

Occlusion of All Aortic Arch Great Vessels: Acute Revascularization to Perform Endovascular Stroke Therapy

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Key words

- Angioplasty
- Aortic arch reconstruction
- Arterial occlusive disease
- Intracranial stenting
- Mechanical thrombolysis
- Stroke

Abbreviations and Acronyms

CCA: Common carotid artery
CT: Computed tomography
ECA: External carotid artery
ICA: Internal carotid artery
MCA: Middle cerebral artery
TIA: Transient ischemic attack



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Citation: *World Neurosurg.* (2014).

<http://dx.doi.org/10.1016/j.wneu.2013.09.019>

Journal homepage: www.WORLDNEUROSURGERY.org

Available online: www.sciencedirect.com

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INTRODUCTION

Steno-occlusive disease of the extracranial vessels can lead to cerebrovascular hypoperfusion or embolic infarcts (3, 6). Remarkably, those patients might remain relatively asymptomatic as long as sufficient collateral circulation has developed (6, 12, 15, 18). Occlusion of multiple extracranial vessels is relatively infrequent (2, 3, 17, 21). To date, there are only a few reports of 4-vessel occlusion (2, 7, 12, 15). We present an exceptional case in which all aortic arch great vessels were occluded at their origins. Prompt endovascular intervention followed by elective cardiothoracic revascularization yielded an excellent outcome.

CASE REPORT

A 46-year-old man presented with symptoms of left hemispheric stroke. On arrival at the emergency department, the patient's National Institute of Health Stroke

■ **BACKGROUND:** We present a case of a patient with a left-sided stroke and occlusion of all aortic arch great vessels who was treated successfully with endovascular intervention followed by delayed cardiothoracic revascularization.

■ **CASE REPORT:** A 46-year-old man presented with acute onset of dense right hemiparesis, facial droop, and aphasia with an initial National Institute of Health Stroke Score of 15. The patient was taken for emergent angiography after failing intravenous tissue plasminogen activator thrombolysis. Dedicated angiography of the aortic arch revealed occlusion of all great vessels, including the right brachiocephalic, left common carotid, and left subclavian artery. Delayed arterial filling of the right brachiocephalic and left subclavian artery by aberrant collaterals was seen. More distally, flow into the bilateral subclavian arteries, right common carotid artery, and left vertebral artery was appreciated. Serial balloon angioplasty of the left common carotid artery origin reconstituted flow. Subsequent selective angiogram of the left internal carotid artery revealed a proximal middle cerebral artery occlusion. Intra-arterial injection of tissue plasminogen activator followed by mechanical thrombectomy and intracranial stenting restored flow in the middle cerebral artery. Two months later the patient underwent aortic arch reconstruction with bifurcated graft to the brachiocephalic artery and left common carotid artery. At 1-year follow-up, the patient's examination revealed almost complete resolution of right hemiparesis with minimal hand weakness and mild expressive aphasia.

■ **CONCLUSIONS:** We report a rare case of occlusion of all aortic arch great vessels. Combined endovascular intervention and surgical revascularization resulted in an excellent durable outcome.

Scale score was 15. He had a dense right hemiparesis with trace movement in the lower extremity, global aphasia, and marked facial droop. Physical examination also revealed a markedly higher blood pressure in lower extremities compared to upper extremities (approximately 100 mm Hg systolic blood pressure differences). His medical history was positive for a 40 pack/year smoking history. Noncontrast head computed tomography (CT) revealed no intracranial hemorrhage and was positive for a dense left middle cerebral artery (MCA) sign (Figure 1). Subsequently, the patient was given intravenous tissue plasminogen activator per the institutional stroke protocol. CT angiography was not performed routinely as part of our stroke protocol algorithm at the time of this case

but has since been incorporated. His neurological examination did not improve significantly; therefore, he was taken emergently for angiography and possible endovascular intervention.

Diagnostic angiography was initiated via a common femoral artery approach. During the diagnostic angiogram, we were unable to pass the guidewire distally into the left common carotid artery to advance the catheter. We do not routinely perform injection of the aortic arch prior to catheterization of the distal vessels. Because of the difficulty with catheterization, a dedicated high-flow injection of the aortic arch revealed occlusion of all 3 great vessels, including right brachiocephalic, left common carotid, and left subclavian artery (Figure 2). There was a delayed arterial



Figure 1. Noncontrast head computed tomography demonstrating a hyperdense left middle cerebral artery sign.

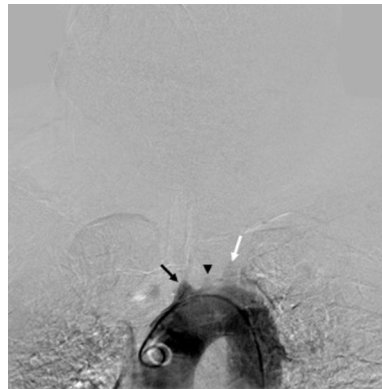


Figure 2. Dedicated angiography of the aortic arch revealed occlusion of all great vessels, including right brachiocephalic (black arrow), left common carotid (arrowhead), and left subclavian artery (white arrow).



Figure 4. Cerebral angiogram demonstrating reconstitution of flow in the left common carotid artery after multiple serial balloon angioplasties. Note that residual stenosis is present at the left common carotid origin even after the multiple angioplasties.

filling of the right brachiocephalic and left subclavian artery by multiple aberrant collaterals (Figure 3). More distally, we observed flow into the right common carotid and the left vertebral artery in a delayed fashion (Figure 3). A 6-F MPC Envoy guide catheter (Cordis Neurovascular, Miami Lakes, FL) was parked in the stump of the left common carotid artery. After multiple attempts, only an Expedian 14 microwire (ev3 Neurovascular, Irvine, CA) was able to be threaded through a portion of the stump into the proximal common carotid artery. Although we certainly had concern for releasing thrombotic material while opening the vessel, a distal protection filter could not fit through the narrow channel. We then proceeded with serial balloon angioplasty of the origin of the left common carotid artery with a Maverick 2 × 12-mm balloon followed by a 4 × 12-mm balloon (Boston Scientific, Natick, MA), which reconstituted the flow (Figure 4). A 2-mm Maverick balloon was selected after unsuccessfully attempting larger options, including an Aviator balloon (Cordis Neurovascular) and larger size Maverick balloons. A selective left internal carotid run demonstrated a proximal left M1 middle cerebral artery occlusion (Figure 5). We then performed transcatheter injection of 8 mg of intra-arterial tissue plasminogen activator with some decrease in clot burden, although the clot remained occlusive. Therefore, we performed mechanical thrombolysis with

a L5 Merci clot retrieval device (Concentric Medical, Mountain View, CA) in the proximal middle cerebral artery. This step was followed by subsequent clot retrievals from M2 branches utilizing the L4 Merci retrieval (Concentric Medical) with improvement of the flow. However, the superior division of the bifurcation, including the angular branch, remained occluded. As a final salvage maneuver, we deployed a 14 × 4.5-mm Enterprise intracranial stent (Cordis Neurovascular) with some

restoration of the flow into the vessel. Final angiography demonstrated a marked increase in the flow through the middle cerebral territory, but with limitation of flow in distal branches and the superior branch of the left MCA (Figure 6). We would like to note that the endovascular stroke therapy devices available at the time of this case are vastly different than our current practice. Devices such as stent retrievers were not approved by the US Food and Drug Administration; they now constitute our first line of intervention. After the procedure, the patient was transferred to the Neuroscience Intensive Care Unit and

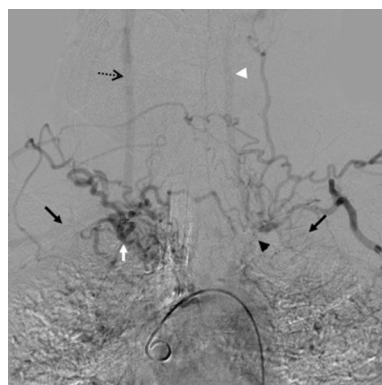


Figure 3. Delayed arterial filling of the right brachiocephalic (white arrow) and the left subclavian artery (black arrowhead) by aberrant collaterals, as well as flow into the bilateral subclavian arteries (black arrows), the right common carotid artery (interrupted black arrow), and the left vertebral artery (white arrowhead).

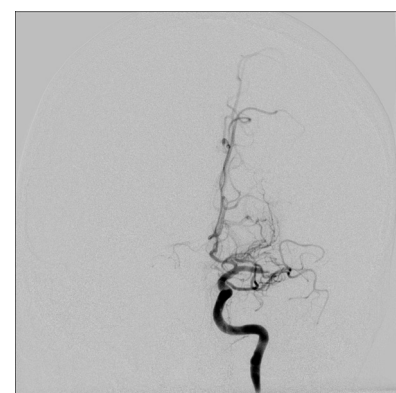


Figure 5. Left internal carotid artery angiogram demonstrating an occlusion of the distal left middle cerebral artery.



Figure 6. Left internal carotid artery angiogram showing restoration of flow in the proximal left middle cerebral artery. Not appreciated in this image is the ongoing stagnation of a few small distal middle cerebral artery branches.

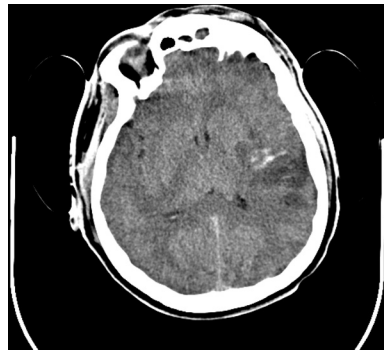


Figure 7. Head computed tomography demonstrating mild hemorrhagic conversion of the ischemic territory and a patchy, contained area of infarct.



Figure 8. Chest computed tomography angiography after aortic arch reconstruction with a bifurcated graft (white arrow) connected to the right brachiocephalic artery (white arrowhead) and to the left common carotid artery (out of plane at this image)

started on aspirin 325 mg and clopidogrel 75 mg daily. He was extubated on post-procedure day 2. Subsequent head CT demonstrated mild hemorrhagic conversion of the stroke; however, the patient continued to do well and follow-up imaging remained stable. The majority of the left hemisphere was preserved (Figure 7). A magnetic resonance image was not obtained due to occupation-related metallic fragments in the orbits. The cardiothoracic surgery service was consulted, and the decision was made to proceed with elective revascularization after the patient recovered from the acute phase of the stroke. A comprehensive evaluation of possible underlying etiologies of his severe vascular disease at an early age revealed smoking, family history, and hyperlipidemia as the primary risk factors; vasculitis workup was negative. The patient was discharged to an inpatient rehabilitation unit with significantly improving right hemiparesis and aphasia. Two months after initial presentation, the patient underwent elective aortic arch reconstruction with a bifurcated graft. One limb connected to the right brachiocephalic artery, and the second limb connected to the left common carotid artery (Figure 8). The left subclavian artery was not revascularized because he had no left arm symptoms nor symptomatic subclavian steal syndrome. Postoperatively, he developed a right coronary occlusion and required a single-vessel bypass. He recovered well from these procedures. Physical

examination at 1 and 3 years after endovascular intervention revealed very mild right hand weakness and mild expressive aphasia. The patient remains independent in all activities of daily living, with a Modified Rankin Scale score of 1. Follow-up CT angiograms at 1 and 3 years revealed patent aortic and intracranial stents as well as the left subclavian artery filling distally from the left vertebral artery.

DISCUSSION

Steno-occlusive disease of the extracranial arteries is a known cause of stroke and cerebral hypoperfusion (6). Although occlusion of a single internal carotid artery or vertebral artery is a frequent finding in patients undergoing cerebral angiography for neurological symptoms, occlusion of multiple extracranial vessels or occlusion of the common carotid artery (CCA) is much less frequent (10, 17). CCA occlusion is present in 0.5% to 5% of cases presenting with stroke or transient ischemic attack (TIA) (4, 8, 14, 20). Similarly, steno-occlusive disease of the brachiocephalic trunk comprises less than 2% of all extracranial causes of cerebrovascular insufficiency (19). Seventy-five percent of patients with flow-limiting atherosclerotic disease involving the subclavian artery have other multiple extracranial lesions (16). If the subclavian artery is occluded, 80% of patients will have involvement of 1 or more additional vessels (5).

There are many case reports of bilateral internal carotid artery (ICA) occlusion as well as a small patient series with short-

term to mid-term follow-up (3, 17). Bogousslavsky et al. also reported on a small cohort of patients with bilateral vertebral artery occlusion (2). Three patients with bilateral CCA occlusion were reported by Zbornikova et al. (21). There is a paucity of case reports of patients with 4-vessel occlusion, and no case series with long-term follow-up (1, 2, 7, 9, 12, 15) (Table 1). None of these cases presented as proximal occlusion of all aortic arch great vessels, as in our patient. Most recently, Kuni et al. presented a case of a 72-year-old man with deep coma (Glasgow Coma Score 4) and hyperthermia who was found to have bilateral ICA and a left vertebral artery origin occlusion. The patient also had a near occlusion of right vertebral artery origin with faint filling of the posterior inferior cerebellar artery and no distal flow. Magnetic resonance imaging showed extensive bilateral strokes, and the patient died 16 hours after admission (7). Previously, Okada et al. reported 2 similar cases (12). The first patient presented with vertigo and was found to have bilateral ICA and bilateral intracranial vertebral artery occlusion. Compensatory pathways included well-developed skull base collaterals from the bilateral maxillary arteries as well as transdural anastomoses from the vertebral arteries. The second patient presented with headaches and hand numbness and was also diagnosed with bilateral ICA and vertebral artery occlusion. This patient had robust

Table 1. Previously Reported Cases of Extracranial Occlusion of Four Vessels (Bilateral Internal Carotid Artery and Vertebral Artery)

Author	Age/Sex	Imaging	Symptoms	Therapy	Outcome
Konaka et al., 2009 (7)	72/Male	Occlusion of bilateral ICA and left VA at origin, critical stenosis of right VA (no flow distal to PICA)	Hyperthermia, deep coma (GCS 4)	Supportive	Death 18 hours after presentation
Okada et al., 1998 (12)					
Case 1	44/Female	Occlusion of bilateral ICA at origins, bilateral VA at skull base	Transient vertigo	Medical management (not specified)	No CVAs for 30 months
Case 2	51/Female	Occlusion of right ICA at C2/3, left ICA at origin, bilateral VA at C1/2	Right hand numbness	Medical management (not specified)	No deficits on discharge
Bogousslavsky et al., 1986 (2)	56/Male	Occlusion of bilateral ICA and VA	Bilateral anterior circulation strokes	Right STA-to-MCA bypass	No CVAs for 8 years
Barnett, 1976 (1)	Not available	Occlusion of bilateral ICA and bilateral distal VA	Three strokes	Not available	Not available
Vitek et al., 1972 (15)	55/Male	Occlusion of left CCA at origin, right ICA at origin and bilateral VA at origins	Mild right hemiparesis and dysarthria	Medical management (warfarin)	Symptoms resolved at 3-month follow-up

ICA, internal carotid artery; VA, vertebral artery; PICA, posterior inferior cerebellar artery; GCS, Glasgow Coma Scale score; CVA, cerebrovascular accident; STA, superficial temporal artery; MCA, middle cerebral artery.

collaterals from meningeal arteries and a vertebrobasilar system mostly fed through a well-developed anterior spinal artery. Both patients suffered small strokes but had very good clinical outcomes with conservative therapy. Vitek et al. presented a case of a 55-year-old man with history of 90% stenosis of the left ICA treated with carotid endarterectomy who was readmitted 9 years later with mild hemiparesis and dysarthria. Cerebral angiography demonstrated occlusion of the bilateral vertebral arteries at the origin, occlusion of right ICA, and occlusion of the left CCA. The whole brain was supplied by bilateral muscular branches from the subclavian arteries feeding the distal right vertebral artery. The patient was treated with anticoagulation (warfarin) and had complete resolution of the symptoms (15). These small case studies noted that smoking is a common finding in patients with multiple extracranial vessel occlusions (6, 11, 14, 17). Our patient had an extensive smoking history and also was diagnosed with hyperlipidemia during the initial admission.

Many of the case reports and small series of chronic multiple extracranial vessel occlusions report either mild symptoms on presentation or good outcomes with

medical management or surgical intervention (3, 6, 12, 15, 18, 21). In a small case series of 3 patients with bilateral CCA occlusion treated medically, Zbornikova et al. noted no new strokes for up to 6.5 years after the initial presentation. One patient suffered TIAs and amaurosis fugax, whereas all patients complained of dizziness and chronic fatigue (21). Bogousslavsky et al. prospectively followed up 7 patients with bilateral ICA occlusion for 14 months. Two of those patients underwent external carotid artery (ECA) endarterectomy, and the other 5 patients were treated conservatively. Of the 5 treated conservatively, 1 patient suffered a minor stroke and 1 patient was symptom free. The other 3 patients had TIAs or amaurosis fugax that resolved completely in 2 patients after adequate anticoagulation was introduced. Despite the events, all of the patients remained alive and at their baseline functional status at final follow-up. The authors concluded that bilateral ICA occlusion might have a benign course when patients are carefully monitored and treated accordingly (3). The same senior author published a prospective case series of 17 patients with bilateral vertebral artery occlusion and

reported a mortality rate of 4.5% per year with a stroke rate of 1.8% per year. Major functional disability was seen in 16.7% of the survivors (2). Karaköse et al. presented a case of a brachiocephalic trunk and left common carotid occlusion in a patient with right arm pain and TIAs. The patient underwent a left common carotid artery embolectomy and Y-graft placement into the brachiocephalic trunk with restoration of flow (6). These small case studies concluded that patients with chronic occlusion of extracranial vessels become symptomatic either from low blood flow or from embolic phenomenon (3). Our patient presented with dense hemiparesis and aphasia related to occlusion of the left CCA at the origin and occlusive thrombus in MCA. He made a remarkable recovery with almost complete resolution of hemiparesis and residual mild aphasia at 1- and 3-year follow-up.

In cases of chronic occlusion of multiple extracranial vessels, development of collaterals and relatively intact intracranial vasculature plays a pivotal role in prevention of cerebral ischemic insults (12, 15). In cases of bilateral ICA occlusion, collateral flow is provided from either the ECA or the vertebrobasilar system (12, 13). If

the vertebral arteries are stenotic or occluded, the flow is usually provided through anastomotic vessels from the occipital artery (or other ECA branches) and thyrocervical trunk. In our case, in which all of the great vessels were occluded at the origins, there was a remarkable network of collateral vessels that filled the right CCA and left vertebral artery in a delayed fashion. Our patient's aberrant collaterals originated from the aortic arch itself; one possibility is that they were intercostal artery collaterals.

We report a rare case of occlusion of all aortic arch great vessels. Our patient presented with stroke symptoms and was treated successfully with a combined endovascular approach and staged surgical revascularization with almost complete resolution of symptoms. Although endovascular intervention provided immediate benefit during the acute stroke period, cardiothoracic surgical revascularization prevented further cerebrovascular ischemic insults related to low flow or embolic phenomena. This case illustrates that patients with chronic multiple extracranial vessel occlusion presenting with acute symptoms can have a good outcome with adequate treatment.

REFERENCES

- Barnett HJ: The pathophysiology of transient cerebral ischemic attacks. *Med Clin North Am* 63: 649-679, 1979.
- Bogousslavsky J, Gates PC, Fox AJ, Barnett HJ: Bilateral occlusion of vertebral artery: clinical patterns and long-term prognosis. *Neurology* 36: 1309-1315, 1986.
- Bogousslavsky J, Regli F: Cerebro-retinal ischemia after bilateral occlusion of internal carotid artery. A study with prospective follow-up. *Neuroradiology* 27:238-247, 1985.
- Collice M, D'Angelo V, Arena O: Surgical treatment of common carotid artery occlusion. *Neurosurgery* 12:515-524, 1983.
- Fields WS, Lemak NA: Joint study of extracranial arterial occlusion. VII. Subclavian steal—a review of 168 cases. *JAMA* 222:1139-1143, 1972.
- Karaköse S, Karabacakoglu A, Solak H: Bilateral common carotid occlusion without neurological deficit. *Australas Radiol* 46:412-415, 2002.
- Konaka K, Miyashita K, Ishibashi-Ueda H, Naritomi H: Severe hyperthermia caused by four-vessel occlusion of main cerebral arteries. *Intern Med* 48:2137-2140, 2009.
- Levine SR, Welch KM: Common carotid artery occlusion. *Neurology* 39:178-186, 1989.
- Miyamoto S, Kikuchi H, Karasawa J, Nagata I, Ikota T: Moyamoya disease associated with bilateral occlusion of the vertebral artery. *Surg Neurol* 22:21-28, 1984.
- North RR, Fields WS, Debakey ME, Crawford ES: Brachial-basilar insufficiency syndrome. *Neurology* 12:810-820, 1962.
- Ogino M, Nagumo M, Nakagawa T, Nakatsukasa M, Murase I: Axilloaxillary bypass for the treatment of subclavian artery stenosis complicated by bilateral common carotid artery occlusion: technical case report. *Neurosurgery* 53:444-447; discussion 447, 2003.
- Okada Y, Shima T, Nishida M, Kagawa R: Magnetic resonance angiography visualization of four vessel (bilateral carotid and vertebral artery) occlusion—two case reports. *Neurol Med Chir (Tokyo)* 38:28-33, 1998.
- Okuchi K, Kamada K, Inui S, Takahashi T, Hiramatsu K, Tanaka Y, Hashimoto H: Bilateral absence of the internal carotid artery—case report. *Neurol Med Chir (Tokyo)* 28:685-689, 1988.
- Podore PC, Rob CG, DeWeese JA, Green RM: Chronic common carotid occlusion. *Stroke* 12: 98-100, 1981.
- Vitek JJ, Halsey JH, McDowell HA: Occlusion of all four extracranial vessels with minimal clinical symptomatology. Case report. *Stroke* 3:462-466, 1972.
- Walker PM, Paley D, Harris KA, Thompson A, Johnston KW: What determines the symptoms associated with subclavian artery occlusive disease? *J Vasc Surg* 2:154-157, 1985.
- Waltimo O, Fogelholm R: Bilateral internal carotid artery thrombosis. Prognosis and risk factors. *Acta Neurol Scand* 51:240-244, 1975.
- Wróblewski L, Korzeniewska A, Barycki J: A case of bilateral internal carotid artery and right vertebral artery occlusion [in Polish]. *Neurol Neurochir Pol* 31:601-606, 1997.
- Wylie EJ, Effeney DJ: Surgery of the aortic arch branches and vertebral arteries. *Surg Clin North Am* 59:669-680, 1979.
- Yamaguchi S, Oki S, Ogasawara H, Sato H, Ito Y, Hibino S: A case of simultaneous bilateral carotid occlusion [in Japanese]. *No Shinkei Geka* 25: 247-251, 1997.
- Zbornikova V, Vorstrup S, Ryding E: Bilateral common carotid artery occlusion with minimal neurological deficit: long term follow up in 3 patients. *Neurol Res* 18:109-116, 1996.

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Received 24 May 2013; accepted 10 September 2013

Citation: World Neurosurg. (2014).

http://dx.doi.org/10.1016/j.wneu.2013.09.019

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