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HEMODYNAMIC PROPERTIES OF FEEDER VESSELS FOR CEREBRAL ARTERIOVENOUS MALFORMATIONS WITH AND WITHOUT ANEURYSMS

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Short Review

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ABSTRACT

BACKGROUND: The blood flow pattern and vessel architecture are disturbed in the neurovascular malformation. The cerebral arteriovenous malformation (CAVM) is a type of cerebrovascular illness characterized by impaired blood flow and a tangle of aberrant blood vessels formed by the direct connection of arteries to veins in the absence of a capillary bed. The term "Nidus" refers to the tangled aberrant vessels that make up the center portion of the deformity. A lot of blood is taken because the AVM-affected area has extremely low pressure. In comparison to normal pressure readings, there is a significant build-up of pressure in the blood vessels, particularly in the veins, because of changes in blood flow patterns. It creates the pressure gradient between veins and arteries.

MATERIAL AND METHODS: The Study was conducted in the Department of Neurosurgery. We follow reporting criteria for CAVM examined a range of CAVM features and Nidus structure types. The AVMs are made up of different permutations of the Nidus complex vascular structure and are found in numerous parts of the human body, including the spine and lungs. For every kind of CAVM, there is a distinct way that Nidus structures form. Because the AVM may burst, the intrusive technique used to get hemodynamic measures for each type of AVM in different organs is challenging for doctors and dangerous for patients.

RESULTS: Absolute mean feeder artery flow (498.6 versus 430.3 mL/min; *P*=0.51) was similar between the 2 groups and vessel diameter (4.0 versus 4.8 mm; *P*=0.24) tended to be lower in feeders with aneurysms, but not significantly. However, WSS (95.7 versus 27.9 dynes/cm^2; *P*=0.04) was significantly higher in feeders with aneurysms.

CONCLUSION: Feeder artery diameter tended to be lower in feeders with aneurysms despite comparable high flows, suggesting that this subgroup of AVM feeders is incapable of compensating for increased blood flow through vascular remodeling.

KEY WORDS: Cerebral Arteriovenous Malformations, Hemodynamics, Feeder Vessels, Internal carotid artery and Wall shear stress

INTRODUCTION

Among the body's vital organs is the brain. The brain's architecture are extremely intricate. Cerebral circulation is explained by the physiology of the brain. The cerebral arterial circle, often known as the Circle of Willis (CoW), is the primary distribution of cerebral blood flow. A collection of arteries close to the brain's base is known as the CoW. The two internal carotid arteries and the two vertebral arteries make up the four primary arteries that deliver blood to the brain. The combinations of arteries implicated determine the clinical outcome of cerebrovascular illness. As a result, the typical brain flow pattern follows a capillary bed from arteries to veins.[1]

The blood flow pattern and vessel architecture are disturbed in the neurovascular malformation. The cerebral arteriovenous malformation (CAVM) is a type of cerebrovascular illness characterized by impaired blood flow and a tangle of aberrant blood vessels formed by the direct connection of arteries to veins in the absence of a capillary bed. The term "Nidus" refers to the tangled aberrant vessels that make up the center portion of the deformity. A lot of blood is taken because the AVM-affected area has extremely low pressure. In comparison to normal pressure readings, there is a significant build-up of pressure in the blood vessels, particularly in the veins, as a result of changes in blood flow patterns. It creates the pressure gradient between veins and arteries.[2] Intracranial hemodynamics are altered by the high-velocity flows through Nidus that follow. Before and after cerebral AVMs radiosurgery, the blood flow and cerebral pressure patterns of the CoW are examined. The Digital Subtraction Angiogram (DSA) is the gold standard for CAVM imaging. Cerebral hemodynamics are impacted by the CAVM, which leads to changes in clinical flow metrics such cerebral blood flow, cerebral pressure, and cerebral velocity.[3]

Most of the research has evaluated cerebral hemodynamics modeling dynamically at several locations. The problem, though, is that they haven't examined more profound structural variation. The studies examined non-stationary multivariate modeling of cerebral autoregulation and patient-specific hemodynamics applied to aortic illnesses, brain disease, and coronary artery disease. Nevertheless, clinical validations place limitations on the models. Because structural differences limit mechanical modeling, the researchers used it to assess the swine AVM model.[4]

Cerebral hemodynamics varies from patient to patient in CAVM patients due to a variety of reasons. Quantification analysis limits research on experimental models to investigative techniques in the field of cerebrovascular analysis. An earlier mathematical AVM model with several arrays of simulated interweaving plexiform and fistulous Nidus vessels was created using electrical network analysis; the only drawback was input parameter uncertainty. The study by Gary et al.[5], shows that in the vessel collapsing model for pressure area analysis, but the drawback is that parameters used in modeling is very less. The human brain's circulation was examined for various stimuli in Krause et al.'s study on feedback structures for loop modeling. This model is constrained by exact parameter values. In another study by Gijsen et al.[6], on bifurcation modeling the model is limited by effects of a variation of the flow pulses.

METHODOLOGY

CAVM structure type simulation:

The Study was conducted in the Department of Neurosurgery. We follow reporting criteria for CAVM examined a range of CAVM features and Nidus structure types. The AVMs are made up of different permutations of the Nidus complex vascular structure and are found in numerous parts of the human body, including the spine and lungs. For every kind of CAVM, there is a distinct way that Nidus structures form. Because the AVM may burst, the intrusive technique used to get hemodynamic measures for each type of AVM in different organs is challenging for doctors and dangerous for patients.

Patient Selection

Clinical information for each patient with a cerebral AVM who had quantitative magnetic resonance angiography performed at our facility, with consent from the institutional review board. Using digital subtraction angiography, AVMs linked to feeder aneurysms—defined as aneurysms originating from arteries supplying the AVM—were found. Feeders with and without aneurysms were divided into two groups. There were no intraanidal aneurysms reported.

Flow, Vessel Diameter and Wall Shear Stress Measurements

Prior to receiving treatment for the AVM, all study participants had quantitative magnetic resonance angiography, which measured the extracranial and intracranial arteries' sizes and flow rates. Additional information is available in the online-only Data Supplement. This method was previously described and confirmed, and it was carried out using the commercial software Noninvasive Optimal Vessel Analysis (NOVA, VasSol Inc, River Forest, IL).[7]

When present in the following anatomic locations, the primary arterial feeders were measured for flow and diameter: internal carotid artery, cervical segment; anterior cerebral artery, A2 segment; middle cerebral artery, M1 segment; and posterior cerebral artery, P2 segment. These feeders are located proximal to the aneurysm or the AVM. The online-only Data Supplement's Figure I provides an example of a case.

Statistical Analysis

Mean flow, diameter, and WSS were compared between the 2 groups using the independent 2-tailed Student *t* test. Exponential regression analysis was used to assess the relationship between blood flow, vessel diameter, and WSS in the 2 groups. All analyses were performed with SPSS (Version 22; IBM Inc).

RESULT

Table 1: Clinical, Anatomic and Hemodynamic Characteristics of Arteriovenous Malformation Feeders with and without Aneurysms

	with Aneurysms	without Aneurysms
Clinical Characteristics		
Mean age (yrs)	44,63±18.32	46.45±17.86
Hemorrhagic Presentation (%)	9.1	
Hemodynamics	N=23	N=12
Feeder artery type (% of feeder vessels)	ICA (36)	ICA (25)
	A2 (27)	A2 (25)
	M1 (23)	M1 (33)
	P2 (14)	P2 (17)

Mean flow, mL/min (Mean ± SD)	430.3±312.6	498.6±249.2
Mean vessel diameter, mm (Mean ± SD)	4.8±1.9	4.0±1.5
Mean WSS, dynes/cm^2 (Mean ± SD)	27.9±18.7	95.7±45.8
ICA=Internal carotid artery, WSS= Wall shear stress		

Absolute mean feeder artery flow (498.6 versus 430.3 mL/min; *P*=0.51) was similar between the 2 groups and vessel diameter (4.0 versus 4.8 mm; *P*=0.24) tended to be lower in feeders with aneurysms, but not significantly. However, WSS (95.7 versus 27.9 dynes/cm^2; *P*=0.04) was significantly higher in feeders with aneurysms.

DISCUSSION

Since Walsh and King[8] described the first clinical case of a cerebral AVM with a feeder aneurysm, numerous studies have confirmed this association and speculated on its pathogenesis. The fundamental cause of aneurysm formation is thought to be high blood flow to the AVM; however, as most AVMs do not generate feeder aneurysms, another hemodynamic mechanism may be at play. Brown et al.[9] found that AVM flow and shunt characteristics, assessed on angiography by the number and size of feeders as well as time between arterial and venous phases, were similar in AVMs with and without feeder aneurysms and instead suggested that higher flow velocities in feeders with aneurysms could result in turbulence and hemodynamic stress.

However, only 1 other study has directly measured and compared hemodynamic parameters in AVM feeder arteries with and without aneurysms. Using time-resolved 3-dimensional magnetic resonance angiography, Illies et al.[10] found no significant correlation between altered transit time and presence of a feeder aneurysm. But their investigation lacked additional assessment of other hemodynamic features. Here, we find that, in comparison to feeders without aneurysms, feeders with aneurysms have equal flow rates but smaller diameters, which results in much higher WSS. Overall, these findings are consistent with WSS being the most likely cause of the pathophysiology of AVM feeder aneurysms. Our results show that WSS may have an effect on cerebral AVM feeder vasculature and support the notion that it is a significant biomechanical stimulus for vascular remodeling.

CONCLUSION

Due to the much higher WSS in cerebral AVM feeder arteries, there are disparities in hemodynamics between those with and without aneurysms. Despite similar high flows, feeder artery width tended to be lower in aneurysm-ridden feeders, indicating that this subset of AVM feeders is unable to adapt their vascular architecture to compensate for higher blood flow.

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