Ruptured fusiform aneurysm of the superior third of the basilar artery associated with the absence of the midbasilar artery

Case report

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A case involving the absence of the midportion of the basilar artery (BA) associated with a ruptured fusiform aneurysm of the superior third of the basilar artery discovered after a subarachnoid hemorrhage is reported. Surgical clipping was precluded by the anatomical conditions. The aneurysm was treated by occlusion (surgical clipping and balloon occlusion) of both posterior communicating arteries to decrease the hemodynamic stress on the aneurysm wall. The pericerebellar arterial network was allowed to supply the distal BA and its collateral vessels indirectly. This treatment proved to be efficient; angiography and magnetic resonance imaging demonstrated shrinkage of the aneurysm cavity. The absence of the midportion of the BA is usually associated with a persisting trigeminal artery (nonexistent in this case) or disclosed in cases of acute BA occlusion in dramatic clinical conditions. A similar anatomical feature has been described only once before. There may be a segmental maldevelopment of the longitudinal neural arteries during embryogenesis or a defect in fusion of these paired structures during the development of the BA itself.

KEY WORDS • basilar artery • basilar artery aplasia • aneurysm • subarachnoid hemorrhage • embolism • posterior communicating artery • thalamus

Parent vessel occlusion is responsible for a major decrease in blood flow within an aneurysm and may be a therapeutic alternative when the malformation cannot be clipped or selectively embolized. We applied such a therapeutic modality by obstructing both posterior communicating arteries (PCoAs) in the case of a ruptured fusiform aneurysm of the superior third of the basilar artery discovered after a subarachnoid hemorrhage. Surgical clipping was precluded by the anatomical conditions. The aneurysm was treated by occlusion (surgical clipping and balloon occlusion) of both posterior communicating arteries to decrease the hemodynamic stress on the aneurysm wall. The pericerebellar arterial network was allowed to supply the distal BA and its collateral vessels indirectly. This treatment proved to be efficient; angiography and magnetic resonance imaging demonstrated shrinkage of the aneurysm cavity. The absence of the midportion of the BA is usually associated with a persisting trigeminal artery (nonexistent in this case) or disclosed in cases of acute BA occlusion in dramatic clinical conditions. A similar anatomical feature has been described only once before. There may be a segmental maldevelopment of the longitudinal neural arteries during embryogenesis or a defect in fusion of these paired structures during the development of the BA itself.

Case Report

This 42-year-old man with no known medical history was admitted to our institution after experiencing a subarachnoid hemorrhage (SAH). On admission the patient was comatose with a Glasgow Coma Score of 8.

Examination. Computerized tomography (CT) scanning revealed significant bleeding at the level of the basilar cisterns, involving mainly the interpeduncular cistern. Cerebral angiography (Fig. 1) demonstrated major abnormalities of the vertebrobasilar system. A large fusiform aneurysm involved the superior third of the BA including the origins of the superior cerebellar arteries (SCAs). The distal BA was supplied exclusively by both PCoAs and opacified the origin of the posterior cerebral arteries (PCAs) and the SCA. The midportion of the BA was not seen to be opacified on vertebral or internal carotid angiograms. Vertebral arteries supplied the posterior inferior cerebellar arteries (PICAs) and the anterior inferior cerebellar arteries (AICAs). The lumen of the inferior third of the BA was irregular. Magnetic resonance imaging was performed 3 weeks after the SAH using flow-sensitive sequences that demonstrated the two components of the lesion: a thrombus and a circulating compartment (Fig. 2). The imaging confirmed the absence of the midportion of the BA. No ischemic lesions were visible within the brainstem. A segmental aplasia of the BA associated with a ruptured aneurysm of the distal BA was diagnosed.
Considering the lack of an aneurysm neck and the proximity to the origin of the PCAs and SCAs, surgical clipping was precluded. Posterior communicating artery occlusion was therefore proposed to decrease the hemodynamic stress applied on the aneurysm wall. Treatment was delayed for 3 weeks because neurological evaluation was impossible as long as the patient was comatose. His clinical status progressively improved, and his neurological responses became normal and reliable, allowing the performance of an endovascular occlusion test. Left and right internal carotid artery (ICA) temporary balloon occlusions, at the level of the PCoAs, were successively performed and were well tolerated.

First Operation. In view of the risks involved with a bilateral surgical approach to the PCoAs, we decided to limit surgery to one side and to perform endovascular occlusion on the opposite side. The choice of the left side for surgery was made with consideration of the origin of the anterior choroidal artery (AChA) from the right PCoA. This latter anatomical variant was judged to be at potentially higher surgical risk than the disposition on the left side. An endovascular approach was therefore made on the right side. Surgical clipping of the left PCoA was performed 24 hours after a temporary balloon occlusion test of the left ICA.

Fig. 1. Upper Left: Left internal carotid artery (ICA) angiogram, lateral view, showing massive opacification of the upper basilar artery (BA) by a patent posterior communicating artery (PCoA). Notice the position of the aneurysm dilation situated immediately below the origin of the PCoAs. Upper Right: Right ICA angiogram, anteroposterior view, demonstrating the bilateral supply of PCoAs and superior cerebellar arteries as well as the irregularly narrowed trunk of the BA below the aneurysm cavity. Lower Left and Lower Right: Lateral and anteroposterior views after vertebral artery injection. Note the lack of opacification of the midthird of the BA in both views.

Fig. 2. Left: Gradient echo magnetic resonance (MR) study performed 3 weeks after hemorrhage: axial slices performed at the level of the aneurysm rupture. Notice the signal intensity of the circulating compartment surrounded by subacute clot and the compression of the brainstem. Right: Gradient echo MR study, axial view, at the level of the pons demonstrating the lack of a vascular signal or hematoma.
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First Postoperative Course. The patient’s postoperative course was marked by thermal and behavioral disorders. Postoperative CT and MR imaging showed anterior thalamic infarction (Fig. 3) probably due to occlusion of the collateral vessels following the clipping of the left PCoA. Follow-up angiography showed patency of the left AChA (Fig. 4). In view of the small caliber of the right PCoA and the potential for catastrophic ischemic complications with intraluminal navigation and deposition of occluding material within this vessel, we considered the occlusion of the ICA at the level of the PCoA to be a safer method, although constituting a heavy vascular sacrifice.

Second Operation. Temporary occlusion of the right ICA was performed by a bilateral femoral approach 2 weeks after the first surgery according to the following protocol: with the patient mildly sedated and receiving heparin, a detachable balloon was positioned at the origin of the right PCoA. The right ICA territory was supplied by the left ICA via the anterior communicating artery.
Clinical testing was performed every 5 minutes. Opacification of the vertebral arteries and of the left ICA allowed us to assess the patency of arterial collateral supplies; the pericerebellar arterial network made it possible to opacify the distal BA and its collateral vessels (Fig. 6). Visual, auditory, and somatosensory evoked potentials were recorded throughout the procedure. After 60 minutes, both neurological status and evoked potentials remained normal. Permanent ICA balloon occlusion was then performed.

**Second Postoperative Course.** To prevent extensive thrombosis within the lumen of the aneurysm, the patient continued to receive heparin therapy for 3 weeks. Six months after the SAH, the patient resumed his normal professional occupation. Six-month follow-up MR imaging demonstrated a decrease of the flow-related signal within the aneurysm cavity (Fig. 7) and angiography performed 1 year posttreatment showed additional shrinkage (Fig. 8).

**Discussion**

The lack of patency of the middle portion of the BA was considered to be secondary to one of the following potential causes: acquired BA occlusion; spontaneous dissection of the BA; or partial agenesis of the BA.

Remote BA occlusion could not be completely ruled out, although MR imaging failed to disclose either ischemic lesions of the brainstem or associated vascular lesions. Similar angiographic features have already been widely described in acquired BA occlusion in children and adults.\(^2,5,7\) In all such reported cases, however, a history of neurological deficit was always evident. Dissection of the middle third of the BA was considered because occlusion has been seen after SAH. However, SAH usually complicates subadventitial dissection, with angiograms showing aneurysm dilation at the site of the lesion.\(^1\) This hypothesis was rejected, however, because MR imaging failed to demonstrate any vascular signal and/or hematoma at the level of the midbasilar artery.

Basilar artery aplasia has previously been described by Lasjaunias, *et al.,*\(^4\) in a 15-year-old patient suffering from hydrocephalus. The BA abnormality was asymptomatic. The distal BA and collateral vessels were similarly supplied by the left PCoA, but the AICAs originated from the distal BA. Unlike in the case reported by Lasjaunias, *et al.,* angiography failed to reveal any moyamoya network in our patient, but it did show a similar irregularly narrowed lumen at the transition zone between the patent and aplastic BA.

The embryological origin of this abnormality remains unknown. The most common cause of BA discontinuity is a persistent trigeminal artery, which was not found in our case. According to Padget\(^8\) and Gillilain,\(^3\) BA discontinuity results from fusion of the longitudinal neural arteries (LNAs). These paired structures are in fact plexiformed by the cranial members of the segmental branches of the ICA and hypoglossal arteries. Longitudinal neural arteries are supplied cranially by the trigeminal division of the carotid artery and variably reinforced by the primitive otic and hypoglossal arteries. After regression of the trigemi-
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Of the arterial supply to the thalamus via the AChA, we can study in a microvascular anatomical study by Percheron. Subarachnoid haemorrhage reported in seven cases. J Neuroradiol 21:1–16, 1994


5. Lasjaunias P, Manelfe C, Roche A, et al: Segmental aplasia of the distal BA. This hypothesis may explain the lack of previous neurological symptoms.

Anterior thalamic artery infarction occurred following surgical clipping. Arterial supply to the thalamus via the PCoA has been demonstrated in 60% to 70% of the brains studied in a microvascular anatomical study by Percheron. The anterior polar thalamic artery, a single well-individualized vessel, is visible in 21.6% of the PCoAs studied. This vessel originates from the middle third of the PCoA, regardless of its size. In case of extensive territory, this artery may supply the reticularis, lateralis and polaris nuclei, the paraventricular area, and a portion of the mamillothalamatic tract. The vascular territory of the anterior polar thalamic artery is compatible with the area of infarction demonstrated on our postoperative MR imaging study. To our knowledge this vessel is not visible on angiography.

The leptomeningeal anastomoses of the cerebellar arteries were clearly demonstrated, indicating collateral blood supply to the distal BA. This relationship has been previously reported in anatomical and clinicangioographic studies. Nevertheless, radiographic demonstration of collateral supply cannot be considered a reliable proof of primary circulation interruption tolerance. Therefore, additional data obtained by a prolonged clinical test and evoked potentials during right ICA occlusion were necessary; they proved to be reliable in view of the follow-up results.

In conclusion, by obstructing the PCoAs, we were able to decrease hemodynamic stress on a ruptured aneurysm that developed at the level of the distal third of the BA under unusual anatomical conditions. The efficiency of anastomoses of the pericerebellar arterial network as well as the shrinkage of the aneurysm cavity were well demonstrated. Nevertheless, the definitive benefit of this therapeutic modality in such a rare situation requires long-term confirmation by means of MR imaging studies and complementary angiography.

References


Manuscript received December 18, 1995.
Accepted in final form May 20, 1996.
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