Thrombosed Basilar Apex Aneurysm Presenting as a Third Ventricular Mass and Hydrocephalus

Case Report and Review of the Literature

James K. Liu, M.D., Oren N. Gottfried, M.D.,
and William T. Couldwell, M.D., Ph.D.

From the Department of Neurosurgery, University of Utah School of Medicine, Salt Lake City, Utah

Please address correspondence to:
William T. Couldwell, M.D., Ph.D.
Department of Neurosurgery
University of Utah School of Medicine
30 North 1900 East, Suite 3B409
Salt Lake City, Utah 84132
Phone: (801) 581-6908
Fax: (801) 581-4385
e-mail: william.couldwell@hsc.utah.edu

Running head: Thrombosed basilar aneurysm presenting as a ventricular mass

Key Words: basilar apex aneurysm, ventricular mass, hydrocephalus, third ventriculostomy

Abstract

Aneurysms presenting as third ventricular masses are uncommon; most are giant aneurysms arising from the basilar apex. We present a case of a thrombosed basilar apex aneurysm presenting as a third ventricular mass and hydrocephalus in a 55-year-old man. The case is unique in the literature as the aneurysm was completely thrombosed and angiographically occult. The lesion was explored to verify the diagnosis and a third ventriculostomy resolved the patient's symptoms. Completely thrombosed aneurysms should be considered in the differential diagnosis of symptomatic third ventricular masses, even when angiographic studies are negative. The literature on aneurysms presenting as third ventricular masses is reviewed.

Introduction

Aneurysms of the basilar apex that present as third ventricular masses are uncommon. Most that have been reported in the literature were giant intracranial aneurysms [1-3,6,8,9,13,17], which are defined as those over 2.5 cm in diameter and comprise about 5% of all verified aneurysms [4,10]. Although not aneurysmal in origin, a severe ectatic basilar artery can also invaginate the floor of the third ventricle and mimic an intraventricular mass [15]. It is not uncommon for these lesions to cause obstruction of cerebrospinal fluid (CSF) flow resulting in hydrocephalus. We present a case of a completely thrombosed, large basilar apex aneurysm presenting as a third ventricular mass associated with hydrocephalus. Our case is unique in that the neuroimaging characteristics did not suggest a giant aneurysm as is typically reported in the literature. The aneurysm was smaller, completely thrombosed, and angiographically occult.

Case Report

History and Physical Examination

This 55-year-old man with hypertension, type II diabetes, and previously treated renal cell carcinoma presented with several weeks of progressive gait instability, urinary incontinence, and memory difficulties. He denied any headaches, nausea, vomiting, or visual disturbances. He had undergone a right nephrectomy three years previously for renal cell carcinoma and had since been in remission. Cranial nerve, motor, and sensory

examinations were within normal limits. The patient exhibited very ataxic gait and he was unable to tandem walk.

Neuroimaging Examination

A magnetic resonance (MR) imaging study revealed a 2-cm lesion located in the anterior aspect of the third ventricle just behind the foramen of Monro causing non-communicating hydrocephalus (Fig. 1). The lesion enhanced slightly with gadolinium and appeared to abut a very high-riding basilar bifurcation. A magnetic resonance angiogram (MRA) did not reveal any aneurysms (Fig. 2). Based on the proximity of the basilar artery and concern about whether this lesion represented a partially thrombosed aneurysm, catheter angiography was performed but did not demonstrate a patent basilar aneurysm (Fig. 3). The differential diagnosis at this time included an intraventricular metastatic tumor, possibly hemorrhagic metastatic disease given his previous history of renal cell carcinoma, or a thrombosed basilar apex aneurysm.

Operation

Given his history of renal cell carcinoma, the patient underwent an exploration of the third ventricular mass through a transcallosal approach for the purposes of diagnosis and CSF diversion with a third ventriculostomy. Resection of the ventricular mass was planned if a tumor was present.

The interhemispheric dissection was approached on the right side. An opening measuring approximately 1.5 cm was made in the corpus callosum. The left lateral ventricle was entered initially as the lesion presented itself more on the left side. CSF was

aspirated within the ventricle, and the hemosiderin-stained lesion was seen bulging in the region of the septum pellucidum and fornix, which grossly appeared consistent with a thrombosed aneurysm.

A very small opening was made at the apex of the lesion and a collection of cholesterol-laden crystals compatible with old hemorrhage as well as thrombus was removed and sent for pathological examination, which confirmed the presence of thrombus and fibrin. The wall of the lesion was consistent with collagenous tissue.

A small 3-mm aneurysm clip was placed across the opening of the thrombosed aneurysm. We then explored anteriorly, traversed the foramen of Monro and located the floor of the third ventricle just anterior to the mamillary bodies, and performed a third ventriculostomy at the region of the tuber cinereum. Free CSF flow was achieved and we could see the anterior aspect of the basilar artery. Closure of the craniotomy was performed in the standard fashion.

Postoperative Course and Follow-up

Postoperatively, the patient was awake with an intact motor, sensory, and cranial nerve examination. He was discharged to inpatient rehabilitation on the second postoperative day. At the 2-month follow-up, his gait had markedly improved and returned to normal, and his urinary difficulties had ceased. A computed tomography (CT) angiogram performed at 4-month follow-up, and subsequently at 12 months, demonstrated stable appearance of the thrombosed basilar aneurysm without evidence of recanalization. The ventricles had decreased in size.

Discussion

Literature Review

A review of the English literature revealed 10 cases (including the present case) of aneurysms presenting as symptomatic third ventricular masses (Table 1) [1-3,6,8,9,13,17]. The mean age of the patients was 62 years (range: 52 to 72 years) with a female-to-male ratio of 7:3. All aneurysms (except the present case) were giant intracranial aneurysms that were angiographically positive. The aneurysms arose mostly from the basilar artery (7 cases), although one case each occurred in the anterior communicating artery (Acomm), posterior communicating artery (Pcomm), and the middle cerebral artery (MCA). Six cases had evidence on neuroimaging of partial thrombosis and 3 cases had none. Our case was unique, having complete thrombosis of the basilar aneurysm. Nine cases presented with signs and symptoms of hydrocephalus, whereas one case presented with a massive intracerebral and intraventricular hemorrhage. The patient with the giant left MCA aneurysm also presented with seizures in addition to hydrocephalus. All patients received a form of CSF diversion. Of the 6 patients who had a good outcome, 5 had CSF diversion procedures without treatment of the aneurysm. There were 4 deaths (2 intraoperative; 2 directly related to aneurysm rupture).

The majority of these patients received a CT scan or MR scan as the initial study after clinical presentation. CT scans can readily demonstrate a third ventricular mass, size of the lesion, hydrocephalus, and sometimes, mural calcification [14]. MR imaging provides superior characterization of thrombus, flow phenomena, precise location within the ventricle, and the relationship of the lesion to the parent vessel [17]. Vascular imaging

studies are imperative to exclude the presence of an aneurysm. Conventional angiography provides reliable delineation of the aneurysmal lumen and its relationship to the parent vessel and adjacent arteries. However, MR angiography and/or CT angiography may provide complementary information in cases of partially thrombosed aneurysms where the mass of the aneurysm is much larger than what is seen on angiographic filling. Diagnosis becomes more difficult in completely thrombosed aneurysms that are angiographically occult. In cases where a third ventricular mass and an aneurysm are anatomically contiguous, the mass should be regarded as part of a partially thrombosed aneurysm until proven otherwise. Bose, et al. [3], reported a case in which failure to recognize a third ventricular mass as a partially thrombosed basilar apex aneurysm resulted in a fatal hemorrhage during operative resection of the mass.

In the present case, the aneurysm was completely thrombosed. Although conventional angiography did not show any remnant of an aneurysm, the third ventricular mass was closely associated with the apex of the basilar bifurcation.

Thrombosed Aneurysms: Persistent Growth and Rupture Risk

Persistent growth of completely thrombosed giant aneurysms has been reported [5,7,16]. Katayama, et al. [7], documented growth of a completely thrombosed vertebral artery–posterior inferior cerebellar artery junction aneurysm on serial imaging. Hirasawa, et al. [5], reported persistent growth of a giant basilar artery aneurysm after complete detachable balloon occlusion. It has been suggested that these giant aneurysms grow through repeated intramural hemorrhages independent of the continuity with the parent artery [16]. Autopsy studies have demonstrated numerous vascular channels and multiple

fresh intramural hemorrhages within the outer margin of the aneurysmal wall resulting in a characteristic onion-skin–like, laminated structure in the wall. It is tempting to hypothesize that the presentation of hydrocephalus in the present case may have been attributed to enlargement of this thrombosed aneurysm.

It has been argued whether intracranial giant aneurysms have less risk of rupturing and instead pose a higher risk of mass effect or ischemia. Some reports show a higher incidence of subarachnoid hemorrhage in giant aneurysms than previously expected [4,9,18]. Near-complete thrombosis of an aneurysm does not protect it from rupturing. Swearingen and Heros [19] reported a case of a patient who died from rupture of an aneurysm that was thought to be completely thrombosed. The angiogram demonstrated a small irregularity at the top of the basilar artery, which may have represented a tiny remnant of the aneurysmal neck. In the present case, the aneurysm was completely thrombosed, and there was no angiographic evidence of an aneurysm. Thus, there may be a low theoretical risk of rupturing.

CSF Diversion

In the review of the literature, 9 patients who presented with hydrocephalus were all treated with a form of CSF diversion. Four of these patients who underwent CSF diversion in the face of an unsecured aneurysm had a good outcome [2,8,13]. One patient hemorrhaged from a giant basilar aneurysm 1 month after having a right vertebral artery occlusion followed by an endoscopic septostomy for unilateral obstructive hydrocephalus [6]. This particular case raises the question whether a change in the CSF pressure from a diversion procedure can induce an unsecured aneurysm to rupture. Although some

evidence indicates CSF diversion may induce a ruptured aneurysm to rebleed [11,12], it is not clear whether the same occurs in unruptured aneurysms.

References

1. Babu VS, Eisen H (1979) Giant aneurysm of anterior communicating artery simulating 3rd ventricular tumor. Comput Tomogr 3: 159-163.
2. Borrie MJ, Campbell AJ, Caradoc-Davies TH (1985) Obstructive hydrocephalus secondary to giant cerebral aneurysm in the elderly: two case reports of functional improvement after ventricular shunting. J Am Geriatr Soc 33: 210-212.
3. Bose B, Northrup B, Osterholm J (1983) Giant basilar artery aneurysm presenting as a third ventricular tumor. Neurosurgery 13: 699-702.
4. Drake CG (1979) Giant intracranial aneurysms: experience with surgical treatment in 174 patients. Clin Neurosurg 26: 12-95.
5. Hirasawa T, Tsubokawa T, Katayama Y *et al* (1992) Growth of a giant aneurysm following complete thrombosis by detachable balloon occlusion. Surg Neurol 38: 283-286.
6. Hongo K, Morota N, Watabe T *et al* (2001) Giant basilar bifurcation aneurysm presenting as a third ventricular mass with unilateral obstructive hydrocephalus: case report. J Clin Neurosci 8: 51-54.
7. Katayama Y, Tsubokawa T, Miyazaki S *et al* (1991) Growth of totally thrombosed giant aneurysm within the posterior cranial fossa. Diagnostic and therapeutic considerations. Neuroradiology 33: 168-170.
8. Koga H, Mori K, Kawano T *et al* (1983) Parinaud's syndrome in hydrocephalus due to a basilar artery aneurysm. Surg Neurol 19: 548-553.
9. Koyama S, Kotani A, Sasaki J (1996) Giant basilar artery aneurysm with intramural hemorrhage and then disastrous hemorrhage: case report.

Neurosurgery 39: 174-177; discussion 177-178.

10. Morley TP, Barr HW (1969) Giant intracranial aneurysms: diagnosis, course, and management. Clin Neurosurg 16: 73-94.
11. Nornes H (1973) The role of intracranial pressure in the arrest of hemorrhage in patients with ruptured intracranial aneurysm. J Neurosurg 39: 226-234.
12. Pare L, Delfino R, Leblanc R (1992) The relationship of ventricular drainage to aneurysmal rebleeding. J Neurosurg 76: 422-427.
13. Piek J, Lim DP, Bock WJ (1983) Obstructive hydrocephalus caused by a growing, giant aneurysm on the upper basilar artery. Surg Neurol 20: 288-290.
14. Pinto RS, Kricheff, II, Butler AR *et al* (1979) Correlation of computed tomographic, angiographic, and neuropathological changes in giant cerebral aneurysms. Radiology 132: 85-92.
15. Rozario RA, Levine HL, Scott RM (1978) Obstructive hydrocephalus secondary to an ectatic basilar artery. Surg Neurol 9: 31-34.
16. Schubiger O, Valavanis A, Wichmann W (1987) Growth-mechanism of giant intracranial aneurysms; demonstration by CT and MR imaging. Neuroradiology 29: 266-271.
17. Smith KA, Kraus GE, Johnson BA *et al* (1994) Giant posterior communicating artery aneurysm presenting as third ventricle mass with obstructive hydrocephalus. Case report. J Neurosurg 81: 299-303.
18. Sonntag VK, Yuan RH, Stein BM (1977) Giant intracranial aneurysms: a review of 13 cases. Surg Neurol 8: 81-84.
19. Swearingen B, Heros RC (1985) Fatal rupture of a thrombosed giant basilar artery

aneurysm. Surg Neurol 23: 299-302.

Table 1

Aneurysms Presenting as Third Ventricular Masses: Review of the Literature

Author	Age, Sex	Aneurysm	Size	Thrombosis	Presentation	Treatment	Outcome
Koga et al.	65, F	BA	Giant	Partial	Hydrocephalus	VP shunt	Good
Piek et al.	60, F	BA	Giant	None	Hydrocephalus	VP shunt	Good
Babu et al.	52, M	Acomm	Giant	Partial	Hydrocephalus	VP shunt, surgical ligation of aneurysm	Death (intraoperative cardiac arrest)
Bose et al.	55, F	BA	Giant	Partial	Hydrocephalus	VP shunt, transcallosal removal of "tumor"	Death (intraoperative hemorrhage)
Hongo et al.	70, F	BA	Giant	None	Hydrocephalus	RVA occlusion, endoscopic septostomy	Death (aneurysm rupture)
Koyama et al.	67, F	BA	Giant	Partial	SAH, IVH	EVD	Death (aneurysm rupture)
Smith et al.	60, F	Pcomm	Giant	Partial	Hydrocephalus	Bilateral EVD/VP shunts, aneurysm clipping/thrombectomy	Good
Borrie et al. (2 cases)	72, F	BA	Giant	None	Hydrocephalus	VP shunt	Good
	70, M	Left MCA	Giant	Partial	Seizures, hydrocephalus	VA shunt	Good
Present case	55, M	BA	Large	Complete	Hydrocephalus	Transcallosal biopsy, third ventriculostomy	Good

BA = basilar artery; Acomm = anterior communicating artery; Pcomm = posterior communicating artery; MCA = middle cerebral artery; VP = ventriculoperitoneal; RVA = right vertebral artery; EVD = external ventricular drain.

Figure Legends

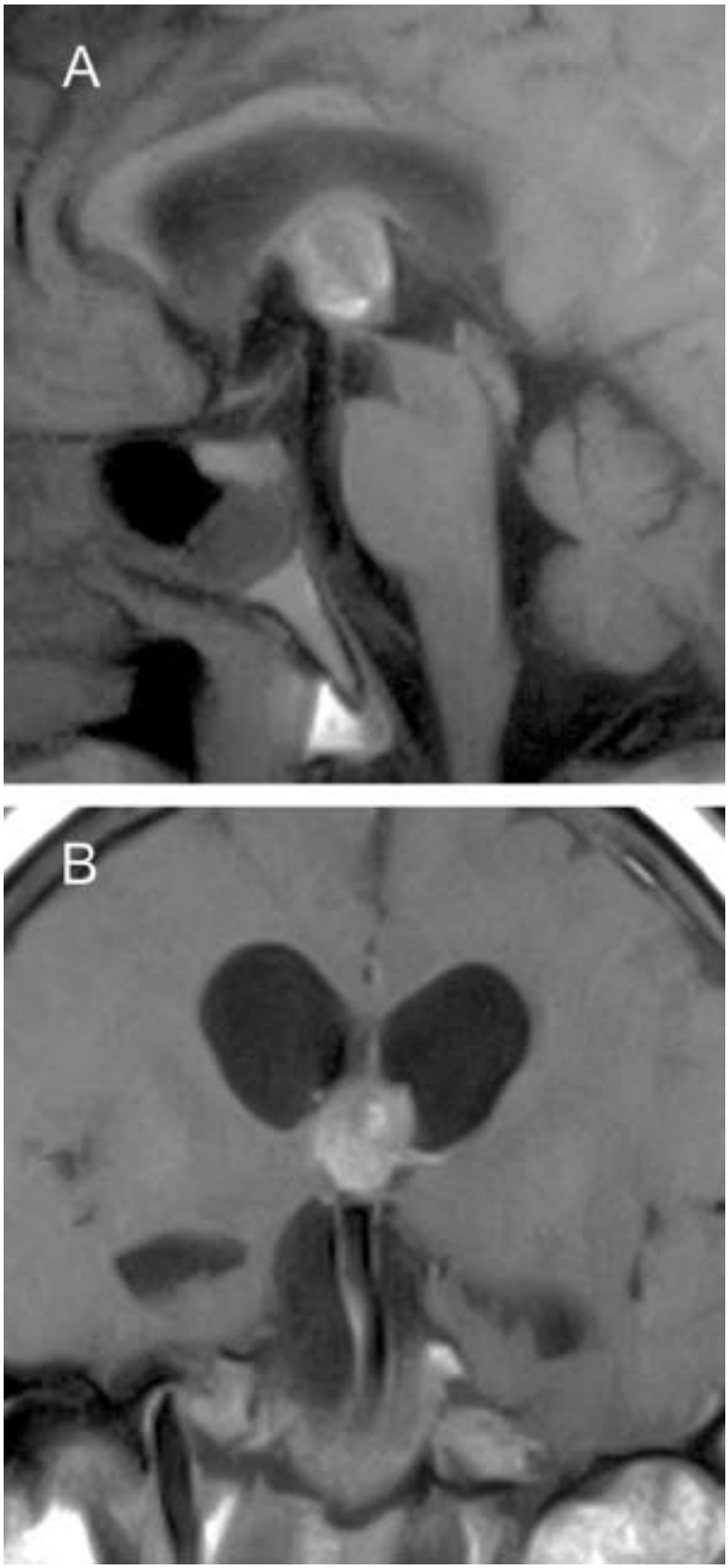

Fig. 1. MR imaging demonstrating mildly enhancing third ventricular mass and non-communicating hydrocephalus. The mass appears to abut the basilar apex on the sagittal and coronal images. A: Sagittal T1-weighted pre-gadolinium MR image; B: coronal T1-weighted post-gadolinium images.

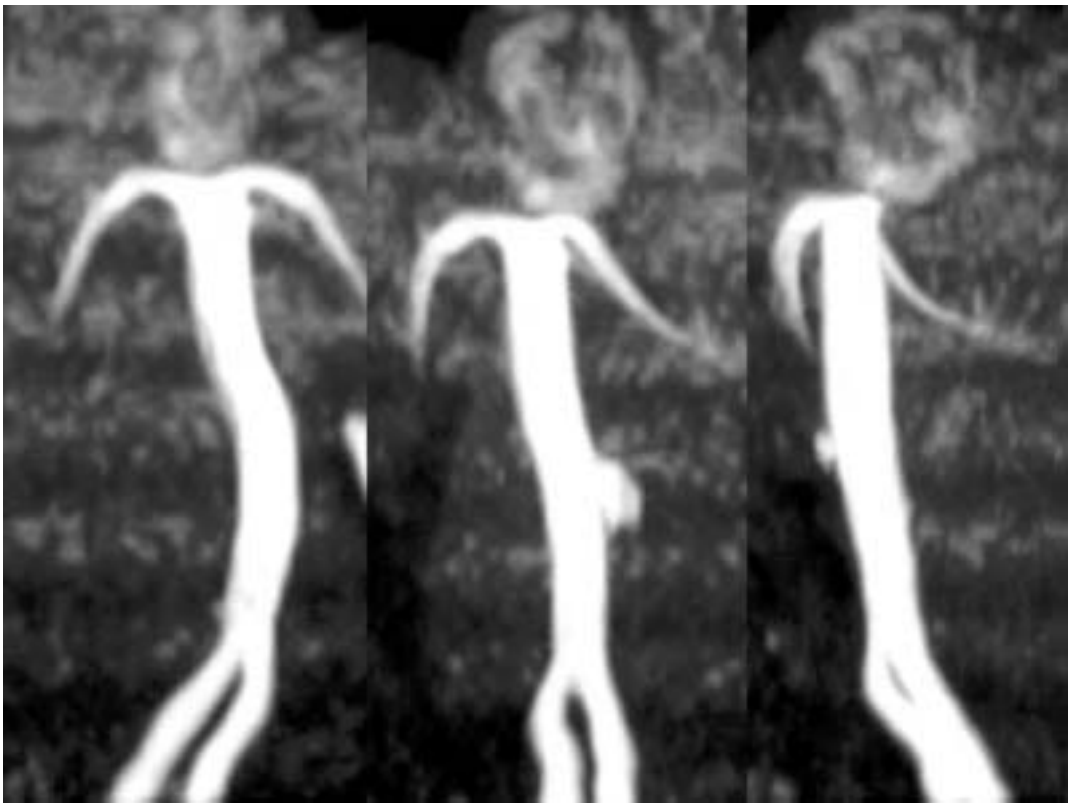

Fig. 2. MR angiogram. There is no evidence of filling of a basilar apex aneurysm. The third ventricular mass is appreciated just above the basilar apex.

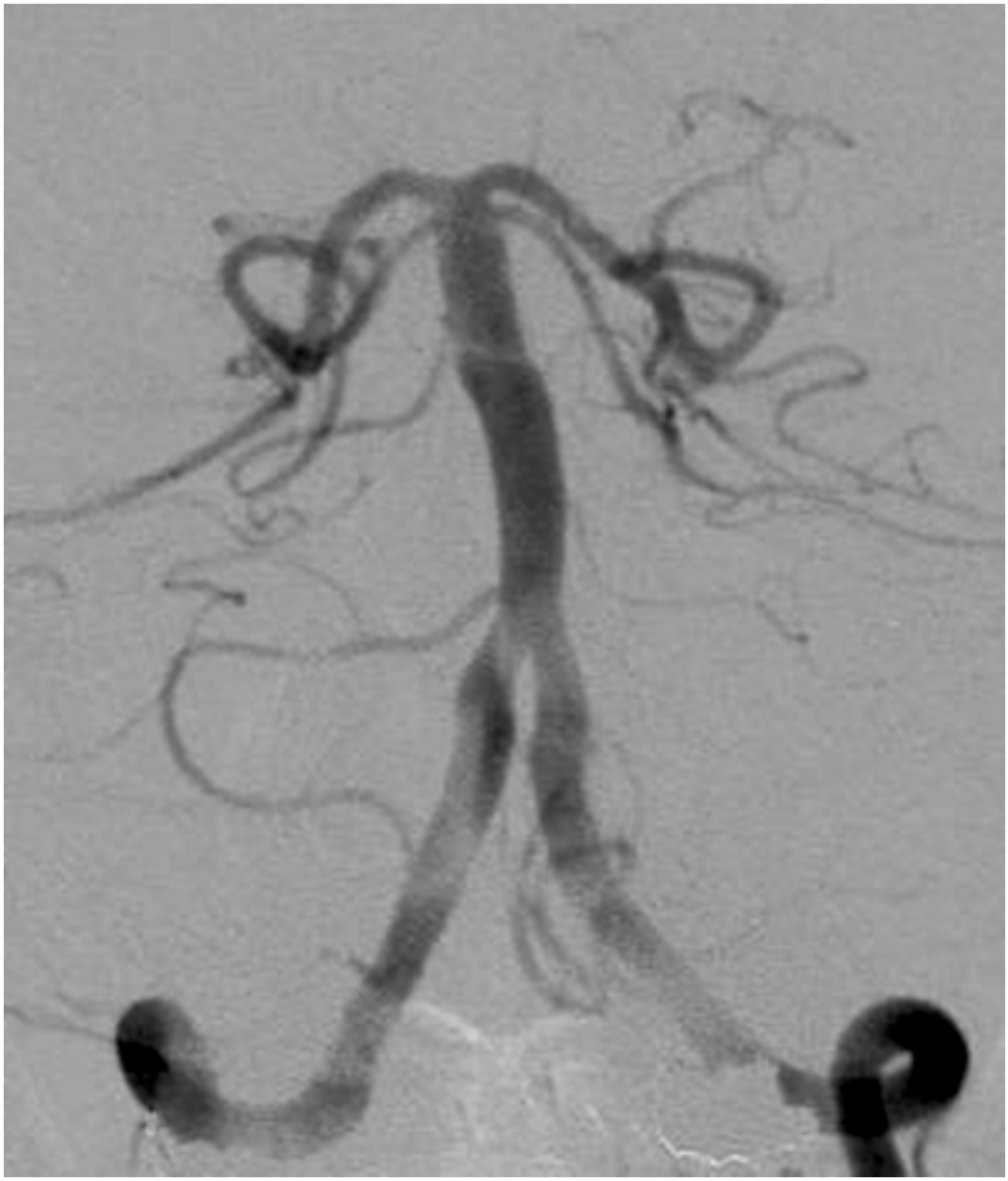

Fig. 3. Cerebral angiogram. There is no evidence of filling of a basilar apex aneurysm.