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Subarachnoid and intracerebral hemorrhage due to the rupture of an arteriovenous malformation and aneurysm in the same patient.

Report of two cases

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Two cases are reported of coexistent intracranial aneurysm and arteriovenous malformation. Both lesions ruptured within one week in one patient and within three months in the other. The rarity of such occurrences is discussed and the surgical operative procedures used are described. One patient is doing well except for some weakness of the right leg and mild dysphasia. The second patient died three months postoperatively, probably because of rupture of the aneurysm. No autopsy was obtained.

A partial review of the literature indicates that the occurrence of an aneurysm and arteriovenous (A.V.) malformation in the same patient is not unusual. But for both lesions to rupture within one week in one patient and within three months in another is certainly rare. Two such cases are reported here.

Vieten in 1955 reported a case of a 46-year-old male, with two episodes of subarachnoid hemorrhage with a saccular aneurysm and an A.V. malformation, both verified at surgery. Paterson and McKissock in 1956 described four patients with an aneurysm and A.V. malformation. However none of them were operated upon. Caram in 1959 reported a case of a 26-year-old white male, with a three-year history of convulsions, who arrived at the hospital with subarachnoid bleeding. Angiograms revealed an angioma of the left temporal lobe and an aneurysm of the anterior communicating artery, filling primarily from the right side. No surgery was advised and the patient was discharged from the hospital.

Locksley in 1966 reported in the Journal of Neurosurgery on the Central Registry of the Cooperative Study of 6,365 cases of aneurysms. In the chapter on relative frequencies of single and multiple aneurysm,
he said "All aneurysm cases in this study (including the A.V. malformations) were analyzed". However, no further information is given in relation to cerebral bleeding produced by the association of these lesions with surgical treatment in the same patient.

Hiroyuki Arai\(^5\) in 1972 reported in the *Journal of Neurosurgery* the case of an infant with a giant aneurysm and a large arteriovenous malformation. However, he did not mention whether or not the lesions were surgically attacked.

**Case reports**

**Case One:**

A 50-year old white woman was admitted to the hospital on December 25, 1975. Six days prior to admission she developed sudden headache, transient unconsciousness, vomiting and weakness of the right leg.

On admission she had another episode of severe headache with vomiting. Weakness of the left arm was noted. At time of examination she was conscious and able to understand simple commands. Mild speech disorder was present. Fundoscopic examination revealed mild venous congestion with early papilledema. The pupils reacted to light and in accommodation. The remainder of the cranial nerves were intact except for a left central facial paresis. No nystagmus or diplopia were present.

The deep tendon reflexes were hyperactive on the right lower extremity. Babinsky and Chaddock signs were present. We found weakness of the right leg and left arm. All modalities of sensation were normal, except for mild hypalgesia of the right leg.

The clinical impression was subarachnoid hemorrhage due to rupture of an aneurysm of the circle of Willis on the right side. We explained the weakness and pyramidal signs of the right leg on the basis of spasm of the left anterior cerebral artery.

Spinal tap revealed a bloody spinal fluid. Right percutaneous carotid angiogram demonstrated an aneurysm of the posterior communicating artery on the right side, with marked spasm of the proximal middle and anterior cerebral vessels. No other pathology was encountered (Figure 1).

Seventy-two hours after admission the patient was taken to the operating room and, under general anesthesia, a right frontotemporal craniotomy was performed. The aneurysm was clearly identified with the use of the microscope and its neck occluded with a Heifitz clip.

Post-operatively she recovered normally with marked improvement of strength in the left arm. We noticed, however that the weakness on the right leg persisted.

Right carotid angiogram on the fifth postoperative day showed that the spasm of the anterior cerebral artery had disappeared. Through this vessel, a large arteriovenous malformation on the left parasagittal region was visualized. The aneurysm was entirely occluded by the clip (figures 2 and 3). Left carotid angiogram showed the malformation also (figures 4 and 5).

The neurological examination at this time was considered normal, except for hyperactive reflexes, pyramidal signs and weakness of the right lower extremity.

On the tenth day after admission the patient was again taken to the operating room and, under general anesthesia, a left frontoparietal craniotomy was done. Under microscope visualization, the malformation was dissected and removed. Evidence of recent bleeding was noted around the lesion (figure 6).

As the patient was taken from the recovery room to the ward, she was able to understand questions and talk with mild dysarthria. This encouraging early response to the surgical removal of the malformation was clouded that evening when we

![Figure 1, Case One](image)

Right carotid angiogram, oblique view. Posterior communicating aneurysm with marked spasm of the anterior and middle cerebral arteries.
found the patient unconscious and with poor response to painful stimuli.

We blamed this adverse reaction on cerebral edema and administered mannitol and steroids; however, since she did not respond in the next 12 hours, she was taken back to the operating room. The bone flap was removed, the dura opened, and severe brain edema was encountered. No intracerebral hematoma was found. The dura was left wide open and the bone flap removed. She was treated in the next few days by medical means.

The histologic report of the malformation showed pigmentation as evidence of recent bleeding around the lesion. The patient continued to have mild motor aphasia and weakness of the right leg.

A follow-up left carotid angiogram on the eighth postoperative day showed no signs of the A.V. malformation but the brain was still protruding (figures 7 and 8). The patient at present is doing well except for some weakness of the right leg and speech difficulties.

Figure 2
Follow up, right carotid angiogram, oblique view, Heifetz clip obliterating the aneurysm. There is no spasm of the anterior cerebral artery, permitting filling of an A.V. malformation on the left parasagittal region.

Figure 3
Follow up right carotid angiogram; A.P. view. Good filling from right to left of parasagittal A.V. malformation.

Figure 4
Left carotid angiogram, showing the A.V. malformation.
Case Two:

A 54-year-old right-handed white male was admitted to the hospital on February 12, 1976. The night before admission he went to bed as usual, but next morning he was found semi-conscious but responsive, complaining of headache. He had vomited several times.

Past history revealed that since the age of 22 he had been having occasional focal motor seizures on the right side and had some difficulty in his speech. At the age of 30 a pneumoencephalogram was normal. He was later seen by a psychiatrist who ordered an electroencephalogram which revealed some paroxysmal activity in the left frontal region. However, no further neurological investigation was considered, and he was treated with phenobarbital.

In 1955 at age 33 he had several seizures. These continued once or twice a year, but he was able to live a normal life working as a business administrator in two different companies.

During our examination he was in a semi-stuporous state but was able to understand simple questions. Motor aphasia was noted. Venous congestion and early papilledema were found on the left side. The left pupil was slightly dilated but reacted to light. Hypalgesia and right facial central paresis were present. The remainder of the cranial nerves were considered normal. He did not cooperate for visual fields. The deep tendon reflexes were active, more so in the right lower extremity where pyramidal signs were present. Spinal tap showed bloody spinal fluid. The diagnosis of ruptured aneurysm was made.

Left percutaneous carotid angiogram revealed an arteriovenous malformation in the left frontal lobe and an aneurysm of the middle cerebral artery (Figures 9 & 10). Marked displacement of the middle cerebral vessels indicated a large intracerebral hematoma. The patient's condition continued to deteriorate.
On the day of admission he was taken to the operating room where a large frontoparietal craniotomy was done. The dura was opened and the malformation identified. The brain was bulging. The cerebral cortex was divided in front of the motor strip and a large intracerebral hematoma was evacuated. The malformation was then dissected and removed under microscopy. Signs of recent bleeding around the lesion were noted. Due to the condition of the patient, no attempt was made to attack the aneurysm. (Figure 11)

Doing well for the next 12 hours, the patient was able to move his right arm and leg and to recognize some of his relatives. He then became unconscious. Due to our previous experience with Case One, we did not hesitate to take him to the operating room, where the bone flap was removed and the dura opened. Severe brain edema was present, but no further intracerebral bleeding was detected. The wound was closed leaving the dura wide open. The bone flap was not replaced.

The patient’s condition gradually improved. A follow-up left carotid angiogram on the seventh postoperative day showed that the malformation had been completely removed, the aneurysm was better seen and the brain was somewhat bulging. (Figures 12 & 13)

He was discharged from the hospital two weeks after admission with a moderate right sided hemiparesis and motor aphasia. He was able to understand simple commands. The relatives refused any further treatment.
Follow up, lateral view. The malformation is not visualized. The aneurysm is better seen. There is good filling of the middle cerebral vessels.

We feel that the death three months after surgery was most likely due to severe bleeding from the aneurysm, but unfortunately an autopsy was not obtained.

These two cases, having associated aneurysm and arteriovenous malformation, are of great interest to us, since we have not been able to find a significant number of cases in our partial review of the literature. Both cases were difficult to handle due to the severe brain edema that developed after the malformations were removed.

**Summary**

Two cases with intracranial aneurysm and A.V. malformation have been presented. The first patient is doing well except for some weakness of the right leg and mild dysphasia. The second patient died three months after the malformation had been removed. We feel that the cause of death was due to rupture of the aneurysm that he had at the trifurcation of the left middle cerebral artery.

**Discussion**

We decided to make a surgical attack on both lesions in the woman in the first case as she was in grade 2 of Botterell’s scale. The A.V. malformation was easily removed. However, she developed severe brain edema after surgery which required removal of the bone flap. We considered the edema to be a result of interruption of venous outflow into the sagittal sinus.

In the second case the large intracerebral hematoma was due to rupture of the malformation; we found evidence of bleeding around this lesion during the dissection. Before operation the patient was in grade 4 of Botterell’s scale but this was due to the presence of the hematoma. This was our indication for taking him to the operating room the day of admission. The aneurysm was not visualized during the exploration and we considered it wise not to continue because of the poor condition of the patient.

**References**


