Pulsatile thrombus evacuation from the false lumen of an occluded carotid artery in acute aortic dissection

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ABSTRACT

Herein, we present a case of aortic dissection with right carotid artery occlusion that was treated successfully with thrombus evacuation from the false lumen of the occluded carotid artery during hemiarch replacement. This procedure is performed with two maneuvers: aggressive retrieval of the thrombus from the innominate artery during circulatory arrest and thrombus evacuation from the false lumen of the right common carotid artery through a right neck incision with the heart beating. In this alternative method, thrombi can be evacuated more naturally and briskly, using pulsatile flow. (J Vasc Surg Cases Innov Tech 2023;9:101264.)

Keywords: Acute aortic dissection; Carotid artery occlusion; Thrombus evacuation; Pulsatile flow; Vascular disease

Type A acute aortic dissection (AAAD) is a lifethreatening disease, of which prompt diagnosis and treatment are essential. In particular, cerebral malperfusion poses a great challenge to surgeons. Even if central aortic repair can prevent critical aortic rupture, cerebral malperfusion can persist. Various procedures have been attempted to achieve better outcomes.¹⁻⁴ We present an alternative procedure that can be applied to selected patients.

CASE REPORT

A 66-year-old woman was transferred to our hospital with reported chest pain. On physical examination, right conjugate deviation of the eyes and left hemiplegia were noted. Enhanced computed tomography (CT) scan revealed an AAAD with right common carotid artery (RCCA) occlusion (Fig 1, A-C). The extent of dissection was limited to the ascending aorta and aortic arch, without any evidence of reentry. The patient was transported to the operating room quickly. Cardiopulmonary bypass was established with right femoral artery perfusion and vena cava drainage. A right-sided neck incision was made during cooling, and the RCCA was continuously monitored using ultrasonography. Regional oxygen saturation (rSO₂) was monitored on the forehead using near-infrared spectroscopy (INVOS 5100C; Medtronic, Minneapolis, MN). The ascending aorta was incised

https://doi.org/10.1016/j.jvscit.2023.101264

during circulatory arrest, when rectal temperature reached 25°C. Retrograde cerebral perfusion was initiated. An intimal tear was observed at the distal site of the ascending aorta. A thrombus in the false lumen of the innominate artery was rigorously removed. The ascending aorta was replaced with a prosthetic graft (J graft 28 mm; Japan Lifeline, Tokyo, Japan). Circulatory arrest time was 35 minutes. After the aortic crossclamp was released, the heart returned to sinus rhythm. Ultrasound examination confirmed that the true lumen of the RCCA was still narrowed by the thrombus in the false lumen. Furthermore, rSO₂ of the right forehead was 69.6 after weaning from cardiopulmonary bypass, which was much lower than that of the left forehead, at 80.9 (Fig 2).

We decided to evacuate the thrombus. An approximately 3cm longitudinal incision was made and restricted to the adventitia layer of the RCCA. Pieces of the thrombus were removed with a gentle maneuver, antegrade blood flow from the false lumen was confirmed, and additional thrombus pieces were removed using pulsatile bleeding (Fig 3, *A*). The adventitia was closed (Fig 3, *B*). Ultrasound examination revealed a widened true lumen of RCCA. Although rSO₂ of the right forehead was still lower than that of the left after thrombus evacuation, that difference decreased over time (Fig 2).

At the end of the operation, the rSO_2 of the right and left forehead was 65.4 and 71.4, respectively. The patient was awake 5 hours after the operation. The postoperative course was uneventful, and the left hemiplegia improved remarkably. Postoperative enhanced CT scans revealed an enlarged true lumen from the innominate artery to the RCCA. However, the false lumen of the distal RCCA exhibited enhanced activity (Fig 4, *A-C*). Subsequent CT scans taken 4 months after then operation revealed that the false lumen had dissipated, and the true lumen of RCCA was widely expanded (Fig 5, *A-E*). Informed consent was obtained from the patient for publication of this case report.

DISCUSSION

AAAD is a life-threatening disease that requires prompt diagnosis and treatment to avoid subsequent organ

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Author conflict of interest: none.

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The editors and reviewers of this article have no relevant financial relationships to disclose per the Journal policy that requires reviewers to decline review of any manuscript for which they may have a conflict of interest.

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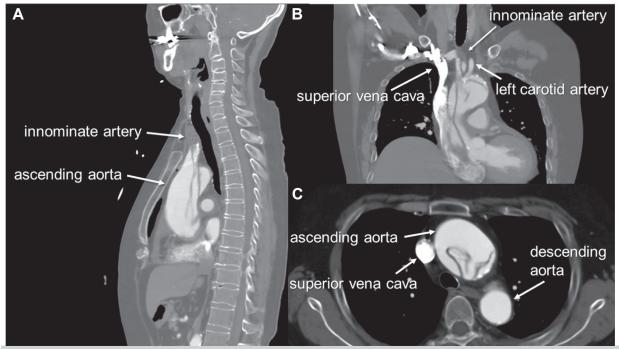


Fig 1. Preoperative enhanced computed tomography (CT) showing aortic dissection with thrombus at bifurcation of innominate artery. (A) Sagittal view, (B) frontal view, and (C) axial view.

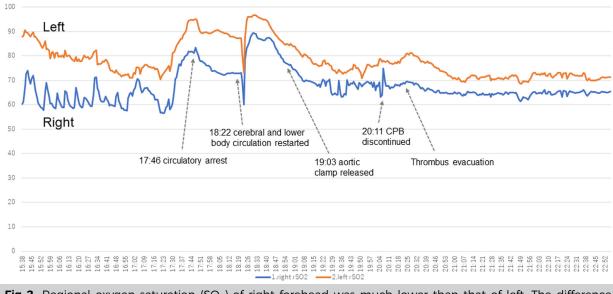
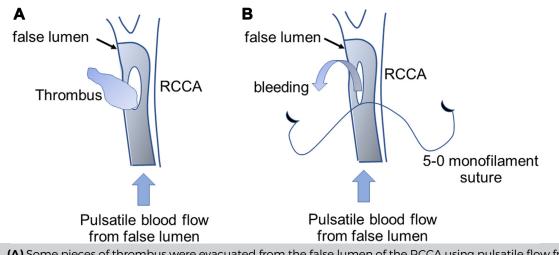
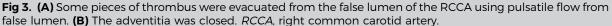


Fig 2. Regional oxygen saturation (SO₂) of right forehead was much lower than that of left. The difference between them decreased after thrombus evacuation. Regional SO₂ of left forehead (*left*); Regional SO₂ of right forehead (*right*). *CPB*, cardiopulmonary bypass.

malperfusion and aortic rupture. Cerebral malperfusion is a particularly critical condition that can cause permanent stroke, and even death. Surgeons might hope that central aortic repair will lead to increased blood flow of the true lumen and resolve cerebral malperfusion. However, this is not always the case. In a recent study, carotid artery stenting was performed in severely symptomatic patients with carotid artery occlusion with AAAD,¹ in whom central repair was not indicated without first improving carotid artery blood flow. Okita et al² proposed a preoperative femoral-to-carotid artery shunt to relieve ischemia using a small roller pump in the emergency room. Other surgeons demonstrated that extraanatomical bypass from the ascending prosthetic graft Journal of Vascular Surgery Cases, Innovations and Techniques Volume 9, Number 3 $\,$





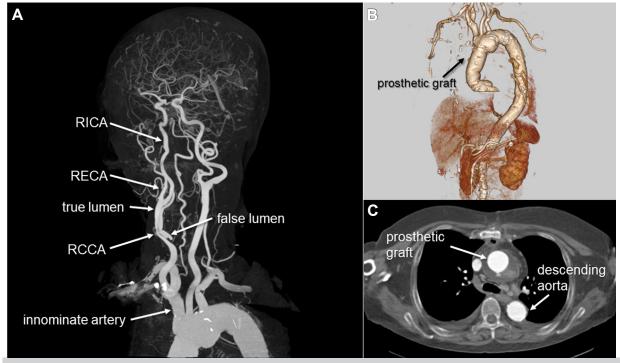
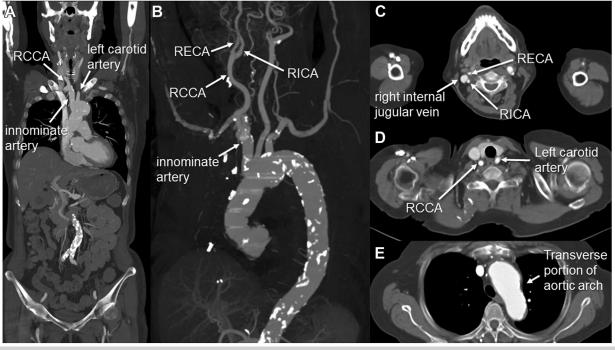
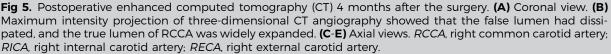


Fig 4. (A) Postoperative enhanced computed tomography (CT) showing widened true lumen of the innominate artery. The false lumen of the right common carotid artery (*RCCA*) exhibited enhanced activity. **(B)** Three-dimensional CT angiography. **(C)** Axial view showed no dissection at descending aorta. *RICA*, right internal carotid artery; *RECA*, right external carotid artery.

to the carotid artery under circulatory arrest achieved good results,^{3,4} but this procedure is time consuming. We hypothesized that, once the patient was cooled to a rectal temperature of 25°C, oxygen demand would decrease, and the brain could better tolerate ischemia, providing that retrograde cerebral perfusion would work as adjunctive therapy.

When opening the aorta, a moderate degree of thrombus was found in the false lumen of the innominate artery, which was evacuated vigorously. At that time, we anticipated that this factor could contribute to widening of the true lumen of the innominate artery and the RCCA. However, this did not occur. Therefore, we added an alternative thrombus evacuation from the





false lumen of RCCA. Our hypothesis proposes that the pulsatile flow observed in the false lumen of the RCCA is caused by the aggregation of blood flow from suture holes located at the distal ascending aorta-prosthetic graft anastomosis during systemic heparinization. We postulate that, although the flow from individual suture holes may be minimal, the collective effect of their outflows could result in a substantial volume, thereby generating sufficient pressure to dislodge the thrombus from RCCA. The cumulative flow from the suture holes is hypothesized to enter the false lumen, subsequently reaching the RCCA. Upon incision of the adventitia of the RCCA, the pressure buildup in the false lumen would generate a pulsatile flow, facilitating the dislodgment of the thrombus. Our hypothesis suggests that this proposed mechanism is likely to be effective during systemic heparinization. Furthermore, we anticipate that it would subside after the administration of protamine, thus preventing a recurrence of innominate artery dissection. However, these small distal entry tears may not always be detected on CT images and they seal in the late postoperative control after true lumen complete pressurization.

Surgeons must ensure that the intima of the carotid artery is not injured when incising the adventitia, which can lead to excessive bleeding that is difficult to repair. Another concern is oxygen saturation. Immediately after thrombus evacuation, the rSO_2 of the right forehead

remained lower than that of the left. That difference decreased over time; however, the interruption of antegrade RCCA blood flow at the onset of AAAD may have a short-term effect on the balance of oxygen demand and its consumption, even if thrombus evacuation was completed. Although we used retrograde cerebral perfusion during circulatory arrest, the difference in rSO₂ values between the left and the right forehead did not decrease throughout its term, which may endorse our hypothesis (Fig 2). We believe that chronological rSO₂ monitoring and direct evaluation of the carotid artery using ultrasonography could contribute to an accurate diagnosis that can lead to prompt treatment. This procedure may not be applicable to all patients with thrombosed carotid arteries in AAAD and should not be used if the intimal tear is located in the carotid artery on preoperative CT scan. In such cases, carotid artery stenting or extra-anatomical bypass to distal carotid arteries would be preferable options.

CONCLUSIONS

Pulsatile thrombus evacuation from the false lumen of the carotid artery is a reasonable option in selected patients. It can be included in a surgeon's armamentarium, alongside thrombus retrieval from the innominate artery during circulatory arrest.

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Submitted Feb 28, 2023; accepted Jun 7, 2023.