



Clinical Study

Relationship of pulsatility and resistance indices to cerebral arteriovenous malformation angioarchitectural features and hemorrhage



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ABSTRACT

The role that hemodynamics plays in the pathophysiology of cerebral arteriovenous malformation (AVM) hemorrhage remains unclear. Here, we examine the relationship of pulsatility and resistance indices to AVM angioarchitectural features and hemorrhage. Records of patients with cerebral AVMs evaluated at our institution between 2007–2014 and with flows obtained before treatment using quantitative magnetic resonance angiography (QMRA) were retrospectively reviewed. Flow volume rate and flow velocity were measured in primary arterial feeders and compared to their contralateral counterparts. Pulsatility index (PI) = [(systolic flow velocity – diastolic flow velocity)/mean flow velocity] and resistance index (RI) = [(systolic flow velocity – diastolic flow velocity)/systolic flow velocity] were calculated for each feeder and compared to the normal contralateral vessel. Relationships between PI, RI and AVM clinical and angioarchitectural features were assessed using linear regression. Seventy-two patients with a total of 101 feeder arteries were included. PI and RI were significantly lower in AVM arterial feeders compared to normal vessels, thereby resulting in significantly higher flow volume rates and flow velocities in feeder vessels. There was no significant association of PI and RI with hemorrhagic presentation, exclusive deep venous drainage, venous stenosis, single draining vein, or deep location. In conclusion, PI and RI can be measured using QMRA and are lower in AVM arterial feeders compared to normal vessels. Although we found no significant correlation between PI, RI, and AVM angioarchitectural characteristics thought to be associated with increased hemorrhage risk, future studies with larger sample sizes may better elucidate this relationship.

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1. Introduction

Cerebral arteriovenous malformations (AVMs) are vascular abnormalities that consist of direct connections from arteries to veins through an intervening network of low resistance vessels rather than through normal capillary beds, subsequently resulting in disrupted hemodynamics and about a 2% to 4% annual risk of intracranial hemorrhage [1–3]. Despite this hemodynamic pathophysiology, current AVM characterization and hemorrhage risk assessment is largely based on anatomical features derived from digital subtraction angiography rather than on flow parameters [4,5]. Specifically, presence of intranidal or feeder artery aneurysms, which are thought to be angioarchitectural surrogates of high AVM inflow, can be predictive of hemorrhage [1]. Additionally, venous outflow obstruction manifested anatomically by venous stenosis, deep venous drainage, deep AVM location, and a single draining vein, has been associated with increased hemorrhage risk [1].

Although AVM hemodynamics have been studied extensively in the past with transcranial Doppler ultrasound [6–9], and more recently with four-dimensional (4D) flow MRI and time-resolved spin-labeled magnetic resonance angiography (MRA) [10,11], pulsatility index (PI) and resistance index (RI) have not been evaluated in a large cohort of patients with cerebral AVMs using quantitative magnetic resonance angiography (QMRA). Moreover, the relationship between PI, RI and AVM clinical and angioarchitectural hemorrhagic risk factors has not been clearly examined. In this study, we measured flow volume rate, flow velocity, PI, and RI in the largest set of AVM feeders to date, using QMRA, and we analyzed the impact of AVM clinical and anatomical characteristics on PI and RI.

2. Methods

2.1. Patient selection

Following Institutional Review Board approval, clinical data for all patients with a cerebral AVM who underwent QMRA at our institution between 2007 and 2014 were collected and reviewed (n = 75). Patients who underwent previous AVM treatment,

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including prior embolization or radiation treatment, were excluded ($n = 3$). A total of 72 patients were included in the study cohort. Among these 72 AVMs, there were a total of 108 arterial feeders. Hemodynamic measurements were available for 101 of these feeders – 30 anterior cerebral artery (ACA), 47 middle cerebral artery (MCA), and 24 posterior cerebral artery (PCA).

2.2. Angiographic evaluation

All patients in this study underwent digital subtraction angiography before any treatment. Angiograms were reviewed by two independent neuroendovascular surgeons. Presence of AVM angioarchitectural features, including venous stenosis, deep venous drainage, and single draining vein, was identified and recorded.

2.3. Blood flow measurements

All patients in this study underwent quantitative flow volume rate (mL/min) and flow velocity (cm/s) measurements of the extracranial and intracranial arteries using QMRA. This technique of blood flow quantification by QMRA has been described previously by Zhao et al. [12] and is currently used in the clinical setting with the commercially available Noninvasive Optimal Vessel Analysis (NOVA) software (VasSol, Inc., River Forest, IL, USA). Specifically, all subjects underwent phase contrast QMRA performed on a 1.5 Tesla or 3.0 Tesla magnetic resonance (MR) system (Sigma VHi, GE Medical system, Milwaukee, WI, USA) using a 4-channel neurovascular coil. A three-dimensional (3D) MRA time-of-flight (TOF) of the head was obtained with the following parameters: repetition time/echo time (TR/TE): 23/3.3 ms; flip angle: 20; field of view (FOV): 220 mm; section thickness: 1 mm; matrix: 512 X256. MRA TOF images were received by the NOVA software on a separate workstation in order to reconstruct a 3D surface-rendering of the vasculature for determining the perpendicular scan plane to vessels of interest. Volume flow measurements based on these positions were performed (TR: 10–15 ms; TE: 4–7 ms; flip angle: 15; NEX: 4; slice thickness: 3 mm for intracranial arteries and 5 mm for neck arteries; FOV: 140 mm for intracranial arteries and 180 mm for neck arteries; matrix: 256 × 192 for intracranial arteries and 256 × 128 for neck arteries). Velocity encoding was automatically adjusted by the NOVA software. All QMRA flow measurements were performed using an oblique two dimensional fast phase contrast sequence with retrospective gating. Flow volume rate and flow velocity in each artery were processed on the NOVA workstation after phase contrast images had been acquired. This QMRA technique has been validated using *in vitro* and *in vivo* models and has demonstrated utility in the hemodynamic evaluation of cerebrovascular pathologies and interventions, including extracranial carotid artery stenosis, intracranial angioplasty/stenting, carotid endarterectomy, and extracranial-intracranial bypass [13–18].

Flows within proximal feeder vessels (ACA: A2 segment; MCA: M1 segment; and/or PCA: P2 segment) and their contralateral counterparts, were obtained.

2.4. Pulsatility index and resistance index calculation

Once flow velocities were measured, PI and RI were calculated for each AVM arterial feeder using the Gosling and Pourcelot equations [19,20]:

$$PI = (\text{systolic flow velocity} - \text{diastolic flow velocity}) / \text{mean flow velocity}$$

$$RI = (\text{systolic flow velocity} - \text{diastolic flow velocity}) / \text{systolic flow velocity}$$

2.5. Statistical analysis

Mean flow volume rate, flow velocity, PI, and RI in ipsilateral versus contralateral feeder arteries were compared using the paired 2-tailed student's *t*-test. Univariate linear regression analysis was used to correlate PI, RI, hemorrhagic presentation, and angioarchitectural features. The independent 2-tailed student's *t*-test was performed to examine the differences in PI and RI relative to hemorrhagic presentation and presence of AVM anatomical characteristics (venous stenosis, deep venous drainage, single draining vein, and deep location). All analyses were performed with SPSS (Version 22; IBM, Armonk, NY, USA).

3. Results

3.1. Patient characteristics

The mean age of the cohort ($n = 72$) was 38 years. 24% of patients presented with hemorrhage. 25% of AVMs were Spetzler–Martin grade I, 34% grade II, 25% grade III, 10% grade IV, and 6% grade V. The mean volume of these AVMs was 11.5 mL (range 0.3–62.8 mL). Patient and AVM characteristics are outlined in Table 1.

3.2. Ipsilateral versus contralateral flow volume rate and flow velocity

Among all AVM feeder arteries, mean flow volume rate (\pm standard deviation) (332.3 ± 244.1 vs. 121.6 ± 66.9 mL/min, $p < 0.001$) and flow velocity (46.1 ± 19.9 vs. 25.8 ± 8.5 cm/s, $p < 0.001$) were significantly higher than in the normal contralateral vessels. Similar results were obtained among each vessel type (Fig. 1) – mean flow volume rate and mean flow velocity were significantly higher in the ipsilateral versus contralateral ACA, MCA, and PCA.

3.3. Ipsilateral versus contralateral pulsatility index and resistance index

Among all feeder vessels, mean PI (0.20 ± 0.12 vs. 0.27 ± 0.13 , $p < 0.001$) and RI (0.18 ± 0.09 vs. 0.23 ± 0.09 , $p < 0.001$) were significantly lower than in their normal contralateral counterparts. Similar results were obtained among each vessel type (Fig. 2) – mean PI and RI were significantly lower in the ipsilateral versus contralateral ACA and MCA. Among the 27 AVMs that had multiple feeders, the PI and RI of the feeders of the same AVM were similar, except one AVM in which the PI and RI of one of the feeders was more than two standard deviations below the mean.

Table 1
Patient and arteriovenous malformation (AVM) characteristics

Patients ($n = 72$)	
Male (%)	56
Mean age, years (range)	38 (16–72)
Hemorrhagic presentation (%)	24
AVM characteristics	
Spetzler–Martin Grade (% of cohort)	1 (25)
	2 (34)
	3 (25)
	4 (10)
	5 (6)
Exclusive deep venous drainage (%)	7
Venous stenosis (%)	18
Single draining vein (%)	33
Deep location (%)	7
Volume, mL (range)	11.5 (0.3–62.8)

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