

Endovascular Treatment of Brain Arteriovenous Malformations in Siriraj Hospital

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Abstract : Objective : To present our experience on endovascular embolization by using N-butyl cyanoacrylate and to demonstrate that this is an effective method in the treatment of brain arteriovenous malformations.

Materials and methods : From 1992 to 1998, 92 patients with brain arteriovenous malformations who having had endovascular embolization as primary treatment was retrospectively reviewed.

Results : 8 patients (8.7%) had completely cure by embolization alone without neurological complications. 17 patients (18.5%) were cured after combined treatment, 9 with surgical removal and the other 8 with radiosurgery. The neurological complications occurred in 17 patients (18.5%) with 3 deaths and 1 vegetative stage.

Conclusion : The endovascular embolization can cure the arteriovenous malformations that smaller than 3 cm, no matter what its location, and it is effective to reduce the nidus volume of the larger lesions, making them suitable for surgical resection or radiosurgery as to achieve the goal of complete eradication of the malformations.

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เรื่องย่อ : การรักษาโรคความผิดปกติของหลอดเลือดสมองโดยวิธีผ่านทางเส้นเลือดในโรงพยาบาลศิริราช

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จุดประสงค์ของบทความนี้เพื่อนำเสนอวิธีการรักษาโรคเส้นเลือดที่ผิดปกติของสมองโดยการใส่สายสวนผ่านเข้าทางหลอดเลือดแดงไปจนถึงตำแหน่งที่ผิดปกตินั้น ๆ แล้วทำการฉีดสารอุด ซึ่งมีชื่อทางเคมีว่า N-butyl cyanoacrylate หรืออาจเรียกว่า "กาว" และแสดงให้เห็นว่าวิธีนี้เป็นวิธีที่ได้ผลดีอีกทางหนึ่งของการรักษา

โดยได้ทำการศึกษาย้อนหลังของผู้ป่วยโรคนี้ที่ถูกส่งมาเพื่อการรักษาโดยวิธีดังกล่าว ณ โรงพยาบาลศิริราช ตั้งแต่ พ.ศ. 2535 ถึง พ.ศ. 2541 ทั้งหมด 92 ราย ผู้ป่วย 8 ราย (ร้อยละ 8.7) หายขาดโดยวิธีการนี้โดยลำพัง ผู้ป่วย 17 ราย (ร้อยละ 18.5) หายเมื่อใช้การรักษาร่วมกับวิธีผ่าตัด (9 ราย) หรือฉายรังสีเฉพาะที่ (8 ราย) โรคแทรกซ้อนทางระบบประสาทที่เกิดจากการรักษา พบ 17 ราย (ร้อยละ 18.5) ในจำนวนนี้ตาย 3 ราย และไม่มีการตอบสนอง 1 ราย

วิธีการรักษาทางหลอดเลือดนี้สามารถรักษาโรคเส้นเลือดที่ผิดปกติที่มีขนาดเส้นผ่าศูนย์กลางไม่เกิน 3 เซนติเมตรให้หายขาดได้โดยไม่ขึ้นกับตำแหน่งของโรค ส่วนในรอยโรคที่มีขนาดใหญ่ก็จะเป็นผลดีในแง่ที่ช่วยลดขนาดก่อนที่จะใช้การรักษาอื่นในวิธีอื่น เช่น การผ่าตัดหรือการฉายรังสีเพื่อทำให้การรักษาหรือการกำจัดรอยโรคมีประสิทธิภาพอย่างสมบูรณ์

INTRODUCTION

The objective of treatment of cerebral arteriovenous malformations (AVMs) is to exclude the lesion from the circulation permanently and preservation of neurological function.¹ Currently, Several treatment options are available, including surgical resection, endovascular embolization, radiosurgery and multimodal treatment. The purpose of this paper is to present our experience in this respect regarding endovascular embolization and to review the recent literature as to determine the value of this technique.

MATERIALS AND METHODS

From January 1992 to December 1998, 92 patients (65 males, 27 females) with cerebral AVMs were referred to Department of Radiology, Siriraj hospital for endovascular embolization. The age distributions, presenting symptoms and locations of AVMs were summarized in table 1-3

All patients had either computerized tomography scan or magnetic resonance imaging scan for diagnosis of the AVMs and probable associated diseases. Complete 4 vessels cerebral angiogram was performed prior to endovas-

cular treatment for angioarchitecture assessment and treatment planing. The AVMs were classified according to the Spetzler and Martin grading (table 4) into 5 groups.¹³ The distributions of these groups were given in the table 5.

All patients were initial proposed endovascular embolization to complete obliteration of the AVM nidus by using the permanent embolic agent N-butyl cyano-acrylate (Histoacryl), so called glue, but except 2 patients in their first session of treatment in our very beginning period that polyvinyl alcohol (PVA or Ivalon) were used. Our planing was to embolize the major feeders and the intranidal aneurysm (figure 2 and 4), if it was identified, first. Several sessions of treatment might be needed in high grade or large AVMs. Although the AVM nidus was not completely occluded, but if further embolization was judged impossible or too risky, the treatment was discontinued. Combined surgery or radiosurgery would then be considered in some cases.

The embolization procedure was performed with the patient under monitoring of an anesthesiologist either by general anesthesia or sedation. An introducer sheath with continuous saline infusion was inserted into the femoral artery. A 5 French or 6 French (F) guiding catheter with continuous heparinized saline was positioned in the access artery (internal carotid or vertebral artery) about the cranial base to facilitate advancement of the microcatheter. Systemic heparinization (5,000 unit bolus) was then intravenous administrated. The microcatheter which was usually a flow-directed standard Magic catheter (Balt, France) 1.8 F or 1.5 F was advanced into a feeding artery of the AVM until its tip reached

or closed to the nidus. The glue and lipiodol mixture were prepared with different concentrations on each time depended on the distance between the catheter tip and the nidus or it was intranidal fistula. The glue mixture was injected after flushing the microcatheter by 5% dextrose solution under filming program at 2 frames per second or 3 frames per second in fistula. The injection was suddenly paused if the glue entered a draining vein or refluxed proximal to the catheter tip. The injecting syringe was aspirated during signal for the assistant to pull out the guide catheter-microcatheter system. Repeated procedure was decided individually in each case. The control angiogram was done to access if there was any change from the baseline. If there was no immediate complication, the patient was discharged on the following day and schedule for repeat treatment in 3-6 months, if it was needed.

RESULTS

The embolization was performed in 92 patients with number of sessions and percentage of AVM nidus reduction shown in table 6 and 7.

Several sessions of embolizations were needed in half of the patients as shown in table 6 which was as many as 9 sessions in 1 patient because of large lesion and finally terminate embolization was decided due to risk of complication is higher than benefit. The patients who had only one session of embolization were including who had complete AVM occlusion, who were waiting for complete treatment and who lost to follow up after initial treatment.

Table 1. Age distribution

Age (years)	No of patients	% of patients
1-10*	2	2.2
11-20	12	13
21-30	33	35.9
31-40	28	30.4
41-50	14	15.2
51-60	2	2.2
>60**	1	1.1
Total	92	100

*The youngest was 6 years

**The oldest was 68 years

Table 2. Clinical presentations

Presentations	% of patients
Headache	57%
Seizure	54%
Hemorrhage	36%
*Neurological deficits	18%
Dizziness	2%
Ataxia	3%
Nystagmus	2%
Dysarthria	2%

*Including motor weakness, facial palsy and visual field defect.

Table 3. Locations of AVMs

Location	No of patients	% of patients
Frontal lobe	21	22.9
Parietal lobe	12	13
Temporal lobe	12	13
Occipital lobe	3	3.3
Temporoparietal lobe	5	5.4
Temporooccipital lobe	3	3.3
Frontoparietal lobe	3	3.3
Parietooccipital lobe	2	2.2
Corpus callosum and cingulate gyrus	11	12
Basal ganglia and Thalamus	6	6.5
Midbrain	2	2.2
Posterior fossa	6	6.5
Total	92	100

Table 4. Spetzler & Martin grading of AVMs

Grade feature	Point assigned
Size of AVM	
Small (< 3 cm)	1
Medium (3-6 cm)	2
Large (> 6 cm)	3
Eloquence of adjacent brain	
Non-eloquent	0
Eloquent	1
Pattern of venous drainage	
Superficial only	0
Deep	1

Table 5. AVM grade

Grade	No of patients	% of patients
1	17	18.5
2	23	25
3	34	37
4	16	17.3
5	2	2.2
Total	92	100

Eight patients were considered to be cure (complete occlusion of the AVM nidus) by embolization alone without any neurological complications (figure 1 and 2). The follow up angiogram at 1 year revealed complete obliteration of the AVM nidus. All of these AVMs were small (less than 3 cm) with few arterial feeders. 6/8 patients had grade 1, (figure 1) and the others had grade 2 (figure 2) and grade 3 respectively.

Seventeen patients had combined treatment after incomplete eradication of the AVM nidus, 9 patients had surgery and 8 patients had radiosurgery (figure 3 and 4). Two of them had neurological complications afterward.

Twenty five patients have been in the follow up program, 13/25 had neurological deficits, 6/25 had finished treatment with incomplete embolization (figure 5) and the other 6/25 are waiting for next embolization. Unfortunately, 38 patients were lost to follow up with incomplete embolization.

The complications occurred in 27 patients (29.3%) with 17 patients (18.5%) had major complications and 10 patients (10.8%) had

minor complications as shown in table 8.

Intracranial hemorrhage was the serious complication including intraventricular, intracerebral, and subarachnoid hemorrhage. These were found in 10 patients (11%), 1/10 had subarachnoid bleeding during the procedure because of rupture of the feeding artery. An extravasation was observed during an attempt to navigate in the right anterior temporal artery. The ruptured artery was glued right away. The patient had seizure for 24 hours which was controllable and recovered on the following day without any neurological deficits. Another 1/10 case had immediate intraventricular hemorrhage within 24 hours after embolization of the splenic portion of corpus callosum and finally died. The other 8/10 patients had delayed intracerebral hemorrhage within months, 5 patients had clinical improvement after conservative treatment and 2 patients were dead, one patient was in vegetative stage.

Hemiplegia had occurred in 2 patients, one had immediate right hemiplegia after embolization of the perforator artery of the midbrain AVM. The other one with AVM in right motor area

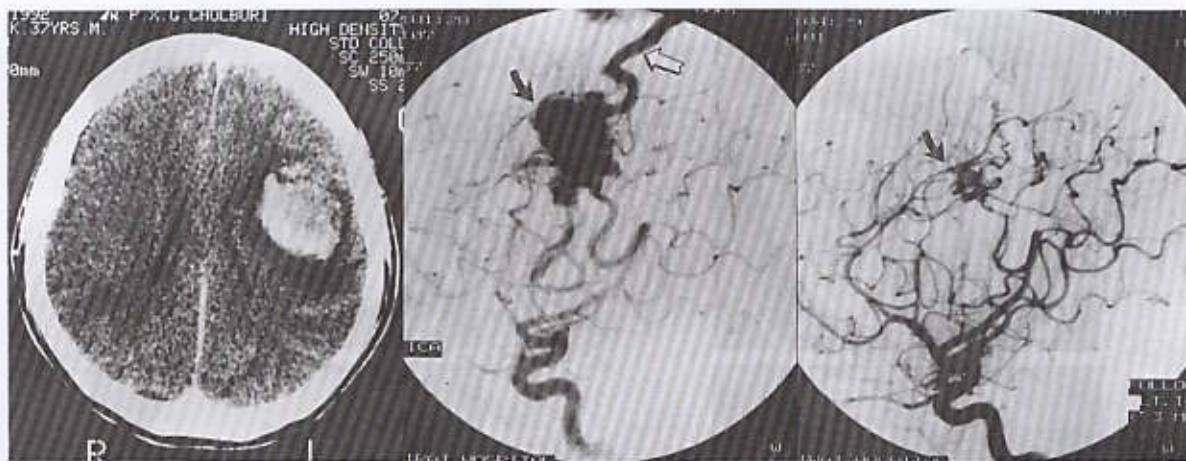


Figure 1. A 37-year-old man presented with first episode of seizure. (A) Axial nonenhanced CT scan revealed hemorrhage in the left frontal lobe. (B) Anteroposterior and (C) lateral views of left internal carotid angiogram demonstrated a left frontal AVM (black arrow) with a single cortical venous drainage (open arrow), to be classified as grade 1 according to Spetzler' classification. (D) and (E) Left internal carotid angiogram which was performed three months after embolization showed complete occlusion of the AVM nidus. This patient was cured.



Figure 2. (A) Anteroposterior and (B) lateral view of left internal carotid angiogram of a 23-year-old woman who had intraventricular hemorrhage (not shown) demonstrated a posterior corpus callosum AVM with intranidal aneurysms (arrow) feeding by pericallosal artery. (C) Post embolization angiogram in lateral view revealed complete obliteration of the aneurysms and the nidus (arrow) and preservation of the distal branches of the pericallosal artery. (D) Glu cast was shown in the lateral scout view. (E) and (F) One year follow up-angiogram showed permanent complete eradication of AVM.

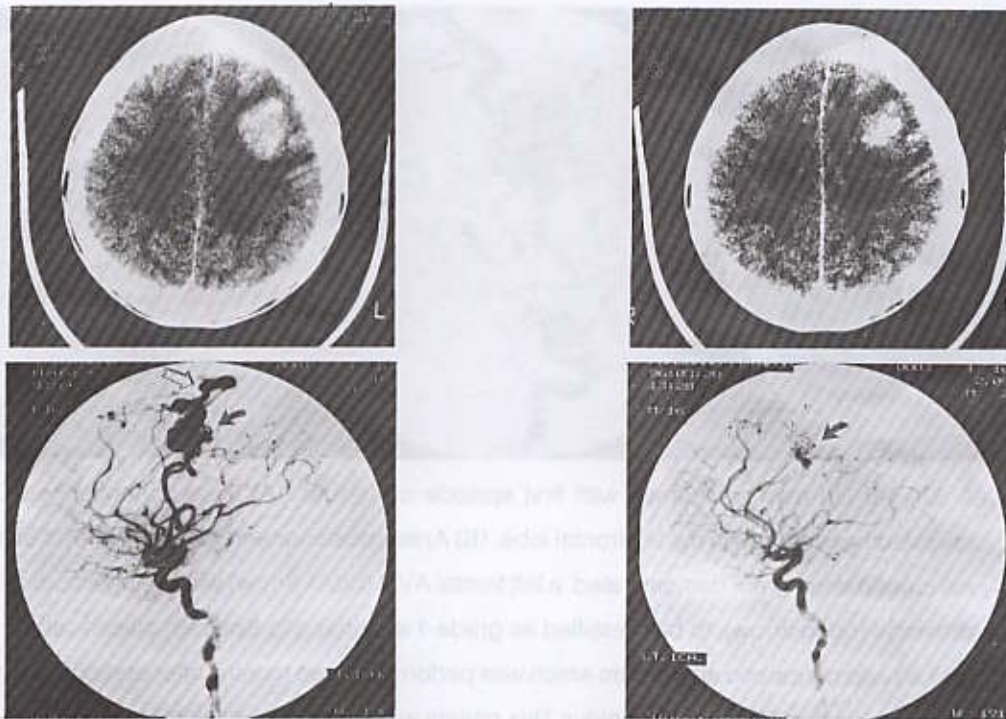


Figure 3. A 19-year-old man who had seizure and weakness of right upper extremity. (A) Axial noncontrast and (B) contrast CT scans showed a hematoma at middle frontal to pre-rolandic area with central enhancement of the nidus. (C) Anteroposterior and (D) lateral views of left internal carotid angiogram revealed a frontal AVM (black arrow) corresponding to the area of hematoma supplied by prerolandic branch of the middle cerebral artery with early cortical venous drainages (open arrow). (E) and (F) Post embolization angiogram demonstrated approximate 90% reduction of the AVM nidus (arrow) with disappearance of the early draining veins. The patient was advised for further radiosurgery to complete treatment.

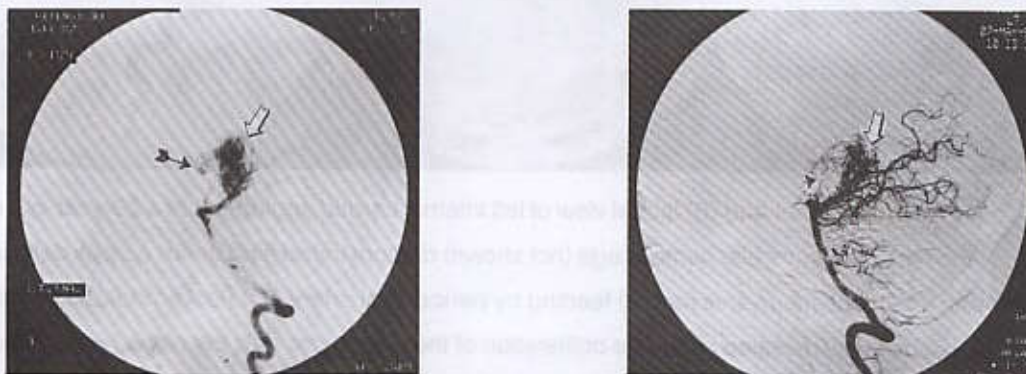


Figure 4. A 17-year-old female with intraventricular hemorrhage and right hemiparesis. (A) Lateral view of vertebral angiogram revealed a choroidal AVM (open arrow) with an intranidal aneurysm (black arrow) which was seen from the thalamoperforating artery. (B) Postembolization angiogram in the same projection showed disappearance of the aneurysm with preserve the parent artery (arrow head). For the residual AVM (open arrow), the patient was sent to radiosurgery. The hemiparesis was significantly improved up to grade 4 after finished embolization (8 sessions).

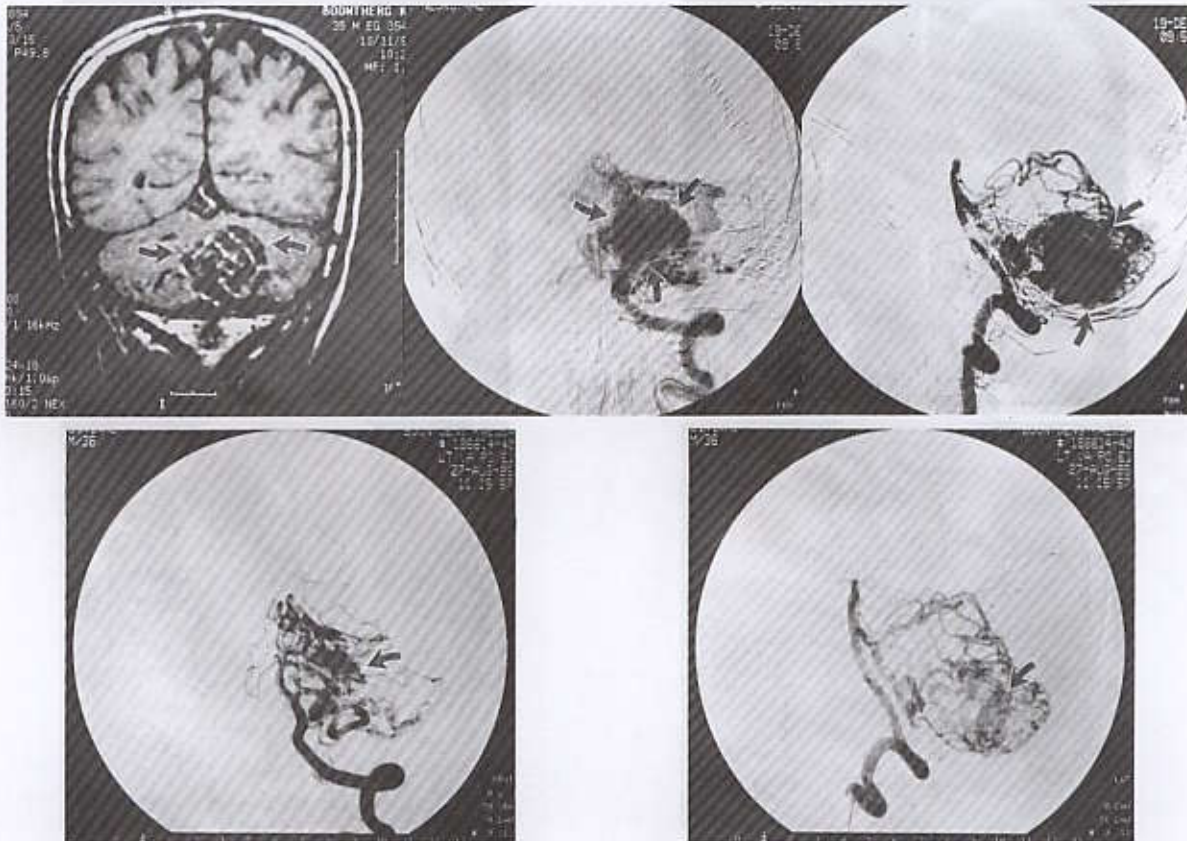


Figure 5. A 35-year-old man presented with dizziness and cerebellar sign positive on physical examination. (A) Coronal view of T1W magnetic resonance imaging revealed an AVM involving left cerebellum and vermis (arrows). (B) Anteroposterior and (C) lateral views of left vertebral angiogram demonstrated that the AVM (arrows) was supplied from all cerebellar arteries, the major feeder was anterior inferior cerebellar artery which took place of the posterior inferior cerebellar artery. (D) and (E) Postembolization angiogram of the same projection as (B) and (C) showed approximate 80% reduction of the size of AVM. The patient symptom was significantly improved. Further embolization was considered to be risky.

developed grade 3 motor weakness of left upper limb after the third embolization.

Homonymous hemianopia type of visual field defect had happened in 5 patients because of refluxed glue into normal territory of calcarine artery, in those AVMs situated at occipital lobe.

Pulmonary emboli was observed in 2 patients of our beginning time by the use of polyvinyl alcohol as embolic agent. However, the patients were free of symptom.

Summary of the patients' outcome were given in table 9.

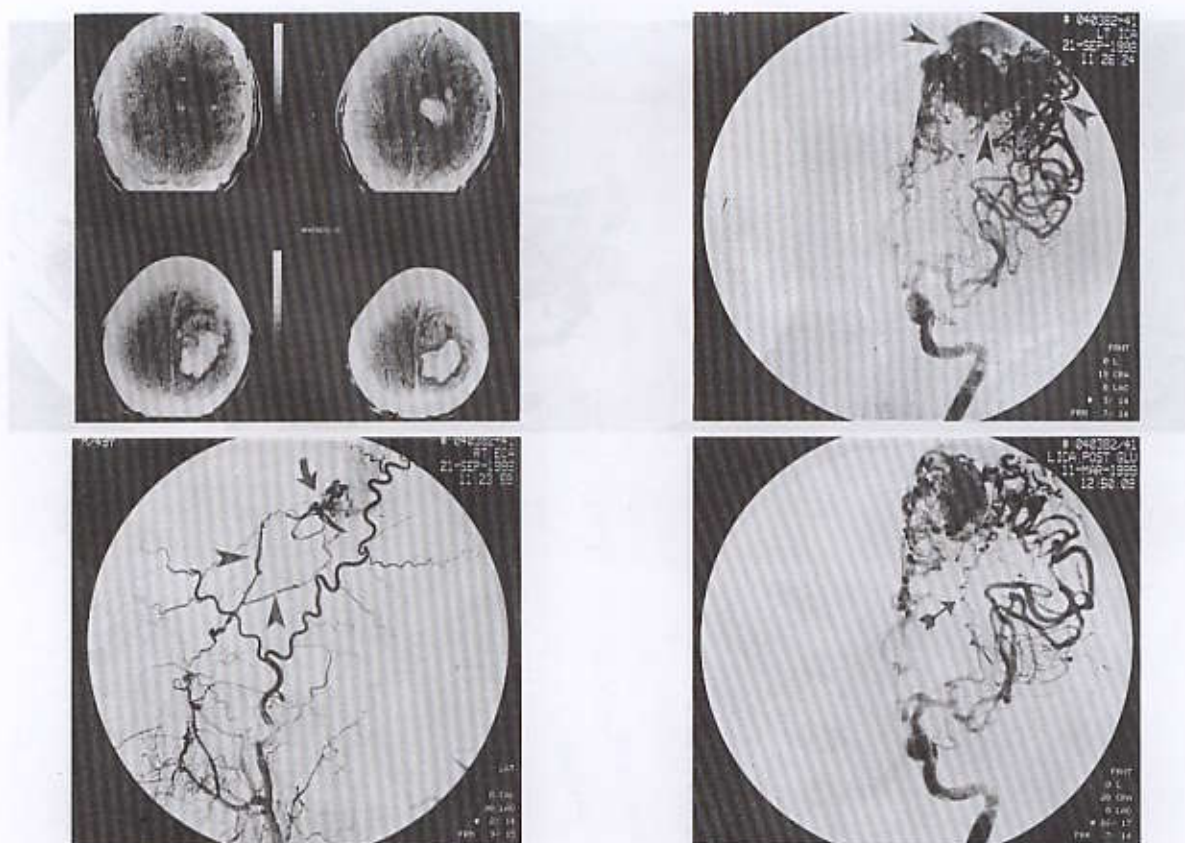


Figure 6. A 49-year-old man who had sudden loss of consciousness and right hemiplegia, the patient was coma on admission. (A) Noncontrast CT scan revealed a large hematoma at high frontoparietal lobes with marked mass effect. The spots of calcifications was also noted anteriorly. (B) Frontal and (C) lateral views of the left internal carotid angiogram after surgical removal of hematoma exhibited a large AVM (arrows) in that area feeding by both anterior and middle cerebral artery circulations. (D) Feeders from left anterior cerebral artery was more clearly seen from frontal projection of the right internal carotid angiogram, passing via the anterior communicating artery. (E) Dural supply of the AVM (arrow) from the middle meningeal artery (arrow heads) was also noted from the lateral view of left external carotid angiogram. (F) and (G) Left internal carotid angiogram which was performed after 3 sessions of embolization demonstrated significant reduction of the nidus, however, it was incomplete. The lenticulostriate artery (arrow in F) was impossible to catheterize. The patient was recovered and full alert. The motor power of right extremities was grade 3.

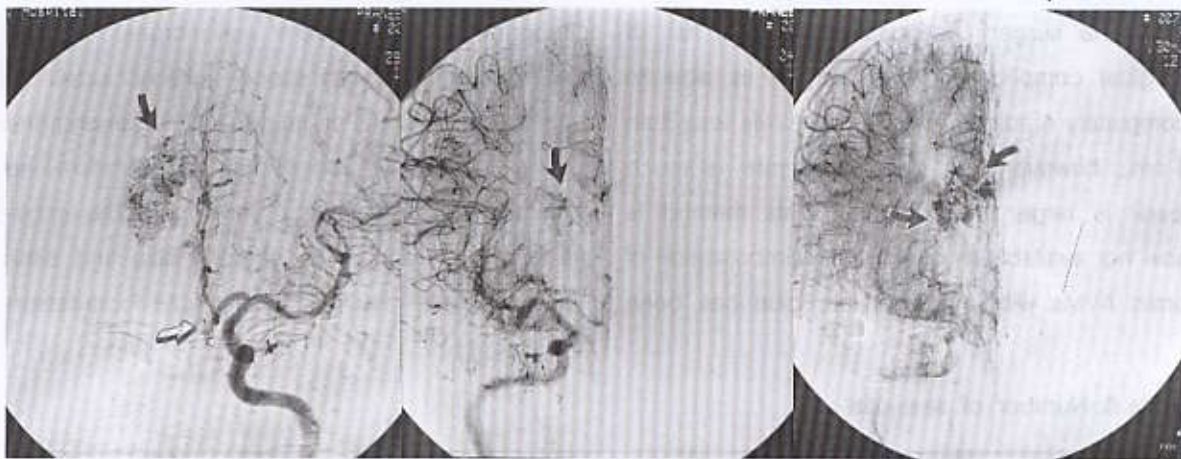


Figure 7. A right frontal AVM of a 29-year-old female who had seizure but never bled was well demonstrated in (A) anteroposterior and (B) lateral views of left internal carotid angiogram (black arrow) being fed by right anterior cerebral artery which was opacified via the anterior communicating artery (open arrow). (C) and (D) Immediate post embolization angiogram revealed nearly complete occlusion of the nidus (arrow). (E) and (F) One year follow up angiogram showed revascularization of the nidus. The patient could discontinued anticonvulsive drug and free of symptom, and because of no angiographic risk of bleeding, further treatment was not offered, the patient was suggested to have clinical follow up once a year.

DISCUSSION

Seizure and headache are common presenting symptoms of the cerebral AVMs. The hemorrhagic events, including intraparenchyma and intraventricular hemorrhages, are the serious complications in the natural history of the disease.^{1,4,9} The deep seated lesions and posterior fossa AVMs are more likely to bleed and have higher risk of rebleeding.^{8,9,12,15} Intracranial aneurysm and central venous drainage are found to correlate with hemorrhage.^{9,10,15} The overall lifetime of hemorrhage is 40%, the annual rate of hemorrhage is from 2-4% and the mean interval between two episodes of bleeding is 7-8 years. The annual rate of morbidity and mortality is 2-3%

and 1% respectively, for both ruptured and nonruptured AVMs.⁴ The risk of untreated lesions is a cumulative 1% mortality and 1.7% morbidity per year.¹

Currently, several treatment options for cerebral AVMs are available, including surgical resection, embolization, radiosurgery and multiple modality methods. For surgical resection of small and medium AVMs, the mortality rate is about 10%.^{4,11} In Siriraj hospital series, the mortality rate is about 6% with 133 cases (unpublished data). The large malformation carries a higher risk of hemodynamic complications during and after surgery. The rate of serious morbidity may reach as high as 50%.^{4,11} The deep seated AVM is more

limited to surgery because of potential risk of surgical complication. Radiosurgery is able to completely eradicate most small AVMs less than 3 cm., however, the obliteration rate is much lower in larger malformation.⁴ This method is now not available in our institute. Embolization of brain AVMs with cyanoacrylate glue has been

reported to have several advantages.^{1,2,3,14} The AVMs that has been successfully occluded by embolization alone is about 11.2% among the lower frequencies reported in the literature, no matter what its location.^{1,14} In large AVMs, embolization can reduce the effective size and diminish the flow, making them suitable for radiosur-

Table 6. Number of sessions

Number of sessions	No of patients	% of patients
1	46	50
2	18	20
3	9	9.7
4	9	9.7
5	6	6.4
6	1	1.1
7	1	1.1
8	1	1.1
9	1	1.1
Total	92	100

Table 7. Reduction of AVM nidus

Estimated reduction (%)	No of patients	% of patients
>20%	10	10.9
21-40%	17	18.4
41-60%	23	25
61-80%	27	29.3
81-90%	7	7.6
cure	8	8.7
Total	92	100

Table 8. Complications of embolization

	Patients
Major	
Intracranial hemorrhage	10
Hemiplegia	2
Visual field defect	5
Total	17 (18.5%)
Minor	
Transient motor deficit	5
Transient dysarthria	2
Pulmonary emboli	2
Urticaria	1
Total	10 (10.8%)

Table 9. Patients' outcome

Outcome	No of patients	% of patients
Cure by embolization alone	8	8.7
Combined with radiosurgery	8	8.7
Combined with surgery	9	9.8
Finished with incomplete embolization	6	6.5
Waiting for complete treatment	6	6.5
Neurological deficits (including hemiplegia, Visual field defect)	13	14
Vegetative stage	1	1.1
Death	3	3.2
Lost to follow up	38	41.3
Total	92	100

gery or reduced surgical morbidity.^{3,4} The complications of embolization are minor morbidity of 10%. Major morbidity of 8% and mortality of 1-1.6%.^{1,4,7}

In this presentation, 8 patients (8.7 %) were completely cured by embolization alone. They had characteristics of small size malformation less than 3 cm with few arterial feeders and draining veins with complete glue casting in the nidus as the technical result (figure 1 and 2). With a good embolization resulting in complete obliteration of the malformation without residual, recanalization does not occur.^{1,6}

On the other hand, in large AVM with multiple pedicles and circulations, the chance of complete obliteration is usually not possible (figure 6). Furthermore, by technique, incomplete occlusion of the AVM is also resulted from inadequate casting of glue in the nidus. This may cause small non-occluded vessels to slowly recruit adjacent collateral circulation (angiogenesis) resulting to revascularization or recurrence.^{1,6} The reconstitution of the nidus has been correlated with the degree of obliteration of the AVM nidus and with the proximity of deposition of the embolic material.⁸ As shown in figure 7, there was incomplete deposition of glue within the nidus but only small residual was observed, on one year follow up, the residual nidus was increased in size. So, if endovascular treatment is decided, embolization has to be as perfect as possible to occlude the nidus totally and those pedicles supplying intranidal aneurysms should be embolized first to potentially reduce the risk of hemorrhage. In those cases that complete embolization is unable to achieve, combined treat-

ment with radiosurgery or surgical resection would be considered to complete cure. However, there has been many reports that partial embolization may be beneficial to prevent breakthrough bleeding and improve regional cerebral blood flow in areas surrounding the AVM.^{1,10}

The neurological complications of embolization are summarized in two categories, hemorrhage and ischemic insults. They are related to procedural technique which result to venous drainage occlusion, arterial rupture or normal brain artery embolization. The risk of embolization increases in the higher grade AVM because it requires many embolization sessions.

Occlusion of the venous drainage may occur if the glue is delivered too fast and it passes through the nidus to enter the draining veins or venous sinus. With continued inflow into the malformation but impaired venous outflow, risk of rupture may increase.⁵ This may cause an immediate or delayed hemorrhagic complication. This happened in our 9 patients resulting to intracerebral and intraventricular hemorrhage with 3 deaths.

Rupture of the feeding artery is the complication that happens during the procedure. We had experience in one case. During catheterization, the tip of the guidewire extended beyond the tip of microcatheter, resulting to subarachnoid hemorrhage, however, the ruptured artery was glued immediately.

Normal brain artery embolization which results to infarction, happens if there is reflux of glue proximal to the tip of the microcatheter into the normal branches. But sometimes the feeding pedicle also nourishes normal parenchyma in

vicinity of the AVM,³ the patient may have neurological complication, even it is good glue injection. We had 7 patients suffering from this kind of complication, 5 of them had partial visual field defect due to embolize normal territory of the calcarine artery in those occipital AVMs. In one case who had midbrain AVM, although no reflux of glue but the patient got immediate hemiplegia after embolization of feeding pedicle from perforator artery.

To our data, the cure rate by embolization alone was 8.7% and the overall permanent neurological complication rate approached 18.4% with 3.2% mortality rate. Our results was not as good as those mentioned in the literatures, but we had the lost follow up cases as high as 41%, in those, complete treatment might be achieved. For the time being, the complication rate tends to decrease as our procedural experience is increase and the endovascular technique is more advanced.

CONCLUSION

Management of brain AVMs requires neurological teamwork with multidisciplinary ap-

proach to make the best strategy of treatment and to maximize the cure rate. The risk to the patients from treatment must be weighted against the benefit they may receive from such treatment. The morbidity and mortality rates from any modality of treatment should not be exceed than those found in the natural history. We proposed endovascular embolization as an initial procedure because the ability to complete cure in small AVMs. The larger AVMs should be embolized as completely as possible and then followed by either surgical removal or radiosurgery. The embolization is advantage for surgery in diminish the arteriovenous shunt and reduce surgical blood loss and help to identify the boundary zone between the AVMs and normal brain.³ Additionally, the embolization can reduce the size of large AVMs making them suitable for radiosurgery. By accurate knowledge of the brain vascular anatomy and its relationship to eloquent area, endovascular embolization in the experienced hand who understand the concept of nidus embolization will provide more cure rate and less complication rate.

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CONCLUSION

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