Carotid Artery Stenting to Prevent Stroke in a Patient with Bilateral Extracranial Internal Carotid Dissection and Vasospasm Following Rupture of an Intracranial Aneurysm

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Summary

The stenting of carotid dissection has been described for the prevention of cerebral ischemia in patients who remain symptomatic despite therapeutic anticoagulation, in those who present contraindications for anticoagulation therapy, or who present a local or extensive stenosis, with an associated pseudoaneurysm. We here report a case associating a high clinical grade aneurysmal rupture with a bilateral extracranial carotid dissection. Because of the haemodynamic risk due to the acute bilateral stenosis-induced dissection and the occurrence of a vasospasm, the carotid dissections were treated with self-expandable stents.

Introduction

Internal carotid artery dissection is an increasingly recognized cause of stroke that accounts for up to 20% of ischemic strokes in young adults^{1,2}. The complication of carotid dissection is generally due to thromboembolic events, and medical therapy is considered to be the first line treatment. In a few patients the haemodynamic effect of the carotid obstacle may lead to a cerebral infarct, and in symptomatic patients, when conservative medical treatment fails, endovascular treatment has been considered.

Arterial vasospasm is present in 40 to 70% of cases after sub-arachnoid haemorrhage, and can be symptomatic in 20 to 30%³. Stroke during vasospasm is explained by arterial narrowing and the plummeting of arterial flow.

The case reported here is the first published associating a bilateral carotid dissection with an angiographic vasospasm due to intracranial aneurysm rupture. The narrowing of the intracranial arteries due to vasospasm, and of the extracranial carotid arteries due to the dissections was considered as a major risk for haemodynamic impairment, and a stenting of both carotid arteries was carried out to prevent a cerebral infarct.

Case Report

Mr G is a 53 year-old-man with no previous history who was found unconscious at home. Hospitalized in intensive care and given respiratory assistance, the patient was given a brain scan which showed a haemorrhage situated mainly in the posterior fossa meningeal spaces, a dilatation of the supratentorial ventricular cavities, and the presence of blood in the fourth ventricle. After placing an external ventricular shunt, cerebral arteriography was performed. This showed the existence of two intracranial aneurysms, one situated on the middle cerebral artery and the other located at the origin of the

Figure 1 Lateral view of the vertebro-basilar system, showing a left PICA aneurysm before and after endovascular coiling.

left postero-inferior cerebellar artery (PICA), and considered to be responsible for the sub-arachnoid haemorrhage (figure 1). Carotid angiography also revealed the presence of staged arterial narrowings visible on the two internal cervical carotid arteries, suggesting an aspect of fibromuscular dysplasia or dissection (figure 2). Angiography additionally revealed hypoplasia of the posterior communicating arteries.

Treatment of the left PICA aneurysm was carried out immediately following arteriography using endovascular access. Considering that both carotid stenoses corresponded to dissections, and that the untreated middle artery aneurysm was asymptomatic, a treatment with heparin followed embolization to avoid thrombo-embolic events.

Follow-up cerebral angiography was performed on the seventh day of bleeding. This showed a significant change of the internal ca-

rotids, with the characteristic appearance of dissection combining a severe bilateral stenosis four centimetres high starting a few centimetres above the bulb and terminating at the intrapetrous segment of the internal carotid arteries with the presence of pseudoaneurysms not visible on the first angiogram (figure 3). In addition, the series of intracranial angiographic images revealed a concentric narrowing of the end of the internal carotid arteries and of the M1 segments of both middle cerebral arteries, secondary to cerebral vasospasm (figure 4). A transcranial Doppler showed average velocities at the M1 segment of the middle cerebral arteries of 150cm/s, but the results obtained were not considered reliable because of upstream carotid obstacles.

Given the presence of vasospasm, the impossibility of appropriate neurological monitoring in a patient receiving respiratory assistance,

Figure 2 Digital subtraction angiography of the right and left common carotid arteries reveals right middle cerebral artery aneurysm, associated with moderate and staged stenosis of both cervical internal carotid arteries diagnosed as dissection or fibromuscular dysplasia.

and above all the radiological evidence of a worsening of the carotid stenoses despite the heparinotherapy, endovascular treatment was indicated.

A 7F catheter (Envoy; Cordis®) was placed in the primitive carotid arteries via a femoral vascular sheath. After passing the stenoses with a 0.014 guide (Transend Boston Scientific®), a self-expanding stent (Wallstent Boston Scientific®) 50 mm long to the right and 40 mm to the left was introduced into the two internal carotids. Angiographic monitoring showed satisfactory placing of these endoprotheses, and a slight residual narrowing of the left side beneath the proximal part of the stent (figure 5).

A scan performed several days later did not show cerebral infarct. On leaving hospital three weeks later, apart from a horizontal diplopia, the patient showed no other symptoms.

Four months later the patient's symptoms had improved, the diplopia being experienced only when looking very sharply to the right. Angiography revealed a satisfactory appearance of the internal carotid arteries, with the persistence of a moderate narrowing on the left side at the beginning of the stent; this stenosis was considered moderate and not significant on a Doppler performed at the same side.

The aneurysm in the left middle cerebral artery was treated surgically three months later, without complication.

Discussion

The annual incidence of spontaneous carotid dissection has been reported to be 2.6 per 100000⁴, and has been estimated to account for up to 20% of strokes in the younger population^{1,2}.

Figure 3 Digital subtraction angiography of both common carotid arteries showed progression of the dissections, despite systemic anticoagulation, with a long-segment stenosis and expansile pseudoaneurysms.

The extracranial internal carotid artery is the most frequent site of spontaneous dissection, with both ICAs involved in a minority of cases ($< \text{or} = 15\%$)⁵.

Trauma and preexisting disease of the artery may predispose to dissection⁶. In our case the origin of the bilateral carotid dissection, associated with intracranial aneurysm rupture, was not clear. The cerebral and renal angiographies did not show features of fibromuscular dysplasia. The patient's loss of consciousness after haemorrhage, and the hypothesis of a possible epileptic seizure, suggested a traumatic origin, but manipulation of the catheter systems during angiography might also be the cause of the dissections.

Most infarcts associated with dissection have been proposed to be embolic in nature, although a non-negligible proportion consists of haemodynamic flow-related infarcts^{7,8}.

The accepted medical standard of therapy has consisted of systemic anticoagulation^{7,9,10}, and has been observed to lead to a significant rate of subsequent resolution and healing^{7,11}. With heparin, the rate of repeat embolization is low, and nothing justifies placement of an intravascular stent as primary treatment. Despite

anticoagulation, a number of patients progress to have haemodynamically significant residual stenosis, or develop pseudoaneurysms leading to a risk of haemodynamically significant stenosis or to a danger of delayed distal embolization. In these symptomatic patients, surgery or endovascular treatment are considered.

Although surgery has been performed for direct repair of carotid dissection, this treatment is not recommended anymore because surgery for carotid dissection is associated with a significantly greater risk than is carotid endarterectomy for atherosclerosis¹², and identification of the inflow zone, and therefore repairing of the entire extent of the dissection, is difficult.

Endovascular stents have been shown to be of benefit for the treatment of coronary atherosclerosis¹³, and for the treatment of carotid atherosclerosis in certain high-risk patient subgroups¹⁴.

Unlike atherosclerotic stenoses, which require higher-pressure angioplasty and necessitate fracturing the plaque-with a risk of distal embolization of atheromatous debris- the stent deployment for dissection is less traumatic, and is performed at a lower pressure because the lesions are more compliant and are not usually atheromatous or calcified. Because stenting allows prompt restoration of the carotid lumen, by providing the necessary centrifugal force to permit apposition of the dissected segment to the vessel wall to obliterate the false lumen and resolve the stenosis¹⁵, endovascular treatment has been proposed for carotid dissection when conservative medical treatment fails¹⁵⁻²⁴.

In the literature, stenting has been indicated for symptomatic patients harbouring a dissection-induced stenosis that has not responded to a course of medical therapy, dissection in a patient with contraindications for anticoagulation therapy, focal or extensive stenosis- with formation of an associated pseudoaneurysm, and in bilateral dissections. In these last cases it was believed that the bilateral nature of the lesions and their severity ($>70\%$) can carry the risk of a severe cerebral haemodynamic impairment with flow restriction and ischemia²⁵. Therefore for many authors, the symptomatic and bilateral nature of the lesions requires a more aggressive management^{19,20,25}.

Despite progress in medical management, vasospasm remains a major cause of mortality and morbidity in patients presenting a sub-

Figure 4 Anteroposterior projection of the right and left internal carotid angiograms showing arterial vasospasm.

arachnoid haemorrhage. This cerebral vasospasm is a frequent and severe complication of aneurysm rupture, and consists of a narrowing of intradural arteries: it produces a decrease of cerebral blood flow in the territory of the spasmed vessel, and can induce transitory or definitive ischemic deficits. More over the self-regulation of the cerebral blood flow is impaired in the territory of the spasmed vessel²⁶, which makes the brain cells situated in this zone very sensitive to reductions in brain perfusion pressure, and to any decrease in oxygen supply.

In the case presented here, the risk of cerebral infarct combined with vasospasm was maximized by haemodynamic repercussions linked to the bilateral carotid stenosis. The placing of carotid stents enabled us to restore normal patency through the dissected carotid arteries, contributing to a greater effectiveness of hypertensive therapies aimed at combatting the fall in regional blood flow caused by the narrowing of

Figure 5 Right and left carotid angiograms realized four months, after stenting, showing good patency of the arterial lumen with a residual stenosis of the left internal carotid artery just beneath the proximal part of the stent.

the middle cerebral arteries. Short-term monitoring of carotid stents has shown perfect stability of carotid calibre.

The long-term efficacy and durability of stent placement remains to be determined, al-

though Liu et al showed a perfect tolerance of these devices, and a complete resolution of dissection-induced stenosis treated with stent after a follow-up monitoring of more than three years¹⁷.

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