Delayed onset of non-ischemic cerebral enhancing lesions is a rare complication of intracranial aneurysms' endovascular therapy (EVT). This complication has been attributed either to foreign body emboli and subsequent granulomatous reaction or cerebral hypersensitivity and nickel allergy. After retrospective review of all patients managed by EVT at our Institution from January 1st 2012 to December 31st 2014, 2 out of 374 patients (0.5%) with such a complication were identified. Patient # 1 developed non-ischemic cerebral enhancing (NICE) lesions 1 month after balloon assisted coiling of a ruptured anterior communicating artery aneurysm. Patient # 2 developed NICE lesions 12 months (the longest delay reported to date for such complication) after the treatment of a right carotidophthalmic aneurysm by loose coiling and flow diversion. Skin patch testing was performed with all endovascular devices used in the 2 patients and with the European baseline series, including nickel. Patient # 2 demonstrated nickel skin reactivity but none of the 2 patients presented allergic reaction to the devices used during interventions. Based on our observations and review of the literature, we hypothesize that delayed non-ischemic cerebral enhancing lesions after EVT are more likely related to foreign body emboli rather than nickel allergy.

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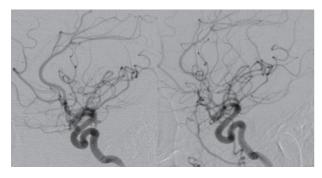
P-021

VERY DELAYED MONOCULAR BLINDNESS FOLLOWING FLOW DIVERSION TREATMENT OF OPHTHALMIC ARTERY ANEURYSM

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Introduction Flow diversion is widely used for the effective endovascular treatment of wide-necked intracranial internal carotid artery (ICA) aneurysms. Ophthalmic artery (OA) occlusion is a known consequence related to flow diversion; however, OA occlusion in this setting usually is without clinical sequela. All reported cases of monocular vision loss after flow diversion have occurred within days to weeks of treatment.



Abstract P-021 Figure 1

Methods We describe a case of monocular blindness due to ophthalmic artery occlusion nearly 3 years after flow diversion treatment of tandem supraclinoid ICA aneurysms. The medical records and images were reviewed for this patient.

Results This 51 year-old woman with history of hypertension was found to have two incidental right supraclinoid ICA aneurysms following headache workup. Cerebral angiography revealed a wide neck 5 mm saccular ophthalmic aneurysm and 3 mm bi-lobed posterior communicating artery (PCoA) aneurysm. Treatment with flow diversion was performed. A 4 × 20mm Pipeline Embolization Device (PED) was deployed from the ICA terminus to the distal genu of the cavernous segment ICA without complication. She had been on dual antiplatelet therapy for 3 months followed by aspirin monotherapy. Six-month follow up angiography revealed obliteration of PCoA aneurysm, but with persistent filling of the ophthalmic aneurysm. Twenty months later, angiography again revealed persistent filling of the ophthalmic aneurysm. Both studies revealed patent right ophthalmic artery.

She presented with acute onset of complete right vision loss 32 months after PED placement. MRI brain showed no acute stroke. Dilated eye exam revealed cherry red spot in macula with attenuated vessels, and retinal whitening, consistent with ophthalmic artery occlusion. Diagnostic cerebral angiography revealed occlusion of the right ophthalmic artery with patent ICA and well apposed PED without in-stent stenosis or thrombosis. The ophthalmic aneurysm was no longer filling. There were no middle meningeal collaterals to the ophthalmic artery. IA tPA was given locally near the ophthalmic artery origin with minimal improvement. She was compliant with her aspirin throughout.

Conclusion Monocular vision loss remains a risk, even months to years following flow diversion of ICA aneurysms. Long term clinical follow-up is necessary to define the incidence of this complication.

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P-022

THE ROAD LESS TRAVELED: TRANSARTERIAL EMBOLIZATION OF DURAL ARTERIOVENOUS FISTULAS VIA THE ASCENDING PHARYNGEAL ARTERY

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Introduction With the introduction of Onyx, transarterial embolization has become the most common endovascular approach for dural arteriovenous fistulas (dAVF)s, often via the middle meningeal or occipital arteries. The ascending pharyngeal artery (APA) is a less frequently explored transarterial route as a result of its small caliber, potential anastomoses to the internal carotid and vertebral arteries, and its vital supply to lower cranial nerves.

Methods We reviewed our endovascular database (January 1996 – March 2016) for cranial dAVFs, evaluating dAVF characteristics and embolization results for those treated transarterially via the APA.

Results Of 267 endovascularly-treated dAVFs, 68 had APA supply (25%). This included all marginal sinus dAVFs (11/11), 43% of transverse/sigmoid (37/86), 26% of tentorial/petrosal

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(12/47), 20% of torcular (3/15), and 8% of cavernous sinus (5/60) dAVFs. Of 68 dAVFs with APA supply, embolization was carried out via this pedicle in eight (12%), and seven were ultimately occluded. There were no complications, including no post-treatment cranial neuropathies or radiographic evidence of nontarget embolization. For five dAVFs, the APA was selected as the initial pedicle for embolization (marginal sinus, n = 2; distal sigmoid, n = 1; cavernous, n = 1, tentorial, n = 1). In 4/5 cases, dAVF occlusion was achieved via the initial APA feeding artery pedicle. In one case, near complete, stagnant occlusion was achieved; adjunctive embolization of a single additional MMA pedicle was performed. In three other cases of complex transverse/sigmoid dAVF, the APA was utilized after multiple attempts via middle meningeal and occipital artery pedicles. Occlusion was not achieved transarterially; two of the three dAVFs were ultimately occluded transvenously.

Conclusion In rare, select cases, the APA is an excellent route for transarterial embolization of cranial dAVFs.

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P-023

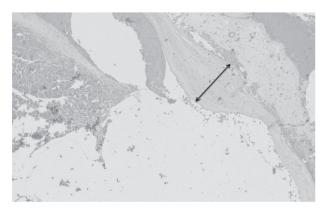
CEREBRAL ARTERIOVENOUS MALFORMATION FLOW IS ASSOCIATED WITH VENOUS INTIMAL HYPERPLASIA

15 Shakur, 1A Hussein, 1S Amin-Hanjani, 2T Valyi-Nagy, 1V Aletich, 1F Charbel, 1A Alaraj. ¹Neurosurgery, University of Illinois at Chicago, Chicago, IL; ²Pathology, University of Illinois at Chicago, Chicago, IL

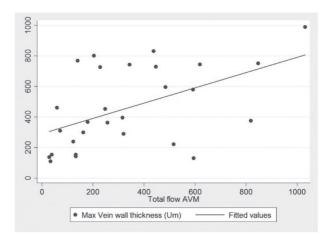
10.1136/neurintsurg-2016-012589.65

Introduction/purpose Histopathological changes in cerebral arteriovenous malformation (AVM) draining veins secondary to chronically high AVM inflow have not been clearly elucidated. Here, we examine the relationship between draining vein wall thickness and AVM flow rate.

Materials and methods Records of patients with cerebral AVMs evaluated at our institution between 2007-2013 were retrospectively reviewed. Patients were included if a surgical specimen of the nidus was available and if flows were obtained before treatment using quantitative magnetic resonance angiography. Specimens were mounted on slides and stained with hematoxylin and eosin as well as elastin special stain. Perinidal veins were identified and the wall thickness of each vein was measured from digitized images of the slides (Figure 1). Maximum vein wall thickness was recorded for each specimen.



Abstract P-023 Figure 1 Example of a perinidal vein with thickened wall. Elastin special stain; original magnification x 10



Abstract P-023 Figure 2 Maximum vein wall thickness (µm) versus total AVM flow (mL/minute) (rho = +0.51, P = 0.006)

Intranidal arteries were also identified and the diameter of each artery was measured. Total AVM flow was estimated as aggregate flow within primary arterial feeders or flow in single draining veins. The relationship between maximum vein wall thickness, total AVM flow, flow per draining vein, flow per unit volume of AVM, and mean intranidal artery diameter

Results 28 patients (20 male, 8 female) with mean age of 37 years (range 16-68 years) were included. Spearman's correlation revealed a statistically significant relationship between maximum vein wall thickness and total AVM flow (rho = +0.51, P = 0.006) (Figure 2) as well as AVM flow per draining vein (rho = +0.41, P = 0.03). However, there was no statistically significant correlation between maximum vein wall thickness and flow per unit volume of AVM (rho = +0.27, P = 0.17) or mean intranidal artery diameter (rho = +0.42, P = 0.24). Mean vein wall thickness was significantly higher in the presence of venous ectasia (562 µm vs. 300 μ m, P = 0.007). Presence of venous stenosis was not significantly associated with age, Spetzler-Martin grade, volume, number of draining veins, deep venous drainage, intranidal fistula, or maximum vein wall thickness.

Conclusion Maximum vein wall thickness is significantly related to total AVM flow and AVM flow per draining vein. This finding implicates chronically high AVM inflow in venous intimal hyperplasia and possible subsequent development of venous outflow stenosis.

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P-024 PROMINENT CONDYLAR VEINS CAUSING PULSATILE TINNITUS: DYNAMIC ANGIOGRAPHIC CONFIRMATION

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Introduction/purpose Numerous processes can cause pulsatile tinnitus (PT), some of which are potentially life threatening. This case series describes a cause of PT - prominent condylar veins - that has undergone little investigation to date. This report characterizes angiographic findings in patients with

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