Carotid Stump Syndrome: Outcome from Surgical Management

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Objectives: in patients with occluded internal carotid arteries the carotid stump is a potential source of microemboli resulting in the persistence of retinal or cerebral ischaemic symptoms. We report 25 patients who had persistent cerebral and retinal ischaemic symptoms with an occluded ipsilateral ICA and a carotid stump who underwent surgical exclusion of the stump.

Methods: between January 1988 and January 1998, 332 patients underwent carotid endarterectomy. Twenty-five patients (20 males; five females; mean age 58.9 (range 44–78 years)) had carotid stump exclusion. Indications for surgery were transient ischaemic attack (22), amaurosis fugax (eight) and cerebrovascular accident (13). Three patients had undergone contralateral carotid endarterectomy and 12 had significant contralateral stenosis. Twenty patients were being treated with aspirin and four with warfarin at the time of presentation.

Results: the diagnosis of carotid stump was made in 22 patients by angiography. In the remaining three patients duplex alone was diagnostic in two patients. In the third case duplex was combined with magnetic resonance angiography (MRA) to confirm the diagnosis. Stump exclusion was carried out by oversewing the ICA origin. All but one patient remained symptom free at follow-up.

Conclusion: carotid stump syndrome should be considered as a likely clinical entity in patients with an occluded ICA and persisting cerebral and retinal microembolic symptoms. Surgical exclusion of the carotid stump is a safe and effective method of treatment.

Key Words: Carotid stump; External carotid artery; Collateral circulation.

Introduction

The overall risk of major stroke or death from ICA occlusion is 30% with an ipsilateral stroke rate of 3–5% per annum.¹ ² ³ Approximately two-thirds of strokes occur ipsilateral to the occluded ICA.² Transient ischaemic attacks (TIA) caused by embolisation from a carotid source should cease when a stenotic lesion in the ICA progresses to complete occlusion. However, a small number of patients with occluded internal carotid arteries continue to experience ocular or cerebral ischaemic symptoms of an embolic nature even when a cardiac source of embolism has been excluded.

Carotid stump syndrome is defined as the persistence of cerebral or retinal ischaemic symptoms after occlusion of an ipsilateral ICA. There is a demonstrable patent proximal remnant of the occluded ICA (carotid stump) in the presence of a patent external carotid artery (ECA) with reverse flow in its collateral circulation.⁴ ⁵ A previous study from this department described the 2-year outcome in a group of six patients with carotid stump syndrome.⁴ Four patients underwent surgery and remained asymptomatic at 2 years. Of two patients treated with aspirin, one remained asymptomatic and the other had a fatal cerebrovascular accident pre-operatively. This report describes the surgical management of a further 25 patients.

Patients and Methods

Patients with carotid stump syndrome who underwent surgical excision were identified from a computerised database. This database was cross-referenced with carotid duplex scan records, theatre log books and angiography records.

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Demographic data

Between January 1988 and January 1998, 332 patients underwent carotid surgery – 307 carotid endarterectomies and 25 carotid stump exclusions. There were 20 male and five female patients (mean age 58.9 years; range 44–78 years).

Presenting symptoms

All 25 patients were symptomatic with amaurosis fugax, transient ischaemic attacks or cerebrovascular accidents (Table 1). At the time of presentation 20 patients were taking aspirin and four patients were taking warfarin. Indications for warfarin included atrial fibrillation (two), arrhythmia with cardiac pacemaker (one) and a past history of pulmonary embolism (one). Three patients had previously undergone contralateral carotid endarterectomy.

Diagnosis

The entire extracranial cervical carotid arterial system was evaluated using a standard 5–8 MHz pulsed wave Doppler probe (Acuson Ltd). Flow reversal in the ophthalmic artery was assessed using a 10 MHz probe over the ipsilateral orbit or by assessing the ipsilateral supraorbital or supratrochlear arteries as an index to the direction of flow. Total occlusion of the ICA was shown in all patients, with the presence of a carotid stump being confirmed by the finding of reverse flow in the cul-de-sac of the occluded internal carotid artery (Fig. 1). Contralateral carotid artery stenosis was diagnosed in 12 patients (four had <50% stenosis; four had 50–79% stenosis and four had 80–99% stenosis.

Twenty-two patients underwent carotid angiography or digital subtraction angiography (DSA). One patient had arch aortography carried out. Nineteen of 22 carotid angiograms identified a carotid stump in the occluded ICA (Fig. 2). Magnetic resonance angiography (MRA) of the carotid arteries was performed in eight patients (Fig. 3). Indications for MRA included failed femoral catheterisation in three patients. In the remaining five cases MRA findings were being correlated with those of conventional angiography as part of an ongoing research study to validate MRA as a modality for evaluating the internal carotid artery. All but two patients had computerised tomographic brain scans. Thirteen infarcts ipsilateral to the carotid stump were diagnosed. Details of these findings are included in Table 1. One patient who presented with a stroke and a normal CT brain scan had a MRI brain scan which showed a cerebral infarct.

All patients had echocardiography and holter monitoring to rule out a cardiac source of emboli. Holter monitoring detected arrhythmias in two patient but neither was clinically significant.

Results

Surgical procedure

All procedures were carried out under general anaesthesia. Following exposure of the common, internal and external carotid arteries and systemic heparinisation, the common and external carotid arteries are clamped. A longitudinal arteriotomy extending from the CCA towards the ECA is performed. The ECA is endarterectomised and the internal carotid stump lumen obliterated with interrupted 6/0 prolene sutures (Fig. 4).

Post-operative complications

One patient developed a right facial paresis and dysarthria immediately post-operatively. CT brain scan showed a new ipsilateral internal capsular infarct. It is possible that this could have arisen during manipulation of the ECA causing embolisation through the ECA if it were a prominent contributor to the affected hemisphere. Evacuation of a wound haematoma was required in another patient. Two patients developed post-operative cranial nerve palsies. One patient died 6 weeks post-operatively following an unrelated surgical procedure.

Follow-up

Patients were discharged from hospital after a mean hospital stay of 8.7 days (range 3–19 days). The mean follow-up period was 11.9 months (range 6 weeks to 36 months). Twenty-three patients have remained asymptomatic since surgery. One patient suffered an ipsilateral deterioration in visual acuity with a retinal infarct 30 months post-operatively.

Discussion

An occasional finding following complete occlusion of the ICA is a patent proximal remnant of ICA seen on
Table 1. Details of 25 patients included.

<table>
<thead>
<tr>
<th>Patient no.</th>
<th>M/F</th>
<th>Age (years)</th>
<th>Medication</th>
<th>Symptoms</th>
<th>Frequency of symptoms</th>
<th>Most recent symptoms</th>
<th>CT scan findings</th>
<th>Outcome</th>
<th>Follow-up</th>
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<tr>
<td>1</td>
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<td>60</td>
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<td>Ipsilateral TIA/AF</td>
<td>2</td>
<td>2 months pre-op</td>
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<td>65 months</td>
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<td>Ipsilateral TIA/AF</td>
<td>2</td>
<td>1 month pre-op</td>
<td>No CT scan performed</td>
<td>No further symptoms</td>
<td>1 month</td>
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<td>Ipsilateral TIA</td>
<td>1</td>
<td>1 month pre-op</td>
<td>Atrophic changes</td>
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</tr>
<tr>
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<td>Immediately pre-op</td>
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<tr>
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<td>Aspirin</td>
<td>TIA/collapse</td>
<td>2</td>
<td>Immediately pre-op</td>
<td>Bilateral frontoparietal infarct</td>
<td>No further symptoms</td>
<td>3 months</td>
</tr>
<tr>
<td>6</td>
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<td>Aspirin</td>
<td>Visual loss left eye</td>
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</tr>
<tr>
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<td>Aspirin</td>
<td>Ipsilateral AF</td>
<td>2</td>
<td>2 months pre-op</td>
<td>Ipsilateral infarct</td>
<td>No further symptoms</td>
<td>12 months</td>
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<td>Ipsilateral AF/RIND</td>
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<td>4 months pre-op</td>
<td>Ipsilateral frontal infarct</td>
<td>RIND 8 months post-op</td>
<td>3 months</td>
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<tr>
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<td>64</td>
<td>Aspirin</td>
<td>TIA/collapse</td>
<td>3</td>
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<td>No CT scan performed</td>
<td>No further symptoms</td>
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<tr>
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<td>TIA/AF</td>
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<td>13 months pre-op</td>
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<td>2</td>
<td>Immediately pre-op</td>
<td>Normal</td>
<td>No further symptoms</td>
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<td>Normal</td>
<td>Recurrent TIA</td>
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<td>AF/CVA</td>
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<td>Normal</td>
<td>No further symptoms</td>
<td>3 months</td>
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<td>TIA/AF</td>
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<td>Normal</td>
<td>No further symptoms</td>
<td>6 months</td>
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<td>Ipsilateral CVA</td>
<td>3</td>
<td>Immediately pre-op</td>
<td>Ipsilateral infarct</td>
<td>No further symptoms</td>
<td>3 months</td>
</tr>
<tr>
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<td>Ipsilateral CVA</td>
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<td>Normal</td>
<td>No further symptoms</td>
<td>1 month</td>
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<td>Ipsilateral</td>
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<td>Ipsilateral infarct</td>
<td>No further symptoms</td>
<td>28 months</td>
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<tr>
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<td>Ipsilateral CVA</td>
<td>1</td>
<td>1 month pre-op</td>
<td>Ipsilateral infarct</td>
<td>No further symptoms</td>
<td>5 months</td>
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<tr>
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<td>M</td>
<td>61</td>
<td>Aspirin</td>
<td>Ipsilateral CVA</td>
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<td>2 months pre-op</td>
<td>Ipsilateral infarct</td>
<td>No further symptoms</td>
<td>46 months</td>
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<tr>
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<td>54</td>
<td>Warfarin</td>
<td>TIA/CVA/ Multiple</td>
<td>1</td>
<td>1 year pre-op</td>
<td>Ipsilateral infarct</td>
<td>No further symptoms</td>
<td>3 months</td>
</tr>
<tr>
<td>23</td>
<td>F</td>
<td>65</td>
<td>Aspirin</td>
<td>TIA/CVA</td>
<td>1</td>
<td>2 months pre-op</td>
<td>Ipsilateral infarct</td>
<td>No further symptoms</td>
<td>6 months</td>
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<tr>
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<td>Aspirin</td>
<td>Ipsilateral</td>
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<td>11 months pre-op</td>
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<td>Wound haematoma</td>
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<tr>
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<td>F</td>
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<td>Aspirin</td>
<td>CVA</td>
<td>1</td>
<td>2 months pre-op</td>
<td>Ipsilateral infarct</td>
<td>No further symptoms</td>
<td>6 months</td>
</tr>
</tbody>
</table>

TIA = Transient ischaemic attack; AF = Amaurosis Fugax; CVA = Cerebrovascular Accident; RIND = Reversible Ischaemic Neurological Deficit.
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Fig. 1. Colour-coded Doppler flow study showing a vortex of blue reversed flow in an internal carotid artery stump.

Fig. 2. Digital subtraction angiogram demonstrating patent common and external carotid arteries. The blind-ending cul-de-sac at the origin of the internal carotid artery is the carotid stump.

carotid duplex scan, carotid angiography or MRA.\textsuperscript{1,2,4,5} This region of the ICA is referred to as the “stump” where turbulent flow causes platelet-fibrin aggregation theoretically leading to microembolisation.

The persistence of cerebral or retinal ischaemic symptoms after occlusion of an ipsilateral internal carotid artery has been attributed to both haemodynamic and embolic factors.\textsuperscript{1,2,8} The haemodynamic

Fig. 3. Magnetic resonance angiogram showing a normal left sided extra-cranial carotid circulation. A carotid stump is clearly seen on the right-hand side with patent common and external carotid arteries.
Fig. 4. Illustration of the steps involved in the surgical procedure to exclude the carotid stump. (1) The common, internal and external carotid arteries are isolated in the standard fashion through a longitudinal neck incision; (2) a longitudinal arteriotomy extending from the common towards the external carotid artery is performed; (3) the external carotid artery is endarterectomised and the internal carotid artery stump obliterated with interrupted 6/0 prolene sutures; (4) the arteriotomy is closed in standard fashion using 5/0 and 6/0 prolene.

mechanism proposes that cerebral hypoperfusion secondary to ICA occlusion may lead to cerebroretinal ischaemic symptoms. Altered cardiac output secondary to changes in cardiac rhythm and postural changes have also been implicated. Several mechanisms have been proposed to explain continuing embolic symptoms in the presence of an occluded ipsilateral ICA. Documented sources of microemboli from the extracranial vascular system include: the ipsilateral CCA or ECA; the carotid bifurcation via a patent ECA, the contralateral carotid vessels via the circle of Willis,7 a patent proximal remnant of the occluded ICA or distal thrombus occluding the ipsilateral ICA. Isolated cases of emboli arising from the distal tail of the thrombus in the occluded ICA have been reported.8–11 This should be considered in cases with occlusion of both the ipsilateral ICA and ECA and where recurrence of ischaemic symptoms following exclusion of the proximal stump occurs.

Intermittent microembolisation from the carotid stump was the most probable cause of persistent ischaemic symptoms in these 25 patients because thrombotic material was found in the base of the stump of the occluded ICA in a number of patients and stump exclusion resulted in cessation of symptoms in all patients.

In the presence of significant contralateral disease embolisation from the contralateral carotid via the anterior communicating artery is possible. Endarterectomy of the contralateral carotid stenosis has been recommended.12,13 This procedure, however, often fails to relieve symptoms because the symptomatic hemisphere does not receive significant collateral blood supply from the contralateral carotid artery.14–16
patients in this study had significant (80–99%) stenosis of the contralateral ICA. Cross-circulation was not demonstrated on carotid angiography. Two patients had contralateral internal carotid endarterectomy 8 days to 1 month before ipsilateral stump exclusion. Both patients continued to have TIAs following the endarterectomy and subsequent stump exclusion prevented further symptoms. The third patient had no further symptoms following stump exclusion and proceeded to have a contralateral internal carotid endarterectomy 1 month after stump exclusion.

If a symptomatic internal carotid artery is accompanied by obstructive lesions in the ipsilateral external carotid artery, as demonstrated by duplex or angiography, external carotid endarterectomy may prove beneficial because the ipsilateral external carotid artery is the major source of blood supply to the symptomatic hemisphere. In the current series four patients had moderate (50–79%) stenosis of the ipsilateral external carotid artery, one patient had severe (80–99%) stenosis of the ipsilateral external carotid artery and a further two patients had mild (16–49%) stenosis of both the ipsilateral external and common carotid arteries. These seven patients had endarterectomy of these atheromatous lesions before carotid stump exclusion.

CT brain scan did not alter the decision to offer or withhold surgery in any of these patients. Six patients had normal CT scans but their continuing TIAs while on antiplatelet or anticoagulant therapy was an indication for angiography.

We describe a little reported syndrome which was managed with minimal morbidity. Carotid stump exclusion poses fewer technical difficulties than carotid endarterectomy. The risk of peroperative embolic stroke should be much smaller. However, theoretically hypoperfusion during the period of ECA clamping could predispose to an ischaemic stroke if that cerebral hemisphere is entirely dependent on the ECA for perfusion. The surgical technique described involves exclusion of the lumen of the ICA via the ECA and requires minimal dissection of the ICA. An alternative approach would be to disconnect the ICA entirely at the bifurcation thereby removing the carotid stump.

It must be borne in mind that one-third of patients who develop ICA occlusion will die or have disabling symptoms. Therefore, a knowledge of the timing between ICA occlusion and the onset of cerebral or retinal embolic symptoms is important to the definitive diagnosis of the carotid stump syndrome. Given the limitations of a retrospective study, it was not possible to ascertain in this report the presence of asymptomatic ipsilateral carotid occlusion before the onset of symptoms. However, the occurrence of symptoms in many of the patients on more than one occasion supports our theory of an embolic phenomenon. Carotid stump syndrome should therefore be considered as a likely clinical entity in patients with an occluded ICA and persisting ipsilateral cerebral and retinal microembolic symptoms.

References


Accepted 9 December 2000