Posterior Cerebral Artery Aneurysms with Common Carotid Artery Occlusion: A Report of Two Cases

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Objective: Common carotid artery occlusion (CCAO) is much rarer than internal carotid artery occlusion (ICAO). Many cases of intracranial aneurysms due to hemodynamic stress caused by ICAO have been reported. On the other hand, only seven cases of intracranial ruptured aneurysms with CCAOs have been reported. We report the two patients with CCAOs and unruptured posterior cerebral artery (PCA) aneurysms.

Case Presentations: The two patients presented CCAOs and unruptured PCA aneurysms. Digital subtraction angiography (DSA) showed that these patients had collateral circulation from the ipsilateral vertebral artery to the internal carotid artery (ICA) via the external carotid artery (ECA). We observed that the collateral circulation could become stagnant due to neck extension in patients with CCAO.

Conclusion: Physicians should be aware of this phenomenon and preoperatively determine whether ischemic symptoms occur under neck extension in patients with CCAOs accompanied by intracranial aneurysms.

Keywords: common carotid artery, occlusion, collateral, aneurysm

Introduction

Compared with internal carotid artery occlusion (ICAO), common carotid artery occlusion (CCAO) is very rare.\(^1\) Many cases of intracranial aneurysms resulting from hemodynamic stress caused by ICAO have been reported.\(^4,5\) On the other hand, only seven cases of intracranial ruptured aneurysms with CCAOs have been reported.\(^1,6-10\) We experienced two cases of posterior cerebral artery (PCA) unruptured aneurysms with CCAOs. In these patients, we observed the flow stagnation of collateral circulation from the vertebral artery (VA) to internal carotid artery (ICA) via the external carotid artery (ECA) occurred due to the neck extension. We report in these cases with some literature review.

Case Presentations

Case 1
A 68-year-old female was diagnosed with left CCAO and an unruptured left PCA aneurysm on MRI performed at another hospital. The patient had a medical history of hypertension, for which she was under medication. She was referred to our hospital for further evaluation and treatment. DSA showed a left CCAO (Fig. 1A), and the left ICA was patent with collateral circulation from the ipsilateral vertebral artery to the internal carotid artery via the external carotid artery (ECA) (Fig. 2A). We decided embolization of the left PCA aneurysm. She underwent general anesthesia and placed in the neck extension position to achieve appropriate projection for embolization. We obtained complete occlusion of the aneurysm (Figs. 1B and 1C). After the procedure, we were aware of disappearance of contrast filling into left ICA through the collateral circulation on left vertebral angiography compared with that in the neutral neck position (Figs. 2A and 2B). Regardless of the head position, flow stagnation of the middle cerebral artery and the anterior cerebral artery through the posterior communicating artery was not
Fig. 1 Case 1: Digital subtraction angiograms. (A) Aortogram showing occlusion of the left common carotid ostium (black arrowhead). (B) Preoperative left VAG showing a left posterior cerebral artery (P1) aneurysm (white arrowhead). (C) Postoperative left VAG shows complete occlusion of the aneurysm. (D–F) The left subclavian artery angiograms showing the collateral circulation between the left ECA (white arrows) and the branches from the thyrocervical trunk. (D) Early arterial phase, (E and F) delayed arterial phase. ECA: external carotid artery; VAG: left vertebral angiogram

Fig. 2 Case 1: (A) Lateral view of the left VAG in the neutral neck position showing the antegrade flow of the left ICA (black arrowheads) from the ipsilateral vertebral artery via ECA. (B) Lateral view of the left VAG in the neck extension position showing disappearance of the antegrade flow of ICA through the collateral circulation. ECA: external carotid artery; ICA: internal carotid artery; VAG: left vertebral angiogram
Case 2

A 53-year-old male patient had been previously diagnosed with CCAO and an unruptured left PCA aneurysm on MRI performed at another hospital. The patient was a two pack-a-day smoker for 30 years. 3D-CT angiogram showed left CCAO (Fig. 3A). DSA revealed a left PCA aneurysm (Fig. 2B) and left CCAO with collateral circulation from the ipsilateral VA to ICA via occipital artery (OA) and ascending pharyngeal artery (Figs. 3E, 3F, and 4A). The collaterals from the ipsilateral ascending cervical artery to the left common carotid artery (CCA) via the sternocleidomastoid branch of the superior thyroid artery were also demonstrated in the left subclavian artery angiogram (Figs. 3C and 3D). In the neck extension position, left vertebral angiography demonstrated that the collateral circulation via OA muscular branch was stagnant without symptoms relative to the flow in the neutral neck position (Figs. 4A and 4B). In a manner most consistent with the patient’s wish, we have continued to follow-up his progress.

Discussion

We experienced two cases of PCA unruptured aneurysms with CCAOs. Only seven cases of intracranial aneurysms with CCAOs have been reported till date (Table 1); almost all cases had enlarged posterior communicating artery as the collateral pathway. All aneurysms were ruptured and arising from the collateral circulation. Major causative factor of PCA aneurysms with CCAO in the present study was hemodynamic stress as recent reports indicate.1,6,8)
Six of seven previous reported cases involved bilateral CCAOs. The patients in the present cases suffered from intracranial aneurysms in the collateral pathway with ipsilateral CCAOs, ipsilateral CCAO can get complicated with intracranial aneurysms.

These two cases showed stagnation of the collateral circulation from the muscular branch of VA to ICA via ECA during neck extension. The phenomenon of reduction of intracranial blood supply caused by neck position is well known as Bow Hunter’s stroke or Powers syndrome that arise from mechanical compression of the VA by head rotation.11,12) In the present cases, the main mechanism of the stagnation of collateral circulation from VA to ICA may be compression of the muscular branches caused by neck extension. The intracranial blood supply may decrease depending on the neck position of patients with CCAO. In the case of CCAO, the evaluation of the collateral circulation to ICA during neck extension may be important. The ECA can remain patent in the presence of CCAO, since many collaterals between branches of the ECA and other extracranial arteries exist, for example, the thyroid arterial network, arteries arising from subclavian artery and vertebral to ECA anastomoses.2) Therefore, we should have also performed angiogram of contralateral VA, bilateral subclavian artery, and contralateral CCA during neck extension.

Neck extension is occasionally made to achieve appropriate projection during embolization for intracranial aneurysms. We experienced stagnation of the collateral circulation from VA to ICA via ECA during neck extension in these cases of CCAO. The stagnation of the collateral circulation can cause thromboembolic complications. Therefore, we should peroperatively administer adequate antithrombotic agents. In addition, insertion of guiding catheter into the VA might also cause flow stagnation because of tortuosity of the origin of the VA in case 1 (Fig. 1A). It may be reasonable to insert guiding catheter into contralateral VA.

Conclusion

The collateral circulation from VA to ICA via ECA can become stagnant because of neck extension in patients with CCAO. Physicians should be aware of this possibility and preoperatively determine whether ischemic symptoms occur under neck extension in patients with CCAO and intracranial aneurysms.

Disclosure Statement

All authors have no conflict of interest.
### Table 1

Cases of intracranial aneurysms with common carotid artery occlusion

<table>
<thead>
<tr>
<th>Authors</th>
<th>Year</th>
<th>Age/Sex</th>
<th>Side of CCAO</th>
<th>Enlarged Pcom</th>
<th>Collateral circulation to ECA</th>
<th>Location of aneurysms</th>
<th>Ruptured/Unruptured</th>
</tr>
</thead>
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<tr>
<td>Kumagai et al.7)</td>
<td>1981</td>
<td>51/F</td>
<td>Bilateral</td>
<td>N/D</td>
<td>VA</td>
<td>Bilateral BA-SCA</td>
<td>Ruptured</td>
</tr>
<tr>
<td>41/F</td>
<td>Bilateral Aortitis</td>
<td>Ruptured</td>
<td>VA</td>
<td>N/D</td>
<td>BA tip, Lt. PCA</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Masuzawa et al.8)</td>
<td>1982</td>
<td>54/F</td>
<td>Bilateral Aortitis</td>
<td>Ruptured</td>
<td>N/D</td>
<td>yes</td>
<td>BA tip</td>
</tr>
<tr>
<td>Kataoka et al.6)</td>
<td>1982</td>
<td>81/F</td>
<td>Bilateral Arteriosclerosis</td>
<td>Ruptured</td>
<td>N/D</td>
<td>yes</td>
<td>BA-AICA</td>
</tr>
<tr>
<td>Araki et al.1)</td>
<td>2002</td>
<td>82/F</td>
<td>Bilateral Arteriosclerosis</td>
<td>Ruptured</td>
<td>VA</td>
<td>yes</td>
<td>Rt. PCA-Pcom, Rt. P2</td>
</tr>
<tr>
<td>Meguro et al.9)</td>
<td>2008</td>
<td>62/F</td>
<td>Bilateral Arteriosclerosis</td>
<td>Ruptured</td>
<td>VA</td>
<td>yes</td>
<td>BA tip, Lt. P1</td>
</tr>
<tr>
<td>Yamao et al.10)</td>
<td>2014</td>
<td>62/F</td>
<td>Right CCA ligation</td>
<td>Ruptured</td>
<td>VA</td>
<td>yes</td>
<td>True Rt. Pcom</td>
</tr>
<tr>
<td>Present cases</td>
<td>2016</td>
<td>68/F</td>
<td>Left Arteriosclerosis</td>
<td>Unruptured</td>
<td>thyrocervical trunk</td>
<td>VA</td>
<td>yes</td>
</tr>
<tr>
<td>53/M</td>
<td>Left Arteriosclerosis</td>
<td>Unruptured</td>
<td>VA</td>
<td>thyrocervical trunk</td>
<td>yes</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

AICA: anterior inferior cerebellar artery; BA: basilar artery; CCAO: common carotid artery occlusion; ECA: external carotid artery; N/D: not described, PCA: posterior cerebral artery; Pcom: posterior communicating artery; SCA: superior cerebellar artery; VA: vertebral artery

### References


