Online September 16, 2021
Journal of Neuroendovascular Therapy 2022; 16: 270–276 DOI: 10.5797/jnet.cr.2021-0040

Case Report

Mechanical Thrombectomy for Bihemispheric Infarction Caused by Acute Unilateral Internal Carotid Artery Occlusion in a Patient with Contralateral Chronic Carotid Occlusion: A Case Report

Hidemoto Fujiwara, Naoto Tsuchiya, Taiki Saito, Ryota Ohkura, and Junichi Yoshimura

Objective: We report a patient with acute bihemispheric infarction who underwent mechanical thrombectomy.
Case Presentation: A 76-year-old man suddenly developed coma and quadriplegia. Brain MRI and MRA revealed acute bihemispheric infarction due to occlusions of both the internal carotid arteries (ICAs). According to the DSA findings, we considered the left ICA as chronic occlusion and the right as acute. Mechanical thrombectomy for the right ICA occlusion was performed. Total recanalization was achieved using a stent retriever 181 minutes after onset. The left hemisphere was perfused by cross circulation through the anterior communicating artery, but the symptoms did not improve. MRI the day after thrombectomy showed extensive bihemispheric infarction. Recanalization for the bilateral hemispheres was maintained, although the left ICA remained occluded. He died 2 months later due to gastrointestinal bleeding.
Conclusion: Acute bihemispheric infarction due to occlusions of both ICAs is a rare entity. The symptoms are very severe and the therapeutic time window is extremely short because of absent collateral pathways. We should consider pre-existing carotid occlusive disease, determine whether the occlusions are acute or chronic, and perform prompt therapy. Further investigation is warranted to obtain a better outcome.

Keywords ▶ mechanical thrombectomy, internal carotid artery, bihemispheric infarction, acute/chronic occlusion

Introduction

Mechanical thrombectomy is the standard procedure for acute ischemic stroke caused by large vessel occlusion (LVO) of the anterior circulation, leading to a favorable outcome.[1] Among patients with LVO stroke, however, multivessel occlusions can occur. These patients develop severe diseases, which result in poor outcomes despite thrombectomy.[2,3] In particular, acute bihemispheric infarction caused by bilateral internal carotid artery (ICA) occlusion leads to the most devastating disease. However, there is limited evidence that demonstrates the effect of thrombectomy for bilateral ICA occlusion.[4–10] We report the case of mechanical thrombectomy in a patient with acute bihemispheric infarction caused by bilateral ICA occlusion.

Case Presentation

The patient was a 76-year-old man with a modified Rankin Scale of 0 prior to the onset. He had a history of hypertension, diabetes, and chronic kidney disease. He fainted while playing golf and was transported to our hospital with an onset-to-door time of 39 minutes. At the time of arrival, he had a blood pressure of 210/113 mm Hg, pulse of 108 beats per minute, and SpO_2 of 98% (O_2 8L). He was

Department of Neurosurgery, Nagano Red Cross Hospital, Nagano, Nagano, Japan

Received: March 13, 2021; Accepted: July 26, 2021
Corresponding author: Hidemoto Fujiwara. Department of Neurosurgery, Nagano Red Cross Hospital, 5-22-1, Wakasato, Nagano, Nagano 380-8582, Japan
Email: hideujisan@yahoo.co.jp

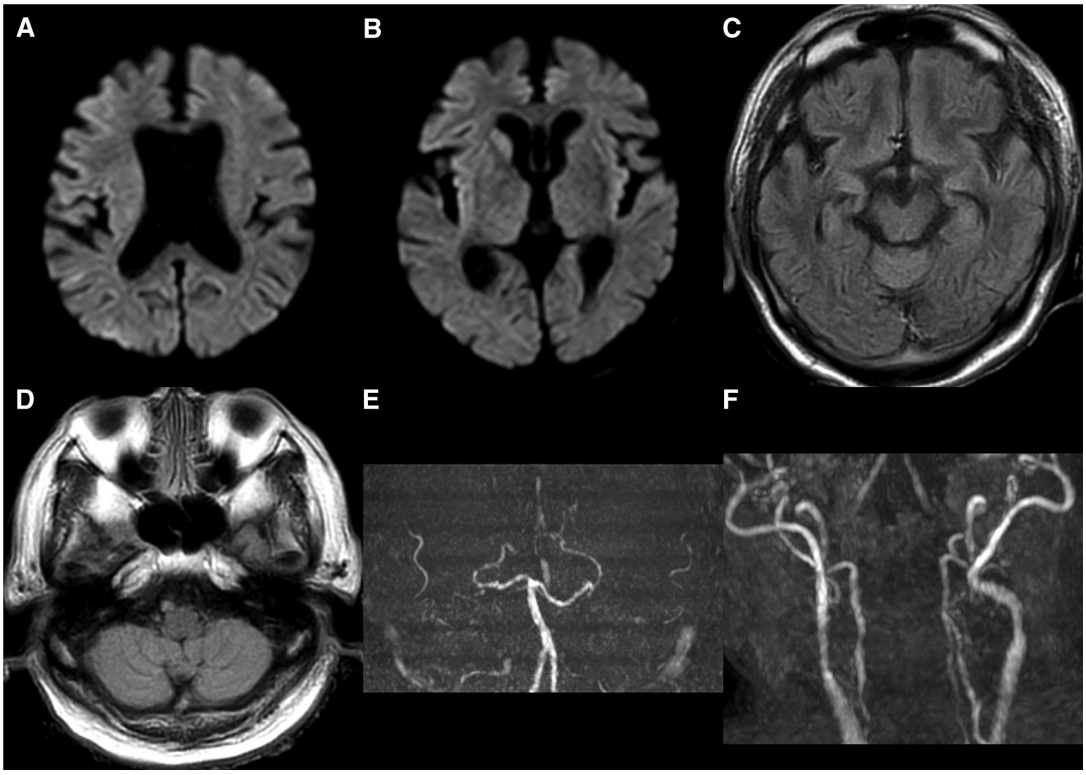

Fig. 1 Brain MRI and MRA about 90 minutes after onset. (**A** and **B**) Diffusion-weighted images showing slightly hyperintense signals in both cerebral hemispheres (ASPECTS+W: R 6, L 8). (**C** and **D**) FLAIR images showing hyperintense vessel signs in bilateral middle and anterior cerebral arteries and also bilateral petrous ICAs. (**E** and **F**) Brain and cervical MRA showing bilateral ICA occlusions. ASPECTS+W: Alberta stroke program early CT score + white matter; ICA: internal carotid artery; L: left; R: right

comatous evaluated as the Japan Coma Scale III-200 with stertorous breathing and roving eye movement. The pupils were the same size, 3 mm in diameter, with normal light reflex. He also presented with quadriplegia and decerebrate rigidity in response to painful stimuli with a National Institutes of Health Stroke Scale of 40. Head CT did not show any signs of bleeding. Chest CT did not show any signs of aortic dissection. On brain and cervical MRA about 90 minutes after the onset, the bilateral ICAs were not visualized from the origin. Diffusion-weighted MRIs showed acute cerebral infarction in both cerebral hemispheres, with an Alberta stroke program early CT score + white matter (ASPECTS+W) of 6 and 8 in the right and left hemispheres, respectively. FLAIR images showed hyperintense vessel signs in the bilateral middle and anterior cerebral arteries. High signal intensity was also identified in the bilateral petrous ICA (**Fig. 1**). Due to a poor respiratory status, perfusion imaging was omitted. An electrocardiogram showed normal sinus rhythm, and a blood test showed the following values: platelets 218000/μL, blood sugar 142 mg/dL, hemoglobin A1c 7.1%, creatinine 2.45 mg/dL, and D-dimer 2.0 μg/mL.

We suspected that occlusion of the ICA occurred either simultaneously in both sides or that the patient had unilateral chronic carotid artery (CA) occlusion and acute occlusion occurred in the contralateral ICA, which perfused both sides of the cerebrum. Intravenous thrombolysis with recombinant tissue plasminogen activator was contraindicated because of significant hypertension. We planned to perform DSA and decide whether revascularization was feasible.

First, the right femoral artery was punctured with a door-to-puncture time of 98 minutes. Left common CA angiograms showed the left ICA occlusion at the origin and a little blood flow into the intracranial ICA via the external CA and the ophthalmic artery (**Fig. 2A**). Right common CA angiograms showed that the contrast agent moved up slowly through the right cervical ICA although mild stenosis at the origin of the right ICA was present (**Fig. 2B**). Collectively, these findings indicated that chronic occlusion was present in the left ICA, while acute

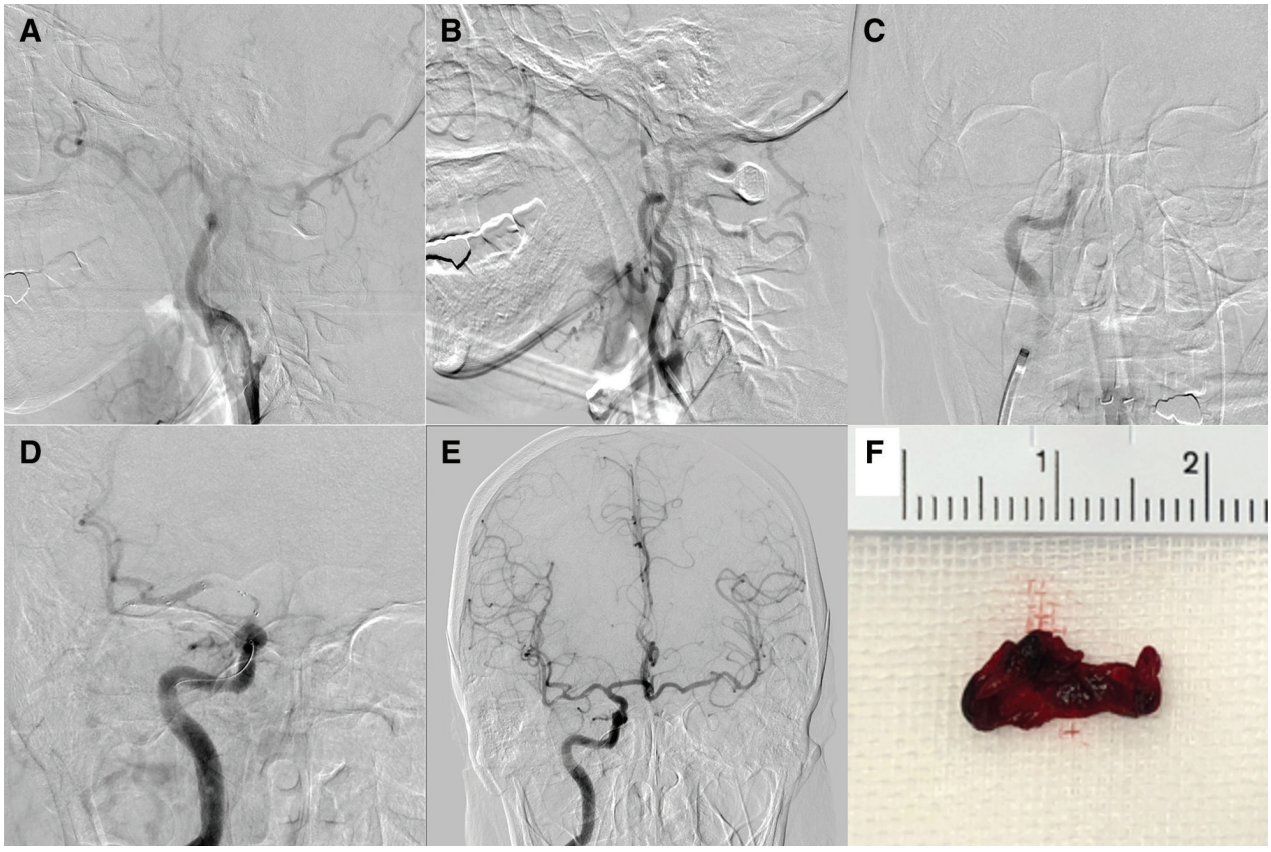

Fig. 2 DSA and mechanical thrombectomy for the right ICA occlusion. (**A**) Left common carotid angiogram (lateral view) showing occlusion of the left ICA at the origin. (**B**) Right common carotid angiogram (lateral view) showing the right ICA occlusion, and the contrast moving slowly upward. (**C**) A 9Fr balloon guiding catheter (Optimo) was placed in the right ICA. Right internal carotid angiogram (anteroposterior view) showing occlusion near the ICA terminus. (**D**) A stent retriever (Solitaire Platinum 6 × 40 mm) was deployed from the right middle cerebral artery (M1 segment) to the right ICA (C2 segment), showing immediate flow restoration. (**E**) Total recanalization (TICI 3) was achieved. The left cerebral hemisphere was perfused by cross flow through the anterior communicating artery. (**F**) Retrieved clot. ICA: internal carotid artery; TICI: thrombolysis in cerebral infarction

occlusion occurred in the right ICA. Thus, we tried to perform mechanical thrombectomy for the right ICA. Placement of a 9Fr Optimo (Tokai Medical Products, Aichi, Japan) in the right ICA showed that the right ICA was occluded around the terminus. Using a Chikai 14 guidewire (Asahi Intecc, Aichi, Japan), a Marksman microcatheter (Medtronic, Minneapolis, MN, USA) was navigated to the right middle cerebral artery (distal M1 segment). Immediate flow was restored after deployment of a Solitaire Platinum 6 × 40 mm stent retriever (Medtronic) from the middle cerebral artery (M1 segment) to the ICA (C2 segment). A large blood clot was collected as the Solitaire Platinum device was removed. Total recanalization for both sides of the cerebrum was achieved via the anterior communicating artery (thrombolysis in cerebral infarction 3), with a puncture-to-recanalization time of 44 minutes and onset-to-recanalization time of 181 minutes (**Fig. 2C**–**2F**).

Neurological symptoms did not improve after operation. Brain MRI and MRA on postoperative day (POD) 1 revealed extensive bihemispheric infarction, mainly in the cerebral cortices and basal ganglia, although the recanalized vessels were patent. The left ICA remained occluded, and black-blood MRI (T1 VISTA) showed a high signal intensity within the entire length of the left ICA (**Fig. 3**). The patient did not develop significant cerebral edema or hemorrhagic infarction. He became bedridden. A transthoracic echocardiogram was normal, and atrial fibrillation was not detected during the postoperative course. A carotid duplex ultrasound showed that the left ICA was totally occluded from the origin, while the right CA had mild stenosis with calcified plaques at the bifurcation. MRA on POD 14 confirmed that the left ICA remained occluded. We supposed that the right acute ICA occlusion was caused by embolic mechanism; however, cardiogenic embolism was less likely. This might be caused by artery-to-artery

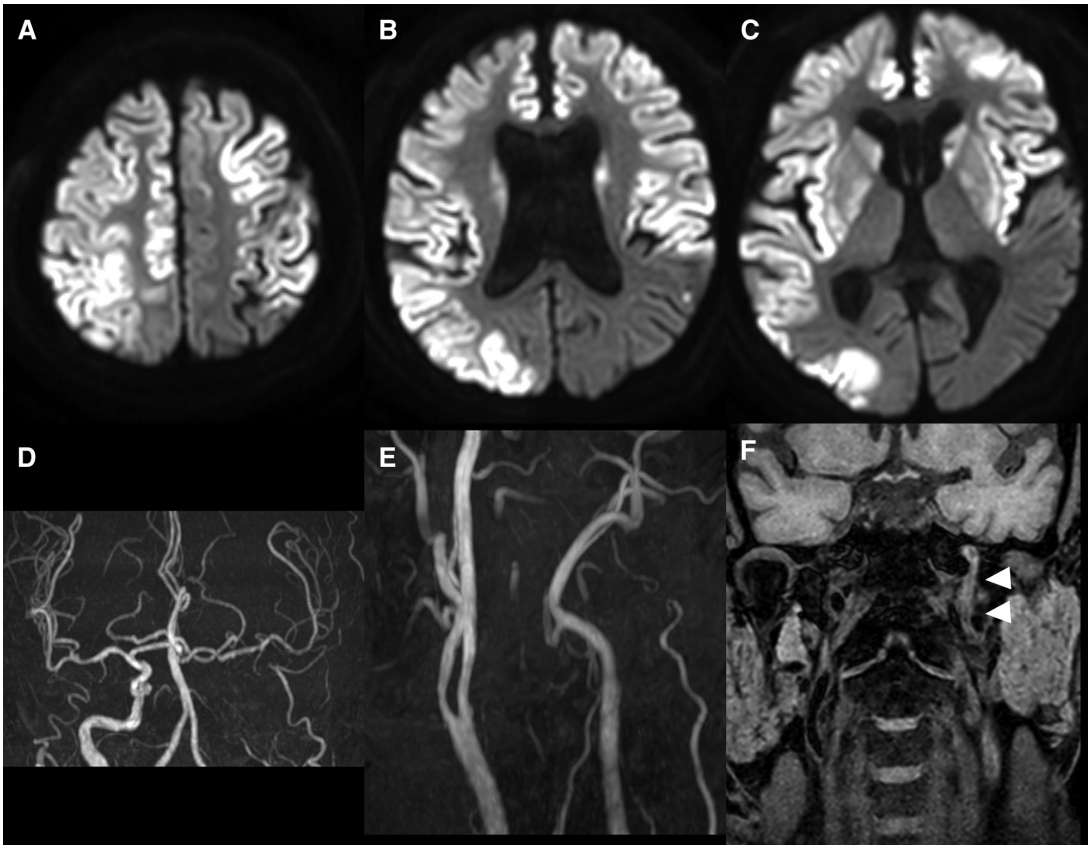

Fig. 3 Brain MRI and MRA the day after mechanical thrombectomy. (**A**–**C**) Diffusion-weighted images showing extensive infarction in the bilateral cerebral hemispheres. (**D**) Brain MRA showing that all intracranial large vessels were patent except for the left ICA. (**E**) Cervical MRA showing that the left ICA remained occluded. (**F**) MR black-blood image (coronal view) showing hyperintense signals in the left cervical ICA (arrowheads), probably indicating chronic occlusion. ICA: internal carotid artery

embolism from the right CA or other embolic sources. Thus, 100 mg of aspirin per day was administered through a nasogastric tube. However, he died on POD 55 due to gastrointestinal bleeding.

Discussion

Approximately 5% of patients with acute cerebral infarction develop bilateral lesions in the anterior circulation.[11] However, acute cerebral infarction caused by bilateral LVO in the anterior circulation is very rare with its incidence ranging between 0.27% and 0.34%.[12,13] Bilateral LVO include bilateral ICA occlusion, bilateral middle cerebral artery (M1) occlusion, and unilateral ICA occlusion plus contralateral middle cerebral artery (M1) occlusion.[4–10,12–15]

Acute bihemispheric infarction caused by LVO is a very severe condition that leads to sudden coma and quadriplegia. While some characteristics of bihemispheric infarction are similar to those of acute occlusion of the basilar artery, patients with bihemispheric infarction do not show anisocoria and have intact brainstem reflexes including pupillary reactivity. They often develop severe respiratory failure and may require intubation. Due to the need for systemic management, it is often difficult to perform enough brain examination within the limited time frame.[5,14]

Compared with bilateral occlusion of the middle cerebral artery,[14] or unilateral ICA occlusion plus contralateral middle cerebral artery occlusion,[13] bilateral ICA occlusion leads to more severe disease with poorer outcome.[5,6,8,12,15] In terms of cerebral hemodynamics, bilateral ICA occlusion is characterized by reduced perfusion of entire hemispheres including territories of the anterior cerebral arteries. Since collateral circulation cannot be established in most cases, bilateral ICA occlusion leads to significant ischemic insult. The therapeutic time window for bilateral ICA occlusion is considered extremely short.

Therefore, rapid diagnosis and treatment is critical for patients with bilateral ICA occlusion. However, it is very challenging to accurately identify the pathological condition of bilateral ICA occlusion. Bilateral ICA occlusion can be caused by a number of mechanisms, including simultaneous occlusion of bilateral ICA and acute occlusion of unilateral ICA with contralateral chronic carotid occlusion. We have to determine whether the occlusion is acute or chronic on each side with the short time frame and limited information. The etiological factors include embolism, atherosclerosis, dissection, and vasculitis, and these conditions may be different on each side. Furthermore, the site of occlusion (i.e., cervical or intracranial) needs to be identified for each side.[16)]

Patients with bilateral ICA occlusion presenting sudden and severe onset were often reported to have simultaneous embolic occlusion of both sides.[5,6,8,9,12)] Bilateral CA occlusion caused by atherosclerosis may only result in a mild disease since collateral circulation develops as stenosis progresses.[17)] CA stenosis and occlusion may also be indicated by the presence of calcified lesions on CT and ultrasound.[16,18)] It is typically accepted that the side with severe symptoms is caused by acute occlusion.[6,9,17)] Hyperdense middle cerebral artery sign on CT and hyperintense vessel sign on FLAIR are considered characteristics of acute occlusions. However, Ota et al. reported a case of acute occlusion that did not show these signs.[5)] Thus, the significance of these signs remains to be elucidated. A study also reported that in the chronically occluded side, the infarct volume is relatively small as a result of watershed shift due to the development of collateral circulation.[15)]

DSA is critical in defining acute vs. chronic occlusion, as well as the site of occlusion, cause of occlusion, and appropriate treatment strategies. Acute occlusion is characterized by an abrupt sharp end, sharp tapered end, and sharp end with embolus, while chronic occlusion is characterized by a round blunt end.[12)] Hong et al. examined site of acute ICA occlusion with CTA and DSA, and demonstrated that a stump pattern was common in the cervical region, while spearhead and streak patterns were common in the cavernous portion and terminus; however, approximately 30% in the terminus showed a stump pattern.[19)] Development of collateral circulation is another indicator to differentiate acute vs. chronic occlusion as well as the site of occlusion, and is associated with the treatment outcome.[16)]

In the present study, the DSA findings were helpful to determine that acute occlusion occurred in the right ICA. Preoperative ASPECTS+W value was higher in the left hemisphere (right: 6, left: 8), and the infarct volume was smaller in the left after thrombectomy, which might reflect watershed shift. Findings of postoperative carotid duplex ultrasound, black-blood MRI, and cervical MRA collectively indicated that the left ICA was chronic total occlusion.[18)]

Table 1 summarizes findings from the literature in which emergency endovascular treatment was performed for acute bihemispheric infarction caused by bilateral ICA occlusion.[4–10)] Acute and simultaneous occlusion of bilateral ICA due to cardiogenic embolism resulted in a very severe disease, and most patients died despite thrombectomy.[5,6,8)] However, the addition of thrombectomy resulted in the survival of a patient with cardiogenic embolism who had atherosclerosis and collateral circulation.[9)] Thrombectomy was typically initiated from the side that was suspected to acute occlusion as well as the side where an extensive penumbral region was present. In one of the cases, thrombectomy was performed simultaneously on both sides by two interventionalists to minimize the time to recanalization.[8)] In two cases, carotid atherosclerosis was considered the cause of occlusions. Agnoletto et al. successfully performed CA stenting for the acute occlusion of the ICA in a patient with contralateral chronic total occlusion and achieved a good outcome.[7)] Huang et al. performed percutaneous transluminal angioplasty for unilateral cervical ICA occlusion and stenting for contralateral intracranial ICA occlusion and obtained significant improvement in the symptoms.[10)] It is important to always consider the possibility of carotid steno-occlusive disease, since appropriate endovascular treatments can improve the outcome. Although we did not find any cases of tandem occlusion in the series, it also should be kept in mind.[20)]

In the present study, we performed thrombectomy with a stent retriever for acute occlusion of the ICA that served the bilateral anterior circulation in a patient with asymptomatic chronic CA occlusion on the contralateral side. To our knowledge, this is the first case who underwent thrombectomy for such hemodynamic condition. Although we achieved total recanalization 181 minutes after the onset, extensive bihemispheric infarction developed, which resulted from rapid progression of ischemia until successful recanalization. Although the patient visited our hospital only 39 minutes after the onset, it took approximately 50 minutes to obtain MRI and MRA. This further delayed the overall process; it took 142 minutes until recanalization. There was still room for improvement in this aspect. Particularly, DSA should be started rapidly to aid in this decision-making process.

Table 1 Summary of reported cases who underwent emergent endovascular therapy for acute bilateral hemispheric infarction due to both ICA occlusions

Author (year)	Age/ sex	Side	Acute/ Chronic	Occlusion site	Etiology	NIHSS	ASPECTS	Endovascular therapy	TICI	PTR (min)	OTR (min)	mRS at 30 days
Ebata et al. (2017)[4]	38/F	R	Acute	Cervical	Takayasu arteritis	NA	NA	(1) PTA + Penumbra	2B	NA	600	3
		L	Acute	Siphon				(2) Solitaire 6 × 30	2B			
Ota et al. (2018)[5]	76/F	R	Acute	Cervical	CES	40	4	(1) ACE60 + Trevo 4 × 20	2B	(1) 32 52	312	6
		L	Acute	Cervical			6	(2) ACE60 + Trevo 4 × 20	3	(2) 20		
Fan et al. (2018)[6]	78/F	R	Acute	NA	CES	40	10	Stent retriever	2C	NA	157 + PTR	6
		L	Acute				10	Stent retriever	2B			
Agnoletto et al. (2019)[7]	54/M	R	Chronic	Cervical	Atherosclerosis	22	10	–	3	NA	NA	0
		L	Acute	Cervical			10	PTA and CAS				
Larrew et al. (2020)[8]	Middle-aged/M	R	Acute	Terminus	CES	40	8	ACE68	3	32 (simultaneous)	NA	6
		L	Acute	Terminus			10	ACE68	2B			
Tsujimoto et al. (2020)[9]	82/M	R	Acute	Cavernous	CES athero-sclerosis as background	20	10	(2) ACE68 + Solitaire 6 × 40	2B	(2) 39 86	276	4
		L	Acute	Cavernous			10	(1) ACE68	3	(1) 47		
Huang et al. (2020)[10]	72/M	R	Acute	Terminus	Atherosclerosis	35	NA	(2) PTA + stent (intracranial)	3	180	770	4
		L	Acute	Terminus				(1) PTA	3			
Present case	76/M	R	Acute	Terminus	Embolism	40	6	Solitaire 6 × 40	3	44 44	181	5
		L	Chronic	Cervical	Atherosclerosis		8	–		–		

ASPECTS: Alberta stroke program early CT score; CAS: carotid artery stenting; CES: cardioembolic stroke; F: female; L: left; M: male; mRS: modified Rankin Scale; NA: not available; NIHSS: National Institutes of Health Stroke Scale; OTR: onset to reperfusion time; PTA: percutaneous transluminal angioplasty; PTR: puncture to reperfusion time; R: right; TICI: thrombolysis in cerebral infarction

Recanalization should be completed as soon as possible; however, there is no evidence to indicate the exact time window for tissue salvage. Some studies to date evaluated cerebral perfusion images before endovascular treatment.[6–9] However, there is little evidence to demonstrate that perfusion images can be used to determine treatment strategies and predict outcomes. Further studies including perfusion analysis should be performed to better understand the disease and to discuss treatment strategies in order to improve outcomes.

Conclusion

We described the case of mechanical thrombectomy for acute unilateral ICA occlusion in a patient with contralateral chronic carotid occlusion. Acute bihemispheric infarction due to bilateral ICA occlusion leads to a very severe disease with an extreme short therapeutic time window. Although this is a rare entity, it is important to recognize the complex disease mechanism, to make an accurate diagnosis, and to perform prompt therapy. Future studies are needed to determine effective treatment strategies in order to improve the outcome.

Disclosure Statement

The authors declare no conflicts of interest.

References

1) Goyal M, Menon BK, van Zwam WH, et al. Endovascular thrombectomy after large-vessel ischaemic stroke: a meta-analysis of individual patient data from five randomised trials. *Lancet* 2016; 387: 1723–1731.
2) Kaesmacher J, Mosimann PJ, Giarrusso M, et al. Multivessel occlusion in patients subjected to thrombectomy: prevalence, associated factors, and clinical implications. *Stroke* 2018; 49: 1355–1362.
3) Kaesmacher J, Meyer L, Styczen H, et al. Primary multivessel occlusions treated with mechanical thrombectomy: a multicenter analysis and systemic literature review. *Stroke* 2020; 51: e232–e237.
4) Ebata T, Uemura J, Yamazaki H, et al. [A case of Takayasu arteritis with acute bilateral occlusion of the internal carotid arteries]. *Brain Nerve* 2017; 69: 665–669 (in Japanese).
5) Ota K, Matsubara N, Takahashi I, et al. A case of acute simultaneous bilateral internal carotid artery occlusion treated by thrombectomy. *JNET J Neuroendovasc Ther* 2018; 12: 386–392.
6) Fan CF, Liebeskind D, Hinman J, et al. Rapid revascularization of simultaneous bilateral ICA occlusions causing coma. *Neurology* 2018; 90 (15 Supplement): P3.252.
7) Agnoletto GJ, Granja MF, Hanel R, et al. Facing the crossroads: acute stroke with bilateral carotid occlusion. *BMJ Case Rep* 2019; 12: e229638.
8) Larrew T, Hubbard Z, Almallouhi E, et al. Simultaneous bilateral carotid thrombectomies: a technical note. *Oper Neurosurg (Hagerstown)* 2020; 18: E143–E148.
9) Tsujimoto Y, Ikeda H, Otsuka R, et al. A survived case of acute bilateral internal carotid artery occlusion treated by mechanical thrombectomy. *JNET J Neuroendovasc Ther* 2020; 14: 195–201.
10) Huang J, Liu H, Chen H, et al. Acute bilateral internal carotid artery occlusion presenting with symmetric cortical infarctions exhibits dramatic improvement after mechanical thrombectomy. *World Neurosurg* 2020; 141: 149–152.
11) Saito K, Moriwaki H, Oe H, et al. Mechanisms of bihemispheric brain infarctions in the anterior circulation on diffusion-weighted images. *AJNR Am J Neuroradiol* 2005; 26: 809–814.
12) Kwon SU, Lee SH, Kim JS. Sudden coma from acute bilateral internal carotid artery territory infarction. *Neurology* 2002; 58: 1846–1849.
13) Pop R, Manisor M, Wolff V, et al. Endovascular treatment in two cases of bilateral ischemic stroke. *Cardiovasc Intervent Radiol* 2014; 37: 829–834.
14) London D, London F, Vandermeeren Y, et al. Successful double mechanical thrombectomy in bilateral M1 middle cerebral artery occlusion. *Acta Neurol Belg* 2020; 120: 211–213.
15) Berican E, Oguz KK, Topcuoglu MA. Bilateral acute internal carotid artery occlusion presenting with sudden coma. *Internal Med* 2009; 48: 1565–1566.
16) Malhotra K, Goyal N, Tsivgoulis G. Internal carotid artery occlusion: pathophysiology, diagnosis, and management. *Curr Atheroscler Rep* 2017; 19: 41.
17) Jadhav AP, Ducruet AF, Jankowitz BT, et al. Management of bilateral carotid occlusive disease. *Interv Neurol* 2016; 4: 96–103.
18) Xu B, Li C, Guo Y, et al. Current understanding of chronic total occlusion of the internal carotid artery. *Biomed Rep* 2018; 8: 117–125.
19) Hong JM, Lee SE, Lee SJ, et al. Distinctive patterns on CT angiography characterize acute internal carotid artery occlusion subtypes. *Medicine (Baltimore)* 2017; 96: e5722.
20) Mayer L, Grams A, Freyschlag CF, et al. Management and prognosis of acute extracranial internal carotid artery occlusion. *Ann Transl Med* 2020; 8: 1268.