Surgical treatment for pseudo-occlusion of the internal carotid artery

Alessandro Desole*, Francesco Campanile, Federico Tosato and Domenico Milite

Operative Unit of Vascular and Endovascular Surgery, S. Bortolo Hospital, Vicenza, Italy

* Corresponding author. Operative Unit of Vascular and Endovascular Surgery, S. Bortolo Hospital, Viale Rodolfi 37, 36100 Vicenza, Italy. Tel: +39-0444-753669; fax: +39-0444-753142; e-mail: al.desole@gmail.com (A. Desole).

Received 14 November 2014; received in revised form 16 December 2014; accepted 27 December 2014

Abstract

OBJECTIVES: Carotid artery pseudo-occlusion is a rare condition and its natural history and clinico-pathological characteristics are not well defined. We reported our 7-year experience in the surgical treatment of carotid artery pseudo-occlusion to determine the real benefit of the surgical option.

METHODS: From January 2006 to December 2013, 1414 patients were treated for high-grade stenosis of the internal carotid artery, 33 (2.3%) presented with a carotid pseudo-occlusion (26 males and 7 females, mean age: 70 ± 10). Nineteen patients were symptomatic, and 14 asymptomatic. Carotid artery pseudo-occlusion was identified by duplex scan (segmental occlusion at the origin of internal carotid artery with very thin distal flow) and the diagnostic confirmation was obtained by angio-computed-tomography (CT) scan. The operation was performed under general anaesthesia and constant Electroencephalography (EEG) monitoring. The follow-up was performed by duplex scan at discharge, 30 days, 6 months and yearly.

RESULTS: Polytetrafluoroetilene (PTFE) patch endarterectomy, eversion endarterectomy and carotid bypass were performed in 20 (61%), 10 (30%) and 3 patients (9%), respectively. No mortality or stroke was observed in postoperative period. Four patients presented with an asymptomatic postoperative thrombosis of the internal carotid artery. No restenosis was observed.

CONCLUSIONS: Surgical treatment for carotid artery pseudo-occlusion is safe and effective.

Keywords: Cerebrovascular disease • Carotid pseudo-occlusion • Carotid stenosis • Carotid endarterectomy • Stroke prevention

INTRODUCTION

Carotid artery pseudo-occlusion (CAPO) was first described by Lippman et al [1] in 1970 as a severe stenosis of the internal carotid artery (ICA) with an extremely narrow residual lumen; angiographically, it looked like a thin string, and a collapsed distal portion was induced by hypoperfusion [2]. CAPO is a rare condition, with an incidence of 0.5–2% [3, 4], and its natural history and clinico-pathological characteristics are not well defined.

Rothwell et al. [5], after the North American Symptomatic Carotid Endarterectomy Trial (NASCET) [6] and European Carotid Surgery Trial [7], analysed the subgroup of the CAPO, and reported that the stroke rate was not higher compared with that of patients with high-grade stenosis of ICA and there were not surgical benefits in this group.

On the other hand, Morgenstern et al. [8] reported that, even if the risk of stroke was lower than that of patients with a severe stenosis of ICA without distal narrowing, the surgical treatment of CAPO reduced the risk of stroke.

Also Terada et al. [9] concluded that even if the risk of stroke was lower than that of patients with a severe stenosis of ICA, CAPO was a surgically treatable lesion with low risk and a high success rate and its treatment may be beneficial in stroke prevention.

Therefore, there is no agreement on the indication, risks and results of surgical treatment in CAPO.

We reported our 7-year experience in surgical treatment of CAPO to determine real benefit of the surgical option.

METHODS

From January 2006 to December 2013, 1414 patients were treated for high-grade stenosis of ICA and 33 of these (2.3%) presented with a carotid pseudo-occlusion. There were 26 males and 7 females with a mean age of 70 ± 10 (range: 31–85 years). The mean follow-up was 32 months (range: 1–6 years).

Pseudo-occlusion was identified by duplex scanning (Philips iu22, linear probe 9-3, 35 Hz) for the presence of a segmental occlusion at the origin of ICA with very thin distal flow, appearance of narrow flow jet (string sign) on colour Doppler images, low velocity in the ICA and/or dampening of common carotid artery spectral waveform [10]. Diagnostic confirmation was obtained in all patients by angio-CT scan in which the segmental occlusion of ICA was noted with reconstitution of filiform flow in the distal portion of the vessel (Fig. 1).

Risk factors were present as given in Table 1.
Nineteen patients were symptomatic and 14 asymptomatic, but all these patients had a non-specific symptom without paraesthesia or amaurosis.

The surgery was performed under general anaesthesia and constant EEG monitoring during all the procedures; from the positioning of patient to the end of surgery, the cerebral activity is recorded with electroencephalography and somatosensory-evoked potential monitoring; all data are analysed in operatory room from a technical neurology specialized in EEG during all the surgery procedures.

The patients were treated with an intraoperative infusion of 5000 UI of heparin before the carotid clamping.

The standard treatment of PTFE patch endarterectomy was performed in 20 patients (61%); the eversion endarterectomy technique was used in 10 patients (30%) due to a kinking of ICA associated with pseudo-occlusion. Three cases (9%) presented a long fibrous plaque in ICA removed with difficulty in the distal portion of the vessel; therefore, it was necessary to perform a great saphenous vein carotid bypass.

During surgical treatment, we used some strategies in order to perform a correct suture in the ICA and to avoid the barotrauma after the unclamping.

The first step is the removal of the plaque in the carotid bifurcation, after the endarterectomy we inserted a dilator in the first part of ICA in order to enlarge the diameter and to able to perform a suture of the distal part of patch without stenosis (Fig. 2).

We used a dilator tin, we have a series with different diameter but in CAPO, because of the thin diameter of ICA, we use only the 3 or 4 mm. After the carotid plaque removal, we inserted the dilator only in the proximal part of ICA (~2 or 3 cm). The insertion had to be very slow and careful so that we could avoid the dissection (we have not had any). There are no scientific data on the use of this technique in CAPO, but we have seen (in our experience) that the use of the dilator slight enlargement of the first part of the ICA, allowing us to enter the shunt and then perform a safe suture.

After the dilator, we inserted a Pruitt-Inahara shunt, not to prevent the clamping ischaemia, but in order to gradually increase the blood flow during the suture of patch and to avoid the barotrauma after carotid unclamping (Fig. 3).

We use a Pruitt-Inahara shunt routinely in high-grade stenosis of ICA in order to avoid neurological damage due to clamping.

**Table 1:** Risk factors in CAPO

<table>
<thead>
<tr>
<th>Variable</th>
<th>No. of patients (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Coronary artery disease</td>
<td>12 (36)</td>
</tr>
<tr>
<td>Hypertension</td>
<td>30 (91)</td>
</tr>
<tr>
<td>Diabetes</td>
<td>10 (30)</td>
</tr>
<tr>
<td>Hypercholesterolaemia</td>
<td>21 (63)</td>
</tr>
<tr>
<td>previous smoker</td>
<td>24 (72)</td>
</tr>
<tr>
<td>Chronic obstructive pulmonary disease</td>
<td>3 (9)</td>
</tr>
<tr>
<td>ASA</td>
<td></td>
</tr>
<tr>
<td>II</td>
<td>20 (60)</td>
</tr>
<tr>
<td>III</td>
<td>13 (40)</td>
</tr>
</tbody>
</table>

**Figure 1:** Angio-CT scan showing segmental occlusion of ICA with reconstitution of filiform flow.

**Figure 2:** Insertion of the dilator in ICA after the endarterectomy.

**Figure 3:** Insertion of the Pruitt-Inahara shunt.
ischaemia, and in CAPO in order to perform a safer suture in the distal part of ICA and to avoid barotrauma after carotid unclamping. Shunt insertion in ICA before plaque removal may have inconveniences such as atheroembolism, intimal dissection and difficulty of endarterectomy; the cerebral parenchyma may tolerate a sufferance due to carotid clamping without neurological deficits for at least 7 min under general anaesthesia. This time is sufficient to perform the most difficult steps of carotid endarterectomy (plaque removal, distal intima checking), allowing shunt insertion in a clean operative field, without inconveniences [11].

On the day of surgery the patients fast, and are treated with electrolyte rehydration (1500 cc in 24 h), antibiotic therapy in order to prevent infections and ‘proton-pump inhibitors’. We also use glycerol (glycerol 10% and sodium chloride 0.9%, 500 cc in 24 h) that increases the plasma osmolality and as a result there is a change in the circulation of water from the extravascular spaces by osmosis; Oppido et al. [12] demonstrated a decrease of brain oedema with an improvement of cerebral compliance. There are no data reported about this therapy in carotid surgery but we use it not only in CAPO but also in high-grade stenosis of ICA with excellent results in preventing cerebral oedema. The follow-up was performed by duplex scan at discharge, 30 days, 6 months and on a yearly basis.

**RESULTS**

EEG monitoring showed no changes during carotid clamping in all patients.

No mortality or minor/major stroke was observed in the short-term and long-term postoperative period. Four patients presented with an early thrombosis of the ICA before discharge (3 after endarterectomy + patch and 1 after eversion endarterectomy), the ICA occlusion was diagnosed by duplex scan at discharge because all the patients were totally asymptomatic; we did not perform intraoperative or postoperative angiography.

We evaluated the flow in the distal portion of the vessel, by an intraoperative flow meter, and we found a very low flow (~3–5 ml/s) prior to endarterectomy and a significant increase to 100 ml/s after revascularization (Fig. 4); it would be interesting to evaluate routinely the intraoperative flow in ICA in order to correlate the increase of flow after endarterectomy with patent ICA during the follow-up.

Two patients presented with a hyperperfusion syndrome that regressed spontaneously in a few days. No restenosis was observed during a short-term and long-term follow-up. Five patients died of cardiac causes few years after surgery. The short-term and long-term follow-up is explained in Table 2.

![Figure 4: Increase of flow postendarterectomy.](https://academic.oup.com/icvts/article-abstract/20/5/636/644152)
DISCUSSION

In our experience in CAPO, the cerebral compensation from the contralateral carotid artery may be weak, and depends on the patient’s general condition; in the past, we have seen in our centre 2 patients with a major stroke 8 months and 1 year after CAPO was carried out, probably due to a hypotension that resulted in a cerebral hypoperfusion; the angio-CT scan showed a still patent distal ICA, in addition to pseudo-occlusion. These cases have changed our attitude towards a more aggressive treatment.

In this study, CAPO is a rare condition with an incidence of 2.3% similar to that of other published studies [3, 4].

The natural history of CAPO is not well known; it is a chronic disease that causes hypoplasia of the ICA but without complete occlusion. The time frame from the diagnosis, by angio-CT scan, to surgical intervention was ~2–3 months and in the meantime we did not find any carotid occlusion. It would be interesting to investigate if in this condition there would be some factors that stabilize the lesion, such as the absence of turbulence on the pseudo-occlusion level or the histopathological composition of the plaque [13].

Some authors said that in CAPO, there is a functional occlusion of the artery without a relevant contribution to the cerebral circulation, without an increased risk of stroke [14] and a benefit from surgical treatment [5, 15, 16]. Conversely, other studies showed that CAPO represents a risk factor for future stroke and the surgical treatment is necessary and effective [3, 9, 17, 18] with a stroke rate reduction from 11.1 to 6.7% per year [8].

Morgenstern et al. [8] reported a rate of ipsilateral stroke of 11.1% per year and Rothwell et al. [5] a risk of 8% every 5 years; Henderson et al. [16] reported a 10.9% risk of stroke every 2 years with collateral flow and 5.3% every 2 years without collateral flow. All these data are smaller than those of high-grade stenosis of ICA without the distal narrowing reported in NASCET.

However, Blaser et al. [19] reported a risk of stroke of up to 27% per month in patients with exhausted cerebrovascular reactivity associated with CAPO.

Carotid surgery on a very thin ICA, such as in CAPO, can be a very complex procedure and sometimes it may be necessary to tie the artery [4]. A strategy we use to facilitate the execution of the suture is, the insertion of the dilator in the ICA after plaque removal in order to enlarge the diameter of the vessel.

Moreover, a dilated ICA allows the insertion of the intraluminal shunt that creates gradual perfusion in the distal ICA during the carotid suture time in order to ‘prepare’ the ICA for unclamping. As a matter of fact, we have never observed EEG abnormalities during carotid clamping time.

Table 2: Follow-up

<table>
<thead>
<tr>
<th></th>
<th>Short-term (30 days)</th>
<th>Long-term (3 years)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mortality</td>
<td>0</td>
<td>5 (15%), cardiac cause</td>
</tr>
<tr>
<td>Minor/major stroke</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Thrombosis</td>
<td>4 (12%), asymptomatic</td>
<td>0</td>
</tr>
<tr>
<td>Restenosis</td>
<td>0</td>
<td>0</td>
</tr>
</tbody>
</table>

CONCLUSION

In our series, CAPO was a rare and chronic condition with poor symptoms probably because of functional occlusion of the ICA. An aggressive approach is correct to prevent brain damage in these patients with mild cerebral perfusion.

A risk of thrombosis of 12% is considerable but all these patients are completely asymptomatic; all the patients with CAPO had a functional occlusion of ICA and the patients with postoperative thrombosis have returned to the preoperative condition without new cerebral lesions. Instead, in 88% of patients, we ‘saved’ a carotid functionally occluded with an effective increase of cerebral perfusion.

In conclusion, surgical treatment is safe and effective even if a considerable risk of early thrombosis (12%) can be possible, without specific symptoms and a low risk of benign hyperperfusion syndrome.

Conflict of interest: none declared.

REFERENCES


