

Giant basilar bifurcation aneurysm presenting as a third ventricular mass with unilateral obstructive hydrocephalus: case report

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Summary We report a rare case of a non-ruptured basilar bifurcation aneurysm presenting as a third ventricular mass producing unilateral obstructive hydrocephalus. This is the first reported case of its kind. A 70 year old woman presented with a giant basilar bifurcation aneurysm in which the aneurysm protruded into the third ventricle as a mass causing unilateral left hydrocephalus. The patient gradually became disoriented and developed a right hemiparesis and global aphasia. The right vertebral artery was occluded by placing coils intravascularly followed by an endoscopic septostomy. The patient's neurological state dramatically improved immediately. One month after the septostomy, however, the aneurysm ruptured and the patient eventually died. Treatment of the hydrocephalus only was selected instead of direct surgery or an intravascular procedure on the aneurysm, which eventually ruptured. The mechanisms for the unilateral hydrocephalus and the rupture of the aneurysm are described. The treatment strategy for these lesions is also discussed. © 2001 Harcourt Publishers Ltd

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INTRODUCTION

A giant basilar artery aneurysm may cause obstructive hydrocephalus;^{1–10} however, there is no report of a giant basilar artery aneurysm causing unilateral hydrocephalus. We present a rare case in which fatal rupture eventually occurred one month after the treatment of the hydrocephalus with an endoscopic septostomy. We speculate on the mechanisms for the rupture of the aneurysm and discuss strategies for treating such cases.

CASE REPORT

This 70 year old, right handed woman developed memory disturbance and dementia one year before admission. The symptoms gradually developed and she was referred to us because a computed tomography (CT) scan disclosed a round mass in the third ventricle and obstructive hydrocephalus. On admission her neurological examination revealed disorientation and memory disturbance, but

focal neurological deficits such as aphasia and motor weakness were not observed. A CT scan and magnetic resonance images (MRIs) taken on admission revealed a round mass, 25 mm in diameter, in the third ventricle with enlargement of the left lateral ventricle (Fig. 1). The mass was of low intensity on both T₁- and T₂-weighted images and was enhanced by gadolinium administration. Vertebral angiography showed a giant aneurysm at the basilar bifurcation, with a maximal diameter of 25 mm, directed antero-superiorly (Fig. 2).

After being admitted to the hospital, she developed a right hemiparesis and global aphasia, which were progressive. Since the aneurysm was very large, with a wide neck where the posterior cerebral arteries originated, direct clipping was considered to be quite hazardous and coil embolisation was not indicated. Endoscopic septostomy was planned to relieve the hydrocephalus. Before the septostomy, in order to decrease the intraluminal pressure of the aneurysm, intravascular coils were placed to occlude the cervical portion of the right vertebral artery, on her non-dominant side. After the proximal occlusion of the vertebral artery, her neurological condition remained unchanged. One week after the intravascular occlusion of the vertebral artery, the septostomy was performed using a neuro-endoscope. A navigation system was used to puncture the right lateral ventricle safely, without injuring the aneurysm dome. The dome of the aneurysm was visualised protruding into the right lateral ventricle through the foramen of Monro (Fig. 3). After the septostomy, there was a marked improvement in the neurological symptoms – her level of consciousness, right hemiparesis and global aphasia. A CT scan showed improvement of the hydrocephalus. Thirty days after the septostomy, however, she suddenly became unconscious, and a CT scan showed a severe subarachnoid haemorrhage in the basal cisterns (Fig. 4). She died 2 days later.

DISCUSSION

Giant aneurysms, defined as those with a diameter of >25 mm, may cause obstructive hydrocephalus, but these are relatively rare occurrences.^{1–10} Basilar bifurcation aneurysm is the predominant location of giant aneurysms reported to cause obstructive hydrocephalus, although giant aneurysms in other locations such as the posterior communicating artery¹⁶ and the internal carotid artery⁹ may also cause obstructive hydrocephalus. There have been several reports of basilar bifurcation aneurysms protruding into the third ventricle mimicking a third ventricular mass.^{1–4,6,9–11} There are, however, no reports of such aneurysms causing unilateral hydrocephalus. Although the aneurysm was round and was located almost in the midline, deviating slightly to the right, the left side of the foramen of Monro was eventually obstructed. The hydrocephalus possibly developed unilaterally because the aneurysm grew asymmetrically. Unilateral hydrocephalus is usually induced by lesions in the lateral ventricle, and this is the first report of a giant basilar bifurcation aneurysm protruding into the third ventricle that induced a significant degree of unilateral hydrocephalus.

CSF shunting is the most frequently performed procedure for treating obstructive hydrocephalus induced by a non-ruptured giant aneurysm.^{2,4–6,9,12} For a ruptured aneurysm, experimental and clinical evidence suggests an increased risk of rebleeding when the pressure gradient across the aneurysm wall is increased.^{13,14} Several reports explain that ventricular drainage is not conclusively associated with an increased incidence of repeated aneurysm rerupture.^{15–18} On the other hand, Pare et al.¹⁹ demonstrated an increased risk of aneurysmal rebleeding in patients undergoing ventricular drainage, particularly in the presence of hydrocephalus. Kawai et al.²⁰ also reported a risk

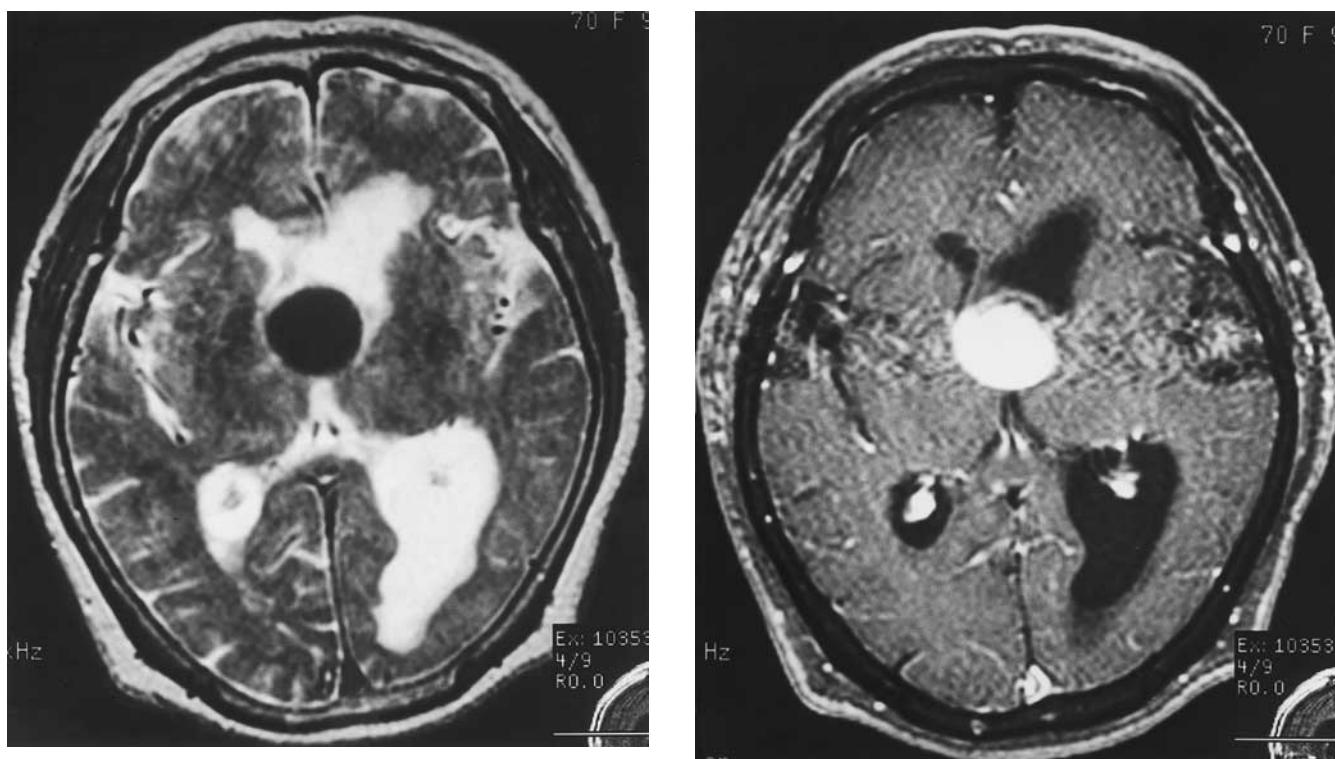


Fig. 1 Magnetic resonance (MR) images on admission showing a 25 mm diameter round mass in the third ventricle with obstructive hydrocephalus. Left: T₂-weighted image. Right: gadolinium enhanced T₁-weighted image.



Fig. 2 Left vertebral angiogram showing a large basilar bifurcation aneurysm projecting anterosuperiorly. Left: anteroposterior view. Right: lateral view.

of ventricular drainage in the case of grade V subarachnoid haemorrhage.

In this case, the aneurysm was initially non-ruptured. Direct surgery on the aneurysm was considered to be quite hazardous and intravascular coil embolisation of the aneurysm was not indicated because the neck of the aneurysm was wide and furthermore

angiograms showed that posterior cerebral arteries originated from the neck of the aneurysm bilaterally. In addition, since the patient's neurological decline was progressive, a procedure for relieving the hydrocephalus was urgently indicated. Even though the aneurysm was non-ruptured, a change in the CSF pressure might have induced it to rupture. The aneurysm was not thrombosed on the

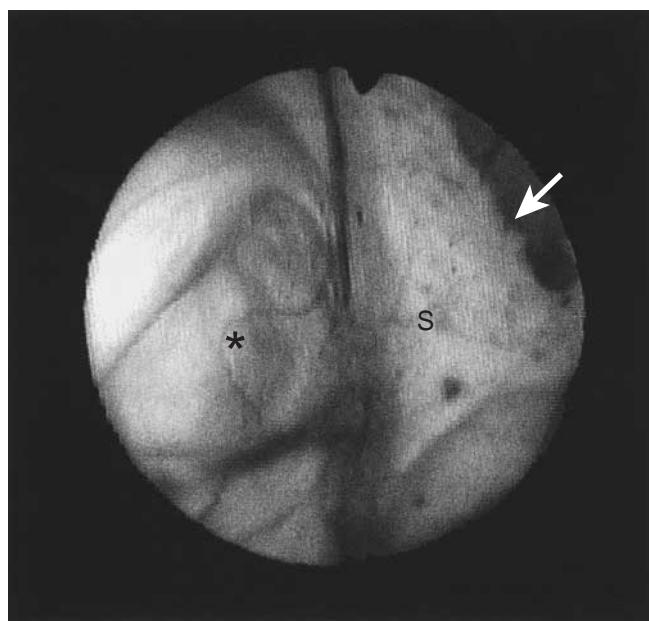


Fig. 3 Endoscopic picture during septostomy. The aneurysm wall (asterisk) is seen protruding into the right lateral ventricle through the foramen of Monro. Septostomy (arrow) is performed. S: septum

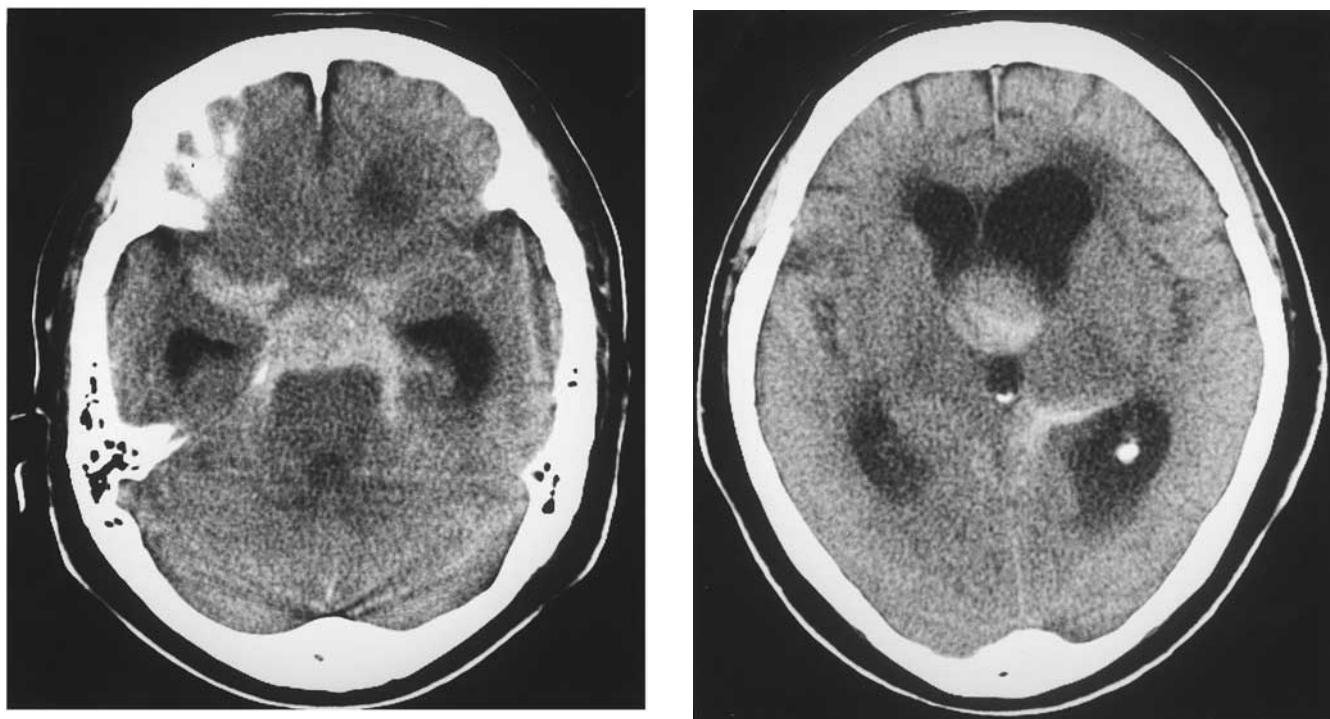


Fig. 4 CT scans 30 days after septostomy showing subarachnoid haemorrhage with obstructive hydrocephalus.

MR images. Therefore, coils were placed to reduce the intra-aneurysmal pressure occluding the vertebral artery on one side, and this was followed by a septostomy. Since the right foramen of Monro was patent, septostomy was thought to be adequate for resolving the unilateral hydrocephalus.

Haemodynamic changes after occlusion of the vertebral artery or a change in the gradient between the intra-aneurysmal pressure and the intracranial pressure may be responsible for the rupture.²¹ It is not clear why the aneurysm ruptured one month after the septostomy, rather than immediately afterwards.

Although the exact cause of the aneurysmal rupture in this patient remains unclear, septostomy or ventricular drainage may

pose some risk for aneurysm rupture. It might be necessary to carefully manage the patient's systemic blood pressure, keeping it low.

REFERENCES

1. Babu VS, Eisen H. Giant aneurysm of anterior communicating artery simulating 3rd ventricular tumor. Computerized Tomography 1979; 3: 159-163.
2. Borrie MJ, Campbell AJ, Caradoc-Davies TH. Obstructive hydrocephalus secondary to giant cerebral aneurysm in the elderly. J American Geriatric Society 1985; 33: 210-212.
3. Bose B, Northrup B, Osterholm J. Giant basilar artery aneurysm presenting as a third ventricular tumor. Neurosurgery 1983; 13: 699-702.

4. Koga H, Mori K, Kawano T, Tsutsumi K, Jinnouchi T. Parinaud's syndrome in hydrocephalus due to a basilar artery aneurysm. *Surg Neurol* 1983; 19: 548-553.
5. Koyama S, Kotani A, Sasaki J. Giant basilar artery aneurysm with intramural hemorrhage and then disastrous hemorrhage: case report. *Neurosurgery* 1996; 39: 174-178.
6. Morota N, Ohtsuka A, Kameyama S, Suzuki Y, Yasukawa K. Obstructive hydrocephalus due to a giant aneurysm of the internal carotid bifurcation. *Surg Neurol* 1988; 29: 227-231.
7. Nishizaki T, Tamaki N, Matsumoto S, Fujita S. Multiple giant fusiform aneurysms with hydrocephalus. *Surg Neurol* 1985; 24: 101-104.
8. Osenbach RK. Giant aneurysm of the distal posterior inferior cerebellar artery in an 11-month-old child presenting with obstructive hydrocephalus. *Pediatr Neurosci* 1989; 15: 309-312.
9. Piek J, Kim DP, Bock WJ. Obstructive hydrocephalus caused by a growing, giant aneurysm on the upper basilar artery. *Surg Neurol* 1983; 20: 288-290.
10. Smith KA, Kraus GE, Johnson BA, Spetzler RF. Giant posterior communicating artery aneurysm presenting as third ventricle mass with obstructive hydrocephalus. *J Neurosurg* 1994; 81: 299-303.
11. Soeur M, Brihaya J, Moerman C. Pseudo-tumoral aneurysm in the third ventricle: report of three cases. *Acta Neurochir (Wien)* 1979; 45: 247-258.
12. Kinoshita K, Sunami N. [Occult hydrocephalus due to unruptured giant basilar bifurcation aneurysm.] *No Shinkei Geka* 1979; 7: 603-608. (Jpn)
13. Nornes H. The role of intracranial pressure in the arrest of hemorrhage in patients with ruptured intracranial aneurysm. *J Neurosurg* 1973; 39: 226-234.
14. Rosenorn J, Westergaard K, Hansen PH. Mannitol-induced rebleeding from intracranial aneurysm. Case report. *J Neurosurg* 1983; 59: 529-530.
15. Hasan D, Vermeulen M, Wijdicks EFM et al. Management problems in acute hydrocephalus after subarachnoid hemorrhage. *Stroke* 1989; 20: 747-753.
16. Sundbarg G, Ponten U. ICP and CSF absorption impairment after subarachnoid hemorrhage. In: Beks JWF, Bosch DA, Brock M (eds). *Intracranial Pressure III*. Berlin: Springer-Verlag, 1976; 139-146.
17. Troupp H. Intracranial pressure in hydrocephalus after subarachnoid hemorrhage. *Zentralbl Neurochir* 1975; 36: 11-17.
18. Voldby B, Enevoldsen EM. Intracranial pressure changes following aneurysm rupture. Part 3: Recurrent hemorrhage. *J Neurosurg* 1982; 56: 784-789.
19. Pare L, Delfino R, Leblanc R. The relationship of ventricular drainage to aneurysmal rebleeding. *J Neurosurg* 1992; 76: 422-427.
20. Kawai K, Nagashima H, Narita K, Nakagomi T, Nakayama H, Tamura A, Sano K. Efficacy and risk of ventricular drainage in cases of grade V subarachnoid hemorrhage. *Neurol Res* 1997; 19: 649-653.
21. Steiger HJ, Poll A, Liepsch D, Reulen HJ. Basic flow structure in saccular aneurysms: a flow visualization study. *Heart Vessels* 1987; 3: 55-65.

Benign segmental myoclonus: electrophysiological evidence of transient dysfunction in the brainstem

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Summary We present a 66-year-old patient with segmental myoclonus evoked by a brainstem infarction. The myoclonus appeared soon after a cerebrovascular accident and it was evident in the soft palate, jaw, neck, shoulders and upper limbs. Brain MRI showed infarction in the left pons and left cerebellum. Small amounts of orally administered clonazepam were remarkably effective. Electroencephalogram (EEG) and auditory brainstem response (ABR) were normal. Somatosensory evoked potential (SSEP) revealed delays in P14 and N19 recorded at C3 by right median

nerve stimulation. These findings were normalized in 4 days. Segmental myoclonus is thought to be evoked by olfactory hypertrophy following cerebrovascular accident in the brainstem and is said to be resistant to medication. The limited involvement of the brainstem in our patient may account for the transient segmental myoclonus. The prognosis for this type of segmental myoclonus is excellent. © 2001 Harcourt Publishers Ltd.

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INTRODUCTION

Myoclonus is defined as abrupt, instantaneous, jerky involuntary movements unassociated with loss of consciousness.¹ Myoclonus can be evoked by various causes,² and is classified by its origin.¹ For example, lesions in the cerebral cortex, brainstem and spinal cord may cause myoclonus. Segmental myoclonus is characterized by rhythmic or arrhythmic involuntary contractions of muscle groups supplied by one or several contiguous segments of the brainstem or spinal cord.³ Palatal myoclonus is a special type of segmental myoclonus and appears exclusively in the soft palate.⁴ We report a patient with segmental myoclonus induced by a brainstem infarction with electrophysiological evidence of transient dysfunction in the brainstem.

CASE REPORT

A 66-year-old man, who had been treated for hypertension, received an operation for a varix in the left leg under epidural anaesthesia. The surgical procedure was successful, and the patient was discharged 5 days later. In the early morning of the first day after discharge, he noticed an abnormal movement in his neck, and visited the emergency room of our hospital. Neurological examination revealed vigorous rhythmic involuntary movement in the jaw, neck and shoulders. These movements were jerky, with rates varying from 120 to 180 oscillations/min. A series of abnormal involuntary movements appeared at a rate of 2-10/min with a duration of 1-2 s per series. These movements, which were distinct from tics, chorea, or other involuntary jerky movements, were diagnosed as myoclonus. Palatal myoclonus (PM) was also present, but negative myoclonus was not apparent. Additional neurological examinations revealed nothing abnormal. Sodium valproate (300 mg/day) was administered and he went home. On the evening of the same day, the involuntary movement became continuous, and the patient was again admitted to the emergency room. The distribution and the rates of myoclonus were unaltered, but the symptoms were aggravated. Myoclonus ceased completely upon intravenous administration of diazepam (10 mg). He continued to receive administration of oral clonazepam (1 mg/day). The next day, intermittent myoclonus was observed, and the day after that, he was admitted to our hospital. Neurological examination revealed a slight terminal tremor of the left arm, accentuation of right knee jerk, and positive Barré's sign in the right leg. The myoclonus had disappeared. Urinalysis and blood laboratory data showed no abnormality. Chest X-ray showed cardiomegaly. ECG revealed a right bundle branch block.