A case of cavernous carotid aneurysm diagnosed when diplopia developed after endoscopic sinus surgery* 

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Abstract

Background: Visual complications of endoscopic sinus surgery usually occur during or immediately after the surgery. We report a case of cavernous carotid aneurysm which developed and gradually worsened after endoscopic sinus surgery was performed.

Case presentation: A 63-year-old woman with chronic rhinosinusitis resistant to conservative treatment underwent endoscopic sinus surgery. Despite the surgery being successful and without complications, diplopia developed 2 weeks later. Intracranial imaging revealed a giant cavernous carotid aneurysm as a likely cause of the diplopia. The patient underwent endovascular stenting treatment, and the diplopia was consequently reduced.

Conclusions: We experienced a rare case of cavernous carotid aneurysm which started to develop 2 weeks after endoscopic sinus surgery. Possible causes of the aneurysm in this patient are an indirect effect of surgery, such as perioperative hypertension, and bacterial sinusitis.

Key words: craniocerebral trauma, nasal surgical procedures, sinusitis, sphenoid sinus

Introduction

Nasal and paranasal diseases can have orbital complications and cause visual impairment owing to their adjacent locations. Surgical treatment for paranasal sinuses can also have visual complications, which rarely occur long after surgery. We report a case of cavernous carotid aneurysm (CCA) diagnosed when diplopia developed and gradually worsened after endoscopic sinus surgery (ESS) was performed.

Case presentation

A 63-year-old woman with no known preexisting illnesses underwent ESS for chronic rhinosinusitis resistant to conservative treatment. Preoperative computed tomography (CT) showed bilateral nasal polyps and opacification in the sinuses (Figure 1). With a biopsy of the nasal polyps, eosinophilic chronic rhinosinusitis was diagnosed. This condition was treated by the department of otorhinolaryngology by means of ESS, septoplasty, and submucosal inferior conchotomy with polypectomy and total removal of sinus laminae. The surgery lasted 2 hours 9 minutes under general anesthesia with desflurane for 2 hours 57 minutes. Vital signs during the surgery were stable; blood pressure was maintained at 80 to 100 over 50 to 70 mm Hg with a total blood loss of 60 ml. No intraoperative trauma occurred, and postoperative endoscopic examinations showed sinus membranes without swelling or infections. Postoperative medications include oral macrolide, carbocisteine, antihistamine, prednisolone 2.5mg and topical steroid.

Two weeks after ESS the patient noticed slight diplopia with far vision. Diplopia due to esotropia was diagnosed 5 weeks after ESS at a nearby private ophthalmologic clinic and was treated with prism correction. The ophthalmologic clinic reported esotropia of 2° (prism dioptres) during the first visit, 10° 7 weeks later (12 weeks after ESS), and 18° 14 weeks later (19 weeks after ESS). The patient had reported the diplopia to our department 7 weeks after ESS; however, we assumed the diplopia was not rela-
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ted to ESS because it had started 2 weeks after ESS and because endoscopic findings of the sinus membranes remained intact postoperatively. She did not report any changes of the diplopia during the following 2 visits to our department (9 and 13 weeks after ESS) but complained of worsening in a later visit (20 weeks after ESS). Therefore, we referred the patient to our hospital's department of neurology to investigate possible intracranial causes.

Magnetic resonance imaging/angiography performed 22 weeks after ESS at our hospital's department of neurology showed multiple intracranial aneurysms. A detailed CT angiographic examination (Figure 2 A,B,C) showed right CCA, which was the largest aneurysm, with a maximum diameter of 17 mm, and was believed to be causing diplopia. A Hess screen test performed 26 weeks after ESS at our hospital's department of ophthalmology suggested esotropia of the right eye (Figure 2 D). Ten weeks the diagnosis of aneurysm (32 weeks after ESS) the patient was admitted to another hospital, where she underwent endovascular aneurysm stenting treatment. After this treatment the diplopia decreased owing to improved eye movement (Figure 2 D). The time course of esotropia and its related events is also shown (Figure 2 E). The patient did not complain of any nasal symptoms in subsequent visits to our department, and no signs or endoscopic findings suggested the recurrence of sinusitis.

Discussion

Visual complications of ESS often arise during or immediately after surgery and are usually due to intraoperative trauma. A meta-analysis in 1994 found that the rate of visual complications after endoscopic surgery was 0.12%[1] and was lower than after traditional surgery (0.47%). Studies in the 2010s have found rates of orbital injury after ESS of 0.07%[2] and 0.09%[3]. Most of these complications were orbital hematomas with only a few cases of extraocular muscle injury (2 of 57 cases of orbital injury) requiring reconstructive surgery[3]. In our patient, intraoperative trauma did not occur, and surgery-related complications typically would have appeared within 2 weeks. Of cerebral aneurysms, fewer than 1% are traumatic intracranial aneurysms[4]. Intracranial carotid aneurysm due to artery injury has been reported after endoscopic endonasal skull base surgery but rarely after ESS. However, at least 4 cases of intracranial carotid aneurysm after ESS have been reported: 1 in the ethmoidal sinus after skull base injury[5], 1 in the sphenoid sinus due to lateral wall injury[6], and 2 with subarachnoid hemorrhage after functional ESS and cosmetic rhinoplasty[7]. Iatrogenic traumatic aneurysms are most often caused by direct arterial injury, which results in the collection of blood leaking from the artery and the formation of pseudoaneurysms. In our patient, preoperative CT examinations of the sphenoid sinus showed partial opacification (Figure 1), and the anterior sphenoid wall was carefully removed with bone punches to avoid cracks of the skull base or the posterior sphenoid wall, which can lead to carotid artery injury. Intraoperative endoscopic examination of the sphenoid sinus showed a nearly intact membrane with little inflammation or swelling; thus, the sphenoid sinus was not surgically manipulated (Figure 3). Because ESS had no complications and produced no marked changes in sphenoid sinus construction, we assume that ESS was not related, at least directly, to the diplopia.

Although the CCA in our patient was not been noticed before ESS, when the level and width of the preoperative CT images had been changed to a soft-tissue window, the aneurysm's contour was detected (Figure 4) and indicated that the aneu-
Figure 2. Image findings of cavernous carotid aneurysms and the Hess chart. (A) Magnetic resonance angiography of aneurysms (bilateral cavernous carotid aneurysms [CCAs] and a right vertebral aneurysm; right CCA: 17 x 13 mm; left CCA: 10 x 8 mm) (B) Computed tomography (CT) with contrast-enhancement of CCA (axial, coronal, and sagittal plane of CCA. Maximum diameter = 17 mm.) (C) Digital subtraction angiography with a CT angiogram of the right internal carotid artery clearly shows an aneurysm of the cavernous sinus and its origin. (D) Hess charts obtained before (left) and after (right) stenting treatment. Diplopia decreased from 12° to 4°.
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A case of carotid aneurysm developed before ESS. However, we did not detect the aneurysm before ESS; if we had, we would have consulted a neurosurgeon. Furthermore, CCA is usually diagnosed with a CT angiographic examination, with some studies using magnetic resonance imaging without contrast enhancement (8) but no studies having supported the use of CT without contrast enhancement (8) but no studies having supported the use of CT without contrast enhancement. The contour of the aneurysm in our patient was detected after ESS, but noticing the aneurysm before ESS was difficult owing to the aneurysm’s low contrast. The natural history of CCA is usually benign (9). The rate of rupture is lower than that of other intracranial aneurysms. Should a rupture occur, it generally forms a carotid artery-cavernous sinus fistula, rarely developing to subarachnoid hemorrhage but occasionally causing secondary epistaxis (10,11). The survival rate of CCA is high, and the main symptom is cranial nerve paralysis via a mass effect. Therefore, CCA is often followed up with imaging studies and treated when cranial nerve symptoms develop. A recent study has found that patients with CCA are more likely to be female and to have a lower incidence of hypertension than do patients with intracranial berry aneurysms and that the risk of growth is associated with aneurysm size (12). According to this study, the aneurysm in our patient, which had a diameter of 17 mm, would be classified as large/giant with a growth risk of 19.2% per patient-year. Therefore, the aneurysm in our patient had a high risk of enlarging and causing cranial nerve symptoms.

Whether the ESS performed in our patient affected aneurysm growth and led to diplopia remains unclear. Hypertension can be a risk factor for aneurysms, but during the surgery the patient’s blood pressure was well maintained and even had to be elevated several times with ephedrine. However, the patient’s mean blood pressure during the 6-day hospital stay of 139/84 mm Hg might suggest previously unnoticed hypertension. Another possible cause of aneurysms is bacterial infection. For example, an internal carotid aneurysm developing after sinusitis and resulting in multiple cranial nerve palsy has been reported.
Many cases of sinusitis-related aneurysm have been reported, but the condition most often related is sphenoid sinusitis. Because the CT and endoscopic examinations in our patient showed no signs of severe sphenoid sinusitis, the likelihood of sphenoid sinusitis-related aneurysm is decreased. Ampicillin was administered as a prophylactic antibiotic, and there were no signs of surgical site infection. Nevertheless, sinusitis was present in the sinuses, and because the surgery itself increased the risk of bacterial infection, which can cause an aneurysm, surgery might have increased the growth of the aneurysm.

Conclusions
We have reported a case of intracranial aneurysm diagnosed when diplopia developed 2 weeks after ESS had been performed. Possible factors in the growth of the aneurysm were an indirect effect of surgery, perioperative or previously unnoticed hypertension, and bacterial sinusitis. Intracranial aneurysms are asymptomatic unless they cause a mass effect or rupture and are difficult to detect with non–contrast-enhanced CT. A thorough examination, including intracranial imaging, should be performed if diplopia develops after ESS.

List of abbreviations
CCA: cavernous carotid aneurysm; ESS: endoscopic sinus surgery; CT: computed tomography; Δ: prism dioptres

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Conflict of interest
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