Massive Epistaxis From a Thrombosed Intracavernous Internal Carotid Artery Aneurysm 2 Years After the Initial Diagnosis
—Case Report—

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Abstract

A 77-year-old woman presented with a rare case of nontraumatic intracavernous internal carotid artery (ICA) aneurysm causing epistaxis. The thrombosed aneurysm was discovered incidentally, and was not treated. However, she suffered massive nasal bleeding 22 months after the initial diagnosis. The lesion was successfully treated by endovascular coil embolization. The present case shows that thrombosed intracavernous ICA aneurysm may still carry the risk of rupture. Radiological evidence of erosion of the sphenoid sinus wall and repeated minor bleeding may be important predicting signs for massive nasal bleeding. Parent artery occlusion including the aneurysm may be the best treatment for intracavernous ICA aneurysms if sufficient collateral blood flow to the territory of the affected ICA is expected.

Key words: thrombosed aneurysm, intracavernous internal carotid artery, epistaxis, embolization

Introduction

Intracavernous internal carotid artery (ICA) aneurysms constitute 1–2% of all intracranial aneurysms and can be divided into traumatic or nontraumatic types.1) The natural history of intracavernous ICA aneurysms can be quite variable, as intracavernous ICA aneurysms usually increase in size, but can spontaneously reduce.10) The overall incidence of bleeding from intracavernous ICA aneurysms is reported to be low.8) Intracavernous ICA aneurysms rarely cause subarachnoid hemorrhage or major neurological morbidity.8) However, a giant intracavernous ICA aneurysm caused epistaxis 11 years after diagnosis.13) Traumatic intracavernous ICA aneurysms causing fatal epistaxis are sometimes encountered.3) However, massive epistaxis from a nontraumatic ICA aneurysm is rare.2,4,6,7,10,12,13,15,18–20,22) Patients with nontraumatic intracavernous ICA aneurysm usually present with symptoms of a space-occupying lesion. We report a patient with a nontraumatic intracavernous ICA aneurysm causing epistaxis during the follow-up period.

Case Report

A 77-year-old woman consulted our hospital complaining of a minor head injury in May 2004. Computed tomography (CT) demonstrated a mass in the left cavernous sinus extending to the sphenoid sinus with erosion of the anterior wall of the sphenoid sinus (Fig. 1A). She had no history of severe head injury. Magnetic resonance (MR) imaging demonstrated a thrombosed aneurysm of the left ICA (Fig. 1B). Angiography showed the thrombosed aneurysm had a small irregular aneurysm cavity (Fig. 1C). Balloon test occlusion evoked no neurological deficit during 20 minutes of ICA occlusion. Collateral blood flow from the right to left side was observed via the anterior communicating artery, so we speculated that the chance of rupture might not be so high because the aneurysm was largely thrombosed. Considering the patient's age, further intervention was not performed and the lesion was scheduled for radiological observation.

CT obtained 11 months later showed the same findings of aneurysm size and erosion of the sphenoid sinus wall (Fig. 2A). One year later, MR imaging demonstrated almost no change in the size of the thrombosed portion of the aneurysm and aneurysm cavity (Fig. 2B). About 14 months after the initial examination, the patient suffered repeated minor nasal bleeding and was treated by a local otolaryngologist for about 4 months. MR imaging in December 2005 showed that the aneurysm size was unchanged (Fig. 2B). The patient had extensive nasal bleeding compared to the episodes in the previous 4 months in January 2006. Nasal bleeding was controlled by compression of the nasal cavity.

The patient experienced massive pulsatile nasal bleeding from both the nasal cavity and mouth and was brought to our hospital by ambulance on March 1, 2006. On admission, blood pressure was 56/39 mmHg and the patient was
in shock. Laboratory examinations demonstrated severe anemia as follows: red blood cell count, $2.33 \times 10^6$ mm$^3$; hemoglobin value, 7.0 g/dl; hematocrit, 20.3%; platelet count, $7.7 \times 10^4$ mm$^3$; and total protein, 2.9 g/dl. The patient required transfusion of 16 units of red blood cells, 20 units of platelets, and 10 units of fresh frozen plasma. Initially, anterior and posterior nasal packing was attempted, but the pulsatile bleeding was not controlled. Therefore, endovascular embolization of the ICA, including the aneurysm, was planned.

The right femoral artery was punctured and a sheath introducer was placed. A 6F-guiding catheter was advanced to the left ICA with an inner 4F catheter. Preoperative angiography showed an aneurysm with a wide neck at the left ICA (Fig. 3A, B). The aneurysm cavity was larger than that observed in 2004. A small protrusion to the sphenoid sinus was faintly opacified. A microcatheter was advanced to the left ICA distal to the aneurysm with the aid of a microguidewire. A Guglielmi detachable coil (GDC) (18 2D, 6 mm in diameter, 20 cm in length) was firstly placed in the ICA distal to the aneurysm. After the first coil, more GDCs (total coil length, 22 cm) were subsequently detached in the frame of the first GDC. At this point, angiography did not show occlusion of either the ICA or the aneurysm (Fig. 3C). After embolization of the distal ICA, the microcatheter was withdrawn to the aneurysm cavity. A GDC (18 3D, 12 mm $\times$ 30 cm) was placed in the aneurysm. More GDCs (total coil length, 235 cm) were added to the aneurysm cavity. The aneurysm cavity was not embolized tightly because of the thrombus. As the aneurysm was embolized, the microcatheter slipped out of the aneurysm cavity into the proximal ICA. At this point, angiography showed that the proximal portion of the aneurysm was not embolized, and contrast medium was leaking into the nasal cavity (Fig. 3D). Pulsatile epistaxis was again observed after the angiography. A GDC (18 2D, 6 mm $\times$ 20 cm) was placed in the ICA proximal to the aneurysm followed by additional GDCs (total coil length, 84.5 cm) in the proximal ICA. Finally, the ICA and aneurysm were embolized with GDCs (total coil length 411.5 cm) and the epistaxis was controlled. Postembolization angiography showed complete obliteration of the ICA and aneurysm (Fig. 3E), and patent collateral flow was demonstrated. Postoperative examination showed that the patient was free of symptoms. Laboratory examinations demonstrated a complete normalization of the blood indices. The patient was discharged 1 week later.

Fig. 1  (A) Computed tomography scans showing an aneurysm protruding into the left sphenoid sinus, with erosion of the bony wall of the sphenoid sinus (arrow). (B) $T_1$-weighted (left), $T_2$-weighted with gadolinium (center), and $T_2$-weighted (right) magnetic resonance images showing a thrombosed mass in the left cavernous sinus. (C) Two- (left) and three-dimensional (right) digital subtraction angiograms of the left internal carotid artery showing an irregular aneurysm at the cavernous portion.

Fig. 2  (A) Computed tomography (CT) scans 11 months after the initial examination showing a bone defect on the wall of the sphenoid sinus as in the first CT. (B) $T_2$-weighted magnetic resonance images showing the lesion had not changed in size at 11 (left) and 17 months (right) after the initial diagnosis.
Fig. 3 (A-C) Left carotid angiograms at endovascular embolization demonstrating enlargement of the aneurysm cavity compared to the first angiogram, with a small protrusion toward the sphenoid sinus (A; arrow), and the distal internal carotid artery (ICA) is embolized with Guglielmi detachable coils (C). (D–F) Left carotid angiograms after embolization of the distal ICA and aneurysm demonstrating leakage of contrast medium from the aneurysm to the nasal cavity (D), complete obliteration of both the aneurysm and affected ICA (E), and sufficient collateral blood flow via the anterior communicating artery (F).

Fig. 4 T$_2$-weighted magnetic resonance images at 3 (A) and 7 months (B) after embolization showing decreased aneurysm size.

blood flow via the anterior communicating artery (Fig. 3F).

The patient regained consciousness the next day and the postoperative course was uneventful. No neurological deficit occurred related to the ICA occlusion. She was discharged on the 14th day after admission. Follow-up angiography performed 14 months after embolization did not show any recanalization of the aneurysm, and indicated sufficient collateral blood flow to the left ICA territory via the anterior communicating artery. MR imaging obtained 3 and 7 months after embolization demonstrated decreased aneurysm size (Fig. 4).

**Discussion**

The present case of thrombosed intracavernous ICA aneurysm was discovered incidentally. Since we thought that rupture of the thrombosed lesion was unlikely, as suggested in previous reports, we decided to simply follow the patient without radical treatment. However, about 2 years after the diagnosis, the patient suffered massive nasal hemorrhage from the aneurysm. Careful examination using CT and MR imaging is necessary for patients with intracavernous ICA aneurysms, especially if bone erosion of the sphenoid sinus is present. Surgical intervention should be considered for patients with intracavernous ICA aneurysms presenting with epistaxis, subarachnoid hemorrhage, or evidence of radiographic enlargement or neurological symptoms.10

Our patient experienced repeated minor nasal bleeding before the major hemorrhage. Epistaxis occurring after aneurysm rupture may recur, so radical treatment is necessary.2,13,15,22 Experience with a case of thrombosed intracavernous ICA aneurysm presenting with repeated epistaxis suggested that the epistaxis from the intracavernous ICA aneurysm tends to become increasingly frequent and severe, and carries a high risk of mortality.15 In our case, repeated minor bleeding was observed before the major bleeding. Minor epistaxis may be a warning sign for massive epistaxis. Therefore, radical treatment is necessary as quickly as possible, even if the bleeding is minor, for patients with intracavernous ICA aneurysms presenting with repeated nasal bleeding.

As many as 66% of cavernous ICA have a bony covering of less than 1 mm and 4% are dehiscent.14 The lateral bony wall of the sphenoid sinus is often less than 2 mm thick, so an aneurysm can easily erode through the bone and appear in the sphenoid sinus.16 Erosion of the sphenoid sinus was observed in our patient, and severe epistaxis developed about 2 years after the diagnosis. However, the bone condition of the sphenoid sinus did not change over 22 months of follow up. The aneurysm could have ruptured, even though the bone erosion was not progressive. Therefore, if CT shows bone erosion of the sphenoid sinus, close observation and radical treatment are required for intracavernous ICA aneurysms.

We initially tried to control the bleeding using a nasal tamponade with gauze in the emergency room, which reduced but did not stop the bleeding. Foley catheters have been used to control nasal bleeding in an emergency situation.17 The use of a urinary balloon catheter was recommended to control bleeding.11 Transient hemostasis using such devices or manual compression of the affected carotid artery at the cervical portion is quite important, because the condition can be fatal. Endovascular embolization or surgical occlusion of the lesion may be the final treatment options to permanently control nasal bleeding.
from an intracavernous ICA aneurysm.\textsuperscript{6,18,19,22} Endovascular embolization may be the best treatment because the lesion can be embolized immediately after diagnostic angiography or after control of bleeding by transient ICA occlusion, including the parent artery. Proximal ligation or embolization of the ICA could be effective for the treatment of unruptured giant or large intracavernous ICA aneurysms.\textsuperscript{31} However, a partially thrombosed ICA aneurysm treated by proximal carotid occlusion subsequently ruptured.\textsuperscript{71} The residual lumen showed growth and massive nasal bleeding occurred 25 months after carotid occlusion. Therefore, the partially thrombosed aneurysm still possessed the capacity for growth and rupture.\textsuperscript{71} Consequently, proximal occlusion of the ICA may not be adequate to prevent rupture of the aneurysm. Emergency ICA occlusion both distal and proximal to the aneurysm is necessary for ruptured intracavernous ICA aneurysms presenting with epistaxis.

In our case, we did not use a guiding catheter with a balloon in the emergency situation when the patient was in shock. A guiding catheter with a balloon should be used in such cases to control ICA blood flow, and could reduce the risk of elevation of intraneurysmal pressure after embolization of the parent artery distal to the aneurysm. Another treatment option is stenting with coil embolization to preserve the parent artery.\textsuperscript{21}

After partial embolization of the ICA and aneurysm in our case, injection of contrast medium resulted in rebleeding from the aneurysm to the sphenoid sinus. The precise site of hemorrhage was identified as the proximal portion of the aneurysm neck, which was not embolized at angiography (Fig. 3D). Injection of contrast medium to the affected artery should be minimized to avoid rebleeding from the aneurysm before complete obliteration. After embolization of the ICA, both distal and proximal to the aneurysm orifice, complete hemostasis was achieved. During the procedure, manipulation of the aneurysm cavity should be minimized to avoid thromboembolism from the preexisting thrombus in the aneurysm.

The present case shows that thrombosed intracavernous ICA aneurysm may still carry the risk of rupture. Therefore, close follow-up observation is necessary. In particular, erosion of the sphenoid sinus wall on CT and repeated minor bleeding may be important predicting signs for massive nasal bleeding. Radical treatment should be considered for such cases. Parent artery occlusion including the aneurysm may be the best treatment for intracavernous ICA aneurysms if sufficient collateral blood flow to the territory of the affected ICA is expected.

References


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