Sylvian arteriovenous malformations (sAVMs) are rare and account for approximately 8%–11% of all intracranial arteriovenous malformations (AVMs). Several classification schemes have attempted to categorize these lesions based on their anatomical location and relationship to eloquent surrounding structures since sAVMs were first described by the Japanese neurosurgeon Kenichiro Sugita in 1987. Sugita divided sAVMs into 4 distinct subtypes based on the location of the nidus: 1) pure sAVM, located in the subarachnoid space around the middle cerebral artery (MCA) and respecting the pial planes separating the frontal and temporal lobes; 2) medial sAVM, located on the medial surface of the sylvian fissure or in the frontal lobe; 3) lateral sAVM, located on the lateral surface of the Sylvian fissure or in the temporal lobe; and 4) deep sAVM, located on the floor of the Sylvian fissure or buried in the insular cortex. In 1988, Yaşargil proposed a different system that classified these AVMs as anterior, middle, or posterior based on their relationship to the insular cortex.

Because of their proximity to eloquent structures such as the motor speech center, the insular cortex, and the internal capsule, microsurgical resection of sAVMs remains a challenging undertaking even for experienced cerebrovascular surgeons (Fig. 1). Despite the anatomical challenges posed by sAVMs, advances in stereotactic...
radiosurgery (SRS) and microsurgical techniques have resulted in excellent neurological outcomes. Yaşargil reported on 23 patients who suffered no neurological complications following microsurgical extirpation;29 Sugita et al. reported on 16 patients with no morbidity and only 1 death after microsurgical obliteration of their sAVMs;25 and on 2 separate occasions Lawton and colleagues reported excellent neurological outcomes after microsurgical resection of medial temporal as well as deep-seated AVMs.11,21

Methods

A total of 600 patients with AVMs were treated with microsurgical resection by the senior author (G.M.M.) at the Henry Ford Hospital between July 2000 and April 2012. Of these, 15 were identified as sAVMs. These AVMs were classified per Sugita’s classification into medial, lateral, pure, and deep after reviewing imaging (CT, MRI, and angiography) and operative notes. Clinical data such as age, sex, symptoms, Spetzler-Martin grade,23 pre- and postoperative embolization, surgery, SRS, and functional status were gathered to assign a modified Rankin Scale (mRS) score to each patient. Follow-up data were also gathered; duration of follow-up along with mRS score immediately postoperatively and at the time of last follow-up visit were also recorded.

Study Selection

PubMed was queried using the terms “arteriovenous malformation,” “AVM,” “perisylvian,” “sylvian,” “insular,” “frontal,” “temporal,” and “mesial temporal” through April 2014. All the studies that mentioned AVMs that could be categorized according to the Sugita classification of sAVMs were included in the study. No restrictions were imposed based on publication dates, types, or language. We also searched the references in the studies to see if they met our inclusion criteria and to ensure completeness of our search. Only AVMs that could be defined as perisylvian AVMs were added to the database from studies that discussed both perisylvian and nonperisylvian AVMs. Review articles and studies lacking information regarding patient demographics or AVM architecture were excluded. One study with the same patient population was represented in the literature multiple times as follow-up accrued.30,21

Data Extraction

From all studies, we extracted mean demographic, symptomatic, and AVM angioarchitectural data including number of patients; mean age; sex distribution; mode of presentation (hemorrhage or seizures); Spetzler-Martin grade; and proportion of patients who underwent preoperative embolization, resection, and postoperative SRS. In studies in which Spetzler-Martin grading was not mentioned explicitly, every attempt was made to derive those data from information that was presented in the study. Mean preoperative and postoperative mRS scores were obtained. Few studies mentioned Karnofsky Performance Scale (KPS) scores and were noted as such. Several studies made no mention of preoperative and postoperative mRS or KPS scores. Complications, both temporary and permanent, were collected as well.

Results

Results of the Literature Review

The initial literature search yielded 153 studies. One hundred three full-text articles were then assessed for eligibility. Seventeen reports comprising 348 patients met inclusion criteria. For these patients, the average age at presentation was 34.64 ± 6.7 years (± SD). There was a slight male predilection (194 males [55.7%] and 154 females). The common presenting symptoms included seizure in 123 patients (37.5%) and hemorrhage (subarachnoid hemorrhage or intracerebral hemorrhage) in 156 patients (47.5%). Twenty-two patients had Grade I sAVMs (7.6%), 81 patients had Grade II (28.2%), 145 patients had Grade III (50.5%), 35 had Grade IV (12.2%), and 4 patients had Grade V sAVMs (1.4%). Three hundred forty patients underwent resection (98%); 1 patient underwent transvenous embolization of the AVM,17 and 7 patients underwent SRS as the primary treatment modality of their AVM. Fifty-one patients (14.6%) underwent preoperative embolization, of which 2 were deemed unsuccessful. One patient underwent preoperative SRS prior to resection and 6 patients (1.7%) received postoperative SRS for residual AVMs. The mean preoperative and postoperative mRS scores obtained from studies that assigned mRS to their patients were 1.497 and 1.419, respectively. Two studies reported KPS scores;26,28 113 of 116 patients had KPS scores > 90. Sugita et al.25 reported subjective outcomes for their 16 patients; 10 had excellent outcomes, 3 patients had good outcomes, 2 had fair outcomes, and 1 fared poorly. Sixty-two patients (18%) suffered from immediate postoperative complications, of whom only 34 patients (9.7%) had a lasting deficit. A total of 6 patients (1.7%) died of related causes within 2 years of intervention (Table 1).

Results of the Institutional Review

Our institutional review revealed 15 patients treated for sAVMs. The mean patient age was 39.6 ± 12.94 years.

A. H. Pabaney et al.
<table>
<thead>
<tr>
<th>Authors &amp; Year</th>
<th>No. of Pts</th>
<th>Age (yrs)†</th>
<th>M/F</th>
<th>No. of Pts (%) Presenting w/ Seizure/Hemorrhage</th>
<th>Spetzler-Martin Grade</th>
<th>Treatment, No. of Pts (%)</th>
<th>Preop mRS</th>
<th>Postop mRS</th>
<th>Complications</th>
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<td>20</td>
<td>39</td>
<td>9/11</td>
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<td>1 (5)</td>
<td>NR</td>
</tr>
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<td>Cannestra et al., 2004</td>
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<td>34</td>
<td>12/8</td>
<td>NR</td>
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<tr>
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<td>24</td>
<td>4/6</td>
<td>3 (30)/6 (60)</td>
<td>I II III IV V</td>
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<td>8 (80)</td>
<td>1 (10)</td>
</tr>
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<td>1 1</td>
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<td>1 (6)/15 (94)</td>
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<tr>
<td>Wang et al., 2011</td>
<td>94</td>
<td>28</td>
<td>60/34</td>
<td>41 (44)/34 (36)</td>
<td>0 16 71 7 0</td>
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<tr>
<td>Yamada et al., 1998</td>
<td>22</td>
<td>9–59</td>
<td>9/13</td>
<td>7 (32)/13 (59)</td>
<td>– – – – –</td>
<td>22 (100)</td>
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<td>4–49</td>
<td>12/11</td>
<td>15 (65)/14 (60)</td>
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<td>32</td>
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<td>8 (100)</td>
<td>3 (38)</td>
<td>0</td>
<td>1.75 0.375</td>
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</table>

* Embo = embolization; NR = not reported; pt = patient; VF = visual field.
† Age at time of presentation. Means are given except for studies that provided a range.
‡ This patient received preoperative radiosurgery prior to resection of the AVM.
§ Sugita et al. reported 10 excellent outcomes, 3 good outcomes, and 2 fair outcomes.
¶ Wang et al. reported a KPS score > 90 in all patients.
** Yamada et al. reported a KPS score > 90 in 19 patients.
postoperatively, 5 patients (33.3%) had an mRS score of 0, and 5 (33.3%) had an mRS score of 1. Immediately postoperatively, 5 patients (33.3%) had an mRS score of 0, and 8 patients (33.3%) had an mRS score of 1. One patient (6.67%) declined from a preoperative mRS score of 1 to an immediate postoperative mRS score of 4 due to a perioperative ischemic stroke resulting in left hemiparesis and third and sixth cranial nerve palsies (Case 2). This patient had a Spetzler-Martin Grade IV sAVM, which had previously been embolized 3 times and was surgically treated due to worsening mass effect and hemorrhage. Four of 8 patients who underwent resection had a decline in their mRS in the immediate postoperative period; however, 2 of those 4 patients regained their preoperative mRS score. There were no complications after preoperative embolization. One patient (6.67%) with a preoperative mRS score of 0 was lost to follow-up (Case 10). On their last follow-up visit, 9 patients remained stable (60%) and 5 patients (33.3%) improved in their functional capacities compared with their immediate postoperative functional status. Overall, the late postoperative mRS score in 9 patients was unchanged from pretreatment (60%), 3 patients worsened, and 2 patients improved (20% and 13.3%, respectively) (Table 2).

Illustrative Cases

Case 3. A 33-year-old man presented to our emergency department with speech arrest and confusion. On examination, he was neurologically intact. Imaging revealed a 3 × 5–cm AVM nidus in the left temporal perisylvian region (Fig. 2A). Cerebral angiography showed a large AVM nidus supplied by the anterior choroidal artery and angular and posterior parietal and temporal branches of the MCA. Venous drainage was superficial to the superficial middle cerebral vein and veins of Trolard and Labbé (Fig. 2B). Preoperative embolization was performed over 4 different sessions using Onyx-18 and N-butylcyanoacrylate (NBCA). Significant reduction in the size of the AVM and shunting was achieved (Fig. 2C). The patient was taken to the operating room 2 days after the last embolization session. Intraoperatively, feeders from the MCA and anterior choroidal artery were identified and coagulated. Draining veins were preserved until all the feeders were divided. Aneurysm clips were placed on the draining veins and then the draining veins were divided. The AVM nidus was then removed. Postoperative angiography revealed complete AVM nidus resection with no evidence of arteriovenous shunting (Fig. 2D). Postoperatively, the patient developed mild right hemiparesis with a strength of 4/5 on Medical Research Council (MRC) scale. The patient continued to have stable right hemiparesis and seizures controlled with 1 medication at his last office visit 14 months after surgery.

Case 13. A 42-year-old woman presented with headaches to our emergency department; the neurological examination disclosed no abnormality. Brain MRI revealed abnormal flow voids suggesting an AVM in the right frontal perisylvian region measuring 2.9 × 2.7 cm (Fig. 3A). Cerebral angiography revealed a Spetzler-Martin Grade II AVM, fed by the branches of the MCA. The AVM drained into the 3 large cortical veins that drained into the superior sagittal and transverse sinuses (Fig. 3B). Surgery was offered but the patient declined resection; hence the patient was referred for SRS. The patient underwent SRS and received 16 Gy to the lesion. Postoperative angiography per-
<table>
<thead>
<tr>
<th>Case No.</th>
<th>Age (yrs), Sex</th>
<th>Presentation</th>
<th>Spetzler-Martin Grade</th>
<th>Sugita Classification</th>
<th>Arterial Feeders</th>
<th>Treatment</th>
<th>Preop mRS Score</th>
<th>Postop mRS Score</th>
<th>Follow-Up (mos)</th>
<th>AVM Obliteration</th>
<th>Complications</th>
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<tr>
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<td>headache, seizures</td>
<td>III</td>
<td>medial</td>
<td>ACA, MCA, PCA</td>
<td>embo w/ resection</td>
<td>0</td>
<td>0</td>
<td>0</td>
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<tr>
<td>2</td>
<td>49, F</td>
<td>headache, hemorrhage, neuro deficit</td>
<td>IV</td>
<td>lateral</td>
<td>MCA, ICA, ECA</td>
<td>embo w/ resection</td>
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<td>4</td>
<td>3</td>
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<td>embo w/ resection</td>
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<td>4</td>
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* CN = cranial nerve; ECA = external carotid artery; LLE = left lower extremity; NA = not applicable; neuro = neurological; RLE = right lower extremity.
formed 18 months later revealed a decrease in the size of the AVM but persistent shunting. Repeat cerebral angiography at 30 months revealed complete obliteration of the nidus with no evidence of arteriovenous shunting (Fig. 3C and D). The patient remained neurologically intact with no decline in functioning at her 36-month follow-up visit.

Discussion

Microsurgical resection of perisylvian AVMs has not gained much popularity historically due to the technical dangers associated with resection of these AVMs. Only in the last couple of decades have cerebrovascular experts dared to venture in this territory with excellent patient outcomes. In addition, technological advancements in our operating rooms—advent of a hybrid operating suite, availability of intraoperative angiography, intraoperative fluorescence technology, improved instrumentation, and advanced imaging technology—have tremendously assisted neurosurgeons in tackling these AVMs in a much safer and controlled fashion and have challenged the belief that such AVMs, especially the deep variant, are deemed “inoperable.”

While SRS has proven efficacious as a stand-alone treatment for AVMs (Fig. 4), the 1- to 3-year latency period following SRS until complete obliteration is achieved does not change the natural history of sAVMs and in fact may predispose patients to higher risk of hemorrhage due to presumed hemodynamic changes. In our case series, 4 of 8 patients treated with SRS had incomplete obliteration at their last imaging session, including 1 who was ultimately treated with surgery to achieve complete obliteration of her AVM. Additionally, due to the unique, oblong angioarchitecture of sAVMs, overlapping isodose centers are required with SRS, and this may predispose patients to heightened risk of radiation necrosis or injury to subcortical structures. We have extensively reviewed the literature regarding management of perisylvian AVMs and present the findings below. In addition, we also share our own experience with these AVMs that have been managed in a multidisciplinary fashion at our institution.

Preoperative and Postoperative Considerations

Diagnosis and management of sAVMs are critically dependent on the patient’s neurological examination. The presence of focal deficits is alarming and should be supplemented with ophthalmological examination and visual field testing, neurocognitive evaluation or memory testing, and speech evaluation with intracarotid sodium amobarbital procedure (WADA) or functional imaging techniques to establish hemispheric dominance and outline neighboring areas of eloquence (Fig. 5). Noncontrast CT and MRI are critical for discovering acute and chronic hemorrhage respectively as well as delineating the surrounding eloquent structures. It is of extreme importance that a solid understanding of the spatial anatomy of the AVM is achieved such that the choice of approach does not disrupt crucial pathways, adding to morbidity. Cerebral angiography remains the gold standard for understanding the
temporal and spatial relationships between the feeding arteries, particularly the MCA and draining veins emanating from the AVM nidus. Depending on the type of sAVM, the MCA can be placed medial (pure AVMs), superior (lateral AVMs), inferior (medial AVMs), or lateral (deep AVMs) to the nidus. The venous drainage pattern also varies between Sugita’s AVM subtypes. Pure sAVMs drain superficially into the middle cerebral veins; lateral AVMs tend to drain inferiorly into the sphenoparietal sinus and vein of Labbé but can also drain medially into the basal vein of Rosenthal. Medial AVMs tend to drain upward into the superior sagittal sinus and vein of Trolard. Deep AVMs typically drain deep into ventricular veins. Early venous drainage, the hallmark of AVMs, as well as high-risk characteristics such as proximal aneurysms or venous outflow obstruction, is best appreciated on cerebral angiography. In the case of large intracranial AVMs, one might consider preoperative embolization as a useful adjunct. The goal of embolization should be to eliminate the arterial feeders that are deep and not readily accessible surgically. This can make AVM resection easier. In addition, in cases of ruptured AVMs, an angiogram should be screened carefully and any flow-related aneurysms should be identified and ideally treated at the same setting. However, embolization has risks of embolic stroke and hemorrhage that have to be weighed carefully in the decision-making process. It is preferred that the ruptured AVMs are not acutely treated; instead, the patient should be brought back after 4–6 weeks for additional angiography once the intraparenchymal clot has resolved, and a treatment decision is formulated at that time. The advent and wide availability of the navigational tools could aid surgeons in narrowing their operative corridor. Postoperatively, patients should be monitored in the ICU setting and hemodynamics should be strictly maintained in a physiological range to avoid breakthrough hemorrhage. Postoperative verification of complete extirpation of AVM by catheter angiography is of paramount importance and should be considered a mandatory component of AVM treatment.

Surgical Approaches and Operative Nuances

The goal of AVM surgery is complete obliteration of the early venous drainage and the nidus to prevent neurological complications related to catastrophic hemorrhage. While perisylvian AVMs pose unique challenges, there are important surgical principles for minimizing complications that need considerable attention. First, judicious preoperative clearance and medical optimization could reduce intraoperative complications related to bleeding or myocardial infarctions. Second, maintenance of intraoperative normotension (120–140 mm Hg), end-tidal carbon dioxide levels, administration of hypertonic solutions or diuretics, and the need for possible blood transfusions should be discussed well in advance with the anesthesia provider.
team. Similarly, a discussion with the operating room staff could alert them to specific surgical equipment necessary to safely carry out the planned operation. Third, minimizing venous outflow obstruction in the neck during positioning and head elevation could reduce venous congestion and facilitate brain relaxation. Fourth, adequate intraoperative exposure that is wide enough to appreciate all cortical draining veins and provides direct visualization of the feeding vessels is important. Dissection of the arterial feeders should be carried out sharply, meticulously, and close to the AVM's gliotic margin to minimize injury to surrounding structures. The venous channels should only be disrupted after all arterial feeders to the nidus have been disconnected to prevent catastrophic intraoperative hemorrhage. In some instances, elimination of major arterial feeders will transform red, “arterialized veins” into blue veins that more closely resemble normal surrounding cortical veins. Every attempt should be made to completely resect the AVM nidus en bloc because incomplete resection does not alter the natural history and, in fact, may predispose patients to higher rates of hemorrhage possibly due to hemodynamic changes. While fluorescein angiography has shown promise as a useful intraoperative guide, intraoperative angiography is limited by low resolution and does not ensure elimination of very small vessels. Therefore, postoperative verification of complete extirpation of AVM by catheter angiography is of paramount importance and should be considered a mandatory component of AVM treatment unless dictated by special circumstances. Fifth, in patients with prior hemorrhage, the hematoma cavity should be used as the main corridor to minimize damage to normal brain tissue. And finally, an honest discussion with family members throughout the postoperative course will ease the patient’s transition from surgery to baseline status.

In addition to the aforementioned principles, certain tenets are specific to the resection of sAVMs. First, it is important to note, though, that many of the vessels encountered in the sylvian fissure are en passage vessels that continue to traverse to normal brain parenchyma. It is of paramount importance during sAVM surgery to ensure that the en passage vessels are not mistaken for feeding arteries and prematurely obliterated. Additionally, the enlarged sylvian veins could make differentiating between en passage vessels or feeding arteries from venous structures difficult. Williamson and colleagues have advocated utilizing temporary clips on veins to aid in differentiation because veins are collapsible and a blue discoloration that will develop distal to the clip can be easily appreciated by the surgeon. Second, Zimmerman et al. have advocated a wide splitting of the sylvian fissure to allow for adequate skeletonization of the MCA branches in addition to maximal maneuverability in the anteroposterior dimension of the sylvian fissure to ensure complete resection of large sAVMs. Various adjuncts such as preoperative embolization, maintaining intraoperative normotension, or even induced hypotension during resection could be used to minimize blood loss. For mesial temporal AVMs, Du and colleagues have advocated a tangential approach through an orbitozygomatic craniotomy to eliminate language deficits secondary to corticectomy, reduce retraction on the temporal lobe, and adequately expose and disconnect all feeding arteries. Experts have also advocated using microretractors to retract the AVM nidus and limit brain retraction while various surgical approaches have been previously described for surgical obliteration of sAVMs, their universal aim is to provide a direct route to the feeding arteries.

Outcomes Based on Literature Review and Institutional Experience

Intracranial hemorrhage from cerebral AVMs is a considerable source of neurological morbidity and mortality. A recent meta-analysis revealed an overall annual hemorrhage rate of 3.0% per year, with an initial annual rupture rate of 2.2% and rerupture rate of 4.5%. Prior hemorrhage, a deep AVM location, exclusively deep venous drainage, and associated aneurysms are significant risk factors for AVM hemorrhage. Multiple short- and long-term reports exist that address the morbidity from AVM-associated hemorrhages. Graf et al. described the outcomes of 191 patients between 1946 and 1980 and found a deficit in 81% of patients immediately after the hemorrhage. Perret and Nishioka found that 58% of patients with an AVM-related hemorrhage had neurological deficits. However, long-term follow-up is not available.

The pooled analysis of sAVMs presented here is the first of its kind. The cumulative temporary and permanent complication rates are 18% and 9.7%, respectively, which are significantly lower than the hemorrhage-associated morbidity. Also, application of multimodal therapy to the treatment of these complex lesions, that is, microsurgery, endovascular treatment, and SRS, is likely to yield even better outcomes with lower complication rates than the disciplines of neurosurgery and biotechnology continue to evolve.

At our institution, we aggressively treat these lesions and aim for complete obliteration, especially in the younger population. We tend to favor resection in patients harboring Spetzler-Martin Grade I and II AVMs located in noneloquent regions of the brain. For patients presenting with higher-grade AVMs or patients with comorbidities that preclude surgical intervention, we recommend multimodal therapy employing expertise from our interventional neuroradiology and SRS colleagues. Also, we take patient preference into consideration when formulating treatment strategies. This is clearly evident in our series as some of our patients underwent radiosurgery despite having Spetzler-Martin Grade I and II AVMs. Three of our 8 patients who underwent SRS refused resection (Cases 8, 14, and 15). One of these 3 patients eventually underwent resection for a residual AVM (Case 8). One patient was offered SRS instead of microsurgical extirpation due to medical comorbidities (Case 12). Four patients were deemed high risk for postoperative deficits due to AVM location in eloquent regions and hence were referred for radiosurgical treatment (Cases 9, 10, 11, and 13). Similarly, 4 of 8 surgically treated patients underwent preoperative embolization of their AVMs to facilitate resection.

Limitations

There are several limitations to our study. First, scant
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data exist for sAVMs, and the reports are not uniformly symmetrical. Data concerning the baseline functional status and functional outcome are missing, especially from older reports. Second, because we could not control the outcome assessment and had to rely on the accurate record keeping of other individuals, our study may suffer from sampling or information bias. We attempted to minimize this by individually reviewing patients’ medical records from the time of their first encounter at our institution and excluding patients who were subsequently diagnosed as having AVMs in nonperisylvian locations. By doing this, we were able to eliminate several patients whose description of their AVM characteristics did not fit the recorded diagnostic code. It is crucial to emphasize that the majority of patients in all reviewed studies had symptomatic AVMs, limiting the external validity of this study to patients with truly incidental AVMs. Given the aforementioned limitations, it may be difficult to generalize our findings to the general populace.

Conclusions

We present the first pooled analysis of the literature of perisylvian AVMs and emphasize the patient characteristics, classification schemes, management paradigms, and outcomes. We realize that by no means does the quality of this report parallel a randomized controlled trial, but it is an effort toward advancing our knowledge of these complex AVMs and a humble attempt to change the old belief that regards these lesions inoperable.

Disclosure

The authors report no conflict of interest concerning the materials or methods used in this study or the findings specified in this paper.

Author contributions to the study and manuscript preparation include the following. Conception and design: Pabaney, Reinard, Malik. Acquisition of data: Pabaney, Reinard, Massie, Naidu, Mohan, Marin. Analysis and interpretation of data: Pabaney, Reinard, Naidu, Mohan, Marin, Malik. Drafting the article: Pabaney, Reinard, Massie. Critically revising the article: Pabaney, Reinard, Mohan, Malik. Study supervision: Marin, Malik.

References

tion surgery. Neurosurgery 67 (3 Suppl Operative)ons237–ons276, 2010

Manuscript submitted May 15, 2014. Accepted July 8, 2014. Please include this information when citing this paper: DOI: 10.3171/2014.7.FOCUS14246.

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