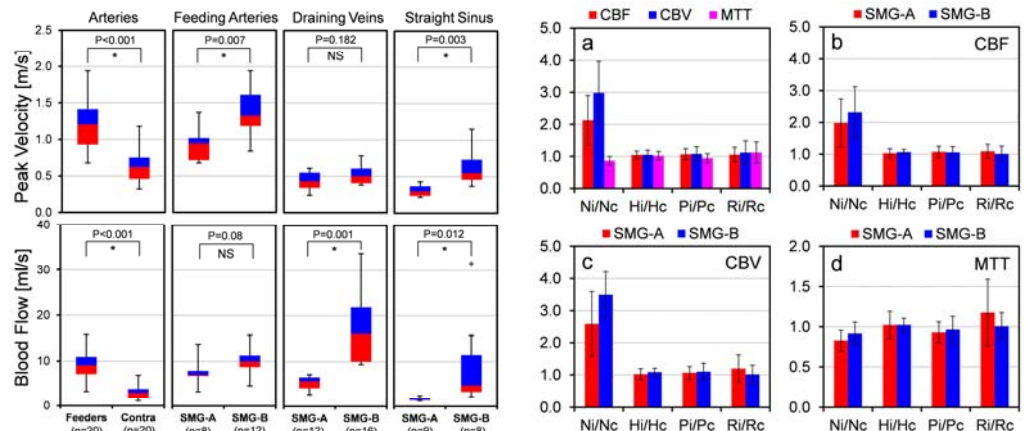


Figure 2: Distribution and comparison of the peak velocities and blood flow in the AVM feeding arteries, contralateral arteries, draining veins, and straight sinus.

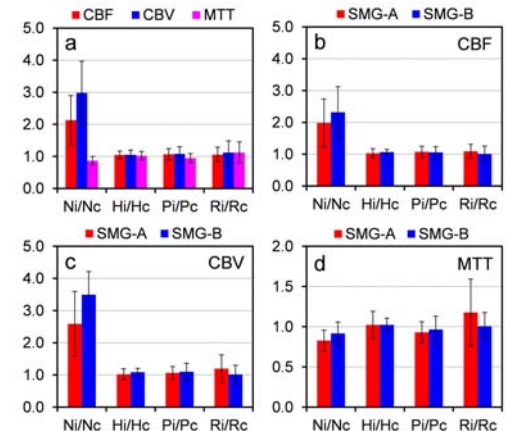


Figure 3: Perfusion ratios (CBF, CBV, and MTT) in four pairs of ROIs (Ni/Nc, Hi/Hc, Pi/Pc, and Ri/Rc) between the affected and nonaffected hemispheres.