Basilar Apex Aneurysm Manifesting as Third Ventricular Mass and Obstructive Hydrocephalus

—Case Report—

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Abstract

A 58-year-old male, with a past history of hypertensive thalamic hemorrhage 12 years before, presented with gradually exaggerating gait disturbance, memory disturbance, and urinary incontinence. On admission, he had gait disturbance represented by petit pas and anteropulsion in addition to significant recent memory disturbance. Cranial computed tomography (CT) revealed a hyperdense mass in the third ventricle with triventricular dilation. Cerebral magnetic resonance (MR) imaging and MR angiography identified the third ventricular lesion as saccular basilar apex aneurysm. No other intracranial abnormal intensity contributing to his clinical symptoms was recognized. Cervical MR angiography showed normal findings. Cerebral blood flow (CBF) measurements revealed diffuse CBF reduction in the cerebral hemisphere. The patient underwent coil embolization which accomplished complete aneurysm occlusion. He showed only slight improvement in his gait disturbance after embolization, and CT following embolization revealed persistent ventriculomegaly. Ventriculoperitoneal shunting was carried out. Intraoperative neuroendoscopy demonstrated cerebrospinal fluid (CSF) obstruction caused by the embolized aneurysm at the level of the third ventricle, with normal CSF findings. Postoperatively his gait disturbance and intellectual impairment showed remarkable improvement. Basilar apex aneurysm associated with obstructive hydrocephalus has complex underlying pathology and should be treated by a combination of definitive aneurysm obliteration and CSF diversion.

Key words: basilar apex aneurysm, normal pressure hydrocephalus, coil embolization

Introduction

Basilar apex aneurysm manifesting as third ventricular mass and obstructive hydrocephalus is rare but clinically extremely important for determining treatment strategy because such pathology usually needs definitive management for both aneurysm obliteration and hydrocephalus. Cerebrospinal fluid (CSF) diversion may carry the risk of inducing aneurysm growth resulting in devastating clinical sequelae, so definitive treatment for the offending cerebral aneurysm is essential before CSF diversion.

Here we present a case of basilar apex aneurysm successfully treated by a combination of coil embolization and ventriculoperitoneal (VP) shunting.

Case Report

A 58-year-old male presented with gradually exaggerating gait disturbance followed by memory disturbance and urinary incontinence. He had suffered hypertensive intracerebral hemorrhage in the left thalamus at age 46 years, from which he had recovered without detectable neurological impairment. He visited the Department of Neurology in our university hospital where he underwent cranial computed tomography (CT) which revealed a third ventricular mass and ventriculomegaly. He was referred to the Department of Neurosurgery on September 21, 2005.

On admission, he had gait disturbance represented by petit pas and anteropulsion in addition to significant recent memory disturbance with Mini-Mental State Examination (MMSE) score of 18 points. Cranial CT showed a round hyperdense mass, with maximal diameter of $15 \times 12$ mm, in the third ven-
tricle, and moderate triventricular dilation with periventricular low density (Fig. 1). Cerebral magnetic resonance (MR) imaging and MR angiography identified the third ventricular lesion as a saccular basilar apex aneurysm. No other intracranial abnormal intensity contributing to his clinical symptoms was recognized (Fig. 2). Cervical MR angiography did not show any stenotic or occlusive lesions. Cerebral blood flow (CBF) measured by N-isopropyl-(123I)-p-iodo-amphetamine single photon emission computed tomography revealed diffuse CBF reduction (not shown). Spinal MR imaging and laboratory tests did not reveal other findings associated with his clinical symptoms. Lumbar spinal tap was not carried out because of the risk of changes in the intra-aneurysmal pressure gradient and brain herniation.

Complete aneurysm occlusion was achieved by coil embolization with the Guglielmi Detachable Coil system (Target Therapeutic, Boston Scientific, Fremont, Calif., U.S.A.) on November 26, 2005, with total coil length of 445 cm and packing density of 29% of the aneurysm (Fig. 3). He showed only slight improvement in his gait disturbance after embolization, and surveillance CT showed persistent ventriculomegaly. Postoperative neuroimaging detected no further enlargement of the obliterated aneurysm. VP shunting was performed on January 25, 2006. Following ventriculostomy in the right frontal horn, a fiberoptic neuroendoscope (Olympus Corporation, Tokyo) was introduced into the third ventricle via the foramen of Monro, which confirmed CSF obstruction by the aneurysm at the level of the posterior third ventricle. Intraoperative examina-
Cerebral T₁-(A) and T₂-weighted (B) magnetic resonance images taken 1 year after ventriculoperitoneal shunting demonstrating resolution of the ventriculomegaly and no additional abnormal findings.

Postoperatively his gait disturbance showed remarkable improvement. His MMSE score improved to 29 points in the 6 months following VP shunting. Cerebral MR imaging taken 1 year after VP shunting revealed resolution of the ventriculomegaly and no additional abnormal findings (Fig. 4). He has been followed up satisfactorily without requiring re-treatment for refilling or coil compaction.

Discussion

In the present case, initial endovascular coil embolization of the offending aneurysm did not resolve the coexisting hydrocephalus, despite no further enlargement of the obliterated aneurysm. Intraoperative examination revealed clear CSF with normal cell count and protein concentration. Then the ventricular catheter was placed in the right frontal horn and a Hakim Programmable Valve (Medos S.A., Le Locle, Switzerland; Codman, Johnson & Johnson Co., Raynham, Mass., U.S.A.) was implanted.

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We should be aware that CSF diversion may carry the risk of inducing aneurysm growth resulting in devastating clinical sequelae. For example, there was a case report that a patient with dissecting basilar artery aneurysm was initially treated conservatively, showed transient clinical improvement after VP shunting, but suffered aneurysm rupture one month after the surgery.

The exact pathophysiology of NPH is also not fully elucidated. Mean CBF has generally been demonstrated to be lower in patients with NPH than in normal subjects. The significant decrease in CBF in NPH patients is considered to result from reductions in cerebral perfusion pressure and appears to be maximal in the paraventricular watershed region. In contrast, significant increases in CBF were recognized after CSF diversion in the left prefrontal dorsolateral areas, right frontal premotor area, right medial prefrontal region, right frontal white matter area, and right basal ganglia. On the other hand, an overlap in the CBF volumes measured between patients with NPH and normal subjects suggested that ischemia may not be a prerequisite for this condition, but instead may reflect increased superficial venous pressure in patients with NPH. Cerebral circulatory disorders in patients with NPH can manifest as either of two pathophysiological conditions: circulatory disorder of the cerebral cortical region, and that of the thalamus-basal ganglia region, and various patterns may develop according to the disease stage. Such discrepant explanations may derive from the absence of comprehensive understanding of NPH, which is actually a dynamic and divergent disease in cerebral venous pulsation which can produce pulsatile gradient of CSF flow, and the water-hammer effect of pulsatile blood flow within the basilar artery. This analysis can be divided into the factors associated with CSF pulsation, pulsatile motion of the offending aneurysm and adjacent ventricular wall, and mutual interference; and the patterns of venous outflow surrounding the third ventricle. Such analysis is likely to better explain the mechanism of the developing hydrocephalus. This analysis may also support the comprehensive treatment of such hydrocephalus by a combination of definitive treatments for the offending aneurysm and the CSF diversion, because all factors can contribute, in different degrees, to the underlying pathophysiology. On the other hand, however, a rare case of thrombosed giant posterior communicating artery aneurysm has been described that presented as third ventricular mass, causing obstructive hydrocephalus, and was successfully treated with clip obliteration and thrombectomy alone.

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Management of the present case with combined endovascular embolization of the aneurysm and VP shunting for the hydrocephalus was successful, but long-term follow up is needed to evaluate the efficacy and validity of the treatment. Further experience coupled with the rapidly evolving technology of endovascular surgery and persistent investigation of hydrocephalus may yield a comprehensive understanding and optimum treatment strategy for an aneurysm occupying the third ventricle and associated with hydrocephalus.

References


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