Original Research Paper

Volume - 11 | Issue - 04 | April - 2021 | PRINT ISSN No. 2249 - 555X | DOI : 10.36106/ijar

Neurosurgery

ENDOVASCULAR MANAGEMENT OF BRAIN ARTERIOVENOUS MALFORMATION, AN OBSERVATIONAL STUDY.

Subhasis Ghosh	Prof. Department of Neurosurgery, Institute of Postgraduate Medical Education & Research Kolkata, West Bengal 700020.
Firdous Ahmad Malik*	Resident Neurosurgery, Institute of Postgraduate Medical Education & Research Kolkata, West Bengal 700020.*Corresponding Author
Sudipto Chatterjee	Prof. Department of Neurosurgery, Institute of Postgraduate Medical Education & Research Kolkata, West Bengal 700020.
Subhomitra Chaudhuri	Assistant Prof Department Of Neurosurgery, Institute of Postgraduate Medical Education & Research Kolkata, West Bengal 700020.
Tapan Dhiber	Prof Department Of Radiology. IPGME&R SSKM HOSPITAL.Kolkata, West Bengal 700020.

ABSTRACT **Purpose:** The aim of the study was to study the clinical presentation, indication for choosing modality of treatment and post endovascular/ post-surgery obliteration rates, intraoperative serious events and postoperative neurodeficit as well as morbidity and mortalityof brain arterio-venous malformations.

Material and methods: Between January2019 –December 2020 twenty patients with intracranial arteriovenous mal- formations were embolised with Onyx with intent to cure. There were six (30%) females and 14 (70%) males with the age from 12 to 60 years (mean 38.91 years, SD 14.82). Inclusion criteria were: patients of cerebral arteriovenous malformation with Spetzler Martin grade 1- 3, symptomatic or incidental detected. Arterio- venous fistulas as well as aneurysms accompanying AVMs were excluded from this study. Clinical presentation was intracranial hemorrhage in eight (40%) out of 20 patients, neurological deficits in three (7%) out of 20 patients, headaches in six (30%) out of 20 patients, and seizures in seven (35%) patients. Based on the Spetzler-Martin score there were four (20%) grade I AVMs, seven (35%) grade II AVMs, and nine (45%) grade III AVM. **Results:** Out of 20 patients, 18 patients underwent embolization only while 1 patient had surgery and 1 patient had both embolization and surgery. Total obliteration was achieved in nine patients (27.8%) with 8 patients of <3 cm and only 1 patient of >3 cm size. The most common reason for initial incomplete angiographic occlusion was unfavourable Angio architectural features of arteriovenous malformations. All patients had follow-up at 1, 3 ,6 months with last follow up at 1 year. The common complication rate was vasospasm in 4 patients (20%). One patient had permanent hemiplegia. There were no deaths.

Conclusions: In conclusion, this study found that endovascular management of brain AVMs as a sole therapeutic modality plays a limited role even in small (< 3 cm) size of AVM with few feeders.

KEYWORDS : AVM -Arteriovenous Malformation, ONYX- 18 Liquid Embolic Agent, NBCA – N -Butyl Cyanoacrylate, DSA- Digital Subtraction Angiography, SM GRADE- Spetzler Martin Grade.

INTRODUCTION

Arteriovenous malformations (AVMs) are vascular abnormalities that consist of multiple fistulous connections between arteries and veins without a normal intervening capillary bed. AVMs are thought to be congenital. 90% of all AVMs are supratentorial[1,2]. The most common presentation is haemorrhage, followed by seizures, headaches and neurological deficits[3-5] . The annual haemorrhage risk is likely 2% to 4% per year for previously diagnosed AVMs[6-8]. Once an AVM ruptures, however, the risk of additional haemorrhage over the first year is increased to 6% to 18%, before returning to 2% to 4%/year[9-11]. Ten percent of patients whose AVM ruptures will die as a result, whereas 20% to 30% will be left with a major disability[10]. The most widely utilized system used to perform a relative surgical risk analysis was reported by Spetzler and Martin[12] in 1986. Their grading system is based on three criteria: AVM size [small (<3 cm), medium (3–6 cm), or large (>6 cm)], pattern of venous drainage (superficial or deep), and neurological eloquence of adjacent brain regions (sensorimotor, language, and visual cortex; hypothalamus and thalamus; internal capsule; brain stem; cerebellar peduncles, and deep cerebellar nuclei are considered eloquent). Points are assigned for each of the aforementioned variables and added for a total score of 1–5 (Grade I and II lesions are generally considered safe surgical lesions with very low incidence of surgically induced neurological deficits, while grade IV and V lesions are frequently accompanied by significant surgically induced neurological deficits. Grade VI lesions are considered inoperable.[12]

MATERIALAND METHODS

Between January2019 –December 2020 twenty patients with AVMs were treated in 18 embolization sessions at our institution. Inclusion criteria were: patients of cerebral arteriovenous malformation with Spetzler Martin grade 1- 3, symptomatic or incidental detected. Arterio- venous fistulas as well as aneurysms accompanying AVMs were excluded from this study. For this prospective study Ethics Committee approval as well as informed patient consent was taken. There were six (30%) females and 14 (70%) males with the age from 12 to 60 years (mean 38.91 years, SD 14.82). Clinical presentation was intracranial hemorrhage in eight (40%), neurological deficits in three (15%), headaches in six (30%), and seizures in seven (35%) patients. AVMs locations were, parieto-occipital region in two (10%) patients, frontal lobe in four (20%) patients, cerebellar hemisphere in three (15%) patients, basal ganglia in one (5%) patient, temporal lobe in two (10%) patients. The size of the AVMs ranged from <3 cm in 12(60%) to >3 cm in 8 (40%) patients. The lesions were fed by post cerebral artery in 7 (35%) out of 20 patients, and by anterior cerebral artery in 6(30%) and middle cerebral arteries in the 5 (25%) remaining ones by anterior inferior cerebellar artery in 2(10%). Based on the Spetzler-Martin score there were four (20%) grade I AVMs, seven (35%) grade II AVMs, and nine (45%) grade III AVM.

TECHNIQUE OF EMBOLIZATION

Digital subtraction angiographies (DSA) performed to evaluate accurately the location of the nidus and the exact anatomy of the feeding arteries and draining veins. Endovascular embolization was the first-line treatment method of all AVMs presented in the current study. All the procedures were done by experienced interventional radiologists, and always after neurosurgical consultations. The patients were treated under general anesthesia. After super selective right transfemoral catheterization of the AVM feeding pedicle, injection of the Onyx-18 was carried out with the intention of reaching the nidus of the AVM only, without occluding the arterial feeders or the draining veins. In cases of AVMs supplied by two or more arterial pedicles staged embolization was performed . During the procedure's heparinized saline was continuously infused into the arterial line. After

embolization, every patient was transferred to the intensive care unit for observation. Patients after uncomplicated treatment were discharged from hospital on postoperative day 1-7.

ANGIOGRAPHIC EVALUATION

A control angiogram was always obtained immediately after the embolization and at minimum follow-up of 4 weeks. The lack of contrast filling within the AVM nidus was classified as a complete occlusion. Patients with incompletely occluded AVMs were offered radiosurgery or surgical therapy. Recanalization was defined as any increase in nidal filling at follow-up. In cases of revascularization following initial complete occlusion of AVMs, the patients were retreated via endovascular approach or were referred to stereotactic radiotherapy. Each time clinical evaluation was recorded on admission to hospital, proper surgical notes were made after endovascular procedure, at the time of discharge from hospital, and during the last angiographic follow-up period.

RESULTS

In the 20 patients analyzed in the study 19 endovascular treatment sessions were performed. The initial complete AVM occlusion was achieved in 09 (45%)cases . The primary incomplete occlusion of the remaining 9 AVMs resulted from difficulties of accessing a nidus through arterial feeding arteries in eight patients (40%) and occurrence of complication. in another one (5%). Two patients (10%) referred for SRS. Angiographic follow-up was achieved in 6 (30%) patients and ranged from 1 to 18 months. Recanalization occurred in three (20%) patients. Of these, one patient was retreated by Onyx embolization therapy without any procedure-related complications and two patients had surgery. Detailed data are presented in Table 1

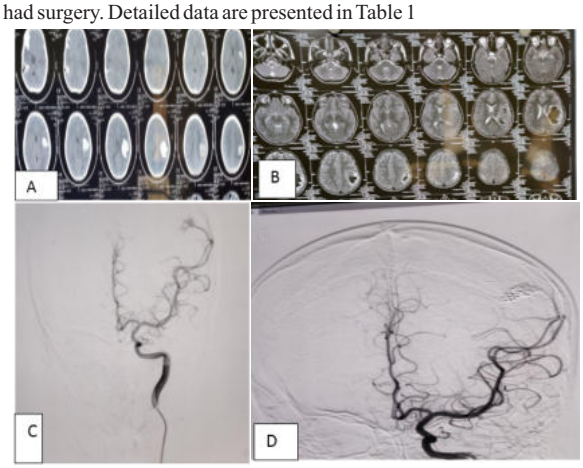

Figure1A NCCT Head Shows Left Temporoparietal ICH
Figure 1B T2WI MRI Brain Shows Left Shows Bleed in Left Temporoparietal Ich.
Figure 1C is Digital Subtraction Angiography Shows Left AVM Feeder from MCA Superior Division.
Figure 1D Is Post ONYX Embolization Of AVM.

Table 1. Angiographic And Demographic Characteristics Of Patients With Arteriovenous Malformations Treated Via Embolization.

Patients			Avm and angiographic outcome							
No	Age/Sex	Clinical presentation	Location	Arterial feeders	Size (cm)	S-M	CLINICALOUTCOME	EMBOLIC AGENTS	COMPLICATION	TREATMENT
1	54/M	ICH	Parietal	PICA	<3	1	Complete	Onyx	None	Embolization
2	14/F	Headache	Frontal	MCA	>3	2	Follow up	Onyx	vasospasm	Embolization
3	38/M	Seizure	Temporal	PCA	<3	3	Follow up	Onyx	None	Embolization
4	59/m	ICH	Occipital	AICA	>3	1	Complete	Onyx	None	Embolization
5	32/F	Seizure	Frontal	PCA, ACA	<3	2	Complete	NBCA	None	Embolization.
6	18/M	Hemiparesis	Cerebellum	MCA	>3	3	Complete	NBCA	None	Embolization
7	30/M	ICH	Frontal-temporal	MCA	<3	1	SRS	Onyx	None	Surgery
8	41/F	Seizure	Frontal	PCA	<3	3	Follow up	Onyx	None	Surgery
9	58/M	Headache	Occipital	SCA, AICA	>3	2	Complete	NBCA	Vasospasm	Surgery
10	28/F	ICH	Corpus callosum	PCA	<3	3	Follow up	Onyx	None	Embolization
11	15/M	Headache	Parieto-occipital	MCA	>3	2	Surgery	Onyx	Vasospasm	Embolization
12	36/M	Headache	Basal ganglia	ACA	>3	3	Complete	Onyx	Temporary deficit	Embolization
13	43/M	ICH +SEIZURE	Temporo-parietal	ACA	<3	2	Complete	Onyx	None	Embolization
14	33/F	Headache	Frontal	PICA	<3	1	Complete	NBCA	None	Embolization
15	40/M	Hemiparesis	Parieto-occipital	PCA	<3	2	Further sessions	NBCA	None	Embolization
16	5O/M	ICH	Frontal	ACA	>3	3	Follow up	Onyx	None	Embolization
17	32/F	Seizure	Temporal	PCA, ACA	<3	3	Complete	Onyx	Temporary deficit	Embolization
18	37/M	ICH	Cerebellum	AICA	<3	2	Complete	Onyx	None	Embolization +surgery
19	46/M	ICH	Parietal	MCA	>3	3	SRS	Onyx	Hemiplegia	Embolization
20	34/M	Seizure	Occipital	PCA	>3	3	Follow up	NBCA	Vasospasm	Embolization

Table 2. Angiographic Outcome /Obliteration rate of AVM.

Fate	N	%
Cured (complete obliteration)	9	45
Follow-up	6	30
Further sessions	1	5
Surgery	2	10
Stereotactic radiosurgery	2	10
Total	20	100

In our study, AVM was completely cured among 45% of the subjects. Follow up and further sessions were required among 30% and 5% of the subjects respectively. Further surgery and stereotactic radiosurgery was required among 10% and 10% of the subjects respectively shown in table 2.

Table 3 . Degree of Obliteration According To Size Of AVM And Postembolization Management.

Degree of Obliteration	Size		Post-Embolization Management (follow-up/radiosurgery/ surgery)
	<3 cm	≥3 cm	
Complete obliteration	8	1	9/0/0
Subtotal	2	5	5/1/1
Partial	2	2	2/1/1
Complete Obliteration Rate	66.67%	12.5%	

Complete obliteration rate was revealed among 66.67% and 12.5% of the subjects having AVM size <3cm and ≥3 cm respectively

COMPLICATION

Immediate clinical outcome was assessed as per Glasgow Outcome Scale score. 15(75%) patients had good recovery (GOS 5), while 5 (25%) patients had GOS score 4.

4 patients (20%) had device induced vasospasm, 2 (10%) patients had temporary neurodeficit which resolved after 2 weeks, 1 patient (5%) had permanent hemiplegia.

DISCUSSION.

Until late 1980's, surgical resection of the cerebral arteriovenous malformation (AVM) was one of the most important subjects in the neurosurgical field. Since the introduction of stereotactic radiosurgery in early 1990's, main stream of AVM treatment was changed to stereotactic radiosurgery due to its low peri-surgical complication rate[13-16]. However, long-term follow-up after stereotactic radiosurgery on AVM resulted in relatively low complete obliteration rate and high treatment-related complication rate[17-21]. According to the long-term results of stereotactic radiosurgery and owing to the modern technical improvement of micro neurosurgery, treatment options for AVM again became controversial. The American Heart Association recommended that surgical extirpation should be considered for the low-graded AVM in the Spetzler-Martin grading and only the lesions in which surgery offers increased risk based on its anatomical feature, stereotactic radiosurgery was recommended[22]. It is also recommended that a combined modality approach with embolization followed by surgery for selected cases of high-graded AVM[23]. Endovascular treatment of patients with Brain AVMs as a monotherapy plays a limited role in managing these lesions [24, 25], which is confirmed by the results of our study. Previous series have shown that the subgroup of patients that may benefit most from this form of therapy is based on morphological characteristics including small size, few supplying pedicles, and accessibility of the nidus [26, 24, 27, 28]. It is also worth mentioning that subtotal occlusion of AVMs achieved by some authors may not be considered therapeutic because it is widely known that it does not confer protection from haemorrhage[26,29, 25] Only total occlusion of the nidus and the arteriovenous shunt constitutes a definitive cure for AVMs [30].

The present non-randomized prospective study comprised of 20 patients diagnosed with brain arterio-venous malformations (BAVM) at clinical OPD and patients for whom endovascular therapy and/or open surgery were preferred as the primary treatment modalities. In the literature, the rate of complete obliteration of AVMs with the use of embolization as one and only therapeutic modality is up to 53.9%, depending on the patient selection, goals of treatment, and operative techniques [31, 27, 30]. The rate of total occlusion achieved by surgical resection and stereotactic radiotherapy is reported to be 57.1-100% and 80%, respectively, although it should be noted that each of the two methods has its own indications and limitations[26]. Also, the AVMs that are most responsive to curative endovascular therapy overlap with those most amenable to surgical excision or radio surgical ablation [31,24, 27]. According to Potts *et al*., patients with contraindications to surgery, those in whom the haemorrhagic risk of radio surgical latency period is too high, or those whose personal preference is embolization are potential candidates for this form of treatment [24].

In our study, 90% of the subjects underwent embolization treatment whereas 1 case each underwent surgery as well combination of embolization and surgery. In our study, AVM was completely cured among 45% of the subjects. Follow up and further sessions were required among 30% and 5%of the subjects. Further surgery and stereotactic radiosurgery was revealed among 10% and 10% of the subjects respectively. Favourable results were found more among subjects having AVM size<3cm as compared to subjects having size ≥3 cm. When impact of embolization was compared according to AVM size, it was found to be statistically significant as $p<0.05$. Complete obliteration rate was revealed among 66.67% and 12.5% of the subjects having AVM size <3cm and ≥3 cm respectively. Complete obliteration was achieved among 75% of the subjects in whom there was only one feeding artery. J.W. PAN et al[33] too revealed similar results.

Mahmouda RN et al[34] in their study found that in the incompletely-cured group, most AVMs were medium-sized or large lesions (3-6 cm or more than 6cm) and most had plexiform nidus.in this group, nine (45%) patients did stereotactic radiosurgery (SRS) two (10%) did surgery and one (5%) was scheduled for further embolization sessions. The obliteration rate is markedly improved in our report compared with previous literature and it based on application of the above-mentioned technology and embolization experience with Onyx[33].

According to GOS scale, good recovery was reported among 75% of the subjects in the present study. Mahmouda RN et al[34] in their study showed that out of 20 patients treated by endovascular embolization, 16(80%) patients had good recovery without neurological deficits at hospital discharge (GOS 5) while four (20%) patients had moderate disability (GOS 4) in the form of hemiparesis. These findings are similar to our study.

In our study; device-induced vasospasm, temporary neurological deficits and hemiplegia was found among 20%, 10% and 5% of the subjects respectively. There was no mortality reported in the present study. Mahmouda RN et al[34] in their study reported that three (15%) patients had complications related to endovascular intervention in the form of arterial device-induced vasospasm. Three (15%) patients had temporary neurological deficits. Two (10%) patient experienced paraesthesia in one side of the body which resolved after 1 day spontaneously. The other (5%) patient had hemiparesis and dysphasia after embolization a parietal AVM. No technique-related mortality was recorded. These results are quite similar to those reported in the previous series of patients with brain AVMs treated with Onyx. The rate of haemorrhagic complications is between 4.0 and 12.2 %, with most series concentrated between 6.0 and 9.0 %. Morbidity is more heterogeneous from one series to another and ranged from 3.5 to 15.5 %. However, in more recent series, including ours, morbidity was between 3.5 and 5.1 %. The mortality rate in BRAVO is slightly higher than that reported in the previous series (0.0 to 3.2%)[31]. Valavanis et al reported 1.5% permanent neurological morbidity rate and 0.4% mortality rate with embolization alone and 2.8% morbidity and 0.2% mortality in the total cure group. They explained morbidity and mortality by pre- or early post-embolization haemorrhage and ischemia. They suggested that immediate surgical removal of the hematoma and neuro-intensive care improve the clinical outcome in these patients[31].AVMs with compressive hematomas, a single-stage treatment with surgery is quite appropriate, suppressing at the same time the brain compression and the bleeding source. In ruptured AVMs without compressive hematoma, the treatment is currently performed a few weeks after the acute phase of bleeding and techniques providing immediate occlusion of the nidus have to be preferred (surgery, embolization or a combination of both). If treatment by a single technique is feasible, it is certainly a good option, but an approach with two techniques will sometimes be necessary[31].Embolization is often the first step employed to obtain a complete occlusion of the nidus or to reduce the size of the nidus for subsequent treatment (surgery or radiosurgery). Embolization is used in two different ways from one centre to another. For some groups, embolization is used and optimized in order to provide the higher possible rate of complete occlusion with embolization alone and to appropriately prepare the incompletely embolized AVMs for further treatment (surgery or radiosurgery). For other groups, embolization is used as a first step treatment to prepare for a second step (surgery or radio surgery).

Our series has several limitations. First, the number of patients included was relatively small. Second, the strategy of treatment, including the fact that embolization was chosen as the first line treatment, is not analysable as the rationale for selecting embolization as the first line treatment option was not requested. Probably the strategy was slightly different from one centre to another, some centre's having as primary objective the complete occlusion of the brain AVM when some others probably just wanted to prepare the patient for surgery or radiosurgery. Third, the final outcome after complementary treatment was not evaluated in our series. Therefore, it was acceptable to evaluate initial results after Onyx embolization. The final outcome as a result of complementary treatment will be evaluated in the future. Fourth, our series is not randomized Nowadays, to occlude AVMs in the mechanism of thrombosis the non-adhesive liquid embolic agent ethylene vinyl alcohol copolymer dissolved in dimethyl sulfoxide, known as Onyx is commonly used during embolization[24,29].The physical characteristics of Onyx compared with previous n-butyl-cyanoacrylate glue allow more prolonged and controlled injections, which has a positive impact on penetration into the nidus [24, 29, 25, 37]. This property, however, pre- disposes to improper reflux of Onyx, which may result in inadvertent occlusion of normal arterial branches or catheter entrapment [38 29, 25]. One of the most important complications of AVM endovascular therapy is haemorrhage, which can be intraoperative and postoperative and may be caused by several factors [40]: AVM bleeding associated with vein

stenosis or occlusion and subsequent active arterial supply to the lesion without adequate venous outflow is one of them [29]. The intracerebral haematoma may sometimes require surgical intervention and the clinical sequelae can be fatal [29, 39]. The haematoma as well as brain edema may also result from the so-called normal perfusion pressure breakthrough syndrome (NPPBS) in which in- creased blood flow towards the normal brain vessels after AVM embolization is observed [24, 29, 40]. To lower the risk of this negative phenomenon, staged embolization of AVMs is considered, in order to ensure the recovery of the brain between procedures, especially in the case of large AVMs [29,40].

In 18 patients demonstrated in this study we performed 19 embolization sessions, and none of the patients developed symptoms of NPPBS. Although Onyx embolization has the potential to be relatively safe, periprocedural complications should not be neglected when selecting this form of treatment embolization is still debatable, and although it is sup- posed to lower periprocedural haemorrhagic complications, leaving a residual shunt after the procedure always harbours a certain risk of subsequent AVM bleeding, especially when the venous outlet is occluded [31,40]. Taking the above into account, in our opinion the need of multiple embolization sessions is a serious limitation of endovascular treatment of AVMs as a monotherapy.

CONCLUSION.

Embolization of intracranial arteriovenous malformations plays a limited role as a sole therapeutic modality even in terms of small lesions with two or fewer arterial feeders. Associated complications are not trivial and should be considered when choosing this form of treatment. ONYX and NBCA is suitable for embolization of AVMs because of its diffuse controllable properties. As per our study observation and results, we suggest clinical follow-up after complete obliteration, additional radiosurgery or angiographic follow- up after subtotal obliteration and additional surgery after partially obliteration. More cases with long-term follow-up are needed to evaluate the long-term prognosis of postembolization management of brain AVM patients.

REFERENCES

1 Stapf C, Mast H, Sciacca RR, Berenstein A, Nelson PK, Gobin YP, Pile-Spellman J, Mohr JP. The New York Islands AVM Study: design, study progress, and initial results. Stroke. 2003;34(5):e29-33.
2 Mahmouda RN, Habibb MA, Hamdanc AR, Abdellatifd AA. Endovascular Management of Brain Arteriovenous Malformation: Safety And Efficacy. 1999;4: 18-24.
3 Tournade A, Riquelme C. Advances in the endovascular treatment of intracranial arteriovenous malformations. Dialogues Clin Neurosci 2000;2: 315-320.
4 Fleetwood IG, Steinberg GK. Arteriovenous malformations. Lancet 2002;359: 863-873.
5 Smith JL, Garg B. Treatment of arteriovenous malformations of the brain. Curr Neurol Neurosci Rep 2002;2: 44-49.
6 Crawford PM, West CR, Chadwick DW, Shaw MD. Arteriovenous malformations of the brain: natural history in unoperated patients. J Neurol Neurosurg Psychiatry. 1986;49(1):1-10.
7 Mast H, Young WL, Koennecke HC, et al. Risk of spontaneous hemorrhage after diagnosis of cerebral arteriovenous malformation. Lancet. 1997;350(9084): 1065-1068.
8 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.
9 Graf CJ, Perret GE, Torner JC. Bleeding from cerebral arteriovenous malformations as part of their natural history. J Neurosurg.1983;58(3):331-337.
10 Itoyama Y, Uemura S, Ushio Y, et al. Natural course of unoperated intracranial arteriovenous malformations: study of 50 cases. J Neurosurg. 1989;71(6):805-809.
11 Jane JA, Kassell NF, Torner JC, Winn HR. The natural history of aneurysms and arteriovenous malformations. J Neurosurg. 1985;62(3):321-323.
12 Ap Simon HT, Reef H, Phadke RV, Popovic EA: A population-based study of brain arteriovenous malformation: long-term treatment outcomes. Stroke 2002; 33: 2794–2800
13 Lunsford LD, Flickinger J, Coffey RJ: Stereotactic gamma knife radiosurgery. Initial North American experience in 207 patients. Arch Neurol 1990;47: 169-175.
14 Lunsford LD, Kondziolka D et Al: Stereotactic radiosurgery for arteriovenous malformations of the brain. J Neurosurg. 1991;75: 512-524.
15 Steiner L, Lindquist C et Al: Clinical outcome of radiosurgery for cerebral arteriovenous malformations. J Neurosurg. 1992;77: 1-8.
16 Friedman WA, Bova FJ: Linear accelerator radiosurgery for arteriovenous malformations. J Neurosurg 1992;77: 832-41.
17 Karlsson B, Lindquist C, Steiner L: Effect of Gamma Knife surgery on the risk of rupture prior to AVM obliteration. Minim Invasive Neurosurg 1996;39: 21-27.
18 Friedman WA, Bova FJ, Mendenhall WM: Linear accelerator radiosurgery for arteriovenous malformations: the relationship of size to outcome. J Neurosurg 1995;82: 180-89.
19 Pollock BE, Flickinger JC: Hemorrhage risk after stereotactic radiosurgery of cerebral arteriovenous malformations. Neurosurgery 1996;38: 652-59.
20 Yamamoto M, Jimbo M. Gamma knife radiosurgery for arteriovenous malformations: long-term follow-up results focusing on complications occurring more than 5 years after irradiation. Neurosurgery 1996;38: 906-914.
21 Yamamoto Y, Coffey RJ et Al: Interim report on the radio surgical treatment of cerebral arteriovenous malformations. The influence of size, dose, time, and technical factors on obliteration rate. J Neurosurg 1995;83: 832- 837.
22 Ogilvy CS, Stieg PE: Special Writing Group of the Stroke Council, American Stroke Association: AHA Scientific Statement: Recommendations for the management of intracranial arteriovenous malformations: a statement for healthcare professionals from a special writing group of the Stroke Council, American Stroke Association. Stroke. 2001;32: 1458-1471.
23 Nagashima H, Hongo K, Kobayashi S, et al. Embolization of Arteriovenous Malformation: Efficacy and Safety of Preoperative Embolization Followed by Surgical Resection of AVM. Interventional Neuroradiology. 2004;10(2_suppl):54-8.
24 Potts MB, Zumofen DW, Raz E, et al. Curing arteriovenous malfor- mations using embolization. Neurosurg Focus 2014; 37: E19.
25 Xu F, Ni W, Liao Y, et al. Onyx embolization for the treatment of brain arteriovenous malformations. Acta Neurochir (Wien) 2011; 153: 869-878.
26 Ogilvy CS, Stieg PE, Awad I, et al. AHA Scientific Statement: Recom- mendations for the management of intracranial arteriovenous mal- formations: a statement for healthcare professionals from a special writing group of the Stroke Council, American Stroke Association. Stroke 2001; 32: 1458-1471.
27 Yu SCH, Chan MSY, Lam JMK, et al. Complete obliteration of in- tracranial arteriovenous malformation with endovascular cyanoacr- ylate embolization: initial success and rate of permanent cure. AJNR Am J Neuroradiol 2004; 25: 1139-1143.
28 Sahlein DH, Mora P, Becske T, et al. Nidal embolization of brain arteriovenous malformations: rates of cure, partial embolization, and clinical outcome. J Neurosurg 2012; 117: 65-77.
29 Katsaridis V, Papagiannaki C, Aimar E. Curative embolization of cerebral arteriovenous malformations (AVMs) with Onyx in 101 patients. Neuroradiology 2008; 50: 589-597.
30 Fournier D, TerBrugge KG, Willinsky R et al. Endovascular treat- ment of intracerebral arteriovenous malformations: experience in 49 cases. J Neurosurg 1991; 75: 228-233.
31 Pierot L, Cognard C, Herbreteau D, et al. Endovascular treatment of brain arteriovenous malformations using a liquid embolic agent: results of a prospective, multicenter study (BRAVO). European radiology. 2013;23(10):2838-45.
32 Bruno CA, Meyers PM. Endovascular Management of Arterio- venous Malformations of the Brain. Interv Neurol 2013; 1: 109-123.
33 Pan JW, Zhou HJ, Zhan RY, et al. Supratentorial brain AVM embolization with Onyx-18 and post-embolization management: a single-center experience. Interventional Neuroradiology. 2009;15(3):275-82.
34 Mahmouda RN, Habibb MA, Hamdanc AR, Abdellatifd AA. Endovascular Management Of Brain Arteriovenous Malformation: Safety And Efficacy. 1999;4: 18-24.
35 Abdulrauf SI, Malik GM, Awad IA. Spontaneous angiographic obliteration of cerebral arteriovenous malformations. Neurosurgery 1999; 44: 280-287; discussion 287-288.
36 Patel MC, Hodgson TJ, Kemeny AA, et al. Spontaneous obliteration of pial arteriovenous malformations: a review of 27 cases. AJNR Am J Neuroradiol 2001; 22: 531-536.
37 Berthelsen B, Löfgren J, Svendsen P. Embolization of cerebral arte- riovenous malformations with bucrylate. Experience in a first series of 29 patients. Acta Radiol 1990; 31: 13-21.
38 Arteriovenous Malformations. In: Harrigan MR, Deveikis JP, eds. Handbook of Cerebrovascular Disease and Neurointerventional Tech- nique [Internet]. Humana Press, Totowa 2009 [cited 2016 Mar 14]. Available from: http://link.springer.com/10.1007/978-1-60327-125-127.
39 Mounayer C, Hammami N, Piotin M et al. Nidal embolization of brain arteriovenous malformations using Onyx in 94 patients. AJNR Am J Neuroradiol 2007; 28: 518-523.
40 Spetzler RF, Martin NA, Carter LP et al. Surgical management of large AVM's by staged embolization and operative excision. J Neu- rosurg 1987; 67: 17-28.