Giant Unruptured Aneurysm of the Vertebral Artery
Presenting with Rapidly Progressing Bulbar Compression
—Case Report—

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Abstract
A 65-year-old male with a giant aneurysm of the vertebral artery, which had caused minimal neurological deficits for a few years, suffered rapid onset of respiratory disturbance followed by coma. Serial neuroimaging studies showed the thrombosed aneurysm continued to enlarge, resulting in increasing compression of the brainstem. Intraoperative observation and histological examination revealed that the aneurysm was composed of totally organized thrombus. Total extirpation of the giant aneurysm resulted in the patient regaining spontaneous respiration and clear consciousness, suggesting that growth of this aneurysm is due to increases in the size and number of the intrathrombotic capillary channels.
Key words: vertebral artery, giant aneurysm, organization, aneurysmectomy, brainstem

Introduction
Large and giant aneurysms of the vertebral artery are challenging vascular lesions for neurosurgeons. In particular, thrombosed vertebral artery aneurysms tend to become giant, causing the symptoms of brainstem compression.
To date, no significant number of patients with this entity have been treated. Moreover, the histological features and growth pattern are poorly elucidated. For this reason, clinical and pathological documentation of an additional single case with this rare lesion seems to be important. Details of this patient, including discussion of therapeutic strategy, are presented.

Case Report
A 65-year-old male, a taxi driver, was admitted to our institute because of gait difficulty and dysphagia on June 7, 1995. His first neurological abnormalities were vertigo followed by altered consciousness in August 1992, when a giant aneurysm of the vertebral artery without subarachnoid hemorrhage was detected at another hospital. About one month later, he became free of neurological abnormalities, except for mild paresis of the right recurrent nerve, and continued to work as a taxi driver. He was referred to our outpatient clinic, and underwent magnetic resonance (MR) imaging studies in February and November 1994, which showed the size of the mass had increased, resulting in marked compression and displacement of the brainstem (Figs. 1 and 2).
Physical examination on admission showed respiratory insufficiency, drowsiness, loss of gag reflex, and bilateral hemiparesis. Computed tomography (CT) without contrast medium delineated a round mass with increased size compared to that on the previous MR images, and appeared to almost replace the brainstem (Fig. 3). Left vertebral angiography and three-dimensional CT angiography showed only filling of the residual lumen around the neck of the thrombosed aneurysm (Fig. 4).
He was conservatively treated, because of the high risk of the surgical intervention relative to his mild neurological manifestations. However, his neurological condition rapidly deteriorated to coma and apnea on June 10 (4 days after admission). He was managed under controlled ventilation, and regained response to painful stimuli. Thereafter, he remained apneic, so surgical intervention was selected after...
consultation with his family.

Proximal clipping of the right vertebral artery with two Weck clips and exploration of the aneurysm was performed via a lateral suboccipital approach on June 21. Postoperatively, he failed to show more than a slight improvement of consciousness, and continued to require ventilator management.

Distal clipping of the right vertebral artery and aneurysm trapping were performed through a subtemporal transpetrosal approach on September 20. At surgery, removal of the aneurysmal content was only partially achieved because of unexpectedly significant hemorrhage from the organized thrombus. He did not show any improvement postoperatively, and remained apneic.

The thrombosed aneurysm was totally removed using the combined suboccipital and subtemporal transpetrosal approach on October 11 (Fig. 5). During excision of the thrombus in the aneurysm, moderately massive hemorrhage was encountered; the most prominent part of the hemorrhage occurred near the inner wall of the aneurysm, which was meticulously coagulated. The outer wall of the aneurysm was associated with fragile vessels which were supplied from surrounding structures. Coagulation of these abnormal vessels facilitated removal of the hemorrhagic thrombus, in a procedure similar to the excision of meningioma.

Postoperatively, facial paresis, hearing disturbance, and lower cranial nerve paresis developed on the right side. Two weeks after the third operation, he regained clear consciousness and was weaned from the ventilator. Thereafter, slow but steady improvement of the cranial nerve paresis and quadriparesis were observed. He is scheduled for transfer to a rehabilitation center.

Histological examination found multiple vascular channels, particularly near the neck of the aneurysm, and in the thrombus (Fig. 6).
Discussion

The brainstem of this patient had been strongly compressed and displaced by the giant thrombosed aneurysm at least for 16 months, but he had suffered minimal neurological deficits, and continued to work as a taxi driver. Unfortunately and unusually
in this patient, the bulbar symptomatology rapidly deteriorated to apnea and coma without subarachnoid hemorrhage. Serial neuroimaging studies in our patient disclosed that the aneurysm, although remaining thrombosed, had progressively enlarged, as described previously. The mechanism of rapid deterioration of the bulbar symptoms is unclear because cerebral blood flow (CBF) study was not performed. However, as in chronic subdural hematoma, a rapid decrease in CBF of the brainstem seems to contribute to the occurrence of this catastrophic event. This may also be supported by the pathological features of the aneurysmal content, which resembles the outer membrane of chronic subdural hematoma. Why this lesion reaches such a large size without rupturing remains unclear. The presence of intrathrombotic vascular channels is probably the main factor in the growth of the thrombosed aneurysm. In our patient, considerable hemorrage during removal of the content of the aneurysm was thought to be caused by highly vascular channels in the aneurysmal wall and organized thrombus. The presence of intramural and intrathrombotic vascular channels was verified by histological examination.

Unlike the saccular, dissecting, or fusiform aneurysms usually found in the vertebral artery, proximal vessel ligation of the thrombosed aneurysm does not achieve a successful outcome in most symptomatic patients. Proximal clipping in our patient did not improve the brainstem compression symptoms. The unfavorable outcome after proximal clipping is closely related to the growth mechanism of the aneurysm. This behaves like an angiomatous mass, so brainstem decompression can only be achieved by extirpation of the aneurysm. The observations of histological examination, although very rarely reported, support this therapeutic strategy. Therefore, recent reports have emphasized the necessity for aneurysmectomy after trapping of the aneurysm with or without bypass surgery. Our patient regained spontaneous respiration only after total aneurysmectomy. However, removal of a large, hardened thrombosed aneurysm with remarkable compression to the brainstem carries a high risk and successful results were reported by the trapping procedure alone (personal communication), so the timing of the surgical intervention is very important. Giant thrombosed aneurysms of the vertebral artery may become large enough to cause brainstem compression. Therefore, spontaneous or therapeutically-induced thrombosis of giant aneurysm of the vertebral artery is not curative, and requires further follow-up with neurological examination and neuroimaging studies.

References


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