CASE REPORT

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CAVERNOUS CAROTID ANEURYSM PRESENTING WITH EPISTAXIS

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Abstract: Background. Carotid artery aneurysms are a rare cause of epistaxis. The most common presentation for non-traumatic cavernous internal carotid artery aneurysms is mass effect, with only 3% presenting with hemorrhage. We present a case of epistaxis caused by a nontraumatic cavernous internal carotid artery aneurysm.

Methods. A 73-year-old white woman was seen with a 1-month history of recurrent right-sided epistaxis. The patient had essential hypertension and a family history of intracranial aneurysm. A complete otorhinolaryngologic, neurologic, and ophthalmologic examinations were normal. Contrast-enhanced CT of the paranasal sinuses revealed a trilobed aneurysm of the cavernous segment of the right internal carotid artery. Coiling embolization of the cavernous aneurysm and right internal artery was performed.

Results. The patient has had no further episodes of epistaxis and has remained neurologically intact.


Keywords: cavernous; carotid; aneurysm; sphenoid, epistaxis

Epistaxis is a common clinical problem encountered by otolaryngologists. Most cases are due to bleeding from the anterior nasal septum and are easily managed with local measures. Mucosal dryness, digital trauma, anticoagulation, and hypertension are common predisposing factors. Posterior epistaxis is more severe, with a distinct source of bleeding often difficult to localize. Patients with epistaxis who fail initial conservative therapy require endoscopic cautery, surgical ligation, and/or transarterial embolization of the nasal cavity vascular supply. Embolization is primarily targeted at the ethmoidal branches arising from the internal maxillary artery. In addition, other branches arising from the external carotid artery need to be evaluated due to collateral supply.

Trauma and vascular abnormalities combined account for less than 5% of all cases of severe epistaxis.1 Carotid artery aneurysms are a rare cause of epistaxis and have an associated mortality rate of 30%.2 This report illustrates a case of epistaxis caused by a nontraumatic cavernous
internal carotid artery (cICA) aneurysm. The presentation, diagnosis, and treatment of carotid artery aneurysms are discussed in this report.

CASE REPORT
A 73-year-old white woman, previously healthy, was initially seen with recurrent right-sided epistaxis over a 1-month period. The most recent event had occurred 1 week before emergency admission and resulted in a significant drop of the hemoglobin count to 4 g/dL. All episodes of bleeding had been controlled with bilateral anterior nasal packing. The patient denied any visual complaints or headaches.

The patient’s family history was significant for an intracranial aneurysm in her mother, and the patient had a history of essential hypertension, which was controlled with an angiotensin-converting enzyme inhibitor. She reported no trauma or history of bleeding disorder. She smokes cigarettes but does not drink alcohol.

A complete otolaryngologic examination did not reveal any abnormalities. Neurologic and ophthalmologic examinations were normal. Admission hematocrit was 27%, and coagulation internal carotid artery (cICA) aneurysm. The presentation, diagnosis, and treatment of carotid artery aneurysms are discussed in this report.

FIGURE 1. CT scans of the paranasal sinuses, axial (A) and coronal (B), with intravenous contrast reveal a trilobed aneurysm of the cavernous segment of the right internal carotid artery with bony erosion through the lateral wall of the sphenoid sinus.

FIGURE 2. Angiograms, coronal (A) and sagittal (B) views, reveal a large, complex, and fusiform aneurysm in the horizontal segment of the right cavernous internal carotid artery, with a pseudoaneurysm extending into the right sphenoid sinus.
studies were normal. CT of the paranasal sinuses with intravenous contrast revealed a trilobed aneurysm of the cavernous segment of the right ICA with bony erosion through the lateral wall of the sphenoid sinus (Figure 1).

An emergent angiogram revealed a large, complex, and fusiform aneurysm in the horizontal segment of the cavernous ICA with a pseudoaneurysm extending into the right sphenoid sinus (Figure 2). A second, 8-mm saccular aneurysm of the right supraclinoid portion of the ICA aneurysm was also identified. Coil embolization of the cavernous aneurysm and the right ICA across the base of the aneurysm were performed after successful balloon occlusion testing with hypotensive challenge (Figure 3).

The patient remained neurologically intact with no significant complaints after the procedure. Intravenous phenylephrine was administered, with a systolic blood pressure parameter of 150 to 160 mmHg, to enhance collateral cerebral circulation for 36 hours. After her blood pressure was gradually returned to baseline levels, with weaning of the phenylephrine drip, she was transferred from the intensive care unit. After being observed on the ward for 2 days, she was discharged home. She has had no further episodes of epistaxis and remains neurologically intact. After the carotid occlusion, the patient received a daily dose of 325 mg of aspirin for 6 months.

A routine follow-up angiogram at 7 months revealed no evidence of ICA recanalization or aneurysm filling.

DISCUSSION

Aneurysms are primarily due to degeneration of the internal elastic lamina and collagen fibers of the arterial wall combined with the fluid mechanics of blood flow. Tobacco, hypertension, and patient age are the greatest risk factors for this vascular disease. Additional risk factors for aneurysms include a family history of intracranial aneurysms, adult polycystic kidney disease, fibrous dysplasia, and coarctation of the aorta. Patients of Finnish or Japanese descent have a higher incidence of aneurysms. Nontraumatic aneurysms are more common in elderly women.

Cavernous carotid aneurysms represent less than 2% of all intracranial aneurysms and are subdivided into traumatic and nontraumatic. History and rapid progression of symptoms result in more rapid diagnosis of traumatic aneurysms compared with true aneurysms. Traumatic aneurysms are typically pseudoaneurysms consisting of a hematoma surrounded by a fibrous layer, rather than a true arterial wall as is seen in nontraumatic aneurysms. Presentation with epistaxis is often delayed, ranging from 5 days to 9 weeks, with an average of 7 weeks. A carotid-cavernous arteriovenous (AV) fistula is a more common presentation of traumatic injury to the cICA.

In a report by Inagawa, during a 10-year period, 22 patients harboring 24 petrous and cavernous ICA aneurysms were observed. All these lesions were unruptured aneurysms. Eighteen of the 22 patients were women, with a mean age of 63 years. Fifty-five percent of these patients harbored multiple unruptured aneurysms. A subgroup of 16 aneurysms with a mean aneurysm diameter of 5 mm were followed for a mean of 5 years. None of these aneurysms ruptured, and 94% remained asymptomatic.

Aneurysms of the cavernous carotid artery typically are seen with the signs and symptoms of a space-occupying lesion. These cavernous sinus syndromes were initially described by Jefferson in 1938. In a study of 87 patients with intracavernous carotid aneurysms, the most common presenting symptom was mass effect (79%), followed by rupture of the aneurysm causing a carotid-cavernous sinus fistula (9%), trauma resulting in a cavernous pseudoaneurysm (8%),
and hemorrhage (3%). Diplopia, secondary to compression of cranial nerves within the cavernous sinus, is the most common sign of mass effect. Epistaxis is an unusual presenting sign and is more commonly associated with major trauma. Nontraumatic cavernous carotid aneurysms presenting with epistaxis are exceedingly rare. Recurrent epistaxis from carotid aneurysms may be due to a small rupture that tamponades from a hematoma in the closed space of the sphenoid sinus.

Cavernous sinus aneurysms typically do not present with a subarachnoid hemorrhage, which classically results in severe headache of sudden onset, meningeval irritation, nausea, and vomiting, because the cavernous portion of the carotid artery is extradural. Rupture of a cavernous carotid aneurysm more typically results in an AV fistula between the ICA and the cavernous sinus. Carotid–cavernous fistulas typically present with severe proptosis, chemosis, injection of the eye, pulsatile tinnitus, orbital bruit, and/or cranial nerve deficit.

The carotid artery has a close relationship with the sphenoid sinus, making it vulnerable to injury. Cadaveric dissection reveals that 71% of cavernous carotid arteries project into the lateral sphenoid sinus, 66% have a bony covering of less than 1 mm, and 4% are dehiscent.

Hahn et al reviewed unruptured giant cavernous carotid aneurysms, which are defined as greater than or equal to 2.5 cm diameter. Approximately 80% of their patients were women, with a mean age of 54 years. Common presenting symptoms were diplopia and retro-orbital pain. Headache, diminished or blurred vision, and photophobia were less common presenting symptoms. More than 90% of their patients had partial or complete ophthalmoplegia. Trigeminal nerve involvement was present in approximately one third of the cases. Other clinical signs included ptosis, decreased visual acuity, proptosis, and visual field defects.

The overall risk of rupture for all aneurysms is approximately 2% per year. Unruptured aneurysms less than 1 cm in diameter have a yearly rate of rupture of 0.05%, with an even smaller risk of rupture for cICA aneurysms. The risk of rupture is higher in women and for aneurysms that are symptomatic, larger than 1 cm, or located in the posterior circulation. Size and location are independent predictors of rupture. A multilobular aneurysm is probably at greater risk of rupture than a unilobular one. The average size of all aneurysms, both ruptured and unruptured, does not increase with age. The growth rate of aneurysms is not predictable. It does not seem that they progress steadily, and some aneurysms may reach a plateau and not progress further. Intracavernous aneurysms may increase in size with time but may also reduce in size. Reduction in size is more likely secondary to partial thrombosis, with true size reduction being rare.

Nasal packing, including posterior packing, is insufficient therapy for acute epistaxis secondary to a ruptured aneurysm. Posterior packing with external digital compression of the ipsilateral carotid artery will decrease blood loss, allowing additional time before definitive therapy.

Initial diagnostic modalities include nasal endoscopy and radiographic imaging. Operative nasal endoscopy provides direct access to most sources of epistaxis, which are subsequently amenable to cauterization. Mass lesions are best defined with a contrast-enhanced CT or MRI of the paranasal sinuses. However, the gold standard for diagnosis of vascular lesions remains digital subtraction angiography.

Open surgical techniques, with clipping of the aneurysm neck or proximal ligation of the ICA, were the standard treatment before the advent of endovascular therapy. Direct exposure of the cavernous carotid artery and aneurysm is a difficult and high-risk procedure. With the development of endovascular techniques, detachable balloons and platinum coils have been used to treat ruptured and unruptured aneurysms. Endovascular therapy is advantageous over traditional surgical therapy because of the difficult exposure presented by limited accessibility to the cavernous sinus. Endovascular options for treatment include primary embolization of the aneurysm or complete occlusion of the ICA.

First, to determine the feasibility of endovascular therapy, a complete four-vessel cerebral angiogram is performed. Special attention is placed on the anatomy of the aneurysm and patency of the circle of Willis. Analysis of the aneurysm is performed to determine whether the aneurysm can be occluded with platinum coils while preserving the carotid artery. The primary determinant of coil embolization will be the neck and shape of the aneurysm. Saccular aneurysms with a small neck (dome-to-neck ratio of 3:1) can generally be directly embolized. If direct embolization of the aneurysm cannot be performed, optimal treatment involves occlu-
sion of the ICA distal and proximal to the aneurysm. Before the ICA is occluded, balloon test occlusion of the ICA to assess functional and anatomic collateral cerebral circulation needs to be performed. In addition, to decrease the risk of late failure after carotid occlusion, provocative hypotensive testing during temporary balloon occlusion should be performed. Patients without anatomic collateral circulation or with a failed balloon test occlusion may require extracranial–intracranial bypass before endovascular therapy.

Endovascular occlusion of the ICA can be performed with detachable platinum coils, detachable balloons, or both. The segment of ICA distal to the aneurysm should be occluded to prevent retrograde filling of the aneurysm from collateral circulation. Coil embolization of the aneurysm sac itself occludes the aneurysm, while leaving the lumen of the ICA intact. A meta-analysis suggests that both balloon occlusion and coil embolization are reasonably safe and result in occlusion of the aneurysm in most patients.25 However, long-term outcomes have not yet been reported.26,27

**CONCLUSION**

Carotid artery aneurysms must be considered in the differential diagnosis of profuse epistaxis. The patient in this case report was initially seen with epistaxis as the first symptom of her aneurysm, a rare finding. The most common presentation for nontraumatic aneurysms is mass effect, with only 3% presenting with hemorrhage. Epistaxis arising from the cavernous carotid artery is commonly due to trauma and often with a delayed presentation. Risk factors of rupture for non-traumatic aneurysms include female sex and aneurysms that are symptomatic, larger than 1 cm, or multilobed. Digital subtraction angiography confirms the diagnosis of an intracranial aneurysm. Endovascular embolization of the aneurysm with detachable platinum coils or ICA occlusion after temporary balloon occlusion testing are safe and effective techniques for both ruptured and unruptured aneurysms.

**REFERENCES**


