Endovascular Treatment of Internal Carotid Artery Pseudo-Aneurysm Presenting with Epistaxis

A Case Report

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Summary

Recurrent epistaxis is a rare presentation of internal carotid artery C4/C5 segment pseudo-aneurysm rupture. We describe a case of a traumatic internal carotid artery pseudo-aneurysm with recurrent epistaxis as a leading symptom that was finally managed with endovascular treatment with stent-assisted coil placement. Clopidogrel and acetylsalicylic acid orally were introduced in the therapy for further stent thrombosis prevention and epistaxis did not recur on six-month follow-up. Endovascular treatment with stent-assisted coil placement seems to be a good method for pseudo-aneurysm treatment while keeping the lumen of the parent artery patent.

Introduction

Internal carotid artery (ICA) C4/C5 segment pseudo-aneurysm rupture can present with recurrent epistaxis. The management options include an endovascular or neurosurgical approach, with no clear recommendations for treatment method selection. Because the neurosurgical approach is difficult for this ICA segment, the endovascular method is a rational alternative. As the pseudo-aneurysm has no stable arterial vessel wall there is often a need for stent placement and antiplatelet therapy, in a patient already at risk of fatal epistaxis. We

describe a case of traumatic ICA pseudo-aneurysm with recurrent epistaxis as a leading symptom that was finally managed with endovascular treatment with stent-assisted coil placement.

Case Report

A 55-year-old white man presented with recurrent epistaxis two months after a major car accident when he was treated conservatively for multiple fractures of the skull base, frontal and ethmoid bone bilaterally, frontal process of maxilla, nasal bone and left temporal epidural haematoma. Epistaxis occurred every seven days for a few minutes and occasionally had to be stopped with nasal packing. Eventually he was hospitalized for presyncope and microcytic anaemia with haemoglobin value 75 g/L requiring a blood transfusion. Brain computed tomography (CT) performed to exclude cranial bone fracture as a reason for recurrent epistaxis revealed a large mass in the cavernous sinus. Cerebral CT angiography (CTA) verified an aneurysm of the right ICA (rICA). Next, digital subtraction angiography (DSA) confirmed the aneurysm of C4/C5 segment of the rICA with its prominence to the sphenoid sinus (Figure 1). Endovascular treatment (Figure 2) with the patient under general anaesthesia followed. At the beginning of the procedure the patient received 4000 IU of heparin intravenously (IV)

Figure 1 Non-subtracted DSA lateral projection image of the rICA C4/C5 segment pseudo-aneurysm with a funnel-shaped neck.

Figure 2 Post-embolization angiogram shows the occluded pseudo-aneurysm sac with a minimal neck remnant and preserved normal flow in the rICA.

then a 6-French guiding catheter was placed through the right femoral artery to the rICA. An Echelon 10 microcatheter (ev3 Endovascular Inc., Plymouth, MN, USA) was placed in the post-traumatic pseudo-aneurysm of C4/C5 rICA segment, assisted by Synchro 14 microguidewire (Stryker, Fremont, CA, USA). During pseudo-aneurysm coiling, despite repositioning and balloon remodelling, coil loops protruded into the parent artery through the irregular and funnel-shaped pseudo-aneurysm neck. Therefore stent deployment was inevitable. The wide neck of the pseudo-aneurysm was crossed with a 4.5×28 mm self-expanding Enterprise stent (Codman Neurovascular, Miami, FL, USA), using the so-called trapping technique. At that time the patient received an IV bolus of 8 ml eptifibatide solution (0.75mg/ml), followed by an infusion of 13 ml/h. The pseudo-aneurysm was occluded by 15 Microplex coils (Microvention, Inc., Aliso Viejo, CA, USA) without complications. The eptifibatide infusion was continued for the next eight hours, when clopidogrel (75 mg) and acetylsalicylic acid (ASA) (100mg) orally were introduced in the therapy for further stent thrombosis prevention. The post-procedural course was uneventful. The patient was discharged five days after the procedure with dual oral antiplatelet therapy (75 mg of clopidogrel and 100 mg of ASA daily). Epistaxis did not recur on six-month follow-up.

Discussion

Diagnosis of ICA dissection in a patient with recurrent epistaxis can be challenging knowing that the time to first clinical presentation of artery dissection can vary from hours to years after trauma. In the majority of cases, epistaxis occurs within one to three months of injury, although delays in presentation of up to 40 years have been seen¹. Usually epistaxis is recurrent and in first appearances can be managed with nasal packs². The first several episodes of nasal bleeding in our patient were also considered plain epistaxis and were successfully treated with nasal packs until the last bleeding episode resulted in syncope. It was then that additional testing showed severe anaemia requiring blood transfusion which led to ordering brain CT that showed a large mass in the cavernous sinus later diagnosed as rICA pseudo-aneurysm. Considering the treatment strategy, the discussion was between parent artery occlusion and preservation. A direct surgical approach for cavernous sinus pseudo-aneurysm is demanding and clip placement requires a stable arterial vessel wall and real aneurysm neck. The option of extracranial to intracranial bypass with ICA occlusion was not considered as this procedure had never been performed in our institution and we agreed that it would pose a great risk for neurological deficit in our patient. The main consideration in

parent artery occlusion by surgical or endovascular intervention is the risk of major stroke if the collateral circulation is inadequate. In order to predict possible stroke after ICA occlusion the carotid artery balloon occlusion test can be performed. Even when patients tolerate this test some studies show that there is also a long-term risk of infarction after ICA occlusion³. The decision was made to perform an endovascular procedure and to exclude the pseudo-aneurysm from the circulation while keeping the rICA patent. Considering the patient's age and no neurological deficit related to rICA pseudo-aneurysm it seemed the best choice. The rICA occlusion was considered a last therapeutic option if the endovascular approach failed. The patient expressed the wish for occlusion to be performed in another session and was fully informed of the possible risks of his decision. Treatment with covered stent placement was considered first. The only available covered stent in our institution was a coronary stent graft (Jostent, Abbott), a rigid stent often not conforming to the vessel wall at this location. Attempts to place such a rigid device may expose the patient to a greater risk of further intimal injury and dissection. Additionally, there would be a risk of occlusion of the ophthalmic artery by a covered stent so we decided not to use it. As the pseudo-aneurysm does not have all normal vessel wall layers, during the procedure the coil loops could not be confined to the pseudo-aneurysm sac. Coil protrusion into the parent artery through the irregular and funnel-shaped pseudo-aneurysm neck made deploy-

ment of the self-expanding Enterprise stent inevitable. In our institution patients usually receive dual antiplatelet therapy with clopidogrel and ASA before the procedure demanding stent placement to prevent in-stent thrombosis. The risk of epistaxis recurrence in this case was estimated as high and according to the literature there was also a possibility of late rebleeding even after covered stent placement⁴. This rebleeding after an endovascular procedure can be explained with the lack of a true vessel wall in a case of pseudo-aneurysm and dissection progression that can lead to coil migration⁵. Some authors stress the risk of thromboembolic accidents and report giving a loading dose of dual antiplatelet therapy at the beginning of the endovascular procedure even in patients presenting with subarachnoid haemorrhage⁶. We decided in this case to give eptifibatide for possible thrombosis prevention given the previous good experience with its administration for stenting of ICA pseudo-aneurysm⁷. The post-embolization angiogram showed the occluded pseudo-aneurysm sac with a minimal neck remnant and preserved normal flow in the rICA (Figure 2). Eight hours after the procedure dual antiplatelet therapy with 75 mg of clopidogrel and 100 mg of ASA was initiated with no epistaxis occurrence on six month follow-up.

In conclusion, repeated epistaxis in a patient with a history of severe craniocerebral trauma should raise a suspicion of possible ICA pseudo-aneurysm. Whenever possible, the first choice of treatment should be pseudo-aneurysm occlusion with parent artery preservation.

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