SHORT REPORT

Primary Stenting for the Acute Treatment of Carotid Artery Dissection

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Objectives. To review and evaluate our experience with carotid artery stenting in the acute treatment of carotid artery dissection (CAD).

Patients and methods. Reviewing the charts of our hospital between 2000 and 2001, we found two consecutive patients who benefited from primary stenting for the acute treatment of spontaneous extracranial internal CAD.

Results. Primary stenting of the internal carotid artery was successful in both cases without any post-operative complications. Clinical and US duplex scan follow-up confirmed the absence of neurological symptoms and the patency of the internal carotid artery with complete disappearance of the dissection at 36 and 42 months after the procedure, respectively.

Conclusion. Despite the small number of patients, primary stenting for acute CAD seems to be safe and effective.

Keywords: Acute; Carotid artery; Arterial dissection; Endovascular procedures; Stenting.

Introduction

Carotid artery dissection (CAD) is a pathology that can occur spontaneously and following trauma or invasive procedures. Up to now, medical treatment with anticoagulation remains the most applied therapy since initial management is mainly focussed on the prevention of cerebral ischemic complications. Surgery is no longer the first line treatment because it is often associated with poor results and is only considered for refractory cases with contraindication to anticoagulants. The aim of this study was to report and evaluate our experience in primary stenting for CAD.

Report

We reviewed the charts of our hospital between 2000 and 2001 and found two consecutive patients that were admitted with the diagnosis of acute spontaneous extracranial internal CAD. Their clinical data are illustrated in Table 1. Both patients were admitted in the few hours following the onset of symptoms and complained of headaches and cervical pain. At the admission, neurological exams were normal. Cervical US duplex scan studies demonstrated bulbar hematoma and an intimal flap of the internal carotid artery. Cerebral CT failed to show any brain infarct. Both patients underwent endovascular repair of the CAD during its acute phase. Intravenous heparin was administered to achieve an ACT of 2–3 times the normal. A Seldinger technique was used to perform selective arch aortography that confirmed CAD in both cases (Fig. 1). A 7-Fr sheath was introduced and a hydrophilic guide wire 0.035 (Terumo) was threaded through the true lumen of the ICA and a 6-mm×2-cm Nitinol self-expandible stent (S.M.A.R.T., Cordis) was deployed at the proximal inflow zone of the internal carotid artery. Post-procedural control angiography confirmed successful placement with extra- and intracranial patency of the internal carotid artery and disappearance of the intimal flap (Fig. 2). Notably, successful stenting led to total disappearance of the
neck pain. Post-operative treatment consisted of ticlopidine for 6 weeks and aspirin (160 mg qd) indefinitely. Serial clinical follow-up studies confirmed the absence of neurological symptoms and US duplex scans showed the patency of the internal carotid artery with no sign of dissection. Both patients are alive and free of symptoms 43 and 49 months after the procedure, respectively.

Discussion

The major concern following internal CAD is stroke which arises in more than half of the cases, affecting predominantly young people, where it is related to a poor outcome with a highly abnormal subsequent quality of life. Cerebral infarction can occur up to 1 month after dissection, with nearly two thirds arising after day 1. Medical management consisting of anticoagulants or antiplatelet drugs are the recommended treatment, even though no clinical trial has proven their relative efficiency. In fact, clinically silent brain microemboli can be detected in medically treated patients. Moreover, recent studies reported a bleeding complication incidence of 22% and complete recovery in less than one third of patients. Surgical management can be complicated by perioperative stroke that occurs in 10% of the cases, its indications being therapeutic failure or contraindicated anticoagulation. Several reports in the literature deal with percutaneous treatment of CAD at a chronic stage with encouraging initial results. But authors are still reluctant to use this technique and favour it only in chronic cases. However, acute treatment of CAD has already been described in seven patients. In five instances stents were placed at the time of an iatrogenic dissection. The two remaining cases were acutely treated in the setting of worsening and fluctuating neurological symptoms. As for the cases described in this study, there was no morbidity nor mortality after the procedure and all authors reported clinical improvement.

Liberal application of this technique is limited by its potential complications which consist of embolic events, thrombosis, and intimal hyperplasia. Distal embolization has been shown to occur at each stage of endovascular procedures for atherosclerotic stenosis. However, dilatation of an otherwise previously normal carotid artery as in CAD differs from the same act performed for highly calcified and atherosclerotic vessel. Indeed, it is less traumatic and requires lower pressures because the lesions are more compliant. Moreover, potential embolic particles are distinct since atheroemboli consist in lipid-rich macrophages and cholesterol clefts while endothelial injuries display thrombi that contain platelets and fibrin. Stent thrombosis and intimal hyperplasia are well-known complication in coronary angioplasty. There is still a need for studies dealing with these complications in endovascular CAD treatments. But the risk of thrombosis can be lowered because of the relatively higher carotid artery diameter and by accurately deploying the stent over the dissection. Clinically significant in-stent recurrent stenoses rate of 6.4% reported in atherosclerosis is expected to be less in CAD since intimal hyperplasia is linked to an underlying inflammatory process.

Table 1. Clinical data of both patients

<table>
<thead>
<tr>
<th></th>
<th>Patient a</th>
<th>Patient b</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>49</td>
<td>64</td>
</tr>
<tr>
<td>Gender</td>
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<tr>
<td>Cardiovascular risk factors</td>
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<tr>
<td>Side</td>
<td>Right</td>
<td>Left</td>
</tr>
<tr>
<td>Type of ICAD</td>
<td>Spontaneous</td>
<td>Spontaneous</td>
</tr>
<tr>
<td>Follow-up (months)</td>
<td>49</td>
<td>43</td>
</tr>
</tbody>
</table>

Fig. 1. Angiography showing the acute bulbar ICAD in patient a.
Immediate post-procedural control angiography in patient a.

References


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