Introduction/Purpose Aneurysmal subarachnoid hemorrhage (SAH) is a devastating condition often complicated by cerebral vasospasm in the days following the initial event. Non-invasive imaging modalities, such as CT angiography and transcranial Doppler, are commonly employed to detect cerebral vasospasm. However, these screening modalities have inherent limitations and may fail to identify hemodynamic compromise in some patients. Quantitative MRA (qMRA) provides direct measurements of cerebral blood flow and may permit a more clinically relevant assessment of ischemia secondary to cerebral vasospasm. We conducted this preliminary study to evaluate the utility of qMRA in the assessment of cerebral vasospasm after SAH.

Materials and Methods We performed a retrospective analysis of a prospectively maintained database of all patients admitted with subarachnoid hemorrhage who underwent a qMRA between post-bleed day 0 and post-bleed day 21. Volumetric flow rates of the A2, M1, and P2 arteries were assessed on qMRA and compared with vessel diameters on catheter-based angiography performed within 24 hours. The sensitivity, specificity, positive predictive value, and negative predictive value of qMRA for detecting cerebral vasospasm was determined by receiver operator characteristic (ROC) curves. Spearman correlation coefficients were calculated for qMRA flow vs. angiographic vessel diameter. Angiographic vasospasm (VS) was defined as a reduction of the diameter of the vessel by greater than 25% between the baseline cerebral angiogram (obtained at the time of the index procedure) and the follow-up cerebral angiogram.

Results Ten patients with 60 vessels were evaluated with qMRA and catheter-based angiography. The median qMRA flow of all vessels found to have angiographic VS (53 mL/min, IQR 34 mL/min) was significantly lower than the median qMRA flow of vessels without angiographic VS (73 mL/min, IQR 52 mL/min) ($p = 0.003$). Angiographic VS reduced qMRA flows by 23 ± 5 mL/min in the ACA ($p = 0.018$), 95 ± 12 mL/min in the MCA ($p = 0.042$), and 16 ± 4 mL/min in the PCA ($p = 0.153$) between the baseline qMRA and spasm period qMRA. Two conditions were modeled using ROC curves based on cutoff points of angiographic VS: greater than 25% and greater than 50%. The overall performance of the two models (AUC) was 0.8325 and 0.8267 for angiographic VS >25% and angiographic VS >50%, respectively. The sensitivity, specificity, positive predictive value, and negative predictive value of qMRA for the discrimination of cerebral vasospasm was 84%, 72%, 84%, and 72%, respectively, for angiographic VS >25% and 91%, 60%, 87%, and 69%, respectively, for angiographic VS >50%. The result of the Spearman correlation indicated a significant association between qMRA flows and vessel diameters ($R = 0.71$, $p < 0.001$). This correlation was further increased when individualized baseline qMRA flows were included ($R = 0.83$, $p < 0.001$).

Conclusion Reduction in qMRA flow is a reliable indicator of angiographic vessel narrowing after SAH and may be useful as a non-invasive imaging modality for the detection of cerebral vasospasm.

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E-017 TOPICAL NITROPASTE IN TRANSRADIAL CEREBRAL ANGIOGRAPHY: EFFECTS ON RADIAL ARTERY CALIBER AND RADIOGRAPHIC VASOSPASM

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Introduction Transradial access for cerebral angiography is gaining popularity and has been associated with fewer serious access site complications and shorter recovery time than transfemoral access. However, the smaller size of the radial artery as compared to the femoral artery presents its own unique challenges. Topical nitropaste applied to the wrist and forearm prior to radial artery access has been shown to increase the radial artery caliber in cardiac procedures. We have incorporated this into our cerebral angiography practice and therefore sought to evaluate its use on radial artery caliber and post-procedure vasospasm.

Materials and Methods We retrospectively reviewed cases of attempted transradial cerebral angiography from May 2020 to March 2022. In each case, topical nitropaste was applied to the right wrist and forearm prior to prepping and draping of the patient. Upon arterial access, intraarterial nitroglycerin (200mcg), verapamil (5mg), and heparin (weight-based dosing) are administered. During a five-month period, we measured radial artery cross-sectional diameters using ultrasound both before nitropaste application as well as afterwards at the time of arterial access. Pre- and post-nitropaste measurements were compared using Welch's t-test. We also routinely perform radial artery angiography after sheath placement and again prior to sheath removal. The presence and severity of radial artery vasospasm was characterized as follows: none, mild (<50% reduction in diameter), moderate (50–70%), and severe (>70%).

Results One hundred seventy attempted transradial diagnostic angiograms were identified. Pre- and post-nitropaste ultrasonography was performed in 47 cases. The mean vertical and horizontal cross-sectional diameters of the radial artery were 0.19cm and 0.23cm, respectively. These increased to 0.22cm and 0.26cm, respectively, after the topical application of nitropaste ($p<0.001$ and $p<0.01$, respectively). The mean cross-sectional area of the radial artery increased from $0.14cm^2$ before nitropaste application to $0.18cm^2$ after nitropaste ($p<0.01$). Overall, 10 cases (5.9%) were converted to transfemoral - eight (4.7%) because the microwire could not be advanced in the radial artery and two (1.9%) because the diagnostic catheter could not be sufficiently advanced due to presumed vasospasm. Final radial artery control angiograms were available for 143 cases (84.1%). Among these cases, 53.1% showed no radiographic vasospasm, 33.6% showed mild spasm, 13.3% showed moderate spasm. There were no cases of severe spasm.

Conclusion The application of topical nitropaste to the wrist and forearm prior to radial artery access significantly increases

the cross-sectional diameter of the radial artery. Its use in conjunction with intraarterial nitroglycerin, verapamil, and heparin upon arterial access is associated with minimal instances of clinically significant vasospasm.

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E-018 EVOLUTION OF TRANSVENOUS EMBOLIZATION IN VEIN OF GALEN MALFORMATION: A REVIEW OF LITERATURE

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Introduction Vein of Galen Malformations (VOGM) comprise approximately 1% of cerebral vascular malformations overall, but a larger percentage of all pediatric VMs. Treatment/total obliteration of the malformation is challenging, but has evolved and improved greatly since the inception of endovascular treatment for VOGM. In our practice, we are able to obtain total obliteration in close to 80% of all cases with transarterial embolization (TAE). The remaining 20% of our cases typically have small arterial contributors that are uncatheterizable. Transvenous embolization (TVE) then becomes an attractive option and several transvenous approaches have been described.

Methods We performed a literature review by parameterizing a search on PubMed with the terms 'Transvenous OR Transtorcular,' 'Vein of Galen Malformation,' and 'Treatment.' The 21 articles chosen for detailed review described the various TVE approaches for VOGM.

Results An unfavorable outcome refers to an incomplete obliteration of the VOGM or long-term sequelae of VOGM treatment. Across the 21 articles reviewed, we identified a total of 107 patients (Figure 1). The ten articles which described a TT approach to treating VOGM were published between 1986 and 2001. The remainder of the articles assessed in the literature review advocated for transfemoral, transjugular, or combination TAE/TVE treatment of VOGMs. The first of these transfemoral approach articles was published in 1989 and the most recent article describing transfemoral TVE was published in 2022. Historically, the TT approach obtained variable levels of success with reports of successful resolution of cardiac failure via TT embolization, but much suggests an association with poor clinical outcomes. Transfemoral TVE therapy has grown and waned in popularity between 1989 and 2021. Endovascular coils were the most commonly deployed embolic agent in TVE cases. Based on our previous experience, we presently favor to begin treatment with staged transarterial embolization (TAE) to reduce flow to the lesion as much as possible and, importantly, to shrink the draining vein. This then allows for the performance of TVE with coils and n-BCA embolization. We translated this technique from Chapot et al.'s use of the procedure to treat an AVM by creating an 'anti-reflux plug' by trapping a detachable-tip microcatheter with coils and glue, followed by embolization with EVOH.

Conclusion In 1986, Mickle first described TVE of VOGM by placement of Gianturco stainless steel coils via surgical exposure over the torcula and direct trans-torcular access to the aneurysmal pouch. Transfemoral TVE with coils was introduced by Van Halbach in 1989 as a less invasive alternative to the TT approach. More recently, we have introduced a TVE approach from a transjugular or transfemoral embolization via the retrograde 'pressure cooker technique' (PCT). PCT uses a combination of coils, liquid embolic agents, and detachable tip microcatheters to retrogradely close the fistulas from the vein.

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E-019 TCAR

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Introduction Transcarotid artery revascularization (TCAR) has emerged as a safe alternative to carotid endarterectomy (CEA) and peripheral carotid stenting for treating patients with carotid artery stenosis. The safety and efficacy of TCAR in patients with carotid stenosis has been established by several studies, beginning with the multicenter ROADSTER trial. However, only 36 of the 141 patients in the ROADSTER study population (26%) were symptomatic, and limited data exist concerning the utility of TCAR for patients with symptomatic carotid stenosis. The objective of this study is to validate the safety and efficacy of TCAR in a single-center cohort of patients with symptomatic carotid artery stenosis.

Methods We identified all patients with symptomatic carotid stenosis who underwent TCAR at Rhode Island Hospital between 11/01/2020 and 11/30/2021. Relevant demographic, comorbidity, and perioperative data were extracted through retrospective chart review. We also evaluated patients using pertinent physiologic and anatomical high-risk criteria as described in the ROADSTER trial. Risk factors were aggregated to form a composite risk total for every patient. The primary outcome of this study was the 30-day adverse outcome rate of stroke, MI, and/or death. Periprocedural stroke was identified by clinical symptoms and radiographic findings. Secondary endpoints included acute device and procedural success, 30-day mortality, 30-day stroke rate, and postoperative complications. All means are reported with standard deviations, and analysis of variance (ANOVA) was used to assess the statistical significance of differences between patients in different composite risk tiers.

Results We analyzed the first 47 patients with >50% symptomatic carotid stenosis who underwent TCAR at our institution. The average age was 71.4 years old (standard deviation = 10.5), and the majority of patients were male (72.3%). High cervical carotid stenosis (34.0%), hostile neck anatomy (12.8%), and restenosis after prior CEA (8.5%) were the most frequent anatomic high-risk criteria which patients in the study fulfilled. The most common physiologic high-risk factors among the study cohort were age >75 years (46.8%), > 2 vessel coronary artery disease with history of angina (12.8%), severe COPD (8.5%), and class III/IV congestive heart failure (8.5%).

The all-cause adverse outcome rate of stroke, death, and MI was 4.3%. Among these patients, one died within 30 days of the procedure from symptomatic intracranial hemorrhage that developed 3 days post-procedure, and the other died of gastroenteritis unrelated to the procedure. No patients