Delayed brain infarction caused by mechanical compression of the anterior choroidal artery after coil embolization for a large internal carotid-posterior communicating artery aneurysm: A case report

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Abstract
A 65-year-old male received coil embolization for a large internal carotid-posterior communicating artery aneurysm. Pre- and postoperative angiography at surgery demonstrated that the ipsilateral anterior choroidal artery branched from the internal carotid artery near the distal side of the aneurysm, and elevated and expanded on the aneurysmal dome, but was clearly visualized. Three days following endovascular treatment, the patient presented hemiparesis on the left side, with brain infarction in the territory of the right anterior choroidal artery despite antithrombotic therapy. The delayed brain infarction was likely caused by a reduction in anterior choroidal artery perfusion caused by mechanical compression following a postoperative increase in internal carotid-posterior communicating artery aneurysmal volume during intra-aneurysmal thrombosis. Transient volume expansion after coil embolization for intracranial aneurysms is rarely reported as a cause of brain infarction. It is important to recognize these arteries as potential postoperative complication risks, and consider the use of open surgery to avoid this risk.

Keywords
Internal carotid-posterior communicating artery aneurysm, anterior choroidal artery, coil embolization, endovascular surgery, brain infarction, mechanical compression

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Introduction
Internal carotid-posterior communicating artery (IC-PC) aneurysms account for 15.5% of all intracranial aneurysms.¹ With the development of endovascular surgery, there is increasing use of coil embolization for treatment of large IC-PC aneurysms. Transient volume expansion after coil embolization for intracranial aneurysms is a common cause of cranial nerve disorder, but rarely reported as a cause of brain infarction. We report a rare case of delayed brain infarction caused by mechanical compression of the anterior choroidal artery (AChA) related to a postoperative increase in IC-PC aneurysmal volume following intra-aneurysmal thrombosis.

Case report
History and examination
A 65-year-old man presented with sudden severe headache. He had a history of hypertension, but denied a history of diabetes mellitus or dyslipidaemia, or a family history of subarachnoid haemorrhage (SAH) or intracranial aneurysm. A right IC-PC aneurysm was detected 8 years prior, but he refused surgical treatment. On physical examination, the patient was alert and oriented, with no limb paralysis. Head computed tomography (CT) revealed an SAH, while CT angiogram demonstrated a large IC-PC aneurysm with a bleb, a foetal type of posterior cerebral artery (PCA) branching the aneurysmal dome, and an AChA branching from the IC artery (ICA) near the distal side of the aneurysm (Figure 1). We proposed direct surgical

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clipping or endovascular coil embolization to treat the ruptured aneurysm. As the patient only agreed to endovascular treatment to prevent rebleeding, we decided to perform coil embolization for the aneurysm.

Interventions

Under general anaesthesia, the right femoral artery was accessed with an 8F sheath. Digital subtraction angiography (DSA) demonstrated a large saccular aneurysm (15.9 × 13.1 × 11.3 mm in size) at the right IC-PC junction. The aneurysm showed a daughter aneurysm as the rupture point, while a foetal type of PCA branched from the contralateral side of the rupture point. A hyperflexed AChA running on the surface of the aneurysm was also observed (Figure 2). No connection between the basilar artery and the right PCA was detected in selective angiography of the left vertebral artery. An 8F Roadmaster guiding catheter (Goodman, Aichi, Japan) was introduced into the right ICA. We initially planned to protect the branching foetal type of PCA by inserting a microcatheter or a microguidewire. However, this was difficult because of the acute branch angle. Thus, we performed coil embolization using the simple balloon stand-by technique.

An occlusion balloon catheter (Shouryu HR 4 × 7 mm; Kaneka Medix Corp., Osaka, Japan) was placed in the aneurysm neck, and an SL-10 microcatheter (Stryker Neurovascular, Fremont, CA, USA) was placed into the aneurysm. A Target XL 360 soft coil (12 mm × 45 cm; Stryker Neurovascular, Kalamazoo, MI, USA) was delivered into the aneurysm cavity, and basket-forming of the coil, with saving of the branch point of the foetal type of PCA, was performed and confirmed with 3D-DSA. Several types of coils were then inserted to pack the first coil. The aneurysm, including the daughter aneurysm as the rupture point, was satisfactorily occluded, while the foetal type of PCA was spared. Postoperative DSA clearly revealed patency of the right AChA, without flow disturbance (Figure 2).

Postoperative course

In the intensive care unit, the patient was started on antiplatelet therapy (aspirin, 100 mg daily) after the operation to
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prevent thromboembolic complications following coil embolization. However, at 3 days after endovascular treatment, he presented hemiparesis on the left side. Diffusion-weighted imaging of the brain showed high-signal-intensity lesions in the right uncus, cerebral peduncle, and the posterior limb of the internal capsule, representing an acute infarction in the territory of the right AChA, but no new high-intensity signals in other arterial territories (Figure 3). Magnetic resonance imaging (MRI; time-of-flight imaging) showed a defect of the right AChA. However, the main trunk of the right ICA, and other branches, including the foetal type of PCA, were observed. Nevertheless, despite continued medical treatment, the motor weakness in his left side remained, and he was transferred to a second hospital to continue rehabilitation.

Discussion

Because of the increase in aneurysmal volume following postoperative intra-aneurysmal thrombosis, external compression of the cranial nerves can occur during the postoperative course of coil embolization for intracranial aneurysms, resulting in neurological disorders. However, this procedure is generally not considered a risk for vessel perfusion abnormalities and brain infarction. Herein, we report a rare case of delayed brain infarction caused by additional mechanical compression to the extended AChA running on the aneurysmal surface following volume expansion of the large IC-PC aneurysm after coil embolization.

Brain infarction caused by perfusion abnormalities of the AChA, such as Abbie syndrome, have been previously reported. The general running course and perfusion territory of the AChA have also been evaluated. The AChA arises from the ICA nearer the origin of the PCA than the carotid bifurcation. The AChA initially runs posteromedially behind the ICA, and reaches the lateral margin of the cerebral peduncle passing below the optic tract. At the anterior margin of the lateral geniculate body, the AChA crosses posterolateral to the uncus and passes through the choroidal fissure to enter the choroid plexus. Thus, the AChA commonly supplies the optic tract, cerebral peduncle, lateral part of the geniculate body, posterior limb of the internal capsule, globus pallidus, and optic radiations. Bain infarctions at these regions can result in characteristic symptoms of contralateral hemiplegia, hemianesthesia, and hemianopsia, as described by Abbie. However, there is large individual variability in the territory distribution of branching and connecting circulations with other vessels, and the infarction area changes case-by-case. In the present case, the right AChA showed a typical running course on preoperative contrast enhanced CT (Figure 4), while brain infarction was observed in specific brain regions corresponding to the clinical symptoms.

For coil embolization in our patient, there was no obvious connection between the PC and the right PCA on angiography, and preservation of the foetal type of PCA was required.
to prevent infarction in the artery territory. Thus, coil embolization was performed disproportionately in the aneurysm to make it tight, particularly around the bleb (the suspected rupture point), but with sparing of the origin of the foetal type of PCA. The right AChA was also not occluded, and was clearly visualized using postoperative DSA (Figure 2).

Considering the partial embolization of the aneurysmal dome in our case, there was a risk of postoperative thrombotic complications. Thus, we initiated postoperative administration of antiplatelet agents. As the suspected flow volumes of the ICA or foetal type of PCA were higher than the smaller branches around the aneurysm, the risk of thrombotic complication was also higher in the territory of the large arteries. However, diffusion-weighted imaging showed no brain infarctions apart from that at the territory of the right AChA.

The course of the right AChA was elevated and markedly expanded by the right IC-PC aneurysm. The artery was firmly fixed to the aneurysmal surface by connective tissue, as commonly observed in surgical clipping for cerebral aneurysms. Compared with the slow expansion of an aneurysm caused by natural growth, the postoperative increase in aneurysmal volume over several days caused by intra-aneurysmal thrombosis following coil embolization was rapid, and is likely related to additional mechanical compression of the attached expanded perforating artery and decreased perfusion, resulting in brain infarction.

Preoperative detection of the potential risk for brain infarction in such vessels can be difficult, depending on their size or course. Furthermore, some patients have a restriction on their choice of treatment; for example, severely ruptured aneurysms requiring rapid decision and treatment, patients unable to receive some therapies because of their condition or comorbidities, or patients who agree only to endovascular therapy, such as the present case. In the present study, our case showed a delayed brain infarction and right AChA observed clearly on immediate postoperative DSA. Thus, reliable prediction of the risk for complication was difficult, even if performed at the end of the coil embolization, while monitoring of motor-evoked potentials would not have been useful. Because of these difficulties in perioperative risk evaluation, it is important to recognize these arteries as potential postoperative complication risks. If preoperative radiology shows these findings, open surgery involving direct surgery of the artery may be considered.

**Conclusion**

We report a rare case of delayed brain infarction caused by AChA compression after coil embolization for a large IC-PC aneurysm. It is important to recognize these arteries as potential postoperative complication risks, and consider the use of open surgery to avoid this risk.

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