Case Report & Case Series

Acute internal carotid artery occlusion after carotid endarterectomy

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ABSTRACT

We report two cases of acute carotid artery (CA) occlusion following carotid endarterectomy (CEA). Case 1: a 58-year-old man was admitted with transient right-sided hemiparesis. Magnetic resonance imaging (MRI) and MR angiography (MRA) revealed cerebral infarction in the left cerebral hemisphere and left CA stenosis. Ten days after admission, he underwent CEA. 24 h after surgery, he developed right hemiplegia. MRI and MRA demonstrated a slightly enlarged infarction and left internal carotid artery (ICA) occlusion. Emergency reoperation was performed and complete recanalization achieved. The patient made a clinically significant recovery. Case 2: a 65-year-old man underwent a right-sided CEA for an asymptomatic 80% CA stenosis. 48 h after surgery, his family noticed he was slightly disoriented. MRI and MRA revealed multiple infarctions and right ICA occlusion. He was treated with antiplatelet therapy without reoperation because sufficient cross-flow from the left ICA through the anterior communicating artery was demonstrated by angiography, and his neurological symptoms were mild. His symptoms gradually alleviated and he was discharged 14 days after surgery. With ICA occlusion after CEA, immediate re-operation is mandatory with severe neurological symptoms, whereas individualized judgement is needed when the symptoms are mild.

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1. Introduction

Carotid endarterectomy (CEA) is safe and effective in reducing the risk of stroke in symptomatic and asymptomatic patients with severe carotid artery stenosis [3,7]. However, perioperative neurological events are reported in 1.5–9% of CEA cases [11]. Particularly, postoperative occlusion of endarterectomized carotid arteries occurs in 0.5% of cases soon after surgery [4]. Management of these complications is an area of controversy. Here, we report two cases of acute internal carotid artery (ICA) occlusion following CEA. The first case was treated operatively and the second case non-operatively, with the relevant literature reviewed.

2. Case report

2.1. Case 1

A 58-year-old man consulted our hospital because he experienced transient right-sided hemiparesis. Magnetic resonance imaging (MRI) revealed cerebral infarction in the left cerebral hemisphere (Fig. 1A). Additionally, magnetic resonance angiography (MRA) and left carotid angiography demonstrated left carotid artery (CA) stenosis (Fig. 1B, C). The degree of CA stenosis was 80% by North American Symptomatic Carotid Endarterectomy Trial (NASCET) criteria [7]. He was admitted and underwent antiplatelet therapy. Recurrence of paralysis was not seen. Ten days after admission, CEA was performed under general anaesthesia by longitudinal arteriotomy to expose the intraluminal atheroma. A Pruitt-Inahara shunt (P-I shunt) was used to maintain cerebral perfusion during the arteriotomy. Complete resection of the plaque with a smooth distal edge was obtained. After endarterectomy, the arteriotomy was closed primarily with 5–0 propylene sutures and the shunt removed before completion of the sutures. His postoperative course was uneventful until 20 h after surgery when he developed right hemiplegia and disorientation. MRI and MRA were performed, with a slightly expanded infarction and left ICA occlusion detected (Fig. 1D, E, F). Emergency re-exploration was performed under general anaesthesia with rapid heparinization. A reddish-black thrombus was identified at the endarterectomy site. Removal of the thrombus resulted in brisk retrograde bleeding. A Fogarty catheter was inserted through the ICA. Clotting of the distal ICA was prevented by aspiration through the Fogarty catheter. Complete recanalization was confirmed by intraoperative angiography, and a P-I shunt inserted to maintain cerebral perfusion. A smooth intima edge was confirmed from the operative findings. The cause of thrombus formation was unknown, however the ICA shape at the endarterectomy site appeared slightly kinked. The arteriotomy was again closed primarily with 5–0 propylene sutures, with no kink detected at the endarterectomy site. The patient made a clinically significant recovery. Fourteen days after reoperation, the patient was transferred to a rehabilitation hospital with mild right hemiparesis. Before discharge from our hospital, he was able to walk with assistance. At his 1-year follow-up evaluation, the patient had a

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modified Rankin Scale (mRS) score of 3 and a patent left CA on MRA (Fig. 2A, B).

2.2. Case 2

A 65-year-old man was admitted with a moderate-size haemorrhage in the putamen and treated conservatively. He was discharged from hospital after 18 days with mild left hemiparesis. During admission, an asymptomatic CA stenosis was detected by MRA (Fig. 3A). The degree of CA stenosis was 75% by NASCET criteria. The patient was readmitted 6 months later and CEA performed under general anaesthesia by longitudinal arteriotomy. During the arteriotomy, a P-I shunt was used to maintain cerebral perfusion. The ICA size was comparatively small, and it was nearly impossible to insert a distal P-I shunt tube. The intraluminal atheroma was exposed and complete resection of the plaque with a smooth distal edge was obtained. After endarterectomy, the arteriotomy was closed primarily with 5–0 propylene sutures, and the shunt removed before completion of the sutures. His postoperative course was uneventful until 48 h after surgery when slight disorientation was noted. MRI and MRA were performed, with multiple infarctions in the right cerebral hemisphere and ICA occlusion detected (Fig. 3B, C). Emergency re-exploration was considered, but sufficient cross-flow from the left ICA through the anterior communicating artery was observed by angiography (Fig. 3D). Moreover, his neurological symptoms were mild, therefore he was treated with antiplatelet therapy. His left hemiparesis did not worsen and his symptoms gradually alleviated. He was discharged 14 days after surgery and lived without worsened symptoms. At her 2-year follow-up evaluation, she had a modified Rankin Scale (mRS) score of 2.

3. Discussion

Several reports have documented that acute thrombus at the endarterectomy site is a common cause of ICA occlusion after CEA [4,11,12]. Complete surgical excision of the carotid plaque during CEA is essential to prevent postoperative thrombus generation [8,10,12]. In Case 1, a smooth intima edge was confirmed during re-exploration, but with a slight arterial kink detected at the endarterectomy site. Correction of kinks in the ICA and common carotid artery (CCA) can help prevent post-CEA thrombosis [9]. Surgeons performing CEA should pay
attention to the shape of the ICA after an arterectomy. In Case 2, the cause of the internal CA occlusion was also unclear. However, the ICA size was comparatively small, and it was nearly impossible to insert the P-I shunt tube. In our institute, arterectomy is routinely closed primarily, but some surgeons recommend a patch closure to reduce the risk of immediate thrombosis, particularly when a small ICA is encountered during endarterectomy [2]. CEA surgeons should pay attention to the ICA size before and during surgery, and determine if patch closure use is appropriate.

When managing acute ICA occlusion after CEA, the surgeon is often preoccupied with determining if the patient should be surgically re-explored. Controversy exists as to which diagnostic test should be performed, and which criteria used for selection of patients to be treated by reoperation. Poor results have been reported if dissection of the distal ICA or intracranial emboli are present [1,4,5,8,10]. Therefore, some surgeons recommend that only patients with suspected artery thrombosis should be re-explored, even when the neurological symptoms are severe [1,4,10]. Nevertheless recently, intraoperative stent placement, accomplished using an endovascular technique or placement of a self-expanding stent under directed vision, was reported as an attractive method to treat dissection after CEA [6]. Moreover, percutaneous transluminal carotid angioplasty with stent deployment has emerged as an alternative strategy for treatment of ICA occlusion after CEA. This treatment is encouraging because a short interval between symptom onset and stent placement can be achieved, with thrombectomy of any intracranial thrombus also possible [1,4]. We believe that in patients with sudden onset of severe neurological deficits or with progressively worsening neurological deficits after CEA, immediate re-exploration with/without a stent or percutaneous transluminal carotid angioplasty with stent placement is mandatory [10,12]. Even if the immediate post-operative course is normal, CEA surgeons should be fully aware that ICA occlusion may occur within several days [8,10].

In contrast, it is particularly difficult to determine whether or not immediate reoperation should be performed when the neurological deficit is mild, as in Case 2 [8,5,10,12]. Rockman et al. found that in 38 cases with neurological events after CEA, patients with more severe events underwent re-exploration, whereas those with less severe deficits were more likely to be managed non-operatively [10]. In Case 2, we also treated the patient non-operatively because of concerns that surgery may cause embolization of a major intracranial artery. However, some surgeons recommend immediate re-exploration because a natural history of asymptomatic postoperative thrombosis after CEA is similar to ICA occlusion without surgical intervention [8]. Moreover, Koslow et al. re-explored three asymptomatic patients and reported no problematic events [5]. When the neurological deficit is mild, individualized judgement by comparing the risk and benefit of reoperation is needed.

Fig. 3. Radiographic findings for Case 2. A: preoperative cervical MRA demonstrated right cervical ICA stenosis. B: three days after CEA, diffusion-weighted MRI demonstrated multiple cerebral infarctions in the right cerebral hemisphere. C: 48 h after CEA, cervical MRA revealed right ICA occlusion. D: left carotid angiography found sufficient left-to-right cross flow via the anterior communicating artery.
References


