A massive postoperative stroke caused by a carotid thrombus that occurred during the surgical repair of an aortic dissection

Hiroko Nemoto, Keiji Uchida, Tomoyuki Minami, Shota Yasuda, Tomoki Cho and Munetaka Masuda

Abstract
A 59-year-old man with no prior neurological deficits developed a massive stroke during the repair of a double-barreled acute type A aortic dissection with major entry in the ascending aorta and an occluded brachiocephalic artery. As right cerebral ischemia was alleviated by the circle of Willis, the patient was alert and conscious preoperatively. Nevertheless, the thrombus in the right carotid artery induced a severe postoperative right cerebral embolism. In conclusion, occlusion of the carotid artery is a risk factor of postoperative severe stroke, even in patients without neurological symptoms preoperatively.

Keywords
Postoperative stroke, carotid thrombus, type A aortic dissection, circle of Willis

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Introduction
Cerebral malperfusion occurs in 6.0%–14% of patients with an acute type A aortic dissection (ATAAD)\(^1\)\(^–\)\(^3\) and is the major factor that worsens a patient’s prognosis. Malperfusion is an ischemic complication, in which the clinical findings match with the imaging findings; however, in cases with cerebral malperfusion, the imaging findings of carotid dissection do not always match the neurological symptoms. Moreover, the mechanisms of stroke associated with ATAAD have not been well researched. In this report, we present a case in which the patient had a massive cerebral infarction of the right hemisphere after central repair. Computed tomography (CT) showed an obstruction in the right carotid artery; however, the patient had no neurological deficits preoperatively. We investigated the mechanism of stroke in this patient.

Case
A 59-year-old man arrived at our hospital by ambulance due to the sudden onset of back pain. Initial evaluation revealed E4V5M6 on the Glasgow Coma Scale (GCS);\(^4\) blood pressure of 83/55 and 121/71 mmHg on the right and left arms, respectively; a regular pulse of 71 beats per minute; and 96% saturation of percutaneous oxygen measurement on 10 L of oxygen per minute. He had no signs of paralysis. Contrast-enhanced CT showed a double-barreled ATAAD with a major entry in the ascending aorta and an occluded brachiocephalic artery (Figure 1(a) and (b)). Carotid ultrasonography revealed a floating ball-shaped thrombus in the true lumen of the right common carotid artery (Figure 1(c)). The patient underwent an emergency central repair operation. Cardiopulmonary bypass was performed with arterial perfusion via the left subclavian and right femoral arteries. After the body temperature was reduced to 25\(^\circ\)C, selective cerebral perfusion (SCP) under circulatory arrest started from the left common carotid artery (250–350 mL/min, 37–78 mmHg) and left subclavian artery (200–300 mL/min, 23–59 mmHg) using 12-Fr cannulas. In the beginning, we avoided inserting the cannula into the brachiocephalic artery. No thrombus was observed in the retrograde backflow from the opened brachiocephalic artery.
brachiocephalic artery; then, a 5-Fr balloon catheter was directly inserted approximately 20 cm from the true lumen of the brachiocephalic artery to the right common carotid artery. However, we could not remove the thrombus. As regional cerebral oxygen saturation (\(\text{rSO}_2\)) on the right side had decreased from 63% to 49%, we had no choice but to start SCP including the brachiocephalic artery with a 14-Fr cannula (450–600 mL/min, 38–61 mmHg), and ascending aortic replacement was performed. The duration of SCP was 52 min. On the following day, left hemiplegia was noted. Magnetic resonance imaging showed a massive cerebral infarction of the right hemisphere with a focus on the right parietal lobe (Figure 2). We started treatment for stroke with intravenous infusion of edaravone. The patient was extubated 2 days after the operation. Following rehabilitation for left hemiplegia, the patient could walk by himself. He was transferred to a different hospital for continuing rehabilitation on postoperative day 33.

**Discussion**

Cerebral malperfusion is associated with ATAAD due to obstruction of the true lumen by the expanded false lumen.\(^5\) It tends to occur on the right side. In general, central repair is prioritized. Recently, urgent central repair and reperfusion of the carotid artery have been reported to provide excellent

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**Figure 1.** Contrast-enhanced computed tomography (CT) shows a double-barreled DeBakey type I acute aortic dissection with major entry into the ascending aorta (a) and an occluded brachiocephalic artery (b). Carotid ultrasonography shows a floating ball-shaped thrombus in the right common carotid artery (c).

**Figure 2.** Magnetic resonance imaging of the patient. A massive right stroke was seen postoperatively (a). The right middle cerebral artery was occluded from the distal part of M1 (horizontal segment) by a thrombus (b).
results, even in cases with severe cerebral malperfusion.6,7 Regarding early reperfusion, Okita et al.8 reported preoperative direct perfusion between the femoral and the right carotid arteries in patients with preoperative neurological symptoms. Furthermore, Gomibuchi et al.9 reported that early reperfusion and extra-anatomic revascularization may reduce the risk of neurological complications in patients with “imaging cerebral malperfusion” (defined as occlusion or severe stenosis of the unilateral or bilateral carotid arteries on contrast-enhanced CT and carotid duplex), with or without preoperative neurological symptoms. Sugiyama et al.10 reported that direct carotid artery perfusion during aortic repair resulted in good postoperative neurological and late mortality outcomes compared with conventional selective cerebral perfusion.

As brain tissues can tolerate ischemia only for a few minutes, it would be too late for patients with an obstructed carotid artery to undergo surgery after arriving at the hospital. However, there are some patients who do not show any preoperative clinical neurological symptoms despite the obstructed carotid artery. In these cases, cerebral ischemia might be alleviated by collateral circulation via the circle of Willis, and ongoing brain ischemia within the ischemic penumbra (the reversible ischemic area) may be relieved by prompt surgery.

Perioperative cerebral infarction occurs by various mechanisms during ATAAD surgery. Malperfusion of the true lumen caused by the expanded false lumen, embolization of the thrombus formed in the false lumen moving into the true lumen through re-entry, inappropriate cerebral protection during the surgery, and air embolisms are the possible causes of perioperative stroke.

We believe that the main cause of the perioperative cerebral infarction in our patient was as follows. This patient did not manifest any neurological abnormalities preoperatively despite complete occlusion of the right carotid artery. Postoperative CT showed recanalization of the right carotid artery and no residual dissection. We observed a floating ball-like thrombus by carotid ultrasonography. As the brachiocephalic artery was occluded, the antegrade flow in the right carotid artery decreased, and cerebral perfusion was preserved by collateral flow from the left carotid artery via the circle of Willis. In this situation, the thrombus might have formed in the stagnant right carotid artery. Thromboembolism likely occurred due to antegrade reperfusion of the right carotid artery when cardiopulmonary bypass was started.

If the thrombus extended to the right internal carotid artery, it would be difficult to remove it directly. Blind thrombectomy using a balloon catheter runs the risk of vascular injury. However, if the thrombus was localized in the right common artery, direct carotid artery thrombectomy and early direct carotid reperfusion might have prevented thromboembolism. We should have performed intraoperative carotid ultrasonography before cardiopulmonary bypass and dealt with the thrombus appropriately, potentially through direct thrombectomy.

**Conclusion**

Oclusion of the carotid artery is a risk factor of postoperative severe stroke caused by thromboembolism, even in patients who have no neurological symptoms preoperatively.

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**ORCID iD**

Hiroko Nemoto [https://orcid.org/0000-0002-3016-4036](https://orcid.org/0000-0002-3016-4036)

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