MICROSURGICAL MANAGEMENT OF THE SUPRATENTORIAL ARTERIOVENOUS MALFORMATIONS (AVMs)

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The treatment of cerebral AVMs has increased in complexity with the development of multimodal approaches. Given the new achievements in the fields of interventional neuroradiology and radiosurgery, the better understanding of the natural history and the risks posed by an untreated cerebral AVM, microsurgery for cerebral AVMs should be selective. Based on existing grading systems, the standard treatment strategy is generally multimodal. However, microsurgical resection with its low rates of mortality and morbidity remains the best option in judiciously selected patients .
Material and method: we report a series of 12 patients with supratentorial cerebral AVMs operated between 2012–2014 in the Neurosurgery Clinic of the National Institute of Neurology and Neurovascular Diseases. 3 patients (25%) had unruptured cerebral AVMs and 9 patients (75%) presented with hemorrhage, 4 of whom were comatose (GCS<9p or mRS-5). For grading cerebral AVMS, we used the Spetzler Martin classification. According to the SM grading scale there were 3 SM grade 1 lesions (25%), 4 SM grade 2 lesions (33,33%), 3 SM grade 3 (25%) and 2 SM grade 4 lesions (16,66%). All the patients underwent surgery, and complete resection was achieved in 10 patients (83,3%%). The mortality rate was 0 and the overall morbidity rate was 33,3%.
Conclusion: Complete microsurgical removal remains the gold standard and the best option for the definitive treatment of cerebral AVMs. Microsurgical resection of the supratentorial cerebral AVMs remains safe and secure in very well selected patients and its benefits must be carefully weighed against the risks and potential postoperative complications. In some cases emergency surgery can be required to remove a life threatening haematoma.

Key words: arteriovenous malformation (AVMs), microsurgery, modified Rankin Scale.

INTRODUCTION

Cerebral AVMs are vascular lesions composed of tortuous arteries and veins without intervening capillaries. Even though cerebral arteriovenous malformations (AVMs) were considered to be congenital lesions, new evidence suggests that they most likely represent an acquired condition and as such, they can undergo a variety of phenomena such as growth, remodeling, regression or hemorrhage[14, 17, 21, 23, 28].

Their prevalence is approximately 0,1% (1) and they are responsible for 1,4%–2 % of all hemorrhagic strokes[26]. The most common age at diagnosis is the third and fourth decade[4].

The most frequently encountered form of presentation is intracranial hemorrhage (aprox 50%), followed by symptomatic epilepsy, with 18–35% of patients diagnosed because of seizures[4]. Intracranial hemorrhage is the most severe clinical presentation[29]. AVMs are the leading cause of nontraumatic intracerebral hemorrhage in young people (< 35 years old) and the most common neurological cause of impairment and death in patients under 20 years[24]. They account for approximately 1,2–2% of all hemorrhagic strokes. 75% of hemorrhagic presentations occur before the age of 50[6]. It seems that posterior fossa AVMs are more likely to present with hemorrhage than supratentorial AVMs[2,3].

The annual rate of rupture in untreated patients is considered to be 2–4 % per year.

Less common symptoms are chronic headaches (6–14% of patients) and focal neurological deficits due to mass effect or hemodynamic changes (3–10% of patients). The number of incidentally found AVMs has been rapidly increasing (10% in contemporary series)[4]. Hemorrhage from an AVM is less hazardous than the rupture of an intracranial aneurysm. Short term case fatality rate in a ruptured AVM is less than 10% per bleeding, compared to 50% in the case of an aneurysmal SAH[7, 12]. Risk factors for hemorrhagic presentation according to univariate and multivariate analyses are: small size, young age, deep venous drainage, non border-zone location, associated aneurysms, deep location[18, 27], high arterial input pressure[11].

The bleeding rate is higher in the first years after diagnosis. Previous rupture is the most consistent factor associated with the increased risk for an AVM rupture[8, 10, 22].

Microneurosurgical resection represents the most important treatment for SM grade 1,2 and in many cases even for SM grade 3 and can be done in numerous combinations with SRS and embolisation. An AVM's microsurgical resection consists of the AVM's exposure, subarachnoid approach, identifying the draining veins, identification of the feeding arteries, pial dissection, parenchimal dissection, "ependymal dissection", nidus resection, interruption of the draining veins[19].

MATERIAL AND METHODS

We report a series of 12 patients that were operated on by the main author in the Neurosurgery Clinic of the National Institute of Neurology and Neurovascular Diseases between 2012–2014. Medical information was retrospectively reviewed. We recorded and analised each patient's medical history, whether or not they had initial hemorrhage, the signs and symptoms at admission, a detailed neurological exam, patient–related factors as well as lesion related-factors, pre and postoperative neuroimaging evaluation (CT scan, MRI and angio MRI, four vessels cerebral angiography), details regarding surgery, clinical outcomes, resection rates, postoperative complications and prognosis.

OUTCOME MEASURES

All the patients underwent a thorough neurological exam both pre- and postoperative until discharge. Neurological follow-up was assessed according to the modified Rankin Scale (mRS).

SURGICAL TECHNIQUES

All the patients were treated by open surgery: subarachnoid and pial dissection, coagulation or clipping of the feeding arteries, circumferential dissection and resection of the nidus, resection or clipping of the flow-related aneurysms, coagulation and transection of the draining veins, ligation of the ECA (one patient), removal of the intracerebral clots. To assess the AVM's obliteration, postoperative angiography was routinely used.

RESULTS

Patients characteristics at admission according to the SM grading system and 3 months follow-up are shown in Table 1.

Of the 12 cases included in this series, 9 patients (75%) had initial hemorrhagic presentation of whom 4 patients were comatose at admission (GCS<9p or mRS-5). 4 patients (33,33%) associated SAH and two patients (16.66%) had intraventricular hemorrhage. 7 patients had motor neurological deficits, 3 presented with visual impairment and 4 patients experienced seizures (complex partial and visual seizures). A single patient had a known history of AVM, diagnosed 10 years before. 7 patients were males and 5 females. The mean age at presentation was 44 years (16–65).

Based on the SM grading scale there were 3 SM grade 1lesions (27,3%), 5 SM grade 2 lesions (45,5%), 3 SM grade 3 (27,3%) and 1 Sm grade 4 lesion.

Angiographic characteristics of the patients AVMs are shown in Table 2.

5 patients had 5 associated aneurysms (3 flow related aneurysms – 2 ACommA aneurysms and one PCA-P3 segment aneurysm and 2 intranidal aneurysms). Deep venous drainage was encountered in 2 patients. 7 patients (58,33%) had high-flow, high arterial input pressure AVMs.

Table 1

Patients characteristics at admission according to the SM grading system and 3 months follow-up

	age	GCS	mRS	Clinical Presentation	SM Scale	3 months follow-up mRS
1	65	8	5	Comatose state, Hemiparesis, orotracheal intubation+mechanical ventilation	4	4
2	65	15	1	Partial complex seizures	1	0
3	53	14	1	Visual field defect, visual seizures	2	0
4	16	12	4	Hemiparesis, altered mental status, ICH syndrome	3	1
5	44	15	1	Visual field defect, visual seizures	2	0
6	33	13	2	Dysphasia, ICH syndrome	2	0
7	50	14	1	Visual field defect, ICH syndrome	2	0
8	47	5	5	Comatose state, Hemiparesis, Orotracheal intubation+mechanical ventilation	2	3
9	25	7	5	Comatose state, Hemiparesis, Orotracheal intubation+mechanical ventilation	2	1
10	30	9	2	Hemiparesis, Altered mental status	2	1
11	56	10	4	Hemiparesis, Altered mental status, ICH syndrome, Aphasia	1	2
12	44	7	5	10 years known history of AVM Comatose state, hemiparesis Orotracheal intubation+mechanical ventilation	4	3

Table 2

Angiographic characteristics of the patients AVMs

	AVM Location	High flow, high arterial input pressure	Feeding arteries	Nidus size (mm)	Associated aneurysm	Draining veins
1	Frontal	yes	M2 segment of MCA	48	ACommA flow related aneurysm	SSS, TS, right sinus
2	Fronto-temporal	no	M3 and M4 segments of MCA, VA via AComP	27	No	SSS, TS, right sinus
3	Occipital	yes	M4 segment of MCA, PCA	35	Intranidal aneurysm	SSS, torcula, TS
4	Fronto-temporal	no	M2 segment of MCA	15	Intranidal aneurysm (3mm)	Torcula, SSS
5	Parieto-occcipital	yes	Posterior temporal artery, P3 segment of PCA	25	No	SSS, TS
6	Frontal	yes	A2 segment	30	No	SSS
7	Occipital	no	P3 and P4 segments of PCA and ICA via ACommP	22	Dissected flow related aneurysm (7mm) in P3 segment	Torcula
8	Temporal	yes	Posterior temporal artery	20	No	TS
9	Fronto-temporal	yes	M2 and M3 segments of MCA	35	No	SSS
10	Frontal	no	M2 segments of MCA	20	No	SSS
11	Parieto-occipital	no	M3 and M4 segments of MCA, P3 segment of PCA	25	No	TS, SSS
12	Frontal	yes	M2 and M3 segments of MCA, pericallosal artery from controlateral ICA via ACommA	60	ACommA flow related	SSS

All the patients in this series underwent surgery, with a 83,3% complete AVM resection rate and a morbidity rate of 33,33%. Temporary and definitive clip application on feeders and less coagulation of the nidus was necessary to control intranidal pressure and to avoid uncontrollable bleeding from

the nidus. Intranidal aneurysms were coagulated and resected. One P3 segment dissected flow related aneurysm was clipped. Postoperative angiography showed residual nidus in 2 cases (1 SM grade 3 patient and 1 SM grade 4 patient). The mortality rate was 0. Outcome was favorable in all cases. 4 patients had minor neurologic deficits upon discharge, with one patient having a severe deficit. Following surgery mRS improved in all patients. One patient experienced postoperative seizures, two patients had a respiratory infection and one patient had a Clostridium Difficile intestinal infection. There was no morbidity directly related to surgery. At three months follow-up, 8 patients (66.66%) were independent in their daily activities. Five patients experienced a permanent neurologic deficit (two cases of hemiparesis, one of aphasia, one of partial visual field defect and one of hemiplegia).

One patient who was admitted in a comatose state (GCS-5 p) and had a life threatening haematoma, was not angiographically investigated immediately after presentation. Cerebral angiography was done 2 days after the emergency surgery for the removal of the haematoma. In the case of the other 3 comatose patients, angiography was quickly performed (between 4–16 hours) and they were operated on 6–24 hours after admission.

CASE 1

A 44 years old male was admitted in our department with visual seizures and transient impressive aphasia. The symptoms had progressively developed 6 months before admission. Contrast-enhanced CT scan showed an unruptured AVM involving the convexity surface of the left posterior temporal and occipital lobes (Fig. 1). Preoperative cerebral angiography showed a high-flow SM grade II posterior temporooccipital AVM with 2 feeding arteries: left posterior temporal artery from the MCA and left P3 segment of the PCA; nidus size 25 mm with no intranidal changes; superficial venous drainage into SSS and TS via vein of Labbe. The patient underwent surgery in a prone position. We performed a left temporooccipital craniotomy over the TS and a transcortical approach through the lateral part of the occipital inferior gyrus and tempororoccipital notch. The complete resection of the AVM is shown in the Fig. 3. At 3 months follow-up, the patient had minor visual field defect.

CASE 2

A 50 years old male was admitted with a sudden onset of visual field defect and ICH syndrome. CT scan showed a right paramedian occipital haematoma (Fig. 4). Four vessels cerebral angiography (Fig. 5) showed a SM grade II right paramedian parietooccipital AVM with feeding arteries from the P3 and P4 segments of the right PCA and from the right ICA via AComm P; 7 mm dissecting flow-related aneurysm located on the right P3 segment; nidus size 24 mm with no intranidal changes; one tortuous draining vein into the torcula. We exposed the AVM through a torcular craniotomy over the SSS, torcula and TS with the patient in the prone position and the nose turned 45 degrees down toward the floor. The AVM was completely resected together with the P3 segment aneurysm as shown in Figure 6.

CASE 3

A 16 years old female presented with altered mental state, GCS-12p, apahasia and right hemiparesis. CT scan showed a frontotemporal haematoma (Fig. 7). Preoperative angiography revealed a SM grade II, left temporal AVM with feeding arteries from the left MCA, nidus size 14 mm, 3 mm intranidal aneurysm; venous drainage into the SSS, important ascending of the MCA (Fig. 8). The AVM was exposed through a pterional approach and completely resected (Fig. 9). The intranidal aneurysm was coagulated and the haematoma was evacuated. At 3 months follow-up she presented mild right hemiparesis and no aphasia.

CASE 4

A 25 years old male was brought to our department in a comatose state, GCS-7p, intubated and mechanically ventilated. CT scan showed a large frontal haematoma with perihemorrhagic edema and mass effect (Fig. 10). Cerebral angiography showed a SM grade II, frontal AVM with feeding arteries from the M3 segment of the MCA, nidus size 25 mm, draining vein into the SSS (Fig. 11). The AVM was completely resected as shown in Fig. 12. At 3 months follow-up, the patient had mild left hemiparesis.

DISCUSSION

Cerebral convexity AVMs account for nearly 30% of all AVMs[19]. Modern treatment of cerebral AVMs consists of multimodal therapies: microneurosurgical resection[15], radiosurgery[5] and endovascular embolisation[30].

Numerous grading systems have been developed in order to achieve the best therapeutic option for each patient's AVM. Two of them, Martin-Spetzler and Lawton supplementary grading system have been validated for their utility in predicting surgical morbidity. The first one includes 3 factors: location, size and eloquence and the former one 3 factors: age, hemorrhage and nidus compactness. The 3-tier Spetzler-Ponce system retains the predictive accuracy of outcomes and simplifies treatment recommendations[25].

The ARUBA trial showed that medical management alone is superior to interventional therapy for the prevention of death or stroke in patients with unruptured brain AVMs[20]. An accurate understanding of the morbidity associated with AVM rupture is important as the number of incidentally found AVMs has been rapidly increasing and more patients with unruptured cerebral AVMs seek consultation.

Since untreated AVM s are associated with increased long term mortality[4], the surgeon's choice for one of the multimodal approaches should be undertaken with a clear understanding of the risk-benefits ratio.

Complete microsurgical removal remains the gold standard and the best option for the definitive treatment of cerebral AVMs[16]. The limitations of microsurgical resection include anatomic accessibility, intraoperative rupture, resection of normal brain tissue, edema from retraction and feeding vessel thrombosis

The primary goal of AVM surgery is to prevent hemorrhage and consequently seizures, neurological deficits, or death[16]. If hemorrhage has occurred, emergency removal of a life threatening haematoma and microsurgical resection of the AVM should be mandatory.

Moreover, AVM hemorrhage is an important factor that facilitates AVM resection by creating a pathway to the nidus and a plane of dissection thus facilitating nidus resection in its parenchimal or "ependymal" sides or even opening up a transcortical pathway to the nidus.

In our series 9 patients (75%) presented with haematoma secondary to a ruptured AVM and complete resection was achieved in 7 patients. 4 of the 5 associated aneurysms were encountered in patients who had hemorrhage at initial presentation, which supports data found in literature stating that associated aneurysms are a significant risk factor for hemorrhage[9,13].

The risk of hemorrhage remains present until complete AVM obliteration is achieved. Therefore partial embolisation of an AVM and radiosurgery, with its latency period, don't completely reduce the risk of hemorrhage to 0. The hemorrhage rate during radiosurgery is no different than the AVMs natural history of 1.9%/year[19]. Multimodality management is the standard for the majority of AVMs. In Lawton's series, 67% of treated AVMs underwent multimodality therapy, in various combinations.

Microsurgical resection alone is utilised in only 8% of AVM patients. In Lawton's series, 14% of patients underwent microsurgery alone.

Despite the fact that the actual grading systems are useful in making treatment strategies, the decision to treat a patient's AVM is a complex process that must take into consideration the individual's neurologic presentation, anatomy of the AVM and biology.

Duong et all suggested that high intranidal pressure is a powerful risk for hemorrhage. In our series 5 (55.5%) of the 9 patients who presented with ruptured AVMs, had high-flow, high arterial input pressure on angiography.

The most common indication for microsurgical resection alone is neurologic deterioration due to intracerebral haematoma. In our series 4 of the 12 patients (75%) underwent microsurgery because of a life threatening haematoma. Microsurgery alone is also indicated in AVMs located distally in a vascular territory with poor endovascular acces[19].

CONCLUSIONS

Multimodality management is the standard for the majority of AVMs

Complete microsurgical removal remains the gold standard and the best option for the definitive treatment of cerebral AVMs. Its benefits must be carefully weighed against the risks and potential postoperative complications.

The most common indication for microsurgical resection alone is neurologic deterioration due to intracerebral haematoma.

ABREVIATIONS

AcommA – anterior communicating artery
ARUBA – a randomised trial of unruptured brain arteriovenous malformations
AVMs – arteriovenous malformations
CT – computer tomography
ECA – external carotid artery
GCS – Glascow coma scale
ICA – internal carotid artery
ICH – intracranian hypertension syndrome
mRS – modified Rankin score
MCA – middle cerebral artery
MRI – magnetic resonance imaging
PCA – posterior cerebral artery
PcommA – posterior communicating artery SM grade-Spetzler Martin grade
SAH – subarachnoid hemorrhage
SRS – stereotactic radiosurgery
SSS – superior sagittal sinus
TS – transvers sinus.

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