Anterior Communicating Artery Aneurysm not Visualized by Angiography. Report of Two Successfully Operated Cases

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Summary

The diagnostic bases for ruptured anterior communicating artery aneurysm, which can not be visualized by repeated bilateral carotid angiographies are discussed in this paper. Direct operations were performed successfully in two such cases based on diagnosis from clinical signs and angiographic findings.

Key words: Anterior communicating artery aneurysm, Cerebral angiography.

Introduction

Neurosurgeons generally do not attempt surgery, unless the aneurysms are visible and are verified by angiography. We believe in some cases however, it is possible to diagnose undetectable ruptured anterior communicating artery aneurysm. Direct operations were performed successfully, in two such cases based on diagnosis from clinical signs and angiographic findings.

Case Report

Case 1

A 50-year-old man was abruptly seized with severe generalized headaches, especially at frontal region, and numbness of extremities on February 28, 1972, and was admitted to a general hospital. A lumbar-puncture yielded hemorrhagic spinal fluid under the initial pressure of 340 mmHg. He vomited several times later, but had clear consciousness. However, he had repeated episodes of same attacks on March 19 and 31, and immediately after the third attack he lost consciousness. After regaining consciousness in late April, he began to complain of gait disturbance, loss of vision and moderate headaches, though his consciousness level improved gradually. Afterwards, his clinical course became stabilized and he was transferred to Tohoku University Hospital for further evaluation on August 25, 1972.

Upon admission, he was a little confused and showed mental disorders such as disorientation, dyscalculia and very poor memory of recent events. He complained of loss of vision in both eyes. The pupils were reactive to light, sluggishly and to convergence, normally. Fundoscopic examination revealed bilateral optic nerve atrophy, right white patches and dustlike opacities in the vitreus which appeared to be the absorbed substance of pre-retinal hemorrhage. Paralysis of bilateral lower extremities was revealed. Muscular strength of upper extremities, however, was not disturbed. Other neurological and physical examinations were normal.

Although repeated serial four-vessel angiographies revealed the huge bowing of the anterior cerebral artery suggesting ventricular enlargement, hypoplasia of left A1 portion and the widening of the gap between the bilateral beginning portion of A2, no aneurysm could be detected. (Fig. 1, 2, 3) As diagnosis of brain tumor could not be excluded, pneumoventriculography was performed, on which no abnormality was revealed except for symmetric enlargement of lateral ventricles. Spinal fluid findings were normal.

According to his clinical course, symptoms and findings of angiography and pneumoventriculography, he was diagnosed as having a ruptured anterior communicating artery aneurysm based on our opinion as stated in the discussion section. A bifrontal craniotomy was performed on September 25. The arachnoid at the base of the brain, especially chiasma region, was thickened, yellowish-brown and turbid, and was adherent to the adjacent brain as evidence of subarachnoid hemorrhage. The
A1 portion of the right anterior cerebral artery showed normal diameter and that of the left one was hypoplastic as seen in the carotid angiogram. An aneurysm of the anterior communicating artery which was about $3 \times 4 \times 4$ mm in size was treated by neck ligation and clipping, and ventriculoperitoneal shunt was performed. It seemed that the blindness was caused by the thickened and adherent arachnoid at the chiasma region. (Fig. 4)
improved gradually. He was discharged on November 7, 1972.

Case 2
A 39-year-old man was abruptly seized with severe headaches and unconsciousness on January 27, 1970 and was admitted to a general hospital. One hour later he began to regain consciousness and had clear consciousness after three days. A lumbar puncture yielded hemorrhagic spinal fluid under the initial pressure of 500 mmH2O on January 28. However, he had a relapse of unconsciousness on February 1, and he was transferred to Tohoku University Hospital on February 4.

Upon admission the patient was in stuporous state with urinary incontinence, mental changes, and he complained of severe headache and nausea. Neurological examination disclosed stiff neck, left hemiparesis which was greater in the lower extremity and bilateral papilledema. Other laboratory findings were normal. After admission his condition improved gradually, and he had almost clear consciousness on February 8.

Repeated four-vessel angiographies did not reveal any intracranial aneurysm. Bilateral A2 portion were filled from left A1 portion and the widening of the gap between the bilateral begininning of A2 with angiospasm was noted on the left carotid angiography. Right A1 and A2 portions were very narrow diffusely due to hypoplasia plus angiospasm on the right carotid angiography. (Fig. 5)

A bifrontal craniotomy was performed under the diagnosis of ruptured anterior communicating artery aneurysm on February 15. An aneurysm of the anterior communicating artery which was about 15 × 15 × 5 mm in size was treated by neck ligation and clipping. The A1 portion of the right anterior cerebral artery was hypoplastic.

Postoperative condition was good and he was discharged on March 4, 1970.

Discussion
It is generally accepted that about 75% of subarachnoid hemorrhages are caused by ruptured intracranial aneurysm, about 10% by hemorrhage from arteriovenous malformations and about 5% by hemorrhage from brain tumors and other diseases which are indications for surgery. The remaining 5 to 10% are unknown in spite of complete examinations, including four-vessel angiographies. Therefore, a procedure to conduct correct diagnosis of these diseases in early stages is essential. However, surgery should be based on a reliable diagnostic method and surgery should not be performed unless angiographic vascular pathological focus is found. It is our belief that some cases of ruptured anterior communicating artery aneurysm, which can not be revealed by repeated angiographies, can be diagnosed from special clinical signs and angiographic findings. Two such cases were treated by direct operations according to the following diagnostic bases.

Up to the end of 1973, we have experienced 336 cases of anterior communicating artery aneurysm including multiple aneurysm cases, and in 320 cases direct operations were performed. Judging from these cases, it is our belief that clinical signs and findings of the ruptured anterior communicating artery aneurysm have such characteristic features as follows:1,5,6,7,9 complaints of headache at onset of subarachnoid hemorrhage are often localized deeply behind both eyes and in the frontal region, papilledema and/or retinal hemorrhage in ocular fundus is observed more frequently than with ruptured aneurysms at the other sites, motor disturbance of bilateral or unilateral lower extremities is apt to occur,
and such a mental disorder as disorientation is more prominent. If these clinical signs and findings are present in the case of subarachnoid hemorrhage, the cause would be presumed as a ruptured anterior communicating artery aneurysm.

Some authors have reported the correlation between aneurysm and anatomical variation of cerebral arteries. We have also reported the correlation between the anterior communicating artery aneurysm and anomalies at the anterior part of the circle of Willis from the operative and angiographic findings in our 68 cases of the anterior communicating artery aneurysm. According to this report, the number of cases with hypoplasia of unilateral A1 portion was 37, equivalent to 54.4%, and the number of cases with anomaly of anterior communicating artery was seven including three cases with a hypoplasia of unilateral A1 portion, equivalent to 10.3%. The number of cases with hypoplasia or aplasia of unilateral A1 portion which was presumed only by angiographies was 13, equivalent to 19.1%. On the other hand, in 80 control cases, consisting of brain tumor, epilepsy and head injury, 28 or 35.1% were unilateral A1 hypoplasia. Consequently, when hypoplasia of unilateral A1 portion and visualization of bilateral A2 portion from dominant A1 are observed on bilateral carotid angiographies, the possibility of the presence of the anterior communicating artery aneurysm are high. In addition, when the widening of the gap between the beginning portion of A2 and angiospasm at the adjacent part of the anterior communicating artery are observed, the presence of an aneurysm on the anterior communicating artery is more likely.

According to the diagnostic bases as mentioned above, two cases of subarachnoid hemorrhage in this report were diagnosed as the ruptured anterior communicating artery aneurysm. In Case 1, severe generalized headache especially at frontal region, retinal hemorrhage in ocular fundus, paralysis of bilateral lower extremities, mental disorder, hypoplasia of unilateral A1 portion and the widening of the gap between the beginning portion of A2 were observed. In Case 2, bilateral papilledema in ocular fundus, left hemiparesis greater in the lower extremity, mental disorder, hypoplasia of unilateral A1 portion, the widening of the gap between the bilateral beginning portion of A2 and angiospasm were observed.

These two patients are now in good health and are working. We believe it is necessary to diagnose ruptured anterior communicating artery aneurysm which cannot be visualized by repeated bilateral carotid angiographies and positively perform direct operation.

Conclusion

The diagnostic bases for the ruptured anterior communicating artery aneurysm, which cannot visualized by repeated bilateral carotid angiographies are as follows: clinical signs are headaches deeply behind both eyes and in the frontal region, papilledema and/or retinal hemorrhage, motor disturbance of bilateral or unilateral lower extremities, and mental disorder as disorientation. Angiographic findings are hypoplasia of unilateral A1 portion and visualization of bilateral A2 portion from dominant A1, the widening of the gap between the beginning portion of A2 and angiospasm at the adjacent part of the anterior communicating artery.

References

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