

Ruptured Internal Carotid Artery Aneurysm at the Origin of a Perforating Artery Associated With a Hyperplastic Anomalous Anterior Choroidal Artery

—Case Report—

Kouichi MISAKI, Naoyuki UCHIYAMA,
Hisashi NITTA*, and Jun-ichiro HAMADA

Department of Neurosurgery, Graduate School of Medical Science,
Kanazawa University, Kanazawa, Ishikawa;

*Department of Neurosurgery, Komatsu Municipal Hospital, Komatsu, Ishikawa

Abstract

A 65-year-old man presented with a hyperplastic anomalous anterior choroidal artery (AChA) associated with a ruptured internal carotid artery aneurysm at the origin of a perforating artery manifesting as sudden onset of headache and vomiting. The aneurysm was too small for endovascular embolization, so we performed open surgery via the left pterional approach. Aneurysm clipping with preservation of the perforator was impossible, so we clipped the aneurysm neck and sacrificed the perforator. We also performed dome clipping because dome puncture resulted in continuous bleeding. Head computed tomography obtained 3 days after the operation showed cerebral infarction at the territory of the sacrificed perforator, but the patient suffered no neurological deficits. This case of internal carotid artery aneurysm with a perforating artery arising from the aneurysm dome shows that sacrifice of the perforator may be necessary to prevent rebleeding.

Key words: hyperplastic anomalous anterior choroidal artery, internal carotid artery aneurysm, perforating artery, subarachnoid hemorrhage, cerebral infarction

Introduction

The anterior choroidal artery (AChA) normally supplies the choroid plexus of the trigone, the anteromedial part of the temporal lobe, and the lateral geniculate body, uncus, and optic tract.^{1,9,10} However, an anomalously enlarged AChA also supplying the temporal, occipital, and parietal lobes is known, and is named the duplication of the posterior communicating artery (PComA) or the second posterior cerebral artery.^{1,9,10,12} This hyperplastic anomalous AChA is characterized by the following angiographic findings: the course of the proximal portion is the same as the cisternal portion of the AChA, a choroidal branch of the anomalous AChA supplies the choroid plexus of the trigone, no other artery originated from the supraclinoid part of the internal carotid artery (a few mm above the origin of the PComA), and the temporal and/or calcarine branches of the posterior cerebral artery are absent or hypoplastic.⁹ Only one such hyperplastic anomalous AChA was encountered among 200 cadaver brain dissections,¹² and the incidence on bilateral carotid angiography is 1.1–2.3%.^{9,10} Anomalous AChA has been associated with intracranial aneurysms.^{1,4,6,8–11}

We treated a patient with a ruptured aneurysm at the origin of a perforating artery arising from the internal carotid artery associated with anomalous AChA, that necessitated sacrifice of the perforator to prevent rebleeding.

Case Report

A 65-year-old man with no history of hypertension was hospitalized for sudden onset of headache and vomiting. He had no neurological deficits except for confused verbal responses (Hunt and Kosnik grade 2). Computed tomography (CT) showed subarachnoid hemorrhage in the basal cisterns and bilateral sylvian fissures (Fig. 1). Cerebral angiography revealed a left saccular internal carotid artery aneurysm measuring 4 mm in maximum diameter, with a fine blood vessel emerging from the aneurysm (Fig. 2A). Development of a treatment strategy required clarification of the distribution of this vessel, so angiography was obtained at a site just distal to the aneurysm orifice, taking care not to rupture the aneurysm. The artery formed an internal loop and divided into several small vessels before changing direction toward the basal ganglia (Fig. 2C, D), which identified the vessel as a perforating branch to the basal ganglia. Another vessel resem-

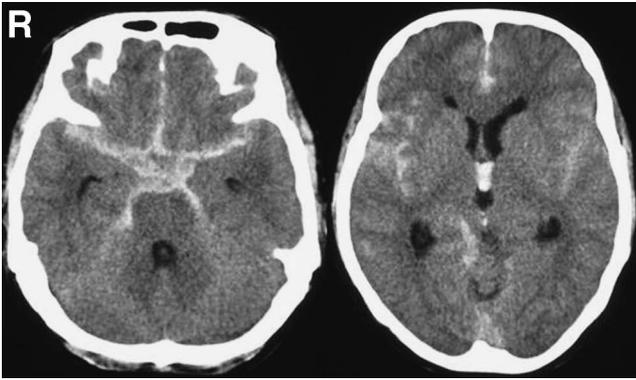


Fig. 1 Head computed tomography scans on admission showing subarachnoid hemorrhage in the basal cisterns and bilateral sylvian fissures, but no low density area in the left internal capsule.

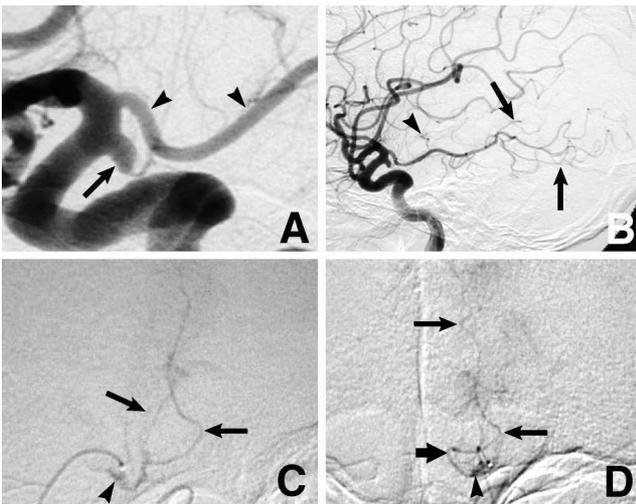


Fig. 2 A, B: Left internal carotid angiograms (lateral view) revealing (A) a sacular aneurysm (arrow) and an anomalous hyperplastic anterior choroidal artery (arrowheads), and (B) the anterior choroidal artery supplying a choroidal branch (arrowhead) and cortical temporo-occipital branches (arrows). C, D: Angiograms of the site just distal to the aneurysm orifice (C: lateral view, D: anteroposterior view) showing perforating arteries branching from the aneurysm dome (arrowheads). The perforator forms an internal loop (thick arrow), then divides into several small vessels and changes direction toward the basal ganglia (arrows).

bling a fetal PComA bifurcated from the internal carotid artery at a point 4 mm distal from the aneurysm, and supplied the choroid plexus and temporo-occipital lobe (Fig. 2B). Three-dimensional CT angiography showed no communication with the ipsilateral posterior cerebral artery (Fig. 3), so we considered this vessel to be an anomalous hyperplastic AChA. Neither left internal carotid nor vertebral angiography under compression of the left cervical carotid artery (Allcock test) depicted the left PComA.

The aneurysm was too small for endovascular emboliza-

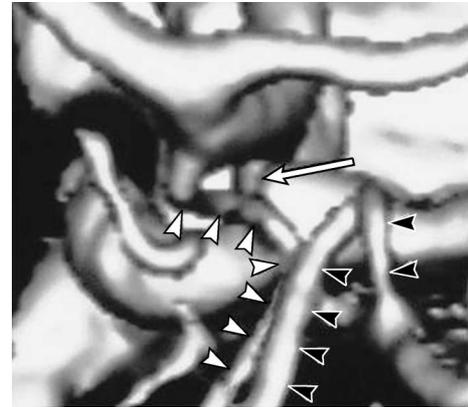


Fig. 3 Three-dimensional computed tomography angiogram demonstrating a saccular left internal carotid artery aneurysm (arrow), and the anomalous anterior choroidal artery (white arrowheads) not communicating with the ipsilateral posterior cerebral artery (black arrowheads).

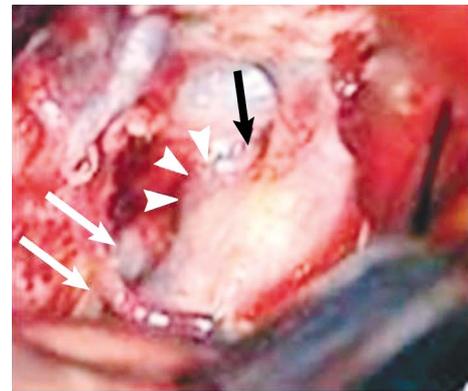


Fig. 4 Operative photomicrograph showing the left internal carotid artery aneurysm (arrowheads), the origin of the perforating artery branching from the aneurysm dome (black arrow), and the anomalous hyperplastic anterior choroidal artery (white arrows).

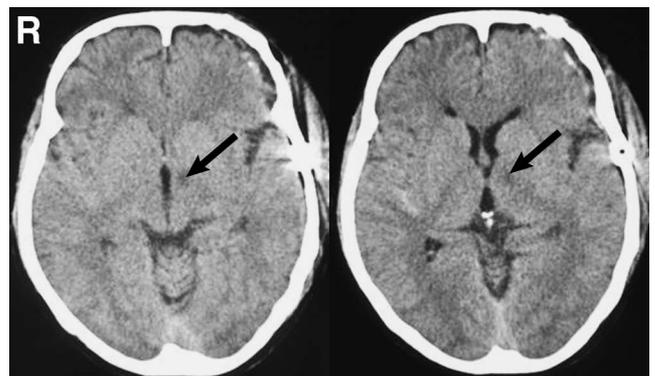


Fig. 5 Head computed tomography scans obtained 3 days after the operation showing cerebral infarction at the left genu of the internal capsule (arrows).

tion, so we performed open surgery via the left pterional approach on the day after onset. A ruptured aneurysm was observed at a site proximal to the origin of the anomalous AChA. There was no vessel between the internal carotid artery and the aneurysm, indicating that aneurysm was an internal carotid artery-perforating artery bifurcation aneurysm (Fig. 4). The perforator originated from the top of the aneurysm dome, so clipping with preservation of the perforator was not possible. Therefore, we applied a clip to the aneurysm neck. Dome puncture resulted in continuous bleeding from the perforating artery, so additional dome clipping was performed. After the clipping, careful observation around the aneurysm could not identify the PComA.

Postoperative angiography demonstrated no aneurysm or perforating artery. Head CT obtained 3 days after the operation revealed cerebral infarction at the left genu of the internal capsule, considered to be the territory of the sacrificed perforator (Fig. 5). However, the patient manifested no neurological deficits and he was discharged 24 days after the onset.

Discussion

The AChA develops at an earlier embryological stage than the PComA, and provides the main cortical supply to the posterior part of hemisphere as well as the choroidal branch at an embryonic crown-rump length of 4–6 mm.^{1,11)} As the PComA develops and the blood flow in the posterior cerebral artery increases, the cortical area formerly supplied by the AChA is fed by these vessels. If the PComA and the posterior cerebral artery fail to take over the supply of the posterior part of the hemisphere, the cortical branches of the AChA may persist as the main feeders of the temporal, occipital, and parietal lobes.^{1,10,11)} This embryological mechanism may explain the presence of the anomalous hyperplastic AChA without obvious PComA in our patient.

Only 9 cases with coexisting aneurysms and confirmed location have been reported (Table 1).^{1,4,6,8–11)} Among the 13 aneurysms associated with anomalous AChA, 6 aneurysms were located at the origin of the hyperplastic AChA, and 7 were located beside this artery. Twenty-five

anomalous hyperplastic AChA were identified in 23 individuals on 2216 carotid angiograms (1.1%)¹⁰⁾; and 6 aneurysms were found in these patients with an anomalous AChA. Four aneurysms were recognized at a site other than the AChA, which indicated that 4 of 23 patients with hyperplastic AChA had remote aneurysms. Hyperplastic AChA seems to be a structural anomaly like persistent trigeminal artery, azygous anterior cerebral artery, and fenestration of the intracranial vessels, which are associated with increased incidence of aneurysm formation even in vessels other than the AChA.^{6,8)} Another presumed mechanism of aneurysm formation is hemodynamic stress. The perforating artery from the aneurysm was clearly depicted by preoperative angiography and had several branches, indicating that the perforator supplied a relatively large area. The relatively abundant blood flow of the perforating artery might contribute to the hemodynamic etiology of aneurysm formation.

No case of aneurysm formed at the origin of an internal carotid artery perforator has previously been reported. Although we have no angiographic or surgical evidence for the presence of a PComA, we think that the perforator originated from the communicating segment of the internal carotid artery that extended from the origin of the PComA to the origin of the AChA. Perforating branches arising from the communicating segment terminate at the optic tract, the floor of the third ventricle, the optic chiasm, infundibulum, and the anterior and posterior perforated substances.^{2,4)} The genu of the internal capsule, the infarcted area in our patient, is irrigated by the perforating artery that passes through the anterior perforating substance.⁷⁾ Therefore, the sacrificed perforating artery presumably supplied a relatively large area, but our patient suffered no obvious postoperative neurological deficits such as contralateral hemiparesis or sensory disturbance. An anatomical study revealed anastomosis in 35.9% of cases with cisternal perforators,³⁾ suggesting that the manifestation of neurological deficits depends on the individual anastomotic variations. In our case, the continuous bleeding from the dome puncture after neck clipping and the small infarction area compared with the estimated total irrigation area of the perforating artery also

Table 1 Intracranial aneurysms associated with hyperplastic anomalous anterior choroidal artery (AChA)

Author (Year)	Age (yrs)	Sex	Side of anomalous AChA	Location of aneurysm
Takahashi et al. (1980) ⁹⁾	43	F	rt	IC-AChA
Takahashi et al. (1990) ¹⁰⁾	—	—	bil	bil IC-AChAs
			rt	AComA
Koyama et al. (1998) ⁴⁾	58	M	rt	IC-PComA
Abrahams et al. (1999) ¹⁾	37	F	rt	IC-AChA
	40	M	lt	BA
Matsumoto et al. (2000) ⁶⁾	55	F	lt	IC-AChA, MCA
Takase et al. (2001) ¹¹⁾	53	F	lt	BA-SCA
Shioya et al. (2005) ⁸⁾	64	F	lt	IC-AChA, IC-PComA
Present case	65	M	lt	IC-perforating artery

AComA: anterior communicating artery, BA: basilar artery, IC: internal carotid, MCA: middle cerebral artery, PComA: posterior communicating artery, SCA: superior cerebellar artery, —: data unavailable for review.

suggest the presence of anastomosis. Intraoperative somatosensory evoked potential and motor evoked potential monitoring are useful for predicting postoperative neurological deficits. However, if the perforator cannot be saved, there is no alternative to aneurysm clipping with sacrifice of the perforator.

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Address reprint requests to: Kouichi Misaki, M.D., Department of Neurosurgery, Graduate School of Medical Science, Kanazawa University, 13-1 Takara-machi, Kanazawa, Ishikawa 920-8641, Japan.
e-mail: misaki@ns.m.kanazawa-u.ac.jp