A Case of Acute Simultaneous Bilateral Internal Carotid Artery Occlusion Treated by Thrombectomy

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Objective: We report a patient who underwent thrombectomy for acute bilateral internal carotid artery occlusion (ICAO).

Case Presentation: A 76-year-old female presented consciousness disturbance. Although warfarin had been administered after prosthetic replacement, it was discontinued due to gastrointestinal hemorrhage. MRI showed bilateral ICAO and right-dominant ischemic changes involving the bilateral hemispheres. Radiological findings indicated acute occlusion of the right internal carotid artery (ICA), and chronic occlusion of the left ICA was primarily considered; however, revascularization was conducted with considering the possibility of bilateral occlusion. Thrombectomy was performed and recanalization of the bilateral ICAs was achieved. However, ischemia progressed, leading to extensive cerebral infarction.

Conclusion: Thrombectomy for bilateral ICAO was performed although the outcome was unfavorable. Acute bilateral ICAO contains poor prognosis although it is indispensable to appropriately diagnose, evaluate, and select therapeutic strategy. A proper management for bilateral ICAO awaits further investigation.

Keywords ► internal carotid artery, occlusion, bilateral, cardio embolism, thrombectomy

Introduction

The efficacy of endovascular revascularization for acute ischemic stroke due to large vessel occlusion was demonstrated,1 and this procedure has been widely performed in various institutions. If the interval until recanalization of a major artery is prolonged, the functional prognosis may deteriorate. Therefore, revascularization should be conducted as promptly as possible, and therapeutic strategies must be managed individually after evaluating the clinical and pathological condition. On the other hand, it is sometimes difficult to access the detailed condition and to decide the strategy of revascularization based on limited information before treatment. In this article, we report a patient with acute embolic bilateral internal carotid artery occlusion (ICAO). Thrombectomy for bilateral ICAO was performed and recanalization was achieved although the outcome was unfavorable. Simultaneous bilateral ICAO is a rare cause of ischemic stroke and we discuss about the course of assessment and therapeutic strategy for revascularization in this patient.

Case Presentation

Patient background
The patient was a 76-year-old female. She had undergone prosthetic replacement for aortic valve disease 10 years previously. Warfarin had been orally administered. She did not have the contributory family history.

Present illness
The patient had been hospitalized and treated in the department of gastroenterology of our hospital due to gastrointestinal hemorrhage for 1 week before onset. On the day of
admission, warfarin was discontinued. She was found developing consciousness disturbance and respiratory failure in the hospital room at night. The interval from time last known well until detection was approximately 2 hours and 50 minutes. Immediately, endotracheal intubation was performed by a duty doctor, and management with an artificial respirator was conducted. As acute stroke was suspected, the patient was referred to our department for a neurologic examination.

**Neurologic findings**

Under respiratory and circulatory cares, cardiorespiratory condition was stabilized. On the initial consultation in our department, it was difficult to properly examine neurologic symptoms because the patient was managed with a respirator after the administration of a sedative. The consciousness level was evaluated as Japan Coma Scale III-200. A decorticate posture was observed. The pupils were the same size and measured 2 mm and the bilateral light reflex was maintained.

**Radiological findings**

Head CT 60 minutes after detection revealed a faint loss of gray-white matter differentiation mainly involving the bilateral frontal lobes (early CT sign). A high-density area (hyper-dense middle cerebral artery [MCA] sign) was noted on the right side (Fig. 1A). Diffusion-weighted image (DWI) on MRI 80 minutes after detection showed high signal intensity areas in the bilateral frontal/parietal lobes (Fig. 1B and 1C). The Alberta Stroke Programme Early CT Score (ASPECTS)-DWI scores for the right and left hemispheres were 4/11 and 6/11, respectively. MRI FLAIR image showed a high signal intensity of cortical vessels along the sulcus in the right hemisphere (intraarterial signal [IAS]) (Fig. 1D). On MRA, the bilateral internal carotid arteries (ICAs) were not visualized (Fig. 1E and 1F). The presence of the posterior communicating artery (PcomA) was not presented even in the source images. Considering the onset at night, severity of symptoms, and time-related restrictions, perfusion imaging was not performed.

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Fig. 1  (A) A high-density area was observed in the right middle cerebral artery (hyper-dense MCA sign) on head CT. (B and C) MRI diffusion-weighted image of onset showing high intensities bilaterally dominantly in the right side. (D) MRI FLAIR demonstrating a faint high-intensity change in the right frontal lobe and intra-arterial signal along the sulcus. (E and F) MRA revealed the absence of bilateral internal carotid artery. MCA: middle cerebral artery
could not be confirmed; it was insufficient to differentiate chronic from acute occlusion (Fig. 2A and 2B). First, thrombectomy was performed in the right ICA, of which acute occlusion was indicated by CT and MRI findings. A 9 Fr balloon guiding catheter (Optimo; Tokai Medical Products, Aichi, Japan) was inserted into the right ICA. The ICA was occluded using a balloon of Optimo, and manually aspirated from a guiding. Subsequently, Penumbra 5MAX ACE (Medtronic, Irvine, CA, USA) was advanced from the proximal ICA to its distal while aspirating thrombi. A large volume of thrombi were collected, and recanalization of the ICA was achieved. However, occlusion was still remained at the right proximal M1 segment (Fig. 3A). After passing CHIAKI 14 guidewire (Asahi Intecc Co., Ltd, Aichi, Japan) and Marksman microcatheter (Medtronic) through the right M1 site of occlusion, Trevo ProVue Retriever 4 mm–20 mm (Stryker, Kalama-zoo, MI, USA) was deployed. After Trevo deployment, immediate flow restoration was noted (Fig. 3B). On angiography 3 minutes after Trevo deployment, there was no reoclusion. Marksman was removed, and an aspiration tube was connected to 5MAX ACE. While conducting pump aspiration, Trevo was retrieved into 5MAX ACE (Solumbra technique). The visualization of the A2 or distal of the right anterior cerebral artery (ACA) was deficient, but recanalization of the right MCA was achieved.

**Assessment and therapeutic strategy**

The right ICA and M1 were regarded as acute occlusion based on the right high-density MCA sign on CT and IAS on MRI FLAIR. Still, left ICA was not exactly diagnosed whether it was in acute or chronic occlusion.

As the distribution of high-intensity areas on MRI DWI was seemed to be smaller considering the severe neurologic symptoms (clinical-diffusion mismatch), revascularization therapy should be indicated. Due to a history of gastrointestinal hemorrhage, neuroendovascular treatment was adapted without considering tissue plasminogen activator (t-PA) administration.

**Neuroendovascular treatment**

As endotracheal intubation had been accomplished, intervention was performed under general anesthesia. The interval from time last known well until arterial puncture of femoral artery was about 4.5 hours, and that from detection until arterial puncture was approximately 90 minutes. Head CT and MRI findings indicated acute occlusion of the right ICA and chronic occlusion of the left ICA was primarily considered; however, treatment was conducted additionally considering the possibility of acute bilateral occlusion.

Initially, bilateral common carotid angiograms were performed. The bilateral ICAs were slowly enhanced from their origins toward intracranially, but the site of occlusion
Acute Bilateral ICA Occlusion Treated by Thrombectomy

The state of recanalization was evaluated as thrombolysis in cerebral infarction (TICI) 2B (Fig. 3C and 3D). The interval from arterial puncture until right-side recanalization was 32 minutes.

On angiography of the right ICA after its recanalization, the left ACA A2 or distal arteries were visualized through the anterior communicating artery (AcomA). However, collateral flow to the left ACA A1 or left MCA was not described. Therefore, left ICA was also considered as acute occlusion and revascularization for it should be performed (Fig. 4). Thrombectomy was performed using procedures similar to those used on the right side. A balloon guiding catheter (Optimo) was navigated into the left ICA. After direct aspiration through Optimo, Penumbra 5MAX ACE was advanced from the proximal to the distal of the left ICA while aspirating thrombi. A large volume of thrombi were collected. Angiography after thrombus aspiration confirmed thromboembolic occlusion involving the ICA top to MCA (Fig. 4A). Penumbra 5MAX ACE and Marksman were guided, and Trevo ProVue Retriever 4 mm–20 mm was deployed. Immediately after Trevo deployment, flow restoration was noted (Fig. 4B). On angiography 3 minutes after Trevo deployment, there was no reocclusion. Marksman was removed, and Trevo was retrieved into 5MAX ACE while aspirating. Recanalization (TICI 3) was achieved (Fig. 4C and 4D). The interval from arterial puncture until left-side recanalization was 52 minutes.

Course after neuroendovascular treatment
Head CT immediately after thrombectomy demonstrated edematous changes and enhancement effects at sites consistent with those on preoperative MRI DWI high intensities. In the left basal ganglia, the leakage of contrast medium was noted. In addition, extensive low-density areas were bilaterally detected in the ICA territories (Fig. 5A and 5B). After intervention, improvements in the neurologic symptoms or consciousness level were not observed. The patient presented dilation of the bilateral pupils the day after intervention, leading to a decrease in the blood pressure and respiratory arrest. Head CT on the same day revealed the
severe infarction of the bilateral hemispheres with marked edematous changes (Fig. 5C). The patient died 2 days after onset.

### Discussion

**Pathological consideration of acute bilateral ICAO**

We presented a patient with acute bilateral ICAO due to cardioembolism. Concerning its etiology, the discontinuation of warfarin used for prosthetic-valve replacement may have led to embolus formation and simultaneous dispersion to the bilateral ICAs. Simultaneous bilateral ICAO is a rare cause of ischemic stroke, whereas, usually, in patients with cardiogenic embolism, small ischemic lesions are scattered in the bilateral hemispheres. Few papers have reported simultaneous embolic occlusion of the bilateral ICAs. Previous reports regarding simultaneous bilateral embolic ICAO are presented in Table 1. The prognosis was unfavorable, and eight of nine patients died. Chisci et al. performed hybrid therapy, consisting of direct surgery and endovascular treatment, for acute occlusion of bilateral common carotid arteries with brachiocephalic artery occlusion, and they reported that the course was favorable. To our knowledge, no study has reported mechanical thrombectomy with a stent retriever and/or Penumbra system for acute bilateral ICAO. In the present case, thrombectomy was performed and recanalization of the bilateral ICAs was achieved; however, this did not lead to a favorable outcome.

**Diagnosis of acute bilateral ICAO**

On diagnosis in the present case, we initially considered acute unilateral ICAO accompanying with chronic contralateral ICAO, considering also the possibility of simultaneous bilateral ICAO. Angiography before endovascular therapy suggested acute bilateral ICAO although a diagnosis was not reached. A preoperative diagnosis of acute occlusion of the bilateral ICAs can be made only when high-density MCA signs are present on the left and right sides or when ischemic areas on MRI DWI are extensive on both sides. However, in a management of acute ischemic stroke, it is required to evaluate the condition in a limited time and immediately to decide the treatment strategy. Thus, sufficient evaluation is sometimes difficult. Thrombectomy

### Table 1 Summary of previous reports on acute bilateral carotid artery occlusion

<table>
<thead>
<tr>
<th>Author (year)</th>
<th>Age (years)</th>
<th>Sex</th>
<th>Cause</th>
<th>Treatment</th>
<th>Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>Browne et al. (1993)</td>
<td>63</td>
<td>F</td>
<td>Atrial myoma</td>
<td>NA</td>
<td>Death</td>
</tr>
<tr>
<td>Yamaguchi et al. (1997)</td>
<td>68</td>
<td>M</td>
<td>Cardioembolic</td>
<td>Medication</td>
<td>Death</td>
</tr>
<tr>
<td>Takahashi et al. (2001)</td>
<td>83</td>
<td>M</td>
<td>Cardioembolic</td>
<td>Medication</td>
<td>Death</td>
</tr>
<tr>
<td>Kwon et al. (2002)</td>
<td>61</td>
<td>F</td>
<td>Cardioembolic</td>
<td>Intra-arterial thrombolysis</td>
<td>Death</td>
</tr>
<tr>
<td>Takahashi et al. (2001)</td>
<td>79</td>
<td>F</td>
<td>Cardioembolic</td>
<td>Intra-arterial thrombolysis</td>
<td>Death</td>
</tr>
<tr>
<td>Kwon et al. (2002)</td>
<td>65</td>
<td>F</td>
<td>Cardioembolic</td>
<td>Intra-arterial thrombolysis</td>
<td>Death</td>
</tr>
<tr>
<td>Zubkov et al. (2007)</td>
<td>72</td>
<td>M</td>
<td>Cardioembolic</td>
<td>rt-PA</td>
<td>Death</td>
</tr>
<tr>
<td>Chisci et al. (2014)</td>
<td>84</td>
<td>M</td>
<td>Unknown</td>
<td>Hybrid surgery</td>
<td>Independent</td>
</tr>
<tr>
<td>Our case (2017)</td>
<td>73</td>
<td>F</td>
<td>Cardioembolic</td>
<td>Endovascular thrombectomy</td>
<td>Death</td>
</tr>
</tbody>
</table>

rt-PA: recombinant tissue plasminogen activator
must be conducted, considering also the possibility of acute bilateral ICAO, as demonstrated in the present case.

**Treatment strategy for acute bilateral ICAO**

In this case, revascularization was initially performed for the right ICA because acute occlusion was indicated by CT and MRI findings and a lower ASPECTS-DWI score was presented in the right side. If a diagnosis of acute bilateral ICAO was made in the initial phase, in another perspective, revascularization could be initially considered on the side with a higher ASPECTS-DWI score, which reflects the limited time window of preserving ischemic penumbra because the rapid progression of cerebral ischemia is expected in bilateral ICAO. However, in the actual clinical setting, properly selecting the therapeutic strategy might be difficult because of insufficient evaluation and time limitation. Therefore, it may be accepted to initially perform the treatment on the side with ischemic image findings and a lower ASPECTS score that definitely indicated acute occlusion.

**Consideration of acute bilateral ICAO and ischemic penumbra**

In the present case, a clinical-diffusion mismatch seemed to be presented in spite of the severe neurologic symptom from the time of onset, and we considered that revascularization therapy should be indicated. Thrombolysis in cerebral infarction 2B-3 recanalization was achieved bilaterally in ICAs, and the time from puncture to recanalization was ≤60 minutes and that from detection to recanalization was ≤140 minutes. However, extensive infarction was appeared involving the bilateral ICA territories. The DWI or CT perfusion assessment with clinical mismatch in the triage of wake up and late presenting strokes undergoing neurointervention with Trevo (DAWN) trial demonstrated that thrombectomy was effective in a time of ≥6 hours from onset when adequately evaluating an ischemic penumbra in patients with large vessel occlusion. However, even if penumbra seems to remain on initial imaging, as demonstrated in the present case, cerebral ischemia may rapidly progress in some cases. For acute revascularization, the therapeutic time window of ischemic penumbra depends on the residual cerebral blood volume/cerebral blood flow. Therefore, occlusion of the bilateral ICAs may have affected collaterals via AcomA or cortical arteries, resulting in the rapid progression of ischemia. It is difficult to evaluate whether the development of the PcomA was hypoplastic or whether embolus-related occlusion was present at the PcomA origin of the ICA. Collateral via PcomA was not evident in the present case, which may have been also involved. In this case, perfusion imaging was not performed and the appropriate evaluation was restricted. However, the residual cerebral blood flow might be very small, and this may have primarily contributed to a poor prognosis. The purpose of revascularization for large vessel occlusion is to adequately evaluate ischemic penumbra and rescue the ischemic brain tissue. However, there are some cases in which recanalization even in a short time could not lead to an improvement in the prognosis, such as a case of simultaneous bilateral ICAO. Although examination-related time loss is a demerit, it is important to evaluate residual cerebral blood flow using perfusion imaging before revascularization for adequate treatment selection. In the future, advances in diagnostic imaging techniques may facilitate the accurate assessment of ischemic penumbra in a short time, making it possible for physicians to determine whether endovascular treatment should be indicated for acute ischemic stroke, including rare condition such as simultaneous bilateral ICAO.

**Conclusion**

We demonstrated a patient with acute simultaneous bilateral ICAO that is a rare condition of acute ischemic stroke. Thrombectomy was performed and recanalization of bilateral ICAs was achieved; however, ischemia progressed, leading to an unfavorable outcome. Acute bilateral ICAO contains poor prognosis although it is indispensable to appropriately diagnose, evaluate, and select therapeutic strategy. A proper management including endovascular treatment for this fatal condition awaits further investigation.

**Disclosure Statement**

There is no conflict of interest for the first author and coauthors.

**References**


