Surgical and Endovascular Treatments of Extracranial Carotid Artery Aneurysms—Report of Six Cases

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Background: Although the natural course of extracranial carotid artery aneurysms (ECAAs) is still unknown, they may cause stroke or cranial nerve dysfunction unless they are treated. In this report, we reviewed the clinical results of 6 patients who underwent endovascular and surgical treatments for ECAAs. *Methods:* A total of 6 patients underwent endovascular and surgical treatments for ECAAs for 9 years. The primary causes of ECAAs included Marfan syndrome (1 patient), infection (1 patient), trauma (2 patients), and unknown (2 patients). All 6 ECAAs were symptomatic. *Results:* One patient underwent surgical resection of the ECAA followed by end-to-end anastomosis of the internal carotid artery (ICA). Another patient underwent proximal ICA ligation combined with high-flow external carotid artery-to-middle cerebral artery bypass using a radial artery graft, because the patient also had a giant thrombosed aneurysm in the cavernous portion of the ipsilateral ICA. Endovascular treatment was selected in the other 4 patients using a covered stent or a bare metal stent combined with coil embolization. Of these patients, one required proximal ICA ligation followed by superficial temporal artery-to-middle cerebral artery anastomosis due to an anatomical problem for stent placement. There was no neurological deterioration at the discharge in all but 1 patient who suffered ischemic stroke during surgery. *Conclusion:* Surgical or endovascular treatment yielded a relatively satisfactory outcome in patients with ECAAs. **Key Words:** Extracranial carotid artery aneurysm—endovascular treatment—covered stent—bypass surgery.

Introduction

Extracranial carotid artery aneurysms (ECAAs) are relatively rare and their incidence ranges from .27% to .6%.[1-5] Even now, the natural course of ECAAs is unknown. Although some investigators have suggested that small asymptomatic ECAAs can be followed up conservatively, others have concluded that ECAAs may cause stroke in more than 50% of patients and may lead to death in 60%-70% when left untreated.[6] Furthermore, large ECAAs are also known to cause cranial nerve dysfunction due to local mass effect. Therefore, large or symptomatic ECAAs are generally considered suitable for surgical treatment, although such opportunity is rare, accounting for .1%-2.0% of all carotid procedures.[3,7-11] In addition, no evidence-based guidelines have been established mainly because of a small number of experiences in each institute. In this report, therefore, the authors review the clinical results

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Table 1. *Summary of clinical features of 6 patients with ECAA on admission*

Case no.	Age (y)/sex	Past history	Presenting symptoms	Etiology	Side	Maximum diameter (mm)	Level of distal end
1	64/F	None	Pulsatile neck mass	Marfan syndrome	R	30	C3
2	78/F	Subarachnoid hemorrhage	Pulsatile neck mass	Unknown	R	30	C2
3	36/M	None	Aphasia, right arm weakness	Trauma	L	18	Just proximal to the carotid canal
4	54/M	CRF, DM, ASO	Hoarseness	Infection	R	36	C4
5	23/M	TAPVC, Fontan operation	Pulsatile neck mass	Unknown	L	49	C2
6	20/M	None	IXth, Xth, XIIth cranial nerve palsy	Trauma	R	42	C1

ASO, arteriosclerosis obliterans; CRF, chronic renal failure; DM, diabetes mellitus; ECAA, extracranial carotid artery aneurysm; F, female; L, left; M, male; R, right; TAPVC, total anomalous pulmonary venous connection.

of 6 patients who were treated for ECAAs with endovascular and surgical treatments.

Methods

The present study included a total of 6 patients who underwent surgical and endovascular treatments for ECAAs in our hospital between January 2007 and September 2016. The clinical features of the patients are summarized in Table 1. There were 4 men and 2 women. The mean age was 45.8 years, ranging from 20 to 78 years. The primary causes of ECAAs included Marfan syndrome (case 1), blunt neck injury due to traffic accident (cases 3 and 6), and infection (case 4), and were unknown in 2 patients (cases 2 and 5). Past history included subarachnoid hemorrhage (case 1); chronic renal failure, diabetes mellitus, and arteriosclerosis obliterans (case 4); and total anomalous pulmonary venous connection (case 5). ECAA-related cranial nerve palsy was observed in 2 patients. Case 4 presented with hoarseness, but case 6 suffered severe cranial nerve palsy including the ipsilateral glossopharyngeal, vagal, and hypoglossal nerves. One patient developed right hemiparesis and motor dysphasia because of thromboembolism, which originated from the ECAA (case 3). Three patients presented with pulsatile mass in the neck (cases 1, 2, and 5). Case 1 was previously reported as a case report in a Japanese-written journal.[12]

On cerebral angiography, all aneurysms originated from the extracranial internal carotid artery (ICA). One patient also had a giant thrombosed aneurysm in the cavernous portion of the ipsilateral ICA (case 2). The mean diameter of the ECAAs was 34.2 mm, ranging from 18 to 49 mm. The distal ends of the ECAAs were located just proximal to the carotid canal in 1 patient, C1 level in another patient, C2 level in 2 patients, C3 level in 1 patient, and C4 level in 1 patient.

Results

Surgical and Endovascular Treatments

All 6 patients underwent surgical or endovascular treatment for the ECAAs. One patient underwent surgical resection of the ECAA (case 1). Under the use of external shunting, the ECAA was resected and the ICA was reconstructed with an end-to-end anastomosis technique (Fig 1). Postoperative course was uneventful. Another patient (case 2) underwent proximal ICA ligation combined with high-flow external carotid artery-to-middle cerebral artery bypass using a radial artery graft, because the patient also had a giant thrombosed aneurysm in the cavernous portion of the ipsilateral ICA. The patient developed ischemic stroke after surgery because of transient graft occlusion during surgery (Fig 2).

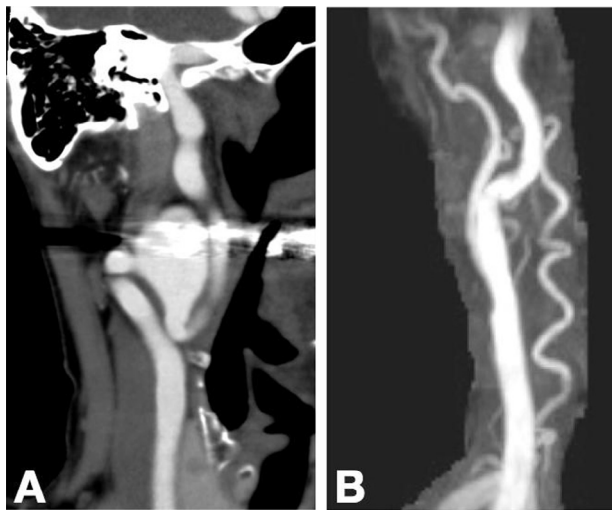

Figure 1. *Three-dimensional computed tomography angiography of the right extracranial carotid artery before (A) and after (B) surgery in case 1.*