Fornix infarction and Korsakoff dementia after coiling of a large anterior communicating artery aneurysm

Pascal John Mosimann, MD
Jean-Pierre Saint-Maurice, MD
Stéphanie Lenck, MD
Francesco Puccinelli, MD
Emmanuel Houdart, MD

Surgical occlusion of the anterior communicating artery (ACoA) can cause Korsakoff-like dementia, characterized by acute confusion, severe anterograde amnesia, and cognitive-behavioral changes, also known as the ACoA syndrome (ACoAS).¹ Surprisingly, this usually does not occur after deliberate endovascular ACoA occlusion, which is occasionally performed to reduce the high recurrence rate of broad-based ACoA aneurysms treated by coiling.² We present a case of bilateral fornix infarction and Korsakoff dementia after coil embolization and parent vessel occlusion of a large ACoA aneurysm. We emphasize the underlying anatomy of a poorly known perforator, the subcallosal artery (SubCA).

Case report

A 47-year-old man was admitted for a Hunt and Hess grade 1 subarachnoid hemorrhage from a large, broad-based ACoA aneurysm at the right A1–A2 junction, which was uneventfully coiled. Six months later, significant recanalization occurred (figure, A). Clipping was refused by the patient, who opted for endovascular retreatment. Given the absence of obvious perforators and well-developed A1 segments, the ACoA was deliberately occluded with a good angiographic result (figure, B–D). Despite the absence of focal neurologic deficits, persistent confusion and memory impairment was observed. MRI showed bilateral infarcts of the anterior fornix, septum pellucidum, and corpus callosum (CC) genu (figure, E and F). Neuropsychological assessment revealed severe anterograde and moderate retrograde amnesia, paramnesias, dysphoria, and a dysexecutive syndrome, characteristic for Korsakoff dementia. Despite intensive rehabilitation, only minor progress was observed 24 months later.

DISCUSSION

The fornix connects the hippocampus to the mammillary body and plays a major role in declarative memory formation and consolidation. ACoA perforator microangiopathy is a rare

Service de Neuroradiologie, Hôpital Lariboisière, Paris, France.

Correspondence to: emmanuel.houdart@lrb.aphp.fr

Figure Pre- and post-therapeutic imaging

(A) Six-month angiogram in frontal view shows significant recurrence after complete coil embolization of a large broad-based anterior communicating artery (ACoA) aneurysm. (B-D) Good angiographic result after retreatment and ACoA sacrifice. (E) Axial diffusion-weighted and (F) fluid-attenuated inversion recovery MRI show acute bilateral fornix infarction extending to the septum and genu of the corpus callosum but sparing the body and splenium, corresponding to the territory of the subcallosal artery.

cause of bilateral fornix infarction and anterograde amnesia.^{1,3-5} Such a complication is rare after endovascular obliteration of a broad-based ACoA aneurysm with intentional ACoA occlusion.

On average, there are 6 ACoA perforators arising from the posterior aspect of the artery. They can be classified into 3 groups according to the territory they supply⁶: 1) chiasmatic, 2) hypothalamic, and 3) subcallosal. The absence of ischemia after occlusion of the 2 former relates to dual supply from branches of the anterior cerebral artery (ACA) and internal carotid artery (ICA). The anterior subcallosal region, on the other hand, has no collateral supply and is vascularized exclusively by a single ACoA perforator, the subcallosal artery (SubCA), which vascularizes the anterior columns of the fornix bilaterally, the rostrum and genu of CC, the

septum pellucidum, and the anterior commissure. One group previously acknowledged its importance by renaming the ACoAS the amnesic syndrome of the SubCA.⁷

The SubCA, despite being the largest perforator, remains a small terminal artery (0.5 mm on average). Although it usually remains angiographically occult, one should always assume it to be present. Situations where preoperative nonvisualization may be reassuring are when a full-sized MACC or azygos ACA are present, since these directly supply the SubCA territory. This relates to the differential development of their common embryologic precursor, the median rostral artery, which basically develops into 1 of 3 mutually exclusive variants: the SubCA, the MACC, or the azygos ACA. Although the 2 former share an identical cisternal course, the MACC is larger and longer and extends more dorsally and posteriorly toward the splenium. Conversely, the SubCA terminates before or at the genu. It can therefore be viewed as a hypoplastic MACC, explaining the more limited, yet functionally catastrophic infarction pattern associated with its occlusion.^{4,5}

The risk of ACoAS is relatively high after surgery of ACoA aneurysms. One consecutive series of 48 patients reports an ACoAS rate >80% when the ACoA is trapped and >15% when the ACoA is preserved and the aneurysm selectively clipped.¹ Intentional endovascular ACoA occlusion, on the other hand, is commonly performed with impunity to treat wide-necked ACoA aneurysms when 2 A1 segments and no ACoA perforators are identified.² The degree of occlusion after endovascular ACoA obliteration, however, is probably not as tight as presumed. Some residual nonopacified flow may still fill the SubCA from the edges of the ACoA bordering the protruding coil mass. In large aneurysms, a more “surgical type” of ACoA occlusion may occur due to a wider prolapsing coil mass, thereby increasing the risk of SubCA infarction. Stenting of the ACoA rather than parent artery occlusion may reduce the risk of SubCA occlusion and Korsakoff dementia, as well as aneurysmal recurrence, although this remains to be shown for broad-based aneurysms >10 mm. Surgery might also be considered when the dome projects anteriorly.

REFERENCES

1. Gade A. Amnesia after operations on aneurysms of the anterior communicating artery. *Surg Neurol* 1982;18:46–49.
2. Fang C, Li MH, Zhu YQ, Tan HQ, Zhang PL, Wang J. Treatment of complex ruptured anterior communicating arterial aneurysms by endovascular coil embolisation of the anterior communicating artery. *Br J Neurosurg* 2011;25:591–595.
3. Adamovich BL, Gualberto G, Roberts T, Haut MW, Gutmann L. Teaching NeuroImages: amnesia due to fornix infarction. *Neurology* 2009;73:e86.
4. Moudgil SS, Azzouz M, Al-Azzaz A, Haut M, Gutmann L. Amnesia due to fornix infarction. *Stroke* 2000;31:1418–1419.
5. Park SA, Hahn JH, Kim JI, Na DL, Huh K. Memory deficits after bilateral anterior fornix infarction. *Neurology* 2000;54:1379–1382.
6. Serizawa T, Saeki N, Yamaura A. Microsurgical anatomy and clinical significance of the anterior communicating artery and its perforating branches. *Neurosurgery* 1997;40:1211–1216; discussion 1216–1218.
7. Moussouttas M, Giacino J, Papamitsakis N. Amnesic syndrome of the subcallosal artery: a novel infarct syndrome. *Cerebrovasc Dis* 2005;19:410–414.

DISCLOSURES

The authors report no disclosures relevant to the manuscript. Go to Neurology.org/cp for full disclosures.