

Clinical Observation and Surgical Treatment of Cerebral Arteriovenous Malformation

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We have had 37 patients with cerebral arteriovenous malformation and subarachnoid hemorrhage, who were admitted to the Department of Neurosurgery, Yonsei Medical Center from 1964 to 1976. All of the cerebral arteriovenous malformations were proved by cerebral angiography. Since the site of the lesions were considered to have a close relation to the neurologic deficit and the results of surgery, various analysis of clinical manifestations and of the cerebral angiographic findings were attempted.

Out of 37 patients, surgery was performed in 28 and the results were analysed according to the various type of surgical procedure.

Arteriovenous malformation(AVM) of the brain was described by Luschka in 1954. Virchow showed in 1863 that arteriovenous malformations are of congenital origin and are not a true tumor. In 1928, the contribution of Cushing. Bailey and Dandy are the classical reviews before the development of cerebral angiography. Since the development clinical reviews on arteriovenous malformation of the brain have been published: Olivecrona and Riives in 1948 reported 60 cases; MacKenzie (1953), 50 cases; Paterson and McKissock (1966), 110 cases; Tonniss *et al.* (1958), 134 cases; Svien and Mcrae (1965), 95 cases; Moody and Poppen (1970), 105 cases; and Forster *et al.* (1972), 150 cases. The report by Perret and Nishioka on the Cooperative Study of Arteriovenous Malformations in 1966 is an excellent compositive review.

Most of the authors follow a classification derived from Bergstrand (1936):

1. Angioma cavernosum
2. Angioma racemosum
 - a. Telengiectasia
 - b. Sturge-Weber disease
 - c. Angioma racemosum arteriole
 - d. Angioma racemosum venosum
 - e. Aneurysma arteriovenosum

Among them, arteriovenous "aneurysms or malformations" are by far the most common and clinically the most important. They comprise approximately 1.5 to 4% of verified intracranial tumor.

In 1957, Hamby published a microscopic dissection of a hemispheric malformation indicating that these enlarged arteries terminated at the malformation by ramifying into multiple smaller interconnecting arteries before passage into a series of tortuous channels. Kaplan *et*

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al. (1961) believed that arteriovenous malformations originate as a congenital maldevelopment of the blood vessels in early embryonic existence and the underlying lesions represented a perpeutation of the primitive arteriovenous communications. The exact diagnosis and treatment were recommended because of subarachnoid hemorrhage (SAH), headache, convulsion, progressive neurological deficit and disturbance of consciousness due to rupture of AVM.

The methods that have been used for their treatment are: symptomatic medical therapy, irradiation, superficial electric coagulation, ligature of the arterial feeding vessels and partial or total resection of the malformation. Leussenhop *et al.* 1960, 1965) have devised and successfully used embolization with small plastic spherules of variable size according to the caliber of the feeding arteries. It was still confirmed that the effect of X-ray therapy and ligature of the feeding arteries or the internal carotid artery were not satisfactory. Now, it has been known that total resection of AVM as possible was the best operative method. Many authors believed that the operation of AVM under microscopic technique was the most nearly safe-method. Recently, stereotaxic clipping was performed successfully under the control of an image intensifier with television monitoring by Kandal and Peresedov in 1973.

This study was based on 37 cases admitted to the Yonsei Medical Center between 1964 and 1976 and by angiography shown to have an intracranial AVM.

MATERIAL AND METHODS

In this study, 37 cases who were diagnosed as having cerebral arteriovenous malformation by cerebral angiography, were admitted to the

Dept. of Neurosurgery of Yonsei Medical College from 1964 to 1976 with headache, subarachnoidal hemorrhage, and seizure attack. Of the 37 patients, surgery was performed in 28 patients. This study was based on the sex, age, symptoms, site of AVM, operative method, preoperative course. Cerebral angiography was performed within 24 hours after admission and we laid stress on the site and number of the feeding arteries and draining veins. Postoperative cerebral angiography was performed in 12 cases (30.3%) among the 28 operated ones.

RESULTS

1. Age and sex

Patient's ages were between 4 and 51, but the 4th. decade (12 cases; 32.4%) was the most frequent, and next was 2nd. decade (11 cases; 29.7%). Most cases (34 cases; 91.8%) were below the age of 40. Twenty-six of the cases occurred in men and eleven in women (2.4:1) (Table 1).

Table 1. Age and Sex Distribution

Age	Male	Female	Total
0 ~ 10	2	1	3
11 ~ 20	9	2	11
21 ~ 30	5	3	8
31 ~ 40	9	3	12
41 ~ 51	1	2	3
Total	26	11	37

The first symptoms appeared most frequently in the fourth decade and next, in order of frequency, the second, third, first, and fifth decade (Table 2).

Table 2. Age & Sex Distribution at Onset

Age	Male	Female	Total
0 ~ 10	4	1	5
11 ~ 20	8	2	10
21 ~ 30	6	3	8
31 ~ 40	8	4	12
41 ~ 51	—	1	1
Total	26	11	37

2. Symptoms and signs

On admission, the main symptoms and signs were SAH (94.6%) and headache (86.5%) and next, in order of frequency, change of consciousness (59.5%), motor weakness (56.8%), nausea and vomiting (56.8%), seizure (45.9%) and stiff neck (37.5%) (Table 3).

Table 3. Symptoms and Signs

Symptoms and signs	No. of cases	%
Headache	32	86.5
Change of consciousness	22	59.5
Nausea & vomiting	21	56.8
Hemiparesis	21	56.8
Seizure	17	45.9
Stiff neck	14	37.8
Facial palsy	8	21.6
Speech disturbance	2	5.4
Ptosis	2	5.4
Visual disturbance	1	2.7

Alternating hemiparesis was experienced in a 4 year old boy who had a giant-aneurysmal AVM in the parietal lobe. It was speculated that such an unusual symptom was probably from cerebral steal syndrome and mass effect.

On spinal tap, increased intracranial pressure was found in 27 cases (73%) and 200-300mm H₂O was most frequent (Table 4). In the

preoperative EKG, left ventricular hypertrophy was seen in 4 cases, myocardial ischemia in 1 case, sinus arrhythmia with premature atrial contraction in 1 case (Table 5).

Table 4. CSF Pressure

CSF pressure (mmH ₂ O)	No. of cases
under 200	10
201 ~ 300	14
301 ~ 400	9
over 400	4
Total	37

Table 5. EKG Findings before Operation

EKG findings	No. of cases
Normal	13
Left ventricular hypertrophy	4
Myocardial ischemia	1
Sinus arrhythmia with premature premature atrial contraction	1
Right axis deviation	1
Total	20

3. Location and arterial distribution to the AVM

The frequency of localization and feeding art. of AVM were analyzed (Table 6). An overwhelming proportion of AVM were located in the parietal lobe (16 cases; 43.2%). But, including the temporoparietal and occipitoparietal AVM, 48.6% was involved in the parietal area.

Feeding arteries arose from the middle cerebral artery (14 cases; 47.8%) and next, the anterior cerebral artery (6 cases; 16.2%). In 9 cases, especially, multiple feeders supplied the arterial connection. Among these, the middle cerebral artery and/or the anterior cerebral artery contributed most frequently.

Table 6. Site and Feeding Arterial Distribution of AVM

Site Feeding Art.	Frontal	Parietal	Temporal	Parieto- temporal	Temporo- parietal	Occipital	Basal Ganglia	Fronto- temporo- parietal	Parieto- occipital	Total
MCA	3	7	3		1					14
ACA	1	5								6
PCA						2				2
ACA/MCA	1	1		1	1			1		5
ACA/PCA						1	1		1	1
MCA/PCA										1
MCA/ACA/PCA		1								1
Ant. choroid	1	1	1							3
Post. choroid		1	1							2
Lent. st/MCA										1
Ant. cho/Lent.st.			1							1
Total	6	16	6	1	2	3	1	1	1	37

MCA : Middle cerebral artery

ACA : Anterior cerebral artery

PCA : Posterior cerebral artery

Ant. choroid : Anterior choroidal artery

Post. choroid : Posterior choroidal artery

Lent. st. : Lenticulostriate artery

Table 7. Distribution of Associated Hematoma

Site of hamatoma	No. of cases
Frontal	4
Parietal	4
Frontoparietal	2
Parietooccipital	2
Temporal	1
Frontotemporal	1
Total	14

4. Location of the associated hematoma

In 14 cases (39.7%) an associated hematoma was found. Frontal lobe (4 cases) and parietal

lobe (4 cases) were most frequent site and in order of frequency, frontoparietal, occipitoparietal, temporal and frontotemporal lobe (Table 7).

5. Operating methods and results

Of 37 patients, 28 cases were operated; total removal in 18 cases, ligature of feeders in 5, electric coagulation of feeders in 3, and ligation of internal carotid artery in 2. Associated hematoma was removed in all cases. In 9 cases, postoperative complications developed (Table 9); homonymous hemianopsia (2 cases), facial weakness, hemiparesis, intracerebral hematoma and gastrointestinal bleedings. In 12 cases, postoperative cerebral angiography was per-

formed on the 10th to 14th postoperative day, and revealed complete removal in 17 cases, subtotal resection in 2 cases, vasospasm in 1 and hematoma formation in 1. Although left ventricular hypertrophy in preoperative EKG was seen in a 4 year-old boy who had a giant-aneurysmal AVM in the left parietal area, it was no longer present in the postoperative EKG.

DISCUSSION

The cerebral arteriovenous malformation, which was named by Mc Cormick (1966), is the most common vascular anomaly in the central nervous system except for the cerebral aneurysm and comprises approximately 1.5 to 4% of the verified intracranial tumors. In "Cooperative Study of Intracranial Aneurysm of SAH" reported by Perret and Nishioka, the ratio of cerebral aneurysm to arteriovenous malformation was 6.5:1, but in this study, the ratio was 4.5:1. Also, they reported that cerebral arteriovenous malformation was often diagnosed below the age of 40, but cerebral aneurysm diagnosed below the age of 40 was only 26%. In this study, most patients of cerebral AVM were below the 4th decade (91.8%), who was diagnosed after attack of SAH.

Lee *et al.* (1976) reported that the Korans who had SAH and were diagnosed as cerebral aneurysm by cerebral angiography was 27.2%. Seizure and SAH in cerebral AVM may develop over the 5th decade but most patients were under the 5th decade. But cerebral aneurysm was noted more often over 5th decade, which seemed to be similar in comparison to other reports.

Other authors (Dandy; 1928, Norman; 1945, Olivecrona and Riives; 1948, Paterson and Mc

Kissock; 1966) agreed that there were grossly as many males as females with AVM. Moody and Poppen (1970) reported that common signs in the patients with cerebral AVM were abnormal reflexes, cranial nerve deficit, and motor weakness, but others (Cushing and Bailey; 1928, Mackenzie; 1946, Olivecrona and Riives; 1948, Luessenhop and Kachman; 1965, Mc Cormick; 1966, Kaplan *et al.*; 1966, Perret and Nishioka; 1966, Hansen; 1976) mentioned that headache, seizure, hemiplegia, SAH, and unconsciousness were common. In this study, bloody CSF was the most common sign (94.5%), and next, in order of frequency, headache, mental change, hemiparesis, and seizure. Thirteen cases among 22 showed temporary mental change. Facial weakness was noted in 8 cases; 4 AVM were located in parietal lobe, 2 in parietotemporal lobe, 2 in parietofrontal lobe.

Speech disturbance developed in 2 cases, in parietotemporal and frontal lobe lesions. Ptosis was noted in 2 cases, one AVM was located in the parietal lobe and the other was in the frontal lobe. Decreased visual acuity was also noted in 1 case, the AVM located in the frontotemporal lobe. Neck stiffness was present in only 14% but in fact, SAH in spinal tap was very common (94.6%). According to this finding, neck stiffness was not always accompanied in SAH in many cases. Mackenzie (1953) mentioned that SAH was not related to the size of cerebral AVM, but seizure was more noted in the large sized AVM.

In this study, cerebral AVM was mostly in the parietal lobe (43.2%) and next, the temporal lobe (6 cases) and the frontal lobe (6 cases). According to Cooperative Study, on overwhelming proportion of AVM are located in the parietal lobe, in 179 cases of 453 cases, frontal in 105, and temporal in 95. Rebleeding developed in 23% and also reported in 23% by Olivecrona and Lanthenheim (1948).

Silverman and associates reported two newborn infants with cerebral arteriovenous fistulas producing congestive failure and cardiomegaly (1955) and Shenkin (1948), Glatt and Rowe (1960) mentioned that cardiomegaly, increase of cardiac output or decrease of diastolic pressure were produced in infants who had cerebral AVM. In this study those who had EKG in the preoperative period, left ventricular hypertrophy was shown in 4 cases and myocardial ischemia, sinus arrhythmia with premature atrial contraction were noted in 1 case each.

In 5 cases out of 70 diagnosed as cerebral AVM, Kelley (1969) found calcification and/or enlargement of the venous channel in the plain skull series, while Olivecrona and Riives (1948) found that calcification was shown in 10-14% and enlargement of venous channel in 2.0%. In this study, calcification was not noted in any case but slight enlargement of the venous channel was noted in 6 cases (16.2%).

Cerebral angiography was most important in the diagnosis of cerebral AVM and pan-cerebral angiography was a required procedure. Recently, Hayward (1976) reported that computerized axial tomography was helpful in the diagnosis of cerebral AVM. A cerebral AVM may be multiloculated and have one or more feeding arteries and confirming the course of the draining vein and the presence or absence of intracerebral hematoma is necessary. Moody and Poppen (1970) found that the arteries feeding the malformation were most frequently seen to come from the middle cerebral artery in more than half while Olivecrona and Riives reported that 28 of 38 cases were supplied from MCA (1948). In this study, AVM supplied by the middle cerebral artery was most common (47.8%), next was from the anterior cerebral artery (16.2%) and the middle cerebral artery cases which showed

multi-feeding arteries occupied in one quarter of the cases.

Ventricular enlargement due to obstruction of basal cistern after SAH was reported by Kibler (1961) and Shulman (1963). In our study, hydrocephalus after SAH was noted in 2 cases. We feel that if pneumoencephalography were performed after SAH at a more later date, hydrocephalus may be found in more cases.

By use of conservative care, Moody and Poppen (1970) reported 17% and Svien and Mc Rae (1965) reported that in 10% in his cases, the clinical signs were aggravated or the patient expired. But Conforti (1971) confirming with cerebral angiography on his patients, that those who were given conservative care, were completely cured after 3 years.

Follow up study was almost impossible and so mortality figures are not accurate. But Pool (1962) reported that if the patients received conservative care 40% of cases were either expired or in worse condition in the follow-up.

It is a relatively recent concept that cerebral AVM is a surgically curable disease. Cushing and Bailey (1928) advised decompressive craniectomy for the purpose of decreasing the intracranial pressure of X-therapy to cause a thrombosis in the cerebral AVM. That had the danger of possibly rebleeding because thrombosis was likely to occur at the site around the draining vein. Dandy (1928) initially performed ligation of the common carotid artery for surgical intervention, and Brooks (1931) successfully treated carotid cavernous fistula by embolization of small pieces of muscle via the internal carotid artery. Lussenhop and Spence (1959) succeeded in the obstruction of the feeding artery of cerebral AVM by the use of plastic spherules. Lussenhop *et al.* said that the method was based on the following mechanism; 1) Blood flow in AVM is increased more than

in the peripheral tissue because the feeding arteries are more enlarged than the normal cerebral arteries and so, peripheral resistance was decreased 2) The angle of the course of the middle cerebral artery is more obtuse than the anterior cerebral artery the chance of passing injected material the middle cerebral artery was ten times as compared with the anterior cerebral artery. But the above embolization method was not popular recently because the complications were vasospasm of the internal carotid artery, embolization and/or thrombosis in normal vessels rather than the target vessels and rupture of accompanied aneurysm etc.

Olivecrona and Riives (1948) found that the ligation of the internal carotid artery was more dangerous than complete resection. Pool (1962), and Moody and Poppen (1970) emphasized that complete resection of the malformation was the best method in surgical procedures, but the results of the surgical procedure were influenced by whether the site of the malformation was resectable, the number and site of feeding arteries and the experience of the surgeon. Poppen and Nischioka (1966) reported that the mortality of the surgical resection of the malformation was 10.9% and Moody and Poppen (1970) reported 12%, but Olivecrona *et al.* (1944) reported that good results from surgical resection was 62% and in recent studies, good results were expected to 80%.

In our study, there was no mortality but 1 case was discharged in a moribund state. In 5 cases, ligation of the feeding arteries was done around the malformation and electric coagulation was done in 3 cases. Left hemiparesis was noted in 1 case among 2 cases who had received ligation of the feeding arteries in the cases in which resection of the malformation site might aggravate the neurologic signs. For the operation of cerebral AVM, arterial configuration in the cerebral angiography was helpful but in some

cases, the venous phase was more helpful. In fact, when the cerebral cortex is exposed, feeding arteries may be noted on the surface of the cerebral hemisphere but in most cases, it was found when exploration of cerebral hemisphere and fissure, but because the draining veins were located on the cerebral surface, it was sometimes a landmark for the finding of the feeding artery and especially, red veins were a cardinal finding in the operative field.

One case for ligation of the feeding arteries was located in the basal ganglia, and had multiple feeding arteries. In the case, ligation was under the control of the image intensifier with TV monitoring and cerebral angiography but complete removal was impossible due to the location. Removal of the intracranial hematoma was performed in 14 cases and 1 in two cases of ligation of the common carotid artery had rebleeding, followed by complete resection and discharged with improvement. Electric coagulation was performed in the deep seated AVM or in the AVM in which a direct approach might be dangerous. This method was based on the thought that further injury of the brain might be prevented by the decrease of cerebral blood flow and among these, visual disturbance was noted in one case as a postoperative complication. Complete resection was performed in 28 cases. To prevent injury to surrounding tissue at operation, feeding arteries were ligated first, as a result, cerebral blood flow to the AVM was markedly decreased. Whether the operation succeeded or not was confirmed by bluish discoloration of the red vein. Especially in the case of a 4 year old child who had a giant aneurysmal AVM, because blood volume in AVM was able to change the systemic or local cerebral blood flow and so, and shock might result, we performed ligation of the feeding arteries in the vascular malformations at first and then, squeezed step by step, which

made the blood volume in the malformation flow into the draining vein and then, finally the draining vein was ligated. Recently, after the introduction of the surgical microscope to neurosurgery field, operation was safer and the possibility of complete resection is increased. In our department, surgical microscopic approach to cerebral AVM was done in 10 cases and Kim *et al.* (1976) has already reported them.

Stereotaxic clipping of an AVM was reported by Kandel and Peresedov (1977), and this may need further study. Complete resection was confirmed by postoperative angiography in 9 of the operative cases. Postoperative angiography is important in that it may give us the information of postoperative change of the malformation as well as hematoma formation in postoperative period or vasospasm.

Among the several procedures, complete resection under the control of surgical microscope was recognized as the best method. By the use of surgical microscope, we think that the feeding arteries may be differentiated correctly and so, surrounding cerebral tissue injury could be minimized during dissection of the AVM. Especially, in the cases of the deep seated cerebral AVM, this may give advantage for the correct information of spread of such AVM comparatively.

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