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

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Objective: We report a survived case of acute bilateral internal carotid artery occlusion successfully treated by mechanical thrombectomy.

Case Presentation: The patient was an 82-year-old right-handed man. Sudden consciousness disturbance and aphasia appeared, and cranial magnetic resonance angiography (MRA) revealed bilateral internal carotid artery occlusion. Cerebral angiography demonstrated occlusion between the cervical and cavernous portions of the bilateral internal carotid artery, and the growth of collateral circulation to the areas where the bilateral internal carotid artery perfused. We performed mechanical thrombectomy first on the left side, which was the main cause of his symptoms and relatively wide ischemic penumbra, and achieved recanalization of the bilateral internal carotid artery and suppressed extensive enlargement of the infarction.

Conclusion: Acute bilateral internal carotid artery occlusion requires an optimal treatment strategy based on the clinical symptoms and preoperative imaging.

Keywords ▶ internal carotid artery, occlusion, bilateral, thrombectomy, acute ischemic stroke

Introduction

Recanalization therapy for major artery occlusions in the anterior circulation is useful because it can improve the outcome of acute unilateral internal carotid artery occlusion. However, the outcome of acute bilateral internal carotid artery occlusion remains unfavorable, and survival cases after bilateral thrombectomy for acute simultaneous bilateral internal carotid artery occlusion are very rare. In this study, we report a survived case of acute bilateral internal carotid artery occlusion treated by mechanical thrombectomy.

Case Presentation

The patient was an 82-year-old right-handed man with a history of rectal cancer. He had smoked 20 cigarettes/day and consumed alcohol every day. He was transferred to our hospital with dyspnea. While preparations were made for admission due to heart failure related to dilated cardiomyopathy, consciousness disorder and aphasia suddenly appeared. The Glasgow Coma Scale score was E2V2M5 and partial conjugate deviation to the left side was noted. The manual muscle testing scores were 3/5 for the bilateral upper limbs and right lower limb and 4/5 for the left lower limb, suggesting incomplete paralysis dominantly in the right side. Severe aphasia and unilateral carelessness were also observed. The National Institute of Health Stroke Scale (NIHSS) score was 20. Diffusion-weighted magnetic resonance imaging (MRI-DWI) at 140 minutes after onset revealed acute cerebral infarction of the area perfused by the bilateral middle cerebral arteries and right posterior cerebral artery. The DWI-Alberta Stroke Programme Early CT Score (ASPECTS) was 10 at both sides (Fig. 1A). Fluid-attenuated inversion recovery (FLAIR) imaging demonstrated little signal changes in the infarction area (Fig. 1B). Although occlusion of the bilateral internal
carotid arteries and right posterior cerebral artery was observed on magnetic resonance angiography (MRA), the peripheries of the bilateral middle/anterior cerebral arteries and bilateral posterior communicating arteries were visualized (Fig. 1C and 1D). Arterial spin labeling (ASL) demonstrated a reduction of the blood flow in the bilateral middle cerebral artery areas dominantly in the left side (Fig. 1E).

### Cerebral angiography

To evaluate the condition, cerebral angiography was performed under local anesthesia. The interval from onset to puncture of femoral artery was 190 minutes. Bilateral common carotid angiography demonstrated bilateral internal carotid artery occlusion at proximal portion exhibiting tapering shape with “to and fro” motion of contrast agent. Internal carotid artery distal from C2 portion and anterior and middle cerebral artery at both sides were perfused from external carotid artery through ophthalmic artery (Fig. 2A–2D). Vertebral angiography demonstrated occlusion of the P3 portion of right posterior cerebral artery, leptomeningeal anastomosis originated from the left posterior cerebral artery, perfusion to the anterior circulation from the bilateral posterior communicating arteries, and perfusion from the posterior pericallosal artery to the anterior cerebral artery region (Fig. 2E and 2F). It was difficult to evaluate whether the occlusion of the bilateral internal carotid arteries was acute or subacute based on angiography, but the clinical course suggested acute occlusion at least on one side. We adopted a strategy to initially perform recanalization therapy on the left side, which was suspected as main cause of consciousness disorder and aphasia and had more extensive area of cerebral blood flow reduction than the right side.

### Endovascular treatment

Under local anesthesia, systemic heparinization was performed 225 minutes after onset. A 9Fr balloon guiding catheter (OPTIMO; Tokai Medical Products, Aichi, Japan)
Acute Bilateral Internal Carotid Artery Occlusion was guided to the origin of the left internal carotid artery. Under the left internal carotid artery occlusion using a balloon of OPTIMO, aspiration was conducted by the guiding catheter. A large volume of fresh thrombus was collected. Angiography showed recanalization of the internal carotid artery with antegrade blood flow and defect of contrast agent by thrombus at cavernous portion of the internal carotid artery. An aspiration catheter (ACE 68; Penumbra, Inc., Alameda, CA, USA) was guided to the proximal site of the thrombus, and thrombectomy with A Direct Aspiration First Pass Technique (ADAPT) was performed (Fig. 3A–3C). The interval from femoral artery puncture to left side recanalization (thrombolysis in cerebral infarction, TICI: 3) was 47 minutes. Angiography after recanalization showed marked arteriosclerotic change in the left internal carotid artery and 40% stenosis at the cavernous portion (Fig. 4A and 4B). Because the symptom did not improve immediately after recanalization of the left internal carotid artery, recanalization therapy to the right side was also subsequently performed.

A 9Fr balloon guiding catheter was guided to the origin of the right internal carotid artery, and aspiration was performed with right internal carotid artery occlusion using the balloon. Thrombus was collected, but recanalization of the internal carotid artery was not achieved. Thrombectomy with ADAPT was conducted through the aspiration catheter guided to the proximal site of occlusion. Angiography revealed the influx of contrast agent into the cavernous portion of the internal carotid artery. When passing a microguidewire through the site of occlusion at the cavernous portion, there was resistance, but it was possible to guide the microcatheter to the distal site of occlusion. A stent retriever (Solitaire Platinum 6 mm × 40 mm; Medtronic, Minnesota, USA), which had been deployed in the site of occlusion at the cavernous portion, was pulled into the ACE 68 that had been aspirated to collect the thrombus (Fig. 3D–3F). Angiography revealed occlusion of the right posterior parietal artery. Recanalization (TICI: 2b) was achieved 86 minutes after femoral artery puncture (Fig. 4C and 4D). Although 70% stenosis remained at the cavernous portion of the right internal carotid artery, there was no serial progression of stenosis during a 20-minute waiting period; therefore, this procedure was completed.

Postoperative course
After surgery, consciousness disorder and aphasia improved, and the NIHSS score improved to 14. On MRI-DWI the day after surgery, the enlarged site of acute cerebral infarction involved the insular cortex and parietal lobe at the right side, and a small area of the left side (Fig. 5). We suspected that dilated cardiomyopathy-related cardiogenic thrombosis led to occlusion of the internal carotid artery with arteriosclerotic change, and started anticoagulant therapy under a diagnosis of cardiogenic cerebral embolism. Rehabilitation was restricted because of heart failure and disuse disorder, and the patient was referred to another...
hospital 43 days after surgery with a modified Rankin Scale score of 4.

Discussion

Acute simultaneous occlusion of the bilateral internal carotid arteries is relatively rare, and its outcome is unfavorable.\textsuperscript{3,4,6–10} Bilateral internal carotid artery occlusion induces coma, causing neurological deficits resembling basilar artery occlusion and leading to a fatal outcome through the rapid progression of ischemia involving an extensive area in some cases.\textsuperscript{9} Therefore, patients in whom bilateral mechanical thrombectomy for acute bilateral internal carotid artery occlusion resulted in recanalization, leading to a favorable outcome, are rare.\textsuperscript{2}

The reasons why thrombectomy led to survival in the present case included ischemia resistance related to the presence of a collateral pathway. In the present patient, arteriosclerosis-related stenosis of the bilateral internal carotid arteries was present as a background factor; cardiogenic embolism associated with dilated cardiomyopathy may have caused bilateral internal carotid artery occlusion. On both sides, stenosis was present at the cavernous portion of the internal carotid artery, and emboli were captured at this site; therefore, a collateral pathway through the ophthalmic arteries and anterior/posterior communicating arteries may have remained, leading to ischemia resistance. For this reason, coma may not have been caused despite acute bilateral internal carotid artery occlusion, with no rapidly progressing ischemic change after onset. This may have resulted in a survival outcome after thrombectomy. Furthermore, treatment was initially started on the left side, on which the penumbra region, as described below, was relatively large, which also may have led to the survival outcome.

Etiological factors for bilateral internal carotid artery occlusion include embolism, atherothrombosis, arterial dissection, vasospasm, and angiitis.\textsuperscript{2,11} In the presence of
Fig. 4  Cerebral angiography findings after thrombectomy (A, C: frontal views, B, D: lateral views). (A and B) Left internal carotid angiography confirmed recanalization (TICI: 3) and 40% stenosis at the cavernous portion (arrow) with arteriosclerotic change in the internal carotid artery. (C and D) Right internal carotid angiography showed recanalization (TICI: 2b) (occlusion of the right posterior parietal artery) and 70% stenosis at the cavernous portion (arrow). TICI: thrombolysis in cerebral infarction

Fig. 5  MRI-DWI the day after surgery. The hyperintensity areas in the insular cortex and parietal lobe of the right hemisphere, and a small area of the left hemisphere were larger than the areas before thrombectomy. MRI-DWI: diffusion-weighted magnetic resonance imaging
acute cerebral infarction, the method and necessity of recanalization therapy depend on the mechanism and timing of occlusion. Therefore, initially, it is necessary to estimate the mechanism (embolism, atherothrombosis, or other factors) and timing (acute/subacute or chronic occlusion) of occlusion on the both sides. In the present patient, acute cerebral infarction was observed in several major cerebral artery areas, suggesting acute or subacute occlusion of the bilateral internal carotid arteries. Furthermore, dilated cardiomyopathy was present as a concomitant disease, suggesting cardiogenic embolism, and the sites of occlusion were from the cervical portion to cavernous portion of the internal carotid artery, suggesting atherothrombosis. In addition, there was a possibility that the pathogenesis of internal carotid artery occlusion differed between the left and right sides. In the present patient, angiography demonstrated the influx of contrast agent with “to-and-fro” motion and tapering at the origins of the bilateral internal carotid arteries; therefore, we considered the cervical internal carotid artery to be patent, ruling out the possibility of chronic occlusion. In addition, it was possible to insert a guidewire into the cervical internal carotid artery, in which contrast agent did not flow, without resistance, and fresh thrombus was able to be suctioned through a guiding catheter. Under a diagnosis of acute occlusion because of these findings, recanalization therapy was accomplished. For recanalization therapy, thrombectomy using a stent retriever is useful in patients with embolism-related occlusion. Balloon angioplasty and stenting are useful in those with atherothrombosis-related occlusion. In the present patient, there was the possibility of atherothrombosis-related occlusion on both sides; therefore, thrombectomy with ADAPT was initially performed, avoiding the traction of a stent retriever in an arteriosclerotic lesion.

When performing recanalization therapy for occlusion of the bilateral internal carotid arteries, it is necessary to evaluate the side on which treatment should be initially performed immediately. Initially, treatment should be started on the side on which the possibility of acute occlusion is higher based on clinical and imaging findings. If internal carotid artery occlusion involves the middle cerebral artery region, occlusion can be evaluated as “acute” by detecting the hyper-dense middle cerebral artery on CT images. However, if occlusion is localized in the internal carotid artery, as in the present patient, it may be difficult to evaluate acute or chronic occlusion. Second, treatment should be prioritized on the side on which the extent of the ischemic area that can be saved by recanalization therapy, the penumbra, is larger, and this may lead to the final suppression of ischemic change. Furthermore, treatment prioritized on the side on which a main etiological factor for neurological deficits is present may lead to a favorable outcome because of amelioration of clinical symptoms. In the present patient, it was impossible to evaluate acute or subacute occlusion on both sides based on angiography. The left penumbra area was relatively large, and symptoms on the left side, suspected as predominant hemisphere, may have caused aphasia and consciousness disorder. Therefore, recanalization therapy for internal carotid artery occlusion on the left side was initially started. Immediately after recanalization of the left internal carotid artery, there was no reduction of clinical symptoms and recanalization therapy on the right side was subsequently performed, considering acute/subacute right internal carotid artery occlusion.

Acute bilateral internal carotid artery occlusion is rare, but physicians will encounter more patients requiring recanalization therapy for acute bilateral internal carotid artery occlusion due to the increasing number of patients undergoing recanalization therapy for acute cerebral infarction. As recanalization therapy leads to a favorable outcome in some patients with acute bilateral internal carotid artery occlusion, it is necessary to evaluate the side on which treatment should be initially performed, based on clinical and imaging (acute cerebral infarction and perfusion assessment) findings. In addition, with understanding of pathogenesis of occlusion, recanalization therapy in accordance with the mechanism of occlusion should be performed.

Conclusion

We reported a case who survived owing to a successful bilateral mechanical thrombectomy for acute bilateral internal carotid artery occlusion. In patients with acute bilateral internal carotid artery occlusion, it is important to decide the order of recanalization side immediately. Based on clinical symptoms and imaging findings, we could estimate the mechanism and the duration after occlusion of each side, and the strategy provided a successful treatment for the patient.

Disclosure Statement

We declare no conflict of interest regarding this article.
References


