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The Brain Aneurysm Institute

Multidisciplinary Care of Patients with Hemorrhagic and Ischemic Stroke

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Neurovascular News

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Dissecting the Risk: A Comparative Study of Management Strategies for Extracranial Carotid and Vertebral Pseudoaneurysms

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Extracranial carotid and vertebral dissections are a rare cause of stroke in the general population, with a yearly incidence estimated to be 2.97/100,000.[1] While an uncommon diagnosis, often overlooked due to the high number of asymptomatic cases, dissections impose a significant burden of disease in young adults.[2] The development of false aneurysms, also termed dissecting or pseudoaneurysm, has been reported to occur in up to 49% of cervical artery dissections. This progression is secondary to the development of a tear in the intimal layer of the vessel and carries a higher risk of thrombus formation within the vessel lumen.[3] That said, unlike their intracranial counterparts, which can rupture and cause devastating subarachnoid hemorrhage, the most feared complication of these lesions is ischemic stroke.[2] The mechanism is thought to be an embolus forming from the aneurysm thrombus. This rationale is the basis for treatment, whether by medical management with anti-platelets, anti-coagulation agents, or more invasively by endovascular or open surgical techniques. The goal is to minimize the likelihood of platelet activation by repairing the injured endothelium or by medically inhibiting platelet activation or coagulation cascades. To note, none of these treatment modalities are without their risks.

There is evidence in the literature to suggest that anti-platelet and anti-coagulation agents offer similar protection against dissection complications.[4,5] Beyond this distinction, the literature is sparse regarding treatment options for extracranial pseudoaneurysms and the role for endovascular and surgical intervention. One of the challenges in studying these lesions is their rarity. In addition, providers have selected to treat more of these lesions endovascularly as the technique has become safer and more effective. The question remaining is whether there is a role for conservative medical therapy in this new age of endovascular intervention.

At the BIDMC Brain Aneurysm Institute we set out to characterize the factors leading to the different management decisions and evaluate clinical and radiological outcomes of patients treated with each modality. We performed a review of medical and treatment records at two institutions and included patients diagnosed with extracranial carotid and vertebral pseudoaneurysms in the study. Patients with vascular disorders were excluded.

Patient and Lesion Characteristics

There was 143 pseudoaneurysms diagnosed in 131 patients between December 2006 and June 2023. The median age was 54 years old with most patients being females (58%). 43.5% of patients had a smoking history, 9.2% had diabetes, 45.8% had hypertension, and 30.5% were on

statins. On initial presentation, 24.4% of patients were asymptomatic while 93.1% were functionally independent. In terms of etiology, 22.9% of patients presented with a major trauma, 9.2% were due to a minor trauma, 9.2% were iatrogenic, and 58.8% presented with unknown etiologies. 16 (11.2%) patients presented with multiple lesions. In terms of lesion characteristics, 51% of lesions were left-sided and 20.3% were in the vertebral arteries. For lesions in the cervical carotid arteries, the majority were distal internal (69.2%), and much less were proximal to the bifurcation (7.7%), involving the common carotid bifurcation (2.1%), and a single lesion was in the common carotid (0.7%). Four lesions (2.8%) were ruptured at presentation, and the most common diagnostic imaging was CTA (71.3%), followed by MRA (17.5%) and DSA (11.1%). The median maximum diameter was 7.3 mm (IQR: 4.68 – 11) across all lesions.

Management Summary

Initial medical regimen was anti-platelets for 84.6%, anti-coagulants for 9.1%, a combination of the two for 1.4%, and none for 4.9%. Of note, many of these lesions presented to our services after they were diagnosed in the acute phase either by the emergency department or other providers. Thus, we were not able to ascertain what medical regimen many of the patients were initially on at the time of diagnosis. The number we collected reflect what the providers in this study prescribed the patients during their initial consultation.

In terms of specific medications, anti-platelets were a mix of aspirin 81 mg and 325 mg at initial follow-up depending on the patient's symptoms. By final follow-up, many of the treatment regimens were de-escalated with 17.4% of lesions were without anti-platelets or anti-coagulants on final follow-up. Of patients remaining on anti-platelet therapy, the majority were on 81 mg of aspirin. The duration patients were on medical therapy varied greatly but occlusion, decrease in size on imaging, and resolution of symptoms for 6-12 months seemed to trigger discontinuation of the medications.

In the endovascular group, the treatments including coiling, stenting, stenting and coiling, flow diversion, and flow diversion and coiling. Flow diversion has been proving to be an effective treatment for these lesions and more providers are electing to use it over traditional stenting. In terms of retreatment, two patients required a second treatment, one of whom required a third treatment.

Of patients on medical treatment alone, 18.7% occluded by final follow-up, 45% got smaller, and 9% stayed approximately the same size. In comparison, 100% of patients who underwent endovascular therapy achieved complete occlusion including retreatments (figure 1). This higher occlusion rate comes with a higher total complication rate of 13.6% in comparison with 1.7% for medically treated lesions. Thromboembolic complications, more specifically, were also significantly higher in the endovascular group (9% vs. 0%). Median follow-up durations did not differ significantly between the two groups (15.6 months for the medical group vs. 11.9 months for the endovascular group).

Management and Occlusion Predictors

We found three significant predictors for patients to undergo endovascular therapy: rupture status of the lesion, initial diagnostic modality being digital subtraction angiography, and a lesion diameter larger than 6 mm. Patients with these characteristics were 35 times, 8 times, and 4 times more likely to undergo endovascular treatment, respectively.

In the medical therapy group, factors that predicted occlusion included: patient age younger than 50 years old and lesion diameter smaller or equal to 6 mm. Patients with these characteristics were 8 times and 10 times more likely to occlude by final follow up, respectively.

Management Flowchart

To sum up our findings, we created a flowchart to aid in selecting the right patients for medical management (figure 2). Patients who do not fit the criteria (i.e. older with larger lesions, symptomatic, growing lesion), should be strongly considered for endovascular or surgical treatment. In terms of imaging, the only recommendation we can make is choosing to perform a DSA only for patients who are truly being considered for intervention. Otherwise, DSA carries with it a specific set of risks, and non-invasive imaging with CTA and MRA perform equally well for these lesions. These two non-invasive techniques have their unique capabilities – CTA is better at visualizing the dissection flap and MRA is better at detecting potential ischemic sequelae – but there is no data to suggest one is better than the other.

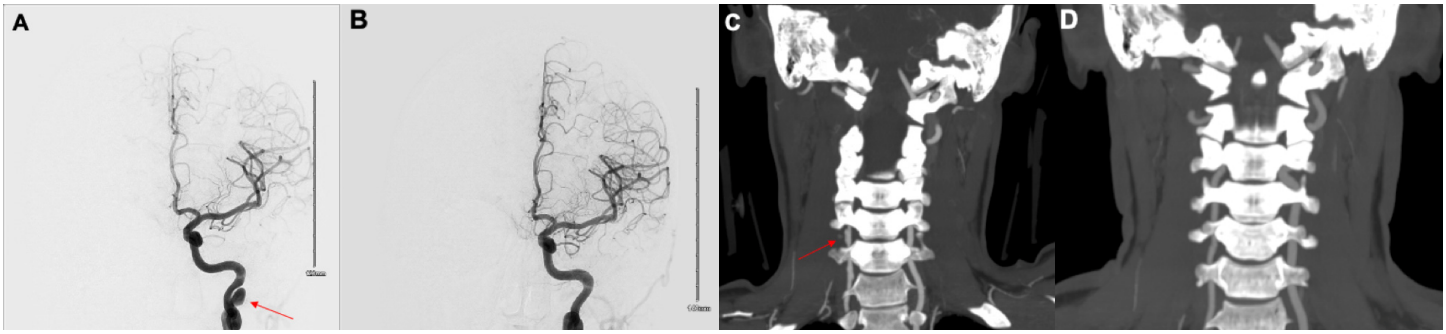

Figure 1: Pseudoaneurysms before and after treatment either with FD (A and B) or medical therapy only (C and D). A: 42-year-old female without any past medical history presented with Horner's syndrome and was found to have a 6 mm pseudoaneurysm in the left distal cervical carotid artery on DSA. B: She was treated with FD because of worsening visual symptoms and a follow-up DSA was obtained one year later which confirmed complete obliteration of the pseudoaneurysm. C: 23-year-old female presented with TBI, C1 fracture, and right vertebral artery pseudoaneurysm (V2 segment) after being struck by a motor vehicle. She required a hemicraniectomy but was discharged in good condition. She was started on Aspirin 81 mg daily for the pseudoaneurysm. D: CTA follow-up three months later showed complete obliteration of the pseudoaneurysm. CTA: computed tomography angiography. DSA: digital subtraction angiography, FD: flow diversion. TBI: traumatic brain injury.

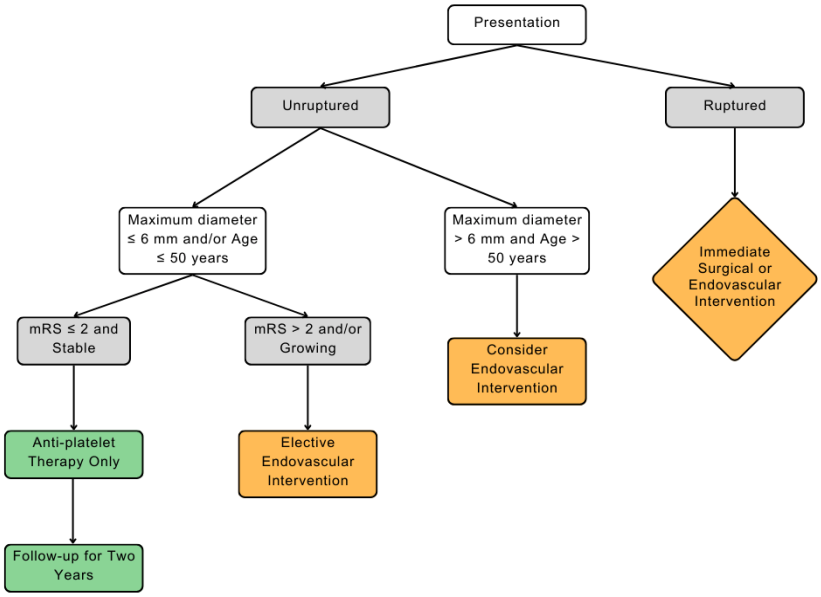

Figure 2: Flowchart for management of extracranial carotid and vertebral pseudoaneurysms. mRS: modified Rankin Scale.

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Flow Diversion in Bifurcation Aneurysms and the Dynamic Adaptability (Plasticity) of the Circle of Willis

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As detailed in previous Neurovascular News editions, flow diversion has emerged as a technique to treat a substantial number of intracranial aneurysms. Flow diverters (FDs) are specialized stents designed with high metal coverage. This design facilitates the redirection of blood flow away from the aneurysm, causing aneurysm occlusion due to flow stagnation and thrombosis. Additionally, FDs serve as a structural scaffold, contributing to the remodeling of vessel anatomy and the proliferation of endothelial cells through the device struts.[1]
In 2011, the FDA approved using flow diversion (FD) for managing large intracranial aneurysms in the distal internal carotid artery (ICA). The positive response observed for aneurysm occlusion in the ICA has motivated clinicians to expand the use of flow diversion to treat aneurysms in the posterior circulation and bifurcation aneurysms, which are subtypes of aneurysms with high rates of incomplete endovascular occlusion and recurrences after treatment.[2]
As with stents in the coronary and peripheral circulation, this technology requires dual antiplatelet therapy (DAPT) for 3 to 6 months to prevent clot formation.

The use of FD at bifurcation sites requires excluding one of the branches, raising concern of a stroke in the territory of the vessel that is covered and excluded from the parent artery. Early observations revealed changes in collateral circulation and vessel size, demonstrating that the circle of Willis had the potential for plasticity in adults. To study this phenomenon in more detail, our research at the Brain Aneurysm Institute performed a retrospective analysis of digital subtraction angiographies (DSA) of aneurysms treated with flow diversion from 2013 to 2023 at Beth Israel Deaconess Medical Center.

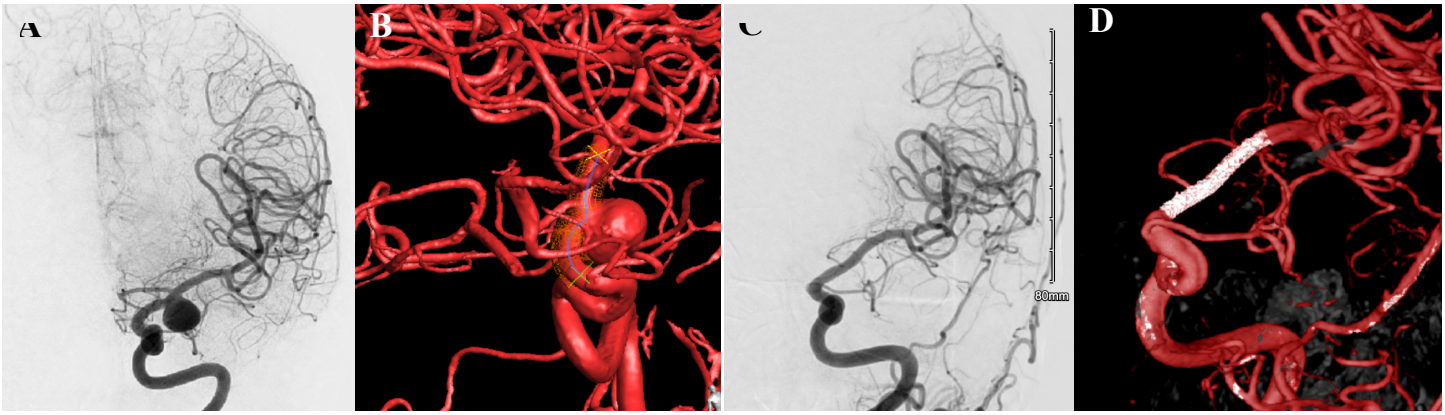

Figure 1 A 67-year-old female with a known terminal ICA aneurysm measuring 12.8x11.5 mm (A) was successfully embolized with a 3.5 x 16 mm pipeline embolization device covering the origin of the left ACA (B). One year after treatment there was lack of flow in the covered vessel evidenced in both angiography (C) and 3D reconstruction images (D).

In the first analysis, we evaluated aneurysms treated in the distal internal carotid artery with and without covering one of the terminal branches of the internal carotid artery (ICA), the anterior cerebral artery (ACA). We included 95 patients with 113 aneurysms treated in 102 procedures, and 58 of those were treated covering the ACA. Thromboembolic events occurred in six patients, all of which were in-stent thrombosis in the ICA; five of which occurred secondary to non-compliance to DAPT, and one which occurred in a patient who was a smoker and with multiple comorbidities. More interestingly, however, we did not see any ischemia or stroke in the distal territory supplied by the covered vessel. In fact, a very high patency was seen in the covered vessels; 43.3% of vessels were unchanged one year after the procedure, 33.3% revealed diminished flow, and 23.3% lacked flow (Figure 1). Those vessels exhibiting a lack of flow were smaller or hypoplastic compared to those with imaging evidence of diminished flow and had received collateral supply from the contralateral side.

In the second analysis, we evaluated aneurysms located in the posterior circulation, specifically in the basilar tip and proximal posterior cerebral artery (PCA), and assessed the rates of ischemic events in patients treated with and without covering the PCA origin with a flow diverter. Out of the total 28 patients included in this analysis, three developed an ischemic event (10.4%), none directly related to the vessel covered by the stent. Good functional outcome (mRS≤2) was reported in 89.3 % of patients in a median clinical follow-up of 5.5 months. Eight of the 15 vessels with angiographic follow-up lacked flow in the imaging follow-up (Figure 2). Still, they were receiving collateral flow through the ipsilateral posterior communicating artery.

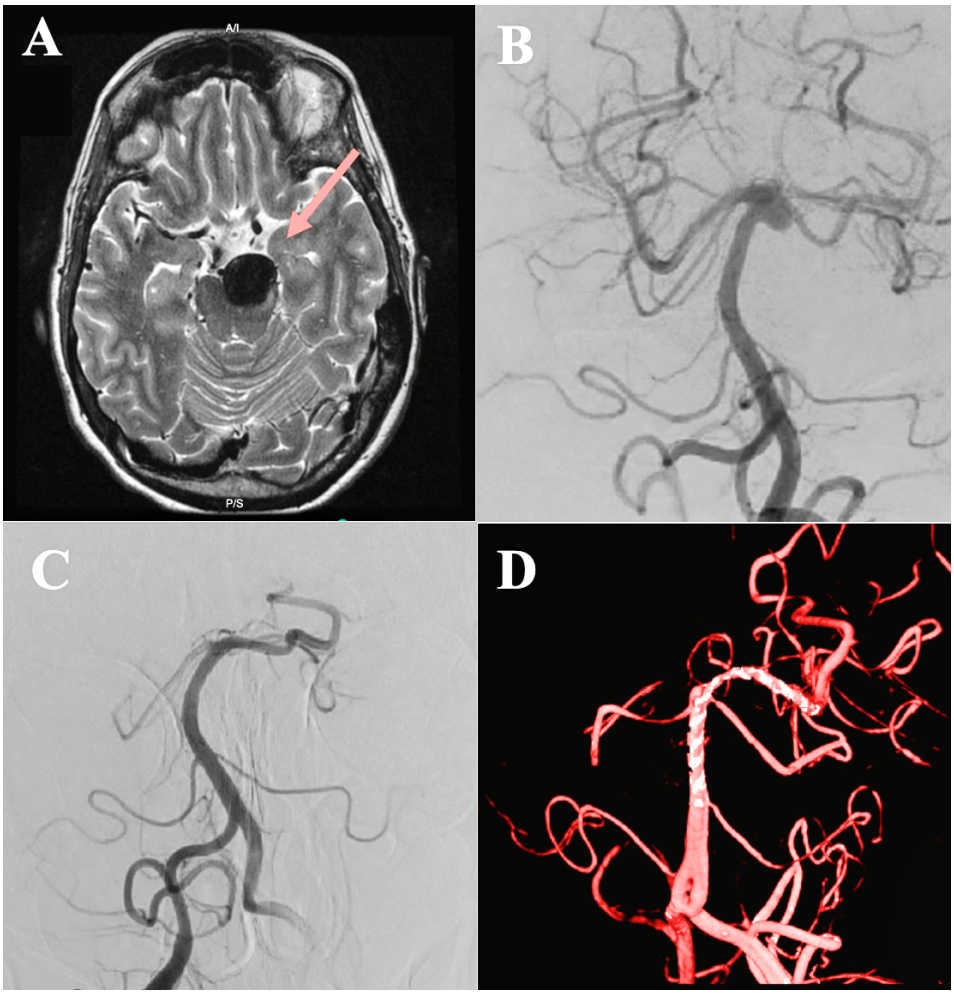

Figure 2 MRI showing a giant, partially thrombosed aneurysm arising at the origin of the left superior cerebellar artery (A). DSA revealed a basilar / left SCA aneurysm measuring 3.5 mm in the greatest diameter (B). The patient was treated with a pipeline embolization device extending from the left PCA into the basilar trunk and covering the origin of the right PCA. One year after the procedure there was angiographic evidence of nonfilling of the covered right PCA and obliteration of the aneurysm (C), also shown in the 3D reconstruction images (D).

These findings challenge the traditional thought of potential poor outcomes due to vessel occlusion after FD coverage. It is now understood that thromboembolic events can result from multiple factors, including device thrombogenicity, aneurysm complexities, technical issues, fluoroscopy time, increasing age (due to increased vessel tortuosity), and patient comorbidities.[3]

Some side branches, such as the ophthalmic artery, anterior choroidal artery, lenticulostriate arteries, terminal branches of the MCA, the posterior inferior cerebellar artery, and the perforator branches of the basilar trunk lack alternative collateral supply. Occlusion of these branches could potentially result in devastating neurological outcomes. However, studies reproducing hemodynamic conditions after FD have revealed minimal changes in flow and pressure on side branches after stent deployment, not reaching the ischemic threshold.[4] The determinant factor of side branch occlusion appears to be the collateral supply of the vessel as it is covered, with higher rates of occlusion reported for those vessels covered and with high distal collateral flow; hence, clinically silent occlusions without impact in patients.

Plasticity of Circle of Willis

The Circle of Willis is a well-described collateral system that provides blood supply to vessels gradually occluded over time or those with anatomical variations, such as a hypoplastic posterior communicating artery—a phenomenon reported in up to 80% of the general population.[5] This factor explains why up to 45% of the population can harbor asymptomatic intracranial atherosclerosis even with critical stenosis.

In our analysis, we identified two patterns of adaptability of the Circle of Willis after treatment with FD. In the first analysis of distal ICA aneurysms treated by covering the ACA origin, we observed that the contralateral A1 and ICA increased in size by 0.45 mm and 0.55 mm one year after treatment, respectively

(Figure 3). Additionally, in patients with basilar tip and proximal posterior cerebral artery (PCA) aneurysms treated by covering the origin of one of the PCAs, there was an increase in the size of the ipsilateral posterior communicating artery (PComA) by 0.44 mm (Figure 4). These patterns illustrate the dynamic changes that occur after treating bifurcation aneurysms with FD, showing an increase in size in the collateral vessels and augmenting the covered vessels' flow. However, high patency of vessels was seen for covered vessels without collateral supply due to tissue demand and well-established gradient pressures.

Future Areas of Research

The illustrated phenomenon of plasticity, which highlights the adaptations that occur in the circle of Willis to fulfill the tissue demands in the distal vasculature of the covered vessel, is not well understood. It has been proposed that both pressure and flow differences can drive these changes, but this area requires further research to understand these compensatory mechanisms fully. However, it is true that there is adaptability and that FD has expanded the use to more IAs, allowing neuro-interventionalists to provide treatment of complex, large, and irregular aneurysms not amenable to treatment using previous endovascular and open surgical techniques.

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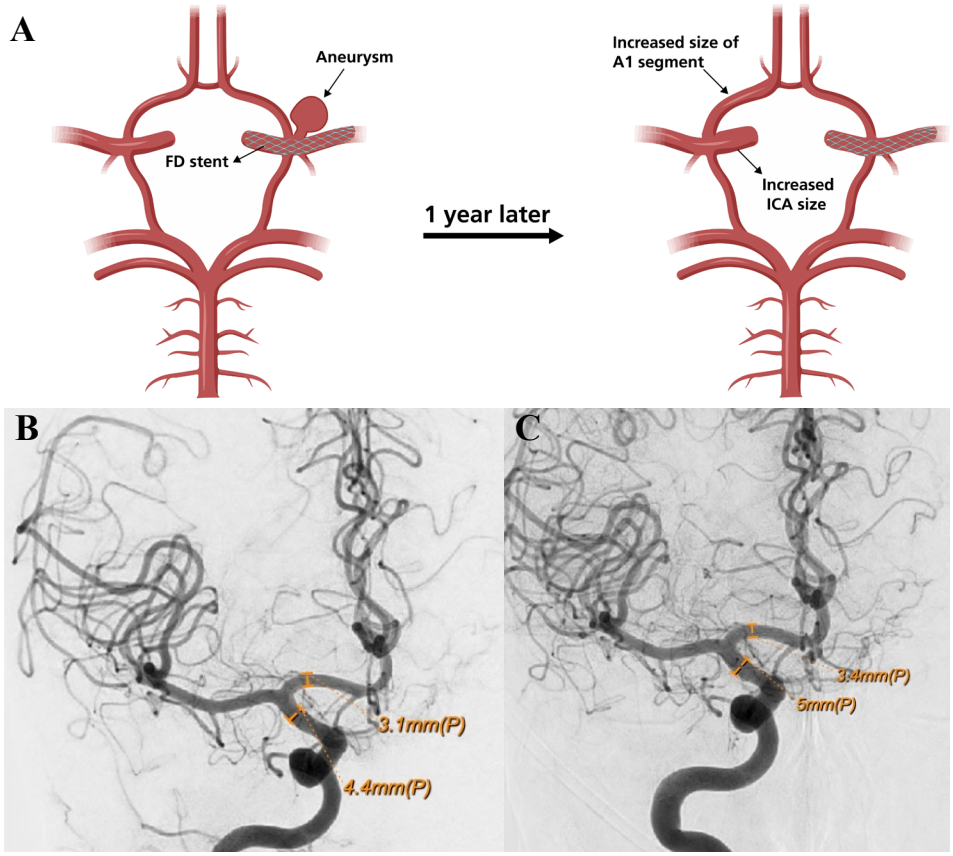

Figure 3 The diagram illustrates the pattern of change that occurs after the treatment with flow diversion covering the origin of the ACA (A). Right carotid angiogram of the patient in figure 1 (B) showing increase in size in both the right ICA and ACA 12 months after (C) covering the left ACA origin with a flow diverter.

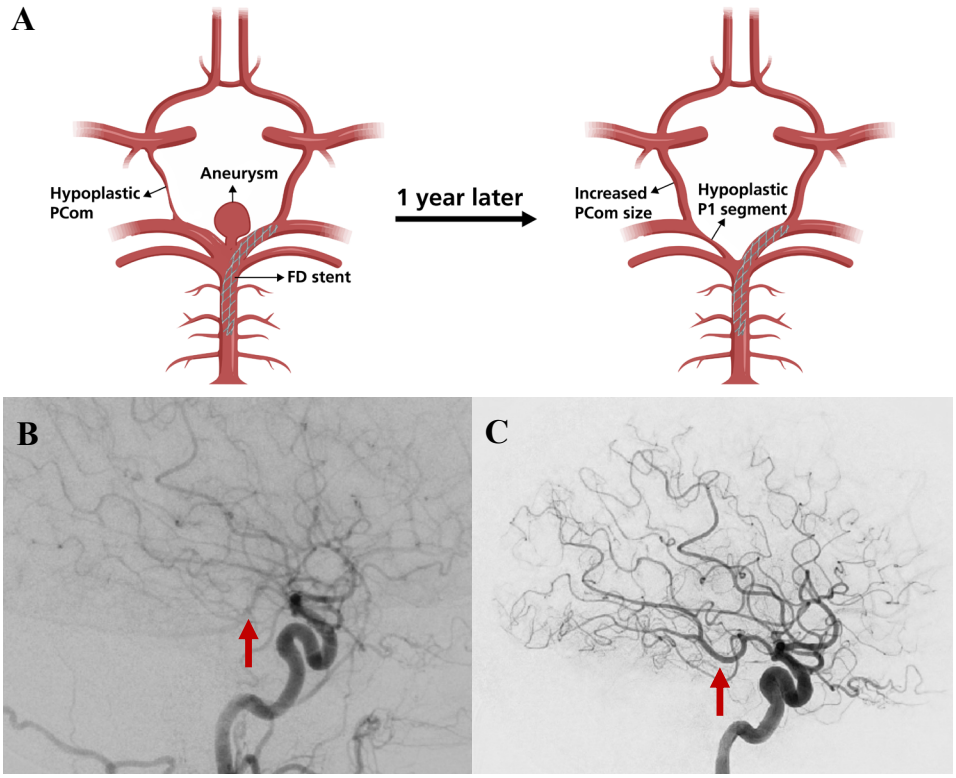

Figure 4 The diagram illustrates the pattern of change in the PCom after treatment with flow diversion, covering the origin of one PCA. Lateral view of the right internal carotid artery of -the patient shown in figure 2 whose aneurysm was treated excluding the right PCA. Note the change in size of the PComA before (arrow) (C) and one year after (D) treatment (arrow).

Trigeminal Neuralgia

Alejandro Enriquez-Marulanda M.D., Daniel Sconzo, B.S., Michael Young, DO., Philipp Taussky, MD., Christopher S. Ogilvy, MD

Trigeminal Neuralgia (TN) is a neurologic condition in which there is severe, unilateral, stimulus-evoked, stabbing facial pain[1,2]. The condition has been attributed to demyelination of the trigeminal nerve at the root entry zone adjacent to the brainstem, mainly due to vascular compression of the nerve by the superior cerebellar artery[3]. Medical management with oral medications such as carbamazepine is considered first-line therapy; however, this medication and other oral medications can have significant side effects. In cases where the pain is refractory to medical management or patients experience side effects of the medication, surgical intervention is an option as the symptoms may be debilitating and affect the quality of life of the patient[3,4]. A large portion of patients with trigeminal neuralgia don't get referred to neurosurgery early enough despite multiple non-operative treatment failures, and it may take 5-10 years after symptom onset. About 70% of post-operative trigeminal neuralgia patients wish they had their surgery done earlier in their disease course. Currently, there are several surgical treatment options, which include invasive (microvascular decompression) and minimally invasive approaches (percutaneous approaches and radiosurgery):

Microvascular decompression (MVD): Microvascular decompression (MVD) is a non-ablative open surgery performed under general anesthesia, in which a retrosigmoid craniotomy is performed to get access to the dorsal root entry zone of the trigeminal nerve. This surgery aims to relieve the compression exerted by a vascular loop, which is commonly produced by the superior cerebellar artery, but other vessels may be involved. Once the area of contact between the vessel and the nerve is identified, gentle dissection is performed to separate and relieve the compression of the offending vessel upon the trigeminal nerve. A small piece of Teflon is then placed between the vessel and the nerve to serve as a cushion and let the nerve heal while the pulsating artery is separated (Figure 1). After this, the surgical site is closed, and the patient is admitted to the hospital for 2-3 days of observation.

Facial pain usually resolves immediately post-procedure, and only surgical incision pain is present. At 4-5 years post-procedure, 61 – 80% of patients have pain relief.[5] This procedure is known to have the lowest failure rate at 4-30%[6] and is a good option for young, healthy patients who have confirmed neurovascular compression on MRI imaging preoperatively. This treatment efficacy, however, decreases when the compression is not present or with subsequent retreatments[6–8].

MVD is the most invasive surgical alternative, and the complication rate is higher compared to other treatment modalities at 8%.[9, 10] Some complications include cerebrospinal fluid leaks, brainstem infarctions, hematomas, meningitis, and sensory loss in part or all trigeminal nerve distribution. Despite this, the average mortality rate is very low (0.3%)[5], and the majority of complications are transient without permanent morbidity or sequelae.

Percutaneous balloon ganglyolysis (PBG): Percutaneous balloon ganglyolysis is an ablative minimally invasive intervention for the treatment of TN that involves the insertion of a 14 gauge Tuohy needle through the foramen ovale to reach the Meckel' 's cave and the trigeminal nerve ganglion (Gasserian ganglion) (Figure 1).[3,7,11–15] To reach the foramen, the needle is placed through the cheek at a point 2.5 cm lateral to the corner of the mouth and 1 cm inferior to the area below the mouth (Hartel technique). During this procedure, the patient is under general anesthesia, so no pain will be felt. Fluoroscopic guidance is used to insert the needle precisely in the correct trajectory. Once inside the gasserian ganglion, a No. 4 Fogarty balloon is inflated, which causes damage to the thick myelinated fibers, which are thought to trigger pain, while leaving the thin unmyelinated fibers unharmed[1,6,11,16]. It takes about an hour to complete the procedure, and this treatment modality has minimal associated risks; commonly, the patient is discharged the same day[12]. The facial pain is relieved in most cases immediately post-procedure, and the pain relief rate at 4 - 10 years of follow-up is about 68% (range 55 – 80%).[5]

Glycerol Rhizotomy: Similar to PBG, a needle is inserted into the Gasserian ganglion using the Hartel technique. Once the needle is in position, anhydrous glycerol (99.5%) is injected to cause an ablative chemical injury to the ganglion. Glycerol is injected slowly and commonly requires less than 0.5 cc total volume (the average volume of trigeminal cistern is 0.25-0.30 cc). The patient needs to be in the same position for about 2 hours to let the alcohol come in contact with the nerve during that time. Glycerol rhizotomy is 90% successful in relieving pain after initial intervention, but it has a pain relief duration of around 4 - 8 years in 28% (range 19 – 58%) of individuals.[5] Additionally, the median time to recurrence of symptoms requiring some degree of medical management is only 2 years[17–19]. Glycerol rhizotomy's

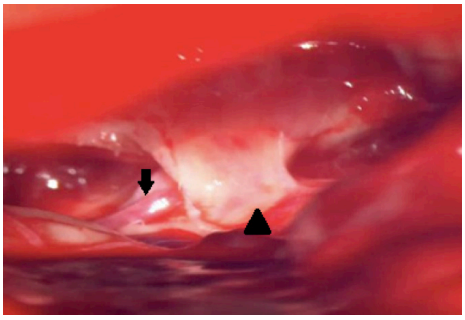

Figure 1A. Surgical microscope view of a retrosigmoid craniotomy visualizing the trigeminal nerve (arrowhead) and a vascular loop from the superior cerebellar artery (arrow), generating pressure into the nerve.

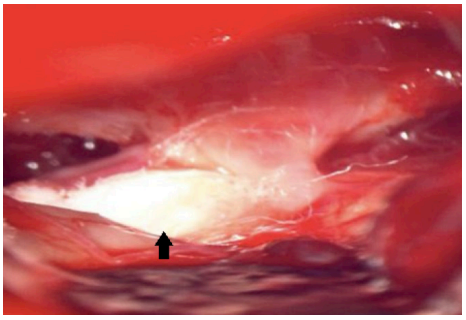

Figure 1B. Surgical microscope view of a retrosigmoid craniotomy. Dissection is performed to separate the vessel from the nerve with the placement of Teflon pledget in between the artery and the nerve.

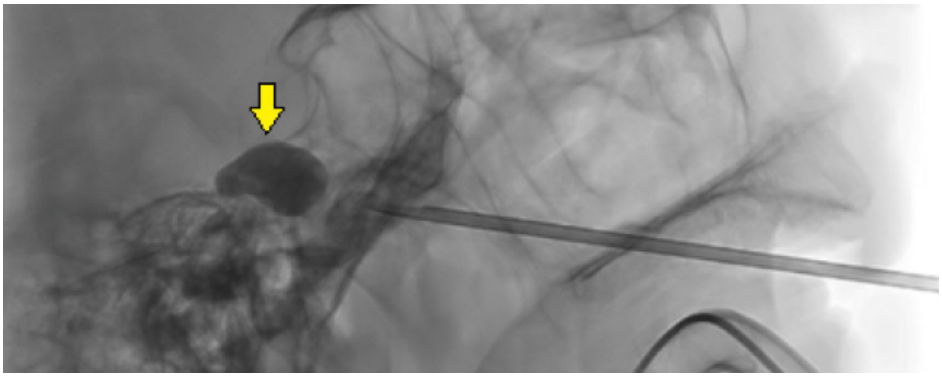

Figure 2. Fluoroscopic images of a female patient with right sided typical TN pain who underwent PBG treatment. Lateral fluoroscopic view showing the inflated balloon (yellow arrow) at the Meckel's cave with pear-like shape. The balloon was kept inflated for 3 intervals of 30 seconds.

effectiveness does not diminish with retreatments, but its increased risk of dysesthesia, corneal hypesthesia, and technical failures makes PBG a more appealing option[20,21].

Radiofrequency rhizotomy: Again, similar to the other percutaneous interventions (PBG and glycerol rhizotomy), an electrode is inserted into the Gasserian ganglion using the Hartel technique. Once the electrode location is confirmed with fluoroscopic images, the lead is heated up to 75 – 80 degrees Celsius for 90 seconds. The patient is awakened a few minutes after completing the lesion to determine if enough pain control and numbness are achieved. The radiofrequency ablation is repeated if needed until the desired numbness is attained.

Radiofrequency thermocoagulation causes non-selective thermal destruction of fibers, regardless of size. This affects pain fibers and potentially can damage fibers responsible for corneal sensation, which can lead to corneal deafferentation and resultant keratitis. To avoid this undesired effect, the probe is directed more inferiorly to prevent damage to the first division of the trigeminal nerve in charge of corneal sensation. Notably, trigeminal sensory deficits are usually transient with PBG or glycerol injections and more severe and lasting after radiofrequency thermocoagulation.[5]

Radiosurgery: This procedure does not need an incision and uses a Cyberknife or Gamma Knife with a stereotactic frame. All patients undergo a stereotactic brain MRI to identify the trigeminal nerve. After this, stereotactic coordinates are calculated for a single 4 mm isocenter placed 2-4 mm anterior to the junction of the trigeminal nerve and the pons.[22] The range of maximum dose used is between 70 to 90 Gy. Once the procedure is completed, the patient may be discharged within 24 hours. The facial pain-relieving effect of radiosurgery takes up to 6-8 weeks to develop, in contrast to PBG, which happens immediately post-procedure.[5] The efficacy of SRS is impacted by the presence of multiple sclerosis, the integral dose, and the mean dose of radiation given[23]. Approximately 24-71% of patients undergo continued pain relief 1-2 years after the procedure[5], but there has been a reported 60% failure rate at 10-year follow-ups for stereotactic radiosurgery[20,23,24]. Facial numbness complicates 16% of cases, and no cases of anesthesia dolorosa, (painful numbness), have been reported.[5]

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NEWS

ERRATUM

The article "Small Aneurysms and Rupture Risk: The Conundrum" contained an error in the SMALLSS scoring table. Patients over 65 years of age were incorrectly given a point meant for those under 65 since they carry increased lifetime rupture risk in the case that they harbor an aneurysm, with more years to live and thus higher chance for aneurysm rupture. The case at the end of the article correctly applied the scoring according to our scale. The corrected SMALLSS scale table displayed here scores a point to patients under 65, indicating higher rupture risk for small aneurysms in patients under 65 years of age.

We apologize for any confusion caused by the initial table in the fall publication 2023, and appreciate the feedback regarding this error.

	SMALLSS	Risk score	No. of cases and percentage
S	Size		
	Small aneurysms < 3.9mm	0	220 (28.5%)
	Small aneurysms 4 - 7 mm	1	551 (71.5%)
M	Multiple aneurysms		
	No	0	407 (52.8%)
	Yes	1	364 (47.2%)
A	Anatomic location		
	Anterior	0	668 (86.6%)
	Posterior	1	103 (13.4%)
L	Lineage (family history of aneurysm)		
	No	0	613 (79.5%)
	Yes	1	158 (20.5%)
L	Lifetime risk (age)		
	<65	1	418 (54.2%)
	>=65	0	353 (45.8%)
S	Smoking history		
	Never smoker	0	326 (42.3%)
	Former or current smoker	1	445 (57.7%)
S	Shape		
	Smooth	0	554 (70.6%)
	Irregular	1	227 (29.4%)

SAVE THE DATE

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