Risk of Rupture of a Second Aneurysm in Patients With Multiple Aneurysms

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There are different opinions about the mortality associated with multiple aneurysms. McKissock, et al., state that multiple aneurysms are associated with a higher natural mortality than single aneurysms. In the Cooperative Study of Intracranial Aneurysms and Subarachnoid Hemorrhage, the multiple aneurysm patients had the same prognosis for survival as those with single aneurysms. According to McKissock, recurrent hemorrhage virtually always occurs from the original lesion. Nishioka found little evidence to suggest that more than one aneurysm would rupture within the follow-up time in the Cooperative Study. Therefore, treatment of the symptomatic lesion is commonly considered adequate.

At the Neurosurgical Clinic of the University Central Hospital during the years 1957–1968, we have operated on 84 patients with multiple aneurysms in whom the ruptured aneurysm was identified with certainty at the operation. Ten of these patients had a recurrent hemorrhage during follow-up periods varying from 4 months to 11 years. In eight of these 10 patients the recurrent hemorrhage was shown to be due to rupture of another previously unruptured aneurysm; in four instances the second hemorrhage was fatal. In one of the remaining patients the second hemorrhage did occur from the original lesion, as shown by autopsy. In the other case, autopsy was not performed, and it is not known which of the aneurysms bled.

Case Reports

Case 1. This 30-year-old woman had a subarachnoid hemorrhage in October, 1956, with residual left hemiparesis. Right carotid angiography showed an aneurysm of the middle cerebral artery. The left carotid angiogram was normal. At operation clear signs of hemorrhage were seen around the aneurysm, which was clipped. Postoperative angiography showed complete obstruction of the aneurysm. The patient was well and fully capable of work for the next 10 years.

In June, 1967, the patient had a second subarachnoid hemorrhage without any neurological deficit. Right carotid angiography showed no aneurysm. Left carotid angiography revealed an aneurysm of the middle cerebral artery. After the angiography, the patient had a third hemorrhage, resulting in deafness, severe dysphasia and mental deterioration. Operation was not performed. At follow-up examination in 1968, 1 year later, she had had no further hemorrhage, but was still deaf, dysphasic, and mentally slow.

The first hemorrhage was caused by rupture of a right middle cerebral aneurysm, the second 10 years later by rupture of a left middle cerebral aneurysm that had not been visible in the 1956 angiograms.

Case 2. This 24-year-old woman had a subarachnoid hemorrhage in August, 1957, without focal signs. Right carotid angiography was interpreted as normal (Fig. 1 upper left). Left carotid angiography disclosed an aneurysm of the posterior communicating artery, which was situated so low that direct intracranial clipping was considered impossible (Fig. 1 upper right). Therefore, the internal carotid artery was ligated in the neck. A week later an intracranial exploration was carried out to ligate the internal carotid artery intracranially as well. However, the artery was found to be thrombosed up to the bifurcation, and ligation was not done. Clear signs of hemorrhage were observed around the aneurysm.

The patient was well and fully capable of work for the next 10 years. In February, 1967, she had a second subarachnoid hemorrhage without any focal signs. Right carotid angiography showed an aneurysm at the bifurcation of the internal carotid artery (Fig. 1 lower left). Upon reexamination of
FIG. 1. Case 2. Upper left: Right carotid angiogram (1957) originally interpreted as normal. Later reexamination showed a small aneurysm at the bifurcation of the internal carotid artery. Upper right: Left carotid angiogram (1957) showing an internal carotid aneurysm. Lower left: Right carotid angiogram (1967) showing the aneurysm at the bifurcation of the internal carotid artery greatly increased in size.

the 1957 angiograms, a small aneurysm could be seen at this site; it had previously escaped notice. The aneurysm was clipped intracranially, and postoperative angiography showed that the cavity had been obliterated. The patient recovered and returned to work.

The 1957 operative findings revealed that the left internal carotid aneurysm had bled. In the intervening years the right internal carotid aneurysm had grown considerably; in fact, in 1957 it was so small that it could scarcely have been clipped.

Case 3. This 41-year-old woman had a subarachnoid hemorrhage in December, 1957, without focal signs, and a second hemorrhage in January, 1958. Left carotid angiography showed an aneurysm in the internal carotid artery at the base of the posterior communicating artery (Fig. 2 upper left) but right carotid angiography disclosed another aneurysm of the middle cerebral artery (Fig. 2 upper right). The internal carotid aneurysm was considered to be the ruptured one, and this was confirmed at operation. Postoperative angiography showed the aneurysm had been obliterated.

The patient was well until January, 1962, when she had another hemorrhage. On admission she was comatose and decerebrate. Right carotid angiography showed that the middle cerebral aneurysm had greatly increased in size (Fig. 2 lower right) and that there was an intracerebral clot in the temporal region. The hematoma was evacuated and the aneurysm clipped. The patient died 2 days after the operation. At autopsy the clips were well in place on both aneurysms.

In this case, the first subarachnoid hemorrhage was shown to be due to rupture of a left internal carotid aneurysm while the bleeding 5 years later was caused by rupture of a right middle cerebral aneurysm.

Case 4. This 30-year-old man developed a left third nerve palsy after 3 weeks of severe headache in October, 1958. The spinal fluid
was clear. Left carotid angiography showed an internal carotid aneurysm just below the bifurcation, while right carotid angiography disclosed a small aneurysm at the base of the posterior communicating artery. The left carotid aneurysm was clipped and clear signs of hemorrhage were observed around it. In a postoperative angiogram the aneurysm no longer filled.

The patient was well and capable of work until November, 1959, when he had a subarachnoid hemorrhage. Left carotid angiography showed no aneurysm. Right carotid angiography revealed that the internal carotid aneurysm had grown considerably. Signs of hemorrhage were seen around the aneurysm which was clipped. Postoperative angiography verified that the aneurysm was now obliterated. Since then the patient has been well and has resumed his previous work.

Thus, in 1958 the left internal carotid aneurysm had ruptured as shown by the third nerve palsy and the operative findings. The second hemorrhage was caused by rupture of the already diagnosed right internal carotid aneurysm, which had greatly enlarged during the intervening year.

Case 5. A 50-year-old man had a subarachnoid hemorrhage in June, 1959, without focal signs, and recurrent hemorrhages 1 and 3 weeks later. Right carotid angiography disclosed an internal carotid aneurysm just below the bifurcation and a smaller aneurysm in the middle cerebral artery. Left carotid angiography was not done. The right internal carotid aneurysm was clipped, and signs of hemorrhage were observed around the aneurysm. Unexpectedly the patient had a subarachnoid bleeding 3 days after the operation and was comatose until his death 2 weeks later. Autopsy showed a ruptured aneurysm situated symmetrically in the left internal carotid artery. The clip was in the neck of the right internal carotid aneurysm, and the middle cerebral aneurysm had not ruptured.
Of course, left carotid angiography should have been performed. The autopsy showed that the right internal carotid aneurysm had ruptured first, while the left internal carotid aneurysm had bled 3 days after the operation. There was a third unruptured aneurysm in the right middle cerebral artery.

**Case 6.** A 37-year-old man had a subarachnoid hemorrhage in October, 1959, without focal signs, and a recurrent hemorrhage 6 weeks later, with right hemiparesis and dysphasia. Left carotid angiography disclosed a large middle cerebral aneurysm, while right carotid angiography showed a small middle cerebral aneurysm. The left middle cerebral aneurysm was considered to be the ruptured one, and this was confirmed at operation. Part of the aneurysm still filled at the time of postoperative angiography.

The patient recovered and returned to work. In July, 1962, he had another subarachnoid hemorrhage. Left carotid angiography showed that the clip had slipped, and the aneurysm was completely filled again. Right carotid angiography showed that the middle cerebral aneurysm was much larger. This aneurysm was operated on but could not be clipped and was therefore wrapped with oxycel. There were clear signs of hemorrhage around the aneurysm. The patient recovered, but died September 9, 1962, after another subarachnoid hemorrhage. Autopsy was not performed.

In this case, the first hemorrhage was due to rupture of the left middle cerebral aneurysm. However, the hemorrhage 3 years later was caused by rupture of the right middle cerebral aneurysm, even though the left middle cerebral aneurysm still filled during angiography.

**Case 7.** This 27-year-old man had a subarachnoid hemorrhage in December, 1961, without focal signs. Right carotid angiography revealed an aneurysm of the anterior communicating artery, and left carotid angiography showed a small aneurysm at the bifurcation of the internal carotid artery. The anterior communicating aneurysm was clipped. There were clear signs of hemorrhage around the aneurysm. Postoperative angiography showed that the aneurysm had been obliterated.

The patient was well until the second hemorrhage in March, 1965, with dysphasia and right hemiparesis. Right carotid angiography then showed no aneurysm, but left carotid angiography disclosed a greatly enlarged internal carotid aneurysm. The aneurysm was considered inoperable because of the wide neck. The patient has been well during the 4 years since the episode and has shown complete recovery from the hemiparesis and dysphasia.

Although the anterior communicating aneurysm had ruptured in 1961, the bleeding 4 years later was caused by rupture of the left middle cerebral aneurysm, as shown by the dysphasia and right hemiparesis.

**Case 8.** This 46-year-old woman had a subarachnoid hemorrhage in November, 1959. Right carotid angiography disclosed an anterior communicating aneurysm, and left carotid angiography, a middle cerebral aneurysm. The larger anterior communicating aneurysm was considered to be the ruptured one, and operation confirmed this. Postoperative angiography showed that the aneurysm had been obliterated.

The patient recovered and was well until she had a second hemorrhage in August, 1968, this time a fatal one. At autopsy there was a left middle cerebral aneurysm 2.5 cm in diameter, which had ruptured. Here the bleeding in 1959 was caused by rupture of an anterior communicating aneurysm, with a fatal second hemorrhage 9 years later due to rupture of the left middle cerebral aneurysm formerly recognized but now greatly enlarged.

**Discussion**

The problem may be stated as follows: Should the other aneurysm or aneurysms also be surgically treated, even if the currently ruptured lesion has been identified? The ruptured aneurysm can be identified with great certainty in most cases. If the aneurysms are operable and situated on the same side so that they can be reached through the same approach, both should be clipped. This situation is found in about one-third of the cases with two aneurysms. However, in the other two-thirds of the cases, the aneurysms are situated on opposite sides and clipping of both aneurysms
means two separate operations. In that case we have to weigh the risk of rupture of the other unruptured aneurysm against the risk of another operation and postoperative morbidity.

In our series of 84 patients, the operative mortality was about 9%. There were 76 patients who lived on under the risk of a second hemorrhage; 10 of them (13%) actually did have a second hemorrhage and eight of these (80%) were shown to be from another previously unruptured aneurysm. Since the second hemorrhage was fatal in three of the cases, the predictable mortality among the original 76 survivors was 4%.

The morbidity in this series was also considerable. There were 66 patients, excluding the operative deaths and those who had a recurrent hemorrhage. Six of the patients have died of causes other than subarachnoid hemorrhage; 21 (35%) of the remaining 60 patients are totally disabled, and 8 (13%) are partially disabled. Naturally, some of the disabilities are due to the subarachnoid bleeding as well as the operation.

We seriously doubt that we could have offered the patient a better prognosis by ligating the unruptured aneurysm at a separate operation. Moreover, at the time of the first operation, the second aneurysm was often so small that it would have been technically impossible to ligate it. Therefore, we feel that only the currently responsible aneurysm should be operated on unless the other one can be reached through the same approach and is technically operable.

**Summary**

We have reported eight patients with multiple aneurysms who had a subarachnoid hemorrhage from a previously unruptured aneurysm after surgical treatment of the first ruptured aneurysm. The total number of patients with multiple aneurysms in whom the responsible lesion was identified and surgically treated was 84; the operative mortality of this group was about 9%. About 10% of the remaining 76 patients subsequently had a rupture of another aneurysm. Three of these cases (4%) were fatal.

Considering the added risk of a second operation and the postoperative morbidity inherent in the procedure, we do not think that we can offer the patient any better prognosis by ligating the unruptured aneurysm at a second operation.

**References**