Spontaneous Petrous Carotid Artery Occlusive Dissection Treated by Local Fibrinolysis and Stent Deployment

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Summary

This paper reports a case of local thrombolytic therapy followed by stenting of the petrous carotid in a young woman with recurrent transient ischemic attacks from spontaneous dissection. A total of four overlapping balloon-expandable stents were delivered in two different sessions one month apart.

The procedure resulted in a potentially efficacious treatment for the prevention or reduction of cerebral damages from ischemia. At follow-up three months later the patient was symptom-free and DSA revealed a delayed proximal small pseudoaneurysm and a carotid-cavernous fistula. At one year follow-up the patient was still symptom-free with unmodified findings at cerebral angiography.

Introduction

Spontaneous dissection of cervical vessels is a well-documented cause of ischemic stroke in young adults (5 to 20%) and of long-term neurologic impairment with poor functional recovery^{1,2}. The cervical vessels are the most frequent site of dissections and only sporadic and scanty case reports are available on patients with intracranial carotid dissections^{3,4}. Dissection is characterized by subintimal (subintimal dissection) or subadvential (subadvential dissection)

penetration of circulating blood causing vessel narrowing and leading to thromboembolism, pseudoaneurysm formation or both. Management of intracranial dissections is complex and still debated. Treatment of cervical vessel dissections has traditionally relied upon anticoagulant/anti-platelet therapy. Surgery is sometimes proposed in symptomatic patients resistant to the medical therapy⁵.

Recently endovascular techniques have been employed with encouraging results in both intra and extracranial arterial dissections and dissecting aneurysms^{3,6-14}. Herein we report a patient with occlusive spontaneous dissection of the left petrous carotid artery treated with local urokinase infusion and stent placement.

Case Report

A 40-year-old woman was admitted for acute onset of motor aphasia, right side hemiparesis and mental confusion. Her medical history was unremarkable. Neither hypertension, trauma, spinal manipulation or smoking were reported. On admission an unenhanced brain computed tomography (CT) scan showed neither ischemic lesions nor intracranial haemorrhage. While the angiographic suite was prepared a cerebral magnetic resonance (MR) examination on a low-field resistive (0.2T) magnet was performed with axial SE proton density and T2

Figure 1 Left common carotid angiograms in the LL view on admission show tapering of the internal carotid artery lumen in the petrous tract with abrupt distal regaining of the vessel diameter consistent with subocclusive dissection.

–weighted (2000/30-100/1 TR/TE/NEX) and axial SE T1-weighted (500/15/2 TR/TE/ NEX) images for a total time of 20 minutes. MRI showed an abnormal signal intensity in the petrous tract of the left internal carotid artery consistent with slow flow or acute thrombosis (medium signal intensity on spin-echo T1-weighted and high signal intensity on proton-density and T2-weighted images) in the absence of any cerebral abnormalities. MR angiography (MRA) was not performed since this technique in our experience yields scarce results on low-field resistive magnet especially at level of the carotid syphon where flow artefacts are redundant. Moreover MRA would have significantly delayed the total length of acquisition time.

The patient subsequently underwent diagnostic four-vessel cerebral digital subtraction angiography (DSA) that showed a subocclusive dissection of the left petrous carotid artery with hypoperfusion in the frontoparietal lobes of the left hemisphere (figure 1). Retrograde flow through posterior or anterior communicating arteries filling the ipsilateral middle cerebral artery was not seen. Both anterior cerebral arteries filled on injection in the right internal carotid artery. At the end of the DSA examination the patient gradually recovered spatial and temporal orientation. The neurological examination revealed an improving residual right upper limb weakness with regression

of aphasia. The patient was put on intravenous heparin infusion (bolus of 2000IU/ followed by infusion of 1000 IU/h) and the neurological deficit completely regressed one and a half hours later.

The following day the patient became suddenly stuporous with recurrence of the right side haemiparesis and aphasia. An unenhanced cranial CT scan was repeated and resulted unchanged. Within four hours from onset of the second episode the patient underwent another cerebral DSA that showed occlusion of the left petrous carotid artery (figure 2A) with scant filling of the ipsilateral middle cerebral artery through the anterior communicating artery. General anesthesia was administered and a 6 Fr groin sheath was inserted into the right common femoral artery. A loading bolus of 5000 IU of heparin followed by infusion of 1000 IU/h was administered. A 6 Fr guiding catheter (Envoy - Cordis Endovascular System) was placed in the extracranial upper tract of the right internal carotid artery and a microcatheter (Prowler .014" Cordis Endovascular System) primed with a 0.010 inches guidewire (Transed EX 10 - Boston Scientific) was subsequently advanced proximal to the occlusion. Local infusion of urokinase (UK) and mechanical manipulation of the thrombus with the guidewire was started. After 1.000.000 units of UK retrograde internal carotid artery filling was restored revealing a lumen stenosis in the distal tract of the dissected portion of the petrous carotid artery (figure 2B).

The guidewire was advanced beyond the stenosis and two overlapping balloon-expandable stents (Seaqueunce Nycomed Amersham 4 x 10 e 4 x 8 mm) were consecutively deployed. Post-stenting angiograms showed restoration of the petrous carotid artery lumen to an almost normal caliber with residual irregularities of the vessel wall in the proximal tract. The right femoral sheath was left in place and intravenous heparin infusion was continued for 48 hours maintaining activated partial thromboplastin time between 70 and 80s. Antiplatelet treatment with aspirin (325 mg daily) and ticlopidine (250 mg twice each day) was also started. A postprocedural cerebral CT did not show ischemic or haemorrhagic lesions.

The patient's outcome was excellent and a cranial CT the following day showed two small cortical ischemic foci in the left anterior aspect

Figure 2 A-B) Left common carotid angiogram in the LL view A) at recurrence of right side hemiparesis and aphasia shows an occlusion of the petrous carotid artery. After local infusion of 1,000,000 IU of UK B) the internal carotid angiogram shows restoration of flow with a stenosis at the distal end of the dissection flap.

of the superior frontal gyrus and sulcus. Since no complications intervened the patient was discharged five days later with a complete clinical recovery. One month later the patient was again admitted because of recurrence of right upper limb paresis despite appropriate assumption of medical therapy. A plain CT scan did not yield new findings. A DSA cerebral examination revealed persistence of an intimal flap with pseudoaneurysmal dilatation of the left petrous carotid artery.

Deployment of two more overlapping balloon-expandable stents (AVE medtronic 4 x 15 mm) with complete remodeling of the vessel lumen was undertaken. At follow-up three months later the patient was asymptomatic and a cerebral DSA confirmed stent patency with pseudoaneurysmal dilatation of the proximal petrous carotid artery and a carotid-cavernous (CC) fistula draining in the inferior petrous sinuses (figure 3). Assumption of ticlopidine and aspirin was continued and at a clinical/ angiographic follow-up one year later the patient was asymptomatic and a control DSA showed spontaneous disappearance of the CC fistula (figure 4).

Discussion

The natural history and management of intracranial dissections are still debated. Contrary to their more frequent cervical counter-

parts intracranial dissections are usually subadventitial and entail a poorer prognosis resulting in vessel rupture and subarachnoid haemorrhage. However, recent reports indicate that unruptured intracranial dissections may have a benign course¹⁵⁻¹⁶.

In occlusive intracranial dissections, as was our case, thromboembolism is presumably the cause of the symptomatology and the first line therapy is pharmacological, primarily consisting of antiplatelet and anticoagulant agents unless there is progression, persistence or recurrence of neurological deficits. Surgical intervention for intracranial carotid dissections has consisted of proximal carotid ligation with or without previous extracranial/intracranial bypass^{4,12}. These procedures are generally technically demanding and yield disappointing clinical results. Endovascular techniques have been described in the treatment of vertebral artery dissections and pseudoaneurysms but this has primarily consisted of balloon occlusion⁶. Successful stenting and coiling of a ruptured fusiform basilar artery aneurysm has been reported⁷.

Elective stenting of extracranial carotid arteries has been found reasonably safe and efficacious in patients with carotid artery stenosis as well as in the treatment of multiple aneurysms⁸, dissecting aneurysms⁹, traumatic dissections and pseudoaneurysms^{10,11} of the extracranial internal carotid artery. Recently several

Figure 3 Left internal carotid angiogram in the oblique left anterior view with a slight cranial tilt one month after the second stenting procedure shows remodeling of the vessel profile with a small pseudoaneurysmal dilatation associated with an early venous drainage in the inferior petrous sinus secondary to a carotid-cavernous fistula.

case reports have been published regarding stenting of intracranial carotid lesions. Dorros G et Al reported successful stenting of the petrous carotid artery in a patient with dissection caused by balloon angioplasty³. Fessler RD et Al described markedly improved cerebral blood flow after balloon-angioplasty and stenting of a petrous carotid atherosclerotic stenosis¹². Feldman et Al reported the use of a

Figure 4 Left internal carotid angiogram in the LL view one year later show spontaneous disappearance of the carotid-cavernous fistula.

petrous carotid stent in a patient with several prior balloon angioplasties of the same lesion¹³ and Al-Mubarak et Al published one case of petrous carotid stenting after balloon angioplasty in a patient with recurrent transient ischemic attacks¹⁴. In our case local fibrinolysis and stenting of the spontaneously dissected petrous carotid artery was prompted after recurrence and worsening of the patient's neurological deficits despite appropriate medical therapy. An aggressive treatment was not attempted from onset since the patient showed a temporary but complete clinical recovery. Most patients, particularly young ones, are capable of perfusing their ipsilateral MCA territory even without an internal carotid artery. It is possible that in our case the acute recurrent symptomatology was related to embolic disease rather than hypoperfusion. In this instance a possible therapeutic option after restoration of flow in the ipsilateral MCA would have been occlusion of the ICA to prevent future complications (emboli, pseudoaneurysm or CC fistula). However the scant collateral flow and the good result of the intra-arterial thrombolytic therapy prompted us to restore the lumen patency by stenting. This proved to be the right choice in the face of the delayed pseudoaneurysm and CC fistula since at one year follow-up the patient was completely asymptomatic on antiplatelet therapy.

The successful albeit difficult deployment of multiple overlapping stents in the petrous segment of the internal carotid artery has potential significance especially considering the need for stent specifically designed for intracranial use. The major difficulty of the procedure consisted in negotiating the guidewire through the true lumen of the dissected tract.

In our case restoration of flow by fibrinolysis allowed differentiation between the true and false lumen. This enabled safe microcatheterization and deployment of the overlapping stents in the true lumen of the dissected artery resulting in restoration of vessel patency and regression of symptoms. However recurrent transitory ischemic attacks from a residual intimal flap acting as a source of possible cerebral emboli (despite assumption of appropriate medical therapy) prompted placement of two more overlapping stents in a subsequent session to complete the remodeling of the vessel lumen.

The patient was symptom-free at three months follow-up and the control DSA revealed a CC fistula as well as a pseudoaneurysm at the proximal end of the stented tract of the vessel.

The CC fistula is a known complication of endovascular procedures involving the intracranial internal carotid artery^{17,18}. It seems to be caused by a microguide-induced perforation of a small branch of the cavernous portion of the internal carotid artery and usually does entails a bad prognosis. In our case it was decided to refrain from interventional procedures in favour of preventive administration of anti-platelet therapy and careful clinical and angiographic monitoring. At one year follow-up the CC fistula had spontaneously disappeared and the patient was completely asymptomatic.

The cause underlying spontaneous intracranial dissection in our patient remains unknown. Fibromuscular dysplasia has been reported in 10 to 20% of patients with extracranial ICA dissection and is more common in patients with bilateral involvement¹⁹. Recently a spontaneous dissection without angiographic evidence of dysplasia that was subsequently pathologically proven has been published²⁰.

Conclusions

Intra-arterial fibrinolysis and stent placement offered a minimally invasive and efficacious treatment for the prevention of cerebral ischemia secondary to occlusive petrous internal carotid dissection. Prospective controlled evaluation of this novel therapeutic modality with long term follow-up is warranted to elucidate its role as potential alternative to surgical intervention in patients who remain symptomatic despite appropriate medical therapy.

Microguide-induced carotid-cavernous fistula draining in the inferior petrous sinuses is a reported complication of endovascular procedures on the intracranial carotid artery and although it usually entails a poor prognosis in our case it spontaneously disappeared at one year follow-up.

The presence of a delayed pseudoaneurysm is of more concern since it may represent a source of emboli; however since the patient was asymptomatic at one year follow-up continuation of the antiplatelet therapy was deemed the only therapeutic option.

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