Temporal Lobe Arteriovenous Malformations: Surgical Outcomes With a Focus on Visual Field Defects and Epilepsy

Pablo Lopez-Ojeda, MD*
Mohamed Labib, MD‡
Jorge Burneo, MD, MSPH*
Stephen P. Lownie, MD, PhD*

*Department of Clinical Neurological Sciences, London Health Sciences Centre, University of Western Ontario, London, Ontario, Canada; ‡Division of Neurosurgery, Department of Surgery, University of Ottawa, Ontario, Canada

Correspondence:
Stephen P. Lownie, MD, PhD,
London Health Sciences Centre,
University of Western Ontario,
London, 339 Windermere Rd,
PO Box 5339, London, Ontario,
Canada N6A 5A5.
E-mail: Steve.Lownie@lhsc.on.ca.

Received, December 9, 2012.
Accepted, July 23, 2013.
Published Online, August 5, 2013.

BACKGROUND: Temporal lobe arteriovenous malformations (AVMs) represent a subgroup of intracranial AVMs with particular characteristics and management issues.

OBJECTIVE: To characterize the surgical outcomes of temporal lobe AVMs with emphasis on visual field deficits (VFDs) and seizures.

METHODS: Between 1992 and 2008, 29 patients were operated on for temporal lobe AVMs. Patient data were retrospectively collected and analyzed.

RESULTS: Twelve of 29 patients (41.4%) presented with seizures and 4 (13.7%) presented with VFDs. Postoperatively, 6 patients (24%) showed new VFDs and 2 improved, with a rate of preservation of full visual fields of 84%. Larger AVMs ($>$ 3 cm) were significantly associated with postoperative VFD (P = .008). Epilepsy outcomes assessed by the Engel scale were as follows: 9 patients (75%) were in class I (seizure free), 1 patient (8.3%) was in class III, and 2 patients (16.6%) were in class IV (no change or worsening). Postoperative modified Rankin Scale outcomes were excellent (grade 0-1) in 18 patients, good (grade 2) in 7, and poor (grade 3-4) in 4. Older age at diagnosis correlated with a worse functional outcome (Spearman $\rho = 0.369$; P = .049). AVMs were totally removed in 27 of 29 patients (93.1%). Complete surgical excision was confirmed with angiography. Two patients needed reoperation for AVM remnant. Three patients had persistent hemiparesis (10.3% permanent morbidity). There was no mortality.

CONCLUSION: Seizure control is usually underappreciated in the surgical management of AVMs. However, in temporal lobe AVMs, good outcomes with low morbidity and good visual field preservation can be accomplished.

KEY WORDS: Arteriovenous malformation, AVM, Seizure, Surgery, Temporal lobe, Visual field deficit

Neurosurgery 73:854–862, 2013 DOI: 10.1227/NEU.0000000000000122 www.neurosurgery-online.com

Temporal lobe arteriovenous malformations (AVMs) are relatively uncommon, accounting for 12% to 16% of intracranial AVMs.[1-4] The literature on the surgical treatment of temporal lobe AVMs is scarce, with only case reports and small series.[3-7] In most instances, the focus has been on surgical management and techniques.[3,5,6] Although seizures and visual field deficits (VFDs) are usual clinical symptoms of temporal lobe AVMs, few attempts have been made to better characterize their relevance and management implications.[4,7] Only 2 articles specifically focus on VFDs as a measurement of surgical outcome,[3,7] and 1 other article addresses both VFD and seizure outcome.[4] Overall, these studies indicate that surgical resection of temporal AVMs can be performed successfully with a good rate of visual field preservation and good seizure control. This is important given the recent controversy concerning surgical indications in unruptured AVMs.[8-10] To better evaluate these features, we performed a retrospective review of all surgically treated temporal lobe AVMs at our center, paying particular attention to VFDs and seizures.

ABBREVIATIONS: AED, antiepileptic drug; **AVM,** arteriovenous malformation; **mRS,** modified Rankin Scale; **VFD,** visual field deficit

PATIENTS AND METHODS

The London Health Sciences Centre AVM database from 1992 to 2008 was reviewed. AVMs were classified according to the Spetzler-Martin classification[11] into grades I through III. Patient demographics, clinical presentation, neuroimaging findings, AVM characteristics, therapeutic management, outcome, and seizure-related history (including seizure onset, seizure type, and seizure control on medication) were collected. VFDs were identified by formal neuro-ophthalmological assessment, including Goldmann field testing. Medically refractory epilepsy was defined as persistence of seizures despite adequate trials of ≥ 2 antiepileptic drugs (AEDs).[12] Epilepsy duration (time from seizure onset to treatment) was classified as < 12, 12 to 24, and > 24 months for statistical analysis. Seizure outcome was evaluated with the Engel Seizure Outcome Scale[13] (class I, free of seizure or residual auras; class II, intermittent, infrequent seizures or relapse after a significant seizure-free period; class III, worthwhile improvement, $> 75\%$ reduction in seizure frequency; and class IV, $< 75\%$ reduction in seizure frequency). The modified Rankin Scale (mRS) was used to estimate both preoperative and postoperative clinical status and neurological disability. AVM location was classified as sylvian fissure, temporomesial, posterior temporal, anterior temporal, and intraventricular or periventricular. AVM size was classified for statistical analysis as < 3, 3 to 6, and > 6 cm.

Statistical Analysis

Data processing and analysis were performed with the SPSS statistical program version 15.0 for Windows (SPSS, Inc, Chicago, Illinois). A descriptive analysis was performed on all study variables using central tendency and dispersion measures for quantitative variables and relative and absolute frequencies for qualitative variables. The χ^2 test was used to compare proportions (applying the Yates correction when necessary) between clinical characteristics and VFDs and seizures. The Spearman correlation coefficient was used to assess the association between age at diagnosis and mRS score. Results were expressed as absolute numbers and percentages, means, standard deviations, odds ratios, and 95% confidence intervals. Statistical tests were considered to be significant at a 2-tailed value of $P < .05$. As an exploratory study, correction for multiple testing was not done.

RESULTS

Ninety-nine patients were identified. Ninety were operated on by the senior author (S.P.L.). Twenty-nine AVMs (in 19 women and 10 men) were located in the temporal lobe. Median postoperative follow-up was 12 months (mean, 28.4 months; range, 1 month-14 years). The mean age was 39.2 years (SD, 16.0 years; range, 7-72 years). Location was perisylvian in 5, mesial in 6, ventricular in 3, anterior neocortex in 3, and posterior neocortical area in 12. The initial presentation was hemorrhage in 15 cases. Of the nonhemorrhagic cases, 12 (41.4%) presented with seizures and 2 with headache. A statistical association was found between hemorrhagic presentation and AVM location (60% had bleeding with posterior location; $\chi^2 = 11.8$; $P = .02$). Nineteen patients (65.5%) had an mRS score of 1 at presentation, 4 (13.8%) had a score of 2, and 6 (20.6%) had a score of ≥ 3. Altogether, most of the patients (23 of 29, 79.3%) presented with a good or excellent preoperative score (≤ 2). With the use of Spetzler-Martin classification, 12 cases (41.4%) were categorized as grade I, 9 (31%) as grade II, and 8 (27.6%) as grade III. Fourteen patients (48.3%) had small AVMs (≤ 3 cm), 13 AVMs (44.8%) were medium (> 3-6 cm), and 2 (6.9%) were large (> 6 cm). Fourteen patients (48.3%) underwent surgical excision with no other procedures, 14 patients received presurgical embolization, and 1 patient (3.4%) received embolization and radiosurgery before the surgical resection (for a detailed overview, see Table 1). Complete excision (confirmed with cerebral angiography) was performed in 27 patients (93.1%). Two patients underwent a second surgical intervention several months after the initial surgery, and complete AVM resection was achieved. One patient demonstrated a small AVM remnant on follow-up angiography, and the other patient bled 15 months after the initial surgery, even though no AVM remnant was seen in follow-up arteriography. At the second surgery a small posterior remnant was confirmed and excised.

No deaths occurred, but VFDs occurred in 8 patients.

Clinical Status and Neurological Morbidity

Preoperatively, hemiparesis was found in 4 patients (13.7%). Three of them had massive hemorrhages, and although 1 patient presented with complex seizures as the initial symptom, a post-embolization hemorrhage caused hemiparesis. Postoperatively, 7 of 29 patients sustained neurological deficits other than an isolated field cut, usually dysphasia or hemiparesis. Most deficits, whether caused by the initial hemorrhage or the surgery, tended to improve significantly or resolved over a 3-month period. Language alterations occurred mainly with left-sided AVMs with hemorrhage and were already present before the surgery. Permanent morbidity (other than isolated VFD) was seen in 3 patients (10.3%). At the last follow-up, these 3 patients still had not fully recovered from their preoperative hemiparesis. Two also had a VFD. These results are comparable to other series with temporal lobe AVMs.[2,3,7] With regard to functional status, 15 of 29 patients (51.7%) had no change in their mRS score, 8 (27.6%) improved after surgery, and 6 (20.6%) had a decline. Only 1 patient had a major worsening, defined as ≥ 2 grades of worsening.

Older age was correlated with a worse final outcome (mRS; Spearman $\rho = 0.369$). mRS score was not statistically associated with size or location of AVM, type of venous drainage, or type of clinical presentation ($P > .05$).

Visual Field Deficits

Preoperatively, VFDs were seen in 4 patients (13.7%). In 3 patients, results of formal visual field testing were not available because of their clinical condition. Postoperatively, VFDs were unchanged in 2 patients, improved in 1 patient, and completely recovered in another patient. New VFDs were present in 6 patients (24%). VFDs were present at follow-up in 5 of these patients. Two cases were nondisabling (upper quadrantanopia) and 3 were disabling (hemianopia). Overall, 8 of 29 patients sustained a permanent VFD (4 quadrantanopia, 4 hemianopia), with 3 of them (3 of 25, 12%) showing a new permanent disabling VFD. The rate of preservation of full visual fields was 84% (21 of 25), with improvement in half (2 of 4) of all patients with preoperative previously known VFD.

TABLE 1. Clinical Features and Outcome of Surgically Treated Cases With Temporal Lobe Arteriovenous Malformation (n = 29)[a]

Case	Age, y	Sex	Site	Side	Spetzler-Martin grade	Presentation	VFD	Postoperative VFD	Engel Seizure Outcome Scale	Preoperative Treatment	Preoperative mRS	Postoperative mRS
1	22	F	IV	L	III	Hemorrhage				E	1	1
2	69	F	S	R	II	Seizure			I		1	1
3	53	F	P	L	I	Hemorrhage					3	3
4	38	F	P	R	II	Hemorrhage	Hemianopia	Quadrantanopia		E	2	1
5	45	F	A	R	III	Seizure			I	E	1	1
6	23	M	P	R	I	Seizure			I	E	1	1
7	47	F	P	L	II	Hemorrhage			I		2	2
8	23	M	A	L	II	Seizure			IV	E	1	2
9	44	M	A	R	II	Seizure			I		1	1
10	12	F	MS	L	I	Hemorrhage					1	1
11	45	F	P	R	III	Hemorrhage	Quadrantanopia	Quadrantanopia		E	1	1
12	31	F	P	R	I	Headache				E	1	1
13	40	M	S	L	II	Seizure			IV	E + RS	1	2
14	28	M	P	R	I	Seizure			I		1	1
15	72	M	P	L	I	Hemorrhage					2	1
16	31	F	S	L	I	Hemorrhage					2	1
17	57	F	MS	R	I	Seizure			I	E	1	2
18	21	F	S	R	I	Headache					1	1
19	53	F	S	R	I	Hemorrhage	Unknown		II		5	4
20	49	M	MS	L	II	Seizure		Quadrantanopia	I	E	1	1
21	25	F	P	R	II	Hemorrhage	Unknown	Hemianopia		E	5	2
22	47	F	IV	L	II	Hemorrhage					1	1
23	48	F	P	L	I	Hemorrhage					1	1
24	61	M	P	R	III	Hemorrhage	Hemianopia	Hemianopia			4	3
25	31	F	MS	R	III	Seizure		Hemianopia	I	E	1	2
26	46	M	MS	R	III	Seizure		Hemianopia	III	E	1	4
27	44	F	IV	L	III	Hemorrhage	Unknown				5	1
28	7	M	P	R	I	Hemorrhage	Hemianopia			E	3	0
29	26	F	MS	L	III	Seizure		Quadrantanopia	I	E	1	2

[a]A, anterior; E, embolization; IV, intraventricular; mRS: modified Rankin Scale; MS, mesial; P, posterior; RS, radiosurgery; S, sylvian; VFD, visual field defect.

The presence of preoperative VFD was associated with hemorrhage as the initial presentation (33.3% vs 0.0%; χ^2 = 5.5; P = .03; odds ratio = 1.5; 95% confidence interval = 1.005-2.238), whereas the presence of postoperative VFD was associated with larger (> 3 cm) AVMs (63.6% vs 7.7%; χ^2 = 8.3; P = .008; odds ratio = 21.0; 95% confidence interval = 1.9-227.2). AVM location was not statistically associated (P > .05) with the presence of VFD (Table 2).

Seizures

Twelve patients (41.3%) presented with seizures preoperatively. The AVMs were located in the mesial region in 5 patients (41.7%),, 3 AVMs (25%) were on the anterior lateral surface, 2 AVMs (16.6%) were perisylvian, and 2 AVMs (16.6%) were on the posterior lateral surface. Two patients had only simple partial seizures, 2 had generalized tonic-clonic seizures, and 8 had complex partial seizures (5 had subsequent secondary generalization). Eight patients (66.7%) progressed to medically refractory epilepsy. Median seizure duration (time from seizure onset to treatment) was 115 months (range, 7 months-46 years). Four patients had magnetic resonance imaging evidence of previous bleeding. Nine patients achieved Engel class I, 1 patient achieved class III, and 2 patients achieved class IV (Table 3). Of the 17 patients who did not experience seizures preoperatively, 2 (11.8%) had new-onset postoperative seizures, and of them, 1 patient became seizure free and the other had seizure reduction on AEDs at the last follow-up.

There was an association between presentation with seizures and with male sex (70.0% vs 26.3%; χ^2 = 5.1; P = .046; odds ratio = 6.5; 95% confidence interval = 1.2-35.5). Seizures were most commonly seen in those patients with a mesial AVM (83.3%) and were less frequent in patients with an AVM in the posterior neocortex (16.7%; χ^2 = 13.7; P = .008). Further analysis was done for any other association with seizure outcome, but the conclusion was that AVM size, type of seizure, and duration of epilepsy did not influence seizure outcome (P > .05; Table 2).

TABLE 2. Visual Field Defect and Seizure Statistical Analysis Summary [a]

	Yes, n (%)	No, n (%)	P
Preoperative VFD			
AVM size			.58
< 3 cm	1 (7.7)	12 (92.3)	
≥ 3 cm	2 (18.2)	9 (81.8)	
AVM location			.17
Anterior	0 (0.0)	3 (100.0)	
Posterior	4 (36.4)	7 (63.6)	
Mesial	0 (0.0)	6 (100.0)	
Sylvian	0 (0.0)	4 (100.0)	
Intraventricular	0 (0.0)	2 (100.0)	
Hemorrhage	4 (33.3)	8 (66.7)	.03
Postoperative VFD			
AVM size			.008
<3 cm	1 (7.7)	12 (92.3)	
≥3 cm	7 (63.6)	4 (36.4)	
AVM location			.06
Anterior	0 (0.0)	3 (100.0)	
Posterior	5 (41.7)	7 (58.3)	
Mesial	4 (80.0)	1 (20.0)	
Sylvian	0 (0.0)	4 (100.0)	
Intraventricular	0 (0.0)	1 (100.0)	
Seizures			
AVM size			.05
<3 cm	4 (28.6)	10 (71.4)	
≥3 cm	8 (66.7)	4 (33.3)	
AVM location			.004
Anterior	3 (100.0)	0	
Posterior	2 (16.7)	10 (83.3)	
Mesial	5 (100.0)	0 (0.0)	
Sylvian	1 (25.0)	3 (75.0)	
Intraventricular	0 (0.0)	1 (100.0)	

[a] AVM, arteriovenous malformation; VFD, visual field defect.

DISCUSSION

Overview

AVMs are challenging lesions to treat, even for experienced vascular neurosurgeons.[7] Temporal AVMs have the additional difficulty of often being in close vicinity to the optic radiations and involving important neurovascular structures. For these reasons, surgical resection is often avoided. In one of the most recent articles concerning temporal AVMs, Boström et al[7] reported a large single-center experience with 44 patients with temporal AVMs who underwent microneurosurgical excision. The results of this series reflect the challenges of treatment, with 7% new permanent hemiparesis and 19% new VFDs. In our series, 24.1% of the patients developed immediate postoperative neurological deficits other than isolated field cuts, mostly dysphasia or hemiparesis. The majority of the deficits improved significantly or resolved within a 3-month period. Such improvement is consistent with the displacement, rather than destruction, of the adjacent white matter tracts by the AVM, hemorrhage, or surgical dissection.[3] At the last follow-up, only 3 patients (10.3%) had persistent hemiparesis. Complete angiographically verified AVM excision was obtained in 93.1% of the patients. Two patients required a second procedure to achieve complete AVM resection. Despite all these challenges, we believe that microneurosurgical treatment remains a viable option, which has the additional advantage of obliterating the lesion immediately in most cases.[1] The probability of complete obliteration with endovascular or radiosurgical treatment is considerably less than 100%.[14-16] Moreover, an incomplete embolization might even increase the risk of AVM rupture, and complete occlusion of AVM after radiosurgery (when achieved) may take ≥ 2 years.[17,18] However, preoperative embolization is helpful, especially in larger AVMs, and was used in 15 of the patients (51.7%). Nevertheless, multimodal treatment should be chosen only when its benefits clearly outweigh the combined risks of multiple treatment sessions.[7]

TABLE 3. Overview of Arteriovenous Malformation Patients With Seizure at Presentation (n = 12)[a]

Case	Age, y	Sex	Site	Side	Type of Seizure	Seizure Frequency	AEDs Preoperatively	Preoperative Seizure Duration	Follow Up Duration	Engel Scale	AEDs Postoperatively
2	69	F	S	R	Simple partial	1-2/mo	> 2 AEDs, refractory	46 y	6 mo	I	2 AEDs, tapering down
5	45	F	A	R	Complex partial	3-5/mo	> 2 AEDs, refractory	21 y	1 y	I	1 AED, tapering down
6	23	M	P	R	Generalized	1-2/mo	1 AED, good control	11 mo	1 y	I	No AEDs
8	23	M	A	L	Complex partial	6-8/mo	> 2 AEDs, refractory	1.9 y	7 y	IV	2 AEDs
9	44	M	A	R	Complex partial	2-3/mo	2 AEDs, refractory	2.8 y	3 y	I	1 AED, tapering down
13	40	M	S	L	Complex partial	6-8/mo	> 2 AEDs, refractory	7 mo	3.6 y	IV	2 AEDs, bad compliance
14	28	M	P	R	Simple partial	2/y	1 AED, good control	1.3 y	2 mo	I	1 AED, tapering down
17	57	F	MS	R	Complex partial	2-4/mo	> 2 AEDs, refractory	11 mo	3.7 y	I	1 AED
20	49	M	MS	L	Generalized	2-4/y	1 AED, partial control	22 y	3 mo	I	1 AED, tapering down
25	31	F	MS	R	Complex partial	3-4/mo	2 AEDs, refractory	6 y	5.4 y	I	1 AED, tapering down
26	46	M	MS	R	Complex partial	1-2/mo	2 AEDs, refractory	11 y	7 y	III	2 AEDs
29	26	F	MS	L	Complex partial	2-3/y	1 AED, good control	1.10 y	1.4 y	I	No AEDs

[a]A, Anterior; AED, antiepileptic drug; MS, mesial; P, posterior; S, sylvian.

Final Clinical Status

Overall, the final functional status (mRS score) was satisfactory, with an excellent or good outcome (mRS score ≤ 2) in 25 patients (86.2%), an improvement rate of 27.6%, and just 1 major worsening, changing from preoperative grade 1 to grade 4 postoperatively, with no patients with a grade worse than 4. Our results, in line with previous studies,[3,7] demonstrate that despite the challenging features of these lesions and their location, good functional outcomes are achieved with surgical treatment.

We also detected a positive and significant moderate association between the final mRS score and age at diagnosis (Spearman ρ = 0.369; P = .049). This linear relation suggests that older age at the time of diagnosis is related to a poorer final outcome, reflecting the importance of avoiding a late diagnosis if possible. This is especially important in patients who present with seizures, normally those who have a more delayed diagnosis after the first onset.[19]

Visual Fields

Preoperative VFDs in surgical series of temporal AVMs were reported by Boström et al[7] in 7 of 44 cases (15.9%). In our series, a preoperative VFD was seen in 4 of 29 patients (13.7%), although in another 3 cases, visual field testing could not be done because of the patients' condition. The majority of these cases, in both series, presented with preoperative VFDs caused by a concurrent hemorrhage as the initial presentation. This was supported by statistical analysis showing a significant association (P = .03) between hemorrhagic presentation and the presence of preoperative VFD. Of the group with preexisting VFD, the VFD was unchanged in 2 cases and improved in 2 other cases (with VFD completely recovered in 1 case) after surgery (Figures 1 and 2). Although it might not be expected that surgery could improve a VFD, we observed this in 2 cases. It is difficult to find a definite explanation. Clinical presentation with homonymous VFD usually portends a permanent visual impairment.[20] The extent of visual recovery correlates negatively with age, probably because of the loss of age-dependent visual plasticity.[21,22] This could have played a role, especially because the patient with complete visual field recovery was by far the youngest in the series (7 years of age). It could also be relevant that both cases were small AVMs (< 3 cm) with hemorrhagic presentation, and the visual impairment could represent indirect compression rather than direct lesioning of the optic pathway. Nagata et al[4] suggested that the interval between the hemorrhage and emergency craniotomy is a significant factor for favorable visual field outcome, reporting 2 cases with improved VFDs after an ultraemergency surgery was performed. However, in our series, the 2 patients with improved VFDs did not

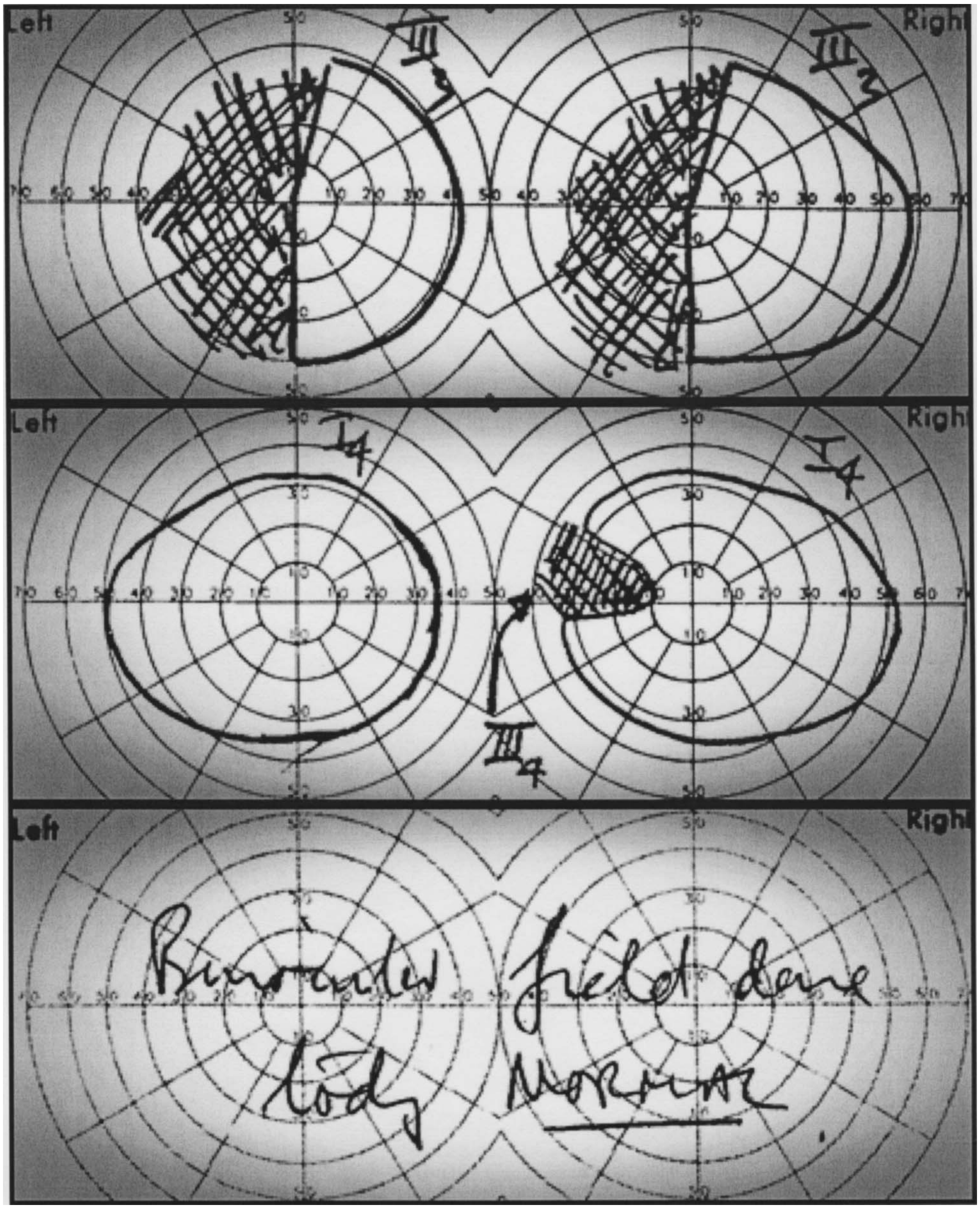

FIGURE 1. *Case 28: visual field deficit improvement. Scotometry 1 week after surgery showed an improvement in the visual field from left hemianopia to upper quadrantanopia (arrow). The patient ultimately had a full visual field by1 month (standard Goldmann visual field for high size target; stimulus intensity, III3 to III4).*

have emergency craniotomy. It is difficult to know whether all these factors played a real role or if visual improvement was a fortunate secondary result of adequate neurosurgical management.[4]

Our rate of preservation of full visual fields of 84% (21 of 25) was similar to that reported by Boström et al,[7] who found new VFDs in 19% of the cases, with a permanent disabling hemianopic deficit in 1 patient. Thus, despite the anatomic proximity to the optic tract, good visual field outcomes could be achieved by experienced vascular neurosurgeons. Because of a correlation we found between the presence of postoperative VFD and AVM size, special consideration should be given to medium and large AVMs because it is probably easier to damage or displace the optic radiations.

Seizures

The second most common presenting symptom of brain AVMs is seizures.[23] With temporal lobe AVMs, seizures have been

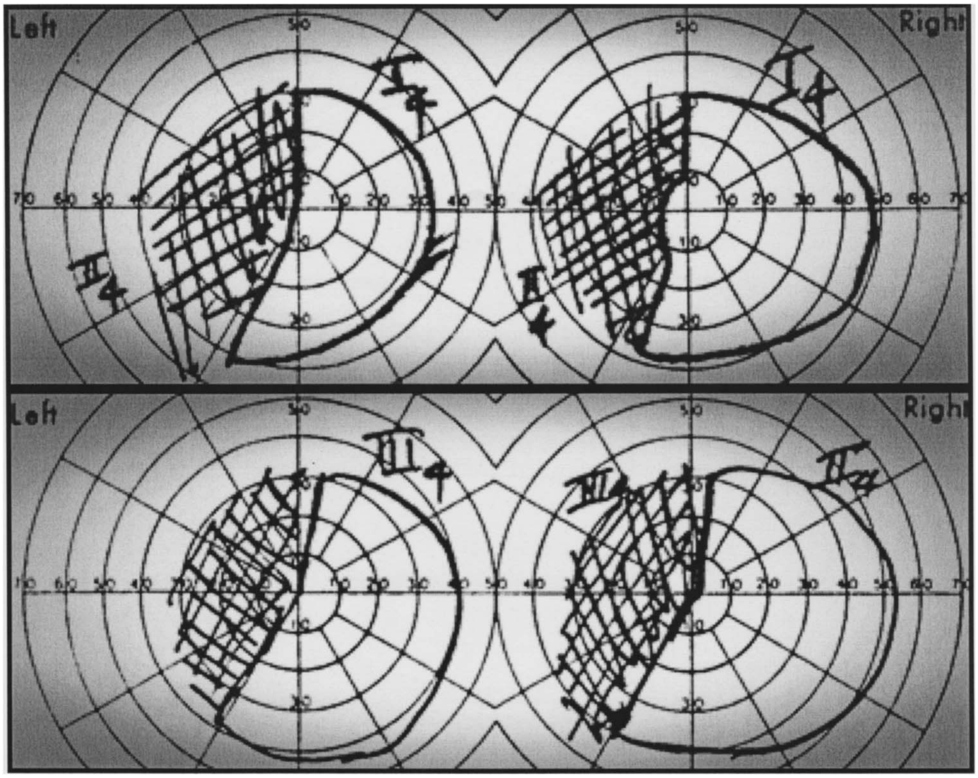

FIGURE 2. *Case 24: persistent visual field deficit. Scotometry after surgery showed no improvement in the visual field deficit with persistence of left hemianopia (standard Goldmann visual field for high test target; stimulus intensity, I-III4).*

reported to be more common, occurring in 46% of patients, compared with 24% in nontemporal AVMs.[3] In our series, the incidence of epilepsy as an initial symptom was 41.3%. Several studies have reported that between 12% and 57% of AVM patients experience seizures, and epilepsy could persist even after surgical resection.[24-27] Epilepsy, particularly when refractory to medical treatment, can lead to significant morbidity and decreased quality of life.[4,24,28-31] The importance of seizure control is usually underappreciated in the surgical management of AVMs because most clinical and neurosurgical practices focus on the risk of bleeding related to these lesions.[32,33] This is particularly notable in A Randomized Trial of Unruptured Brain Arteriovenous Malformations (ARUBA)[8,34-37] in which seizure outcome was considered a secondary neurological adverse event.

In our series, 66.7% of patients with seizures as the initial presentation progressed to medically refractory epilepsy. Our outcomes show that good control could be achieved in patients with refractory and disabling, complex partial seizures. It is known that temporal lobe epilepsy, particularly when lesional (eg, low-grade gliomas),[38-41] responds better to a surgical management than extratemporal epilepsy. The few studies[3,4] that address epilepsy in temporal AVMs show good postsurgical seizure outcomes. Although recent studies suggest that interventional treatment of any kind does not influence the chances of achieving seizure freedom compared with conservative management in AVM patients,[9,10] our results indicate that in the cases of temporal AVMs, the situation is different. Considering those results and the impact of the surgery on temporal lobe lesional epilepsy, we believe that surgery is the most reliable option for these patients. We considered, in most cases, that seizure semiology and preoperative electroencephalogram supported the AVM as the seizure focus, and in general, we did not perform any specific workup to define the extension of the epileptogenic zone. We planned pure lesionectomy alone (AVM resection) in most cases except for the anteriorly located AVMs, for which an extended anterior lobectomy was the usual surgical procedure. However, some authors[42,43] have described that the remote epileptic activity is particularly prominent in AVMs of the temporal lobe and usually involves the medial temporal structures. Nagata et al[4] showed 2 cases (2 of 11, 18.2%) of refractory seizure postoperatively with remote seizure foci that needed epilepsy surgery a few years after AVM surgery. In our series, the 2 patients (2 of 12, 16.7%) with refractory seizures (Engel class IV) after surgery showed some epileptic activity outside the AVM resection in follow-up studies. One of these patients, several years after the AVM surgery, underwent epilepsy surgery with a good outcome (seizure free, Engel class I), and in the other patient, although epilepsy surgery was considered, it was finally rejected given the patient's alcohol abuse. Therefore, we think that the surgical approach to temporal AVMs should be planned not only as a vascular procedure but also taking into account the value of excision of the epileptic focus, considering the definition of the epileptogenic zone limits. Hence, epilepsy presurgical evaluation (epilepsy consult and video-electroencephalogram) should be considered.

Limitations

This study has limitations. It was retrospective in nature with a relatively small number of patients. Statistical correction for multiple testing could not be performed. However, we made important findings not previously reported in the literature.

CONCLUSION

Surgical management of temporal lobe AVMs remains challenging. However, good outcomes with low morbidity and good visual field preservation can be accomplished, especially if the surgery is performed by experienced neurovascular surgeons. Although prevention of intracranial hemorrhage or rehemorrhage remains the most important operative consideration, our data suggest that good seizure outcome can be achieved in patients with refractory seizures. It is concluded that patients with temporal lobe AVMs and refractory seizures benefit from surgery.

Disclosures

This work was supported in part by the Fundación Alfonso Martin Escudero grant for international research 2011, as part of Dr. Lopez-Ojeda Fellowship funding, with no conflict of interest concerning this publication. Dr Burneo has received financial support from UCB Canada for educational activities and research with no conflict of interest concerning this publication. Dr Labib is an active consultant for NICO Co with no conflict of interest concerning this publication. The authors have no personal financial or institutional interest in any of the drugs, materials, or devices described in this article.

REFERENCES

1. Brown RD, Weibers DO, Forbes G, et al. The natural history of unruptured intracranial arteriovenous malformations. *J Neurosurg.* 1988;68(3):352-357.
2. Drake CG. Cerebral arteriovenous malformations: considerations for and experience with surgical treatment in 166 cases. *Clin Neurosurg.* 1979;26:145-208.
3. Malik GM, Seyfried DM, Morgan JK. Temporal lobe arteriovenous malformations: surgical management and outcome. *Surg Neurol.* 1996;46(2):106-114.
4. Nagata S, Morioka T, Matsukado K, Natori Y, Sasaki T. Retrospective analysis of the surgically treated temporal lobe arteriovenous malformations with focus on the visual field defects and epilepsy. *Surg Neurol.* 2006;66(1):50-55.
5. Heros RC. Arteriovenous malformations of the medial temporal lobe: surgical approach and neuroradiological characterization. *J Neurosurg.* 1982;56(1):44-52.
6. Oka N, Kamiyama K, Nakada J, Endo S, Takaku A. Surgical approach to arteriovenous malformation of the medial temporal lobe: report of three cases. *Neurol Med Chir (Tokyo).* 1990;30(12):940-944.
7. Boström A, Schaller K, Seifert J, Schramm J. The place for surgical treatment for AVM involving the temporal lobe. *Acta Neurochir (Wien).* 2011;153(2):271-278.
8. Stapf C, Mohr JP. Unruptured brain arteriovenous malformations should be treated conservatively: yes. *Stroke*. 2007;38(12):3308-3309.
9. Josephson CB, Bhattacharya JJ, Counsell CE, et al. Seizure risk with AVM treatment or conservative management: prospective, population-based study. *Neurology*. 2012;79(6):500-507.
10. Wedderburn CJ, van Beijnum J, Bhattacharya JJ, et al. Outcome after interventional or conservative management of unruptured brain arteriovenous malformations: a prospective, population-based cohort study. *Lancet Neurol.* 2008;7(3):223-230.
11. Spetzler RF, Zabramski JM. Grading and staged resection of cerebral arteriovenous malformations. *Clin Neurosurg.* 1990;36:318-337.
12. Kwan P, Arzimanoglou A, Berg AT, et al. Definition of drug resistant epilepsy: consensus proposal by the ad hoc Task Force of the ILAE Commission on Therapeutic Strategies. *Epilepsia*. 2010;51(6):1069-1077.
13. Engel J, Van Ness PC, Rasmussen TB, et al. Outcome with respect to epileptic seizures. In: Engel J, ed. *Surgical Treatment of the Epilepsies*. 2nd ed. New York, NY: Raven Press; 1993:609-621.
14. Panagiotopoulos V, Gizewski E, Asgari S, Regel J, Forsting M, Wanke I. Embolization of intracranial arteriovenous malformations with ethylene-vinyl alcohol copolymer (Onyx). *AJNR Am J Neuroradiol.* 2009;30(1):99-106.
15. Valavanis A, Yaşargil MG. The endovascular treatment of brain arteriovenous malformations. *Adv Tech Stand Neurosurg.* 1998;24:131-214.
16. Viñuela F, Duckwiler G, Guglielmi G. Contribution of interventional neuroradiology in the therapeutic management of brain arteriovenous malformations. *J Stroke Cerebrovasc Dis.* 1997;6(4):268-271.
17. Schauble B, Cascino GD, Pollock BE, et al. Seizure outcomes after stereotactic radiosurgery for cerebral arteriovenous malformations. *Neurology*. 2004;63(4):683-687.
18. Yang SY, Kim DG, Chung HT, Paek SH. Radiosurgery for unruptured cerebral arteriovenous malformations: long-term seizure outcome. *Neurology*. 2012;78(17):1292-1298.
19. Al-Shahi R, Warlow C. A systematic review of the frequency and prognosis of arteriovenous malformations of the brain in adults. *Brain*. 2001;124(pt 10):1900-1926.
20. Zhang X, Kedar S, Lynn MJ, Newman NJ, Biousse V. Natural history of homonymous hemianopia. *Neurology*. 2006;66(6):901-905.
21. Pambakian ALM, Kennard C. Can visual function be restored in patients with homonymous hemianopia? *Br J Ophthalmol.* 1997;81(4):324-328.
22. Huxlin KR. Perceptual plasticity in damaged adult visual systems. *Vision Res.* 2008;48(20):2154-2166.
23. Thorpe ML, Cordato DJ, Morgan MK, Herkes GK. Postoperative seizure outcome in a series of 114 patients with supratentorial arteriovenous malformations. *J Clin Neurosci.* 2000;7(2):107-111.
24. Englot DJ, Young WL, Han SJ, et al. Seizure predictors and control after microsurgical resection of supratentorial arteriovenous malformations in 440 patients. *Neurosurgery*. 2012;71(3):572-580.
25. Heros RC, Korosue K, Diebold PM. Surgical excision of cerebral arteriovenous malformations: late results. *Neurosurgery*. 1990;26(4):570-577; discussion 577-578.
26. Hoh BL, Chapman PH, Loeffler JS, Carter BS, Ogilvy CS. Results of multimodality treatment for 141 patients with brain arteriovenous malformations and seizures: factors associated with seizure incidence and seizure outcomes. *Neurosurgery*. 2002;51(2):303-309; discussion 309-311.
27. Josephson CB, Leach JP, Duncan R, et al. Seizure risk from cavernous or arteriovenous malformations: prospective population-based study. *Neurology*. 2011;76(18):1548-1554.
28. Murphy MJ. Long-term follow-up of seizures associated with cerebral arteriovenous malformations: results of therapy. *Arch Neurol.* 1985;42(5):477-479.
29. Cascino GD. When drugs and surgery don't work. *Epilepsia*. 2008;49(suppl 9):79-84.
30. Cramer JA, Mintzer S, Wheless J, Mattson RH. Adverse effects of antiepileptic drugs: a brief overview of important issues. *Expert Rev Neurother.* 2010;10(6):885-891.
31. Kraemer DL, Awad IA. Vascular malformations and epilepsy: clinical considerations and basic mechanisms. *Epilepsia*. 1994;35(6):30-43.
32. Nakaji P, Spetzler RF. Indications for surgical treatment of arteriovenous malformations. *Neurosurg Clin N Am.* 2005;16(2):365-366.
33. Ondra SL, Troupp H, George ED, Schwab K. The natural history of symptomatic arteriovenous malformations of the brain: a 24-year follow-up assessment. *J Neurosurg.* 1990;73(3):387-391.
34. Cockroft KM. Unruptured brain arteriovenous malformations should be treated conservatively: no. *Stroke*. 2007;38(12):3310-3311.
35. Fiehler J, Stapf C. ARUBA: beating natural history in unruptured brain AVMs by intervention. *Neuroradiology*. 2008;50(6):465-467.

36. Mohr JP, Moskowitz AF, Stapf C, et al. The ARUBA trial: current status, future hopes. *Stroke*. 2010;41(8):537-540.
37. Mohr JP, Moskowitz A, Ascheim D, et al. The ARUBA Trial: A Randomized Trial of Unruptured Brain Arteriovenous Malformations (ARUBA). 2006. http://arubastudy.org/. Accessed October 2012.
38. Englot DJ, Berger MS, Barbaro NM, Chang EF. Predictors of seizure freedom after resection of supratentorial low-grade gliomas: a review. *J Neurosurg*. 2011;115(2):240-244.
39. McIntosh AM, Kalnins RM, Mitchell LA, Fabinyi GCA, Briellmann RS, Berkovic SF. Temporal lobectomy: long-term seizure outcome, late recurrence and risks for seizure recurrence. *Brain*. 2004;127(pt 9):2018-2030.
40. Mirsattari SM, Chong JJR, Hammond RR, et al. Do epileptic seizures predict outcome in patients with oligodendroglioma? *Epilepsy Res*. 2011;94(1-2):39-44.
41. Salanova V, Markand O, Worth R. Temporal lobe epilepsy surgery: outcome, complications, and late mortality rate in 215 patients. *Epilepsia*. 2002;43(2):170-174.
42. Yeh HS, Privitera MD. Secondary epileptogenesis in cerebral arteriovenous malformations. *Arch Neurol*. 1991;48(11):1122-1124.
43. Yeh HS, Tew JM Jr, Gartner M. Seizure control after surgery on cerebral arteriovenous malformations. *J Neurosurg*. 1993;78(1):12-18.

Acknowledgments

We gratefully acknowledge the contributions of the neurologist Secundino Lopez-Pousa and biostatistician Josep Garre Olmo from the Institut d'Assistència Sanitària (IAS) Research Unit, Salt (Girona), Spain, for their assistance with various aspects of statistical data analysis and for review of the manuscript. We wish also to acknowledge with gratitude Dr Warren Blume, epileptologist, who referred many of the patients with AVMs and seizures contained within this report, with regard to the potential role of AVM surgery in seizure reduction or elimination.

COMMENT

The authors describe seizure control and outcome of visual function in patients with temporal arteriovenous malformations (AVMs). The article emphasizes that in any patient with seizures related to a structural lesion that requires surgical intervention, there is a unique opportunity not only to ameliorate the consequences of the lesion but also to remove the seizure focus.

A recent study compared the prospective risk of developing seizures and epilepsy in patients with newly diagnosed AVMs and did not find a difference between conservative management and interventional treatment.[1] This likely reflects the fact that only about half of AVMs are epileptogenic, and medication often works well in newly diagnosed epilepsies (as does surgery). In contrast, the present study included 8 of 12 patients with seizures who already failed >2 antiepileptic drugs and had developed intractable epilepsy at the time of surgery. Six of the 8 did extremely well (Engel class I). Particularly in patients with temporal lobe epilepsy, a detailed seizure semiology and interictal and ictal video electroencephalogram is likely able to delineate if the epileptogenic zone extends anterior, mesial or posterior to the AVM or involves remote structures, and a presurgical epilepsy evaluation might be able to further improve outcome in these patients.

Aside from visual decline, patients who undergo temporal lobe surgery are at risk for postoperative memory impairment, particularly if the lesion is not already scarring the hippocampus and if it involves the hemisphere dominant for language. In the present report, 8 of the 12 patients had seizures arising from the right temporal region, and only 4 had presumably dominant temporal lobe epilepsy. Memory outcome was not further discussed, and there is very limited literature in that regard when it comes to vascular malformations. Going forward, it will be important to systematically evaluate not only visual but also memory function preoperatively and postoperatively to better estimate the risk of functional decline.

Stephan U. Schuele
Chicago, Illinois

1. Josephson CB, Bhattacharya JJ, Counsell CE, et al. Seizure risk with AVM treatment or conservative management: prospective, population-based study. *Neurology*. 2012;79(6):500-507.

CME Questions:

1. What does Engel Class 1 outcome represent in terms of seizure frequency as compared to pretreatment status?
   A. Reduction in seizure frequency by 25%
   B. Reduction in seizure frequency by 50%
   C. Increase of seizure frequency by 50%
   D. Absence of disabling seizures or auras

2. What is the most common presentation of unruptured temporal AVMs?
   A. Visual field deficits
   B. Seizures
   C. Memory disorder
   D. Motor deficits
   E. Personality changes

3. Which location in the figure corresponds to Meyer's loop, the optic radiation in the temporal lobe?
   A. A
   B. B
   C. C
   D. D