Reminder of important clinical lesson

Two different cases of postoperative symptomatic common carotid artery involvement in type A aortic dissection

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Postoperative common carotid artery occlusion after reconstruction for type A aortic dissection can lead to major neurological morbidity. Surgical strategy to re-establish the cerebral perfusion depends on the time of onset of neurological deficits in this otherwise life-threatening disease. We present two cases with neurological deficits after replacement of the ascending aorta for a type A dissection treated with two different surgical strategies. In both cases, prompt surgical interventions improved neurological outcome.

BACKGROUND

In contrast to preoperative massive cerebral infarction due to the obstruction of the common carotid artery by extended acute aortic dissection,^{1–3} postoperative occlusion can be monitored (eg, by new diagnosed neurological deficits or duplex sonography) and diagnosed early. Therefore a therapeutical approach with reconstructive surgery should be considered as first-line treatment of postoperative common carotid occlusion in contrast to the damage-control attitude in case of preoperative common carotid artery occlusion with unknown duration of neurological deficits. In the literature, different surgical techniques are described to maintain cerebral perfusion in case of aortic dissection. On the other hand, Sartipy *et al*⁴ reported an aorto-carotid bypass in a situation of cerebral malperfusion due to progressive dissection of the carotid arteries after aortic dissection surgery.

CASE PRESENTATION**Case 1**

A 58-year-old man with a sudden onset of chest pain was referred from a peripheral hospital with the CT diagnosis of a type A aortic dissection. Initially, a replacement of the ascending aorta and a double aortocoronary bypass was performed. Twelve hours postoperatively in the intensive care unit, the patient developed an anisocory with a non-reacting pupil on the right side. In the cranial CT scan, an occlusion of the nearly complete right common carotid artery was diagnosed (figure 1A: arrow), and consequently, a carotid–carotid bypass was performed (figure 1B).

Case 2

A 58-year-old man presented to the emergency ward with an acute onset of chest pain radiating into the left arm and left limb. At the time of admission, no neurological deficit was present. The initial CT scan showed a type A dissection of the aorta with a dissection of the right carotid artery (figure 2: arrow). Treatment consisted of reconstruction of the ascending aorta. The patient was

only extubated 3 days postoperatively and then a paresis of the left arm became apparent. A CT scan at that time revealed frontal and infratentorial ischaemic lesions on the right side. A conservative treatment with oral anticoagulation and aspirin was started. Eight days after the first operation, the paresis of the distal left arm worsened. Duplex sonography of the right carotid artery showed the well-known dissection with diminished flow (figure 3: arrows) and new an intracranial left to right cross-flow could be demonstrated. The cranial CT scan at that time did not reveal any new lesions.

Indication for surgery was based on increasing neurological deficits, failed conservative treatment and no new lesion in the CT scan. We performed a formal carotid endarterectomy with proximal resection and distal tack-down of the dissection membrane. The incision of the carotid artery was closed with a Dacron patch. In the postoperative Duplex Ultrasound, intracerebral cross-flow had disappeared, but the blood flow in the right internal carotid artery was still diminished owing to persistent dissection of the proximal common carotid artery.

OUTCOME AND FOLLOW-UP**Case 1**

Postoperatively, the anisocory and non-reacting pupil on the right side disappeared completely within hours. Three months after the operation, the patient was alive without neurological symptoms.

Case 2

Postoperatively no new deficits occurred and the paresis of the left arm improved during follow-up. At 3-month and 6-month follow-up, the patient did well with only very minor persistent weakness of the left arm.

DISCUSSION

Neurological deficits occur in about 5–15% of patients with type A dissection.³ In a case series, fast replacement of the aortic branch has been recommended for type A

Figure 1. (A) CT scan showing occlusion (arrow) of the right carotid artery due to type A aortic dissection. (B) Carotid-carotid bypass because of occlusion of the right carotid artery after type A aortic dissection.

dissection because the redirected blood flow into the true aortic lumen helps to restore cerebral perfusion. However, this work mainly focuses on the indication of aortic replacement in case of preoperatively existing neurological

Figure 2 Type A aortic dissection of the aorta with dissection of the right carotid artery (arrow).

deficits.³ In another work published by Gaul *et al*, 44 of 102 patients with type A aortic dissection had a supraaortic vessel involvement. There was no significant correlation between occurrence of postoperative neurological deficits and supraaortic vessel involvement. Interestingly, in 14.9%, postoperative neurological complications were due to nerve compression and not to ischaemia or bleeding. Finally, no correlation between occurrence of postoperative neurological complications and death could be observed by this group.⁵

In the two cases presented above, neurological symptoms occurred only after aortic replacement, and to our knowledge, there is no standard procedure for this clinical situation. We are fully aware that factors other than vascular involvement, such as hypothermic circulatory arrest, microembolism and perioperative hypotension, can lead to postoperative neurological deficits after aortic replacement in case of type A aortic dissection. However, this fact does not help a surgeon's decision-making process in this situation.

The evidence, based on few case reports, suggests a classification into preoperative and postoperative neurological deficits, meaning symptoms of unknown or known

Figure 3 Duplex sonography of the right carotid artery showing the dissection with diminished flow (arrows).

duration. Shimazaki⁶ presented a case with left hemiplegia and extended aortic dissection into both common carotid arteries. Surgical procedure consisted of replacement of the aorta, ligation of the right common carotid artery and reconstruction of the truncus brachiocephalicus, the subclavian arteries and the left common carotid artery. Clinical and radiological follow-up showed neither an increase of the infarction nor a cerebral haemorrhage. Postoperative consciousness improved to a Glasgow Coma Scale of 15, but a left hemiplegia persisted. Matsuyama⁷ reported a similar case without a lesion in the initial cranial CT scan, the right common carotid artery was cannulated to maintain cerebral perfusion. A postoperative CT scan showed a cerebral infarction of the right parietal lobe which correlated clinically with a left hemiparesis that was treated conservatively. Sartipy⁴ published a case with intermittent neurological symptoms after surgery for a type A aortic dissection. Progressive dissection of the carotid arteries was diagnosed and treated with an aorto-carotid bypass. Consequently, all neurological symptoms resolved. In contrast to preoperative cerebral infarction where surgery seems to have mainly a role for damage-control, postoperative common carotid artery occlusion with consequent neurological deficits can be treated therapeutically. If neurological deficits occur in a short-time interval or worsen despite conservative treatment reconstructive surgery (bypass, thrombendarterectomy) should be taken into consideration and could result in a good outcome. However, the short time-interval was defined in a different therapeutic setting in which studies demonstrated that thrombolytic therapy for ischaemic stroke should be performed within 3 h, otherwise, severe brain damage due to reperfusion would occur.^{8–10}

The classification into preoperative and postoperative onset of neurological deficits has to be taken with caution and put in a context with perioperative complications such as pneumonia and myocardial infarction that can be treated conservatively and significantly increase the morbidity and mortality risk in case of a reoperation.

It is controversial for how long patients should be ventilated after aortic dissection surgery and what neurological monitoring is necessary during the postoperative period on the intensive care unit. Our experience and the little evidence in the literature support early postoperative extubation with neurological monitoring in very short-time intervals, especially in the case of absence of preoperative neurological deficits. In this situation, postoperative sonographic control of blood flow in the carotid arteries, in short-time intervals, would further help to early identify cases that could benefit of an additional surgical intervention.

In conclusion, we present two cases with different kind of supra-aortic vessel involvement in case of type A aortic dissection. However, both patients suffer from neurological complications following aortic replacement, and in both cases, prompt surgical interventions improved neurological outcome. Decisions for or against an operation have to be made individually, based on neurological evolution, time of onset of symptoms and other perioperative complications such as pneumonia or myocardial infarction.

Learning points

- Classification into preoperative and postoperative onset of neurological deficit in case of aortic replacement for type A dissection helps to decide about future surgery.
- Early extubation after aortic replacement for type A dissection is crucial for a good neurological monitoring.
- Postoperative ultrasound of the carotid arteries is recommended after replacement for type A aortic dissection.

Competing interests None.

Patient consent Obtained.

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