Vertebral Artery Dissecting Aneurysm Rebleeding After Proximal Occlusion
—Case Report—

Nobuyuki TAKAI, Isamu EZUKA, Takatoshi SORIMACHI, Takashi KUMAGAI and Katsuhiro SANO

Department of Neurological Surgery, Niigata Rosai Hospital, Joetsu, Niigata

Abstract
A 38-year-old male presented with vertebral artery dissecting aneurysm manifesting as subarachnoid hemorrhage. An attempt at trapping the aneurysm failed, so the vertebral artery could only be clipped proximally. Rebleeding occurred, resulting in death, probably due to excessive length of the dissection requiring thrombosis and/or retrograde dissection due to back pressure from the contralateral vertebral artery.

Key words: dissecting aneurysm, subarachnoid hemorrhage, vertebrobasilar system

Introduction
Intracranial dissecting aneurysms manifest clinically as ischemic symptoms in the anterior circulation, while vertebral artery (VA) aneurysms are generally identified after subarachnoid hemorrhage. VA dissecting aneurysms have two contrasting pathological features: occlusion of the arterial lumen by mural dissection and hemorrhage from the disrupted adventitia. Conservative treatment is therefore not adequate for such complex aneurysms. Intracranial proximal occlusion of the VA has been advocated, but rebleeding after surgery may occur.

Here we describe a patient with a VA dissecting aneurysm manifesting as subarachnoid hemorrhage, which rebled even after proximal clipping of the VA.

Case Report
A 38-year-old male was well until October 24, 1990, when he suffered sudden headache followed by loss of consciousness and generalized convulsions. He was admitted to a hospital in a restless condition and received 10 mg of diazepam intravenously. Respiratory arrest occurred and he was intubated. He was transferred to our hospital with suspected subarachnoid hemorrhage.

Neurological examination revealed isocoric reactive pupils with right adducting eye, tetraplegia, and indefinite spontaneous respiration. Computed tomographic (CT) scans showed clots in the perimesencephalic, vermian, basal and Sylvian cisterns (Fig. 1 left). The lateral ventricles were expanded by packed clots in the aqueduct and third ventricle. His respiration became normal 2 hours after ictus but without improvement in the other neurological symptoms.

Bilateral carotid angiograms on October 26 demonstrated no abnormalities except enlargement of both lateral ventricles. Neither posterior communicating artery was developed well. Right vertebral angiograms revealed an irregular wall of the intracranial right VA, beginning at the third segment and extending just distal to the origin of the posterior inferior cerebellar artery (PICA), where a fusiform aneurysmal dilatation was seen. The distal vertebrobasilar complex was not filled (Fig. 2). Left vertebral angiograms were normal with reflux of contrast medium to the narrow distal portion of the right VA (Fig. 3 left), which was considered indominate at that time. The diagnosis was subarachnoid hemorrhage due to a ruptured VA dissecting aneurysm.

Right ventricular drainage was performed to cor-
Postoperatively, his consciousness improved gradually; he was able to open and close his eyes, protrude his tongue incompletely, flex the elbows weakly, and draw up his knees in response to verbal commands. Right vertebral angiograms on November 8 revealed that the aneurysm had grown, accompanied by recanalization of the distal VA. Pooling of contrast medium within the false lumen also occurred in the venous phase (Fig. 4). Left vertebral angiograms showed marked vasospasm in the vertebrobasilar system. The reflux to the contralateral VA was similar to the previous angiogram (Fig. 3 right). A second hemorrhage occurred on November 11. CT scans showed clots extending from the fourth ventricle to both lateral ventricles, and swollen brain with multiple low-density areas (Fig. 1 center).

Suboccipital decompressive craniectomy exposed the right VA with him in the lateral position on November 13. The VA was sclerotic with a yellowish discolored wall accompanied by marked neovascularization. The fusiform segment, enlarged and purplish in color, was just distal to the origin of a small artery feeding the medulla oblongata. The distal end of the aneurysm and the origin of the PICA could not be observed due to obstruction by the fusiform enlargement. The planned attempt to trap the aneurysm was abandoned. The VA was clipped proximally at the junction between the aneurysm including the origin of the PICA and the yellowish sclerotic portion, sparing the small feeder to the medulla oblongata.

Postoperatively, his condition remained unchanged even after a ventriculoperitoneal shunt was emplaced on November 29. On December 6, he suddenly fell into irreversible respiratory arrest. CT...
scans revealed a third hemorrhage (Fig. 1 right). He died on December 17, 54 days after onset. No autopsy was performed.

Discussion

The incidence of VA dissecting aneurysm presenting as subarachnoid hemorrhage has increased recently, and the incidence of rebleeding is 18-33%. Mortality among patients with dissecting aneurysm in the posterior circulation receiving conservative treatment was 90%. Dandy carried out the first ligation of the VA at the cervical portion in 1928. Guthkelch first ligated the intracranial VA for dissecting aneurysm in 1949. Yonas et al. then used this procedure for VA dissecting aneurysm manifesting as subarachnoid hemorrhage, and proximal clipping of the affected VA has frequently been used. However, proximal occlusion of the VA is not always safe, causing 5-10% of reported deaths. The indications that occlusion is possible are: 1) absence of sclerotic changes in the contralateral VA with the diameter the same or greater than the artery to be occluded, 2) the aneurysm should be associated with a well-developed posterior communicating artery at least, and 3) anastomosis between the superior cerebellar artery and the PICA should be confirmed.

The most important issue is whether proximal occlusion alone can prevent further dissection. Bakay and Sweet found no measurable drop in pressure in the VA or PICA distal to occlusion of unilateral VA trunk at the atlas, so occlusion of the VA is unlikely to prevent the development of aneurysms on vessels supplied by the VA. Shibata et al. reviewed 24 cases of proximal ligation, finding two cases of rebleeding (8.3%). Only three cases of rebleeding after surgery has been reported, so this case is the fourth. Certainly, proximal ligation is not always a satisfactory method. Trapping of the aneurysm combined with vascular reconstruction seems reasonable, but should be avoided except when the segment to be isolated is very short and devoid of perforating vessels.

In our case, angiography showed that bilateral proximal VAs were equal in caliber, and the dissection extended beyond the origin of the PICA with no filling from the contralateral VA or backflow via the PICA. The first intention was to trap the aneurysm, but only proximal clipping was possible because the distal end of the aneurysm could not be seen. Postoperatively, the aneurysm did not bulge, suggesting that satisfactory oblitative thrombosis of the dissected segment was achieved. The causes of rebleeding were as follows: 1) the dissected segment including the orifice of PICA was too long to be thrombosed; and 2) retrograde dissection due to back pressure may have begun at the re-entry because the back pressure of the contralateral VA had not decreased, suspected from the second vertebral angiography. The balloon occlusion test would have confirmed that the aneurysm was not filled by the backflow, and trapping the aneurysm combined with vascular reconstruction surgery may have achieved a better outcome.

We emphasize that in the treatment of VA dis-
secting aneurysms, proximal clipping alone does not always prevent aneurysm rupture and dissection.

References


Address reprint requests to: N. Takai, M.D., Department of Neurological Surgery, Niigata Rosai Hospital, 1–7–12 Toun-cho, Joetsu, Niigata 942, Japan.