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CASE REPORT

Immediate Catheter Directed Thrombolysis for Thromboembolic Stroke During Carotid Endarterectomy

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Background: Carotid artery endarterectomy (CEA) is a common procedure undertaken by vascular surgeons with over 5,000 procedures performed annually worldwide. Published rates of perioperative stroke range from 1.3% to 6.3%.

Case report: A case is presented in which on-table intra-cranial angiography and catheter directed thrombolysis were used for a thromboembolic occlusion of the distal internal carotid artery (ICA) and proximal middle cerebral artery (MCA). An 83-year-old lady developed a dense right hemiparesis while undergoing a CEA under local anaesthetic (LA). Immediate re-exploration of the endarterectomy did not reveal technical error. Intraoperative duplex scanning of the internal carotid artery revealed no detectable diastolic flow. On-table angiogram showed complete occlusion of the distal ICA and proximal MCA. Catheter directed administration of TPA was undertaken. The entire ICA and MCA were completely clear on a completion angiogram. The patient made a full neurological recovery.

Discussion and conclusion: Prompt diagnosis and treatment with intraoperative catheter directed thrombolysis can resolve thromboembolic occlusion of the ICA/MCA. It is argued that performing CEA under LA is useful for immediate recognition of perioperative stroke. Furthermore, the advantage is highlighted of vascular surgeons having both the resources and skillset to perform on-table angiography and thrombolysis.

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INTRODUCTION

There is strong evidence to support carotid artery endarterectomy (CEA) for the prevention of stroke in patients with symptomatic or moderate to high grade carotid stenosis.[1] Unfortunately, a well-recognised complication of CEA is the very disaster the operation sets out to prevent: stroke. Reported rates range from 1.3% to 6.3%, depending on the degree of carotid stenosis, patient selection, and operative technique.[2] The cause of intraoperative stroke is multifactorial, with the most common cause being perioperative thromboembolism. The presence of a neurological deficit in the intraoperative or immediate postoperative period, with a suspicion of thromboembolism, is an indication for immediate re-exploration.[3] This traditionally involves re-exploring the endarterectomy site with the potential to proceed to thrombectomy. However, this method does not address the possibility of a more distal arterial occlusion, either within the distal cervical or intracranial vessels. A case is presented in which a patient developed a dense right hemiparesis while undergoing an elective left CEA. Management with on-table angiography and catheter directed thrombolytic therapy is described.

CASE REPORT

An 83-year-old woman (164 cm/74.5 kg) was admitted for a left CEA with a history of a transient ischaemic attack (TIA) affecting the patient's speech and right arm two weeks prior to surgery. Carotid duplex scanning demonstrated dense calcified atheroma at the origin of the left internal carotid artery (ICA) with high grade stenosis (>70%). CT angiogram of the carotids confirmed this, and also demonstrated severe stenosis of the right vertebral artery. Pre-existing medical conditions included hypertension, hyperlipidaemia, impaired glucose tolerance, and obesity. Preoperative antiplatelet medication consisted of clopidogrel 75 mg once daily.

The carotid endarterectomy and patch graft closure was performed under local anaesthetic (LA). LA was administered by the consultant surgeon under ultrasound guidance for both a superficial block (20 mL 1% lidocaine with adrenaline) and a deep cervical block (20 mL 0.5% chirochaine).

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Heparin (5,000 units) was administered prior to clamping the carotid artery. The patient became unresponsive after clamping the common carotid, 1 hour and 5 minutes from the initiation of surgery. An arteriotomy and shunt insertion were performed, improving the patient's condition to baseline, which, combined with flushing of the stent, confirmed the stent patency. Systolic blood pressure was maintained between 115 and 130 mmHg throughout the procedure. The operation continued as usual and endarterectomy and patch graft closure (Dacron) were performed.

Shortly after completion of the patch and 2 hours and 5 minutes into the surgery, the patient developed a dense right hemiparesis and dysphasia before becoming unresponsive with seizures. The arteriotomy was re-opened and extended. No arterial dissection flap or clot was found, although feeble backflow bleeding was noted. Intraoperative duplex revealed no detectable diastolic flow in the ICA. The arteriotomy was closed and an on-table cerebral angiogram demonstrated complete occlusion of the left distal ICA and proximal middle cerebral artery (MCA) (Fig. 1A). During the above events, which prolonged the surgical time to over 2 hours, the patient was given a further 3,000 units of heparin.

A 3 Fr vertebral catheter was inserted over a 0.018 Terumo guidewire into the proximal ICA under fluoroscopic guidance. A total of 35 mg of recombinant tissue plasminogen activator (rt-PA) was administered by the vascular surgeon via the catheter in 5 mg boluses every 10 minutes with repeated angiograms (Fig. 1B). The patient was assessed by the anaesthetist, who confirmed that the neurological status had returned to baseline (the patient remained under LA), and a completion angiogram demonstrated a fully patent ICA and MCA (Fig. 1C,D). The final operative time was 4 hours and 10 minutes.

The patient was transferred to the high dependency unit (HDU) post operatively. A computed tomography (CT) scan of the brain the following day demonstrated no newly ischemic regions. The patient made a full neurological recovery, determined by a consultant neurologist, and was discharged 1 week following surgery.

DISCUSSION

Common postoperative causes of stroke include thromboembolic event and hyperperfusion leading to cerebral haemorrhage.[4] Intraoperative neurological deficit usually results from ischaemia after cross-clamping of the carotid, and is exacerbated by poor collateral vessels. Thromboembolic events can also occur intraoperatively, especially during manipulation of surrounding structures or during insertion of shunts. Determining the cause of stroke will dictate further management. The following discussion

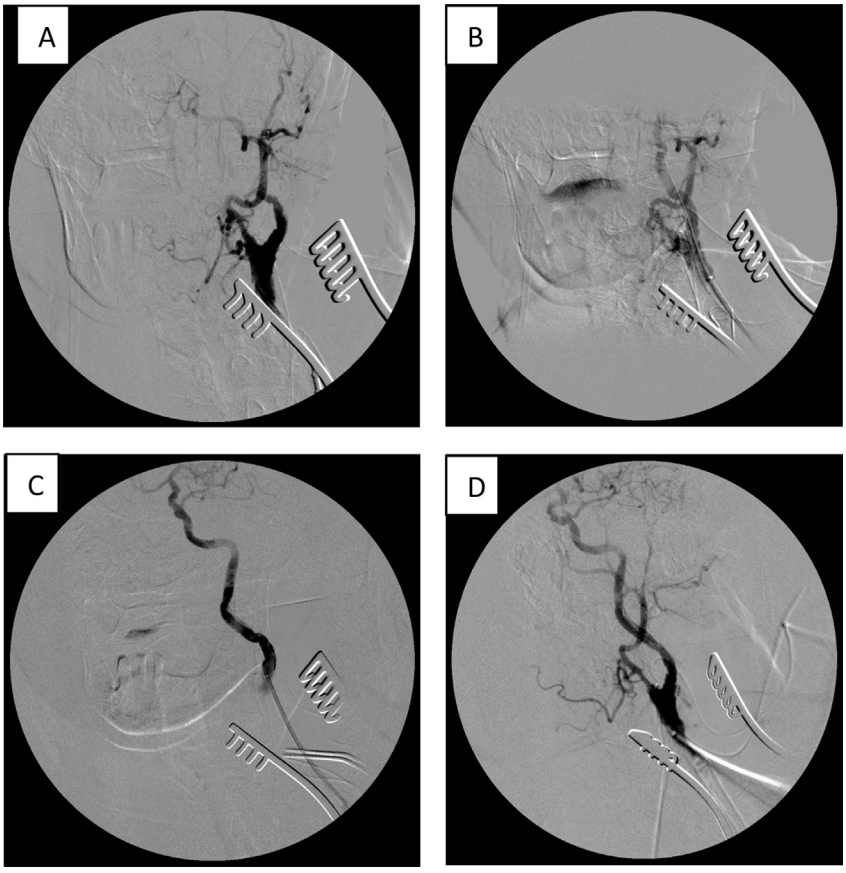

Figure 1. Sequential on-table intraoperative angiograms. (A) Initial angiogram demonstrating occlusion of distal ICA. (B) Mid-procedural angiogram after commencing treatment with rt-PA. (C) Isolated ICA angiogram at end of treatment showing newly patent ICA. (D) Completion angiogram at end of treatment.

focuses on management of thromboembolic stroke during, or immediately following CEA.

Rapid recognition and thus management of perioperative stroke improves outcome.[5] It is argued that performing CEA under LA is advantageous: it allows for effective neurological assessment in a conscious patient and is easy to administer. Alternative methods in the anaesthetised patient include near-infrared spectroscopy (NIRS), somatosensory evoked potentials (SSEP), electroencephalography (EEG), carotid artery stump pressures, and transcranial doppler (TCD). These have varying advantages and disadvantages,[6] but none is superior to LA when considering immediate recognition of perioperative stroke. However, it is worth noting that some authors advocate the use of these monitoring methods even during LA, especially EEG and TCD, as they can help elucidate the underlying pathological mechanism of the stroke and therefore guide immediate management.[7]

Although there has never been published guidance, the common historical approach to perioperative thromboembolic stroke in CEA has been re-exploration and thrombectomy.[3] The focus has been on correction of technical error and removal of thrombus either at, or just distal to the surgical site. The possibility of a more distal occlusion was not usually addressed and neurological recovery was poor. It is worth noting that this management option refers to immediate thromboembolic stroke specifically, rather than perioperative stroke of alternative aetiology. In contrast, there are now a number of imaging modalities that can be used for the investigation of perioperative stroke, such as angioscopy, angiography, intravascular ultrasound (IVUS), or intraoperative duplex scanning. The authors prefer the last of these as it provides immediate evidence of intracerebral occlusion in the absence of diastolic flow, and further information can then be provided by on-table angiography. This method provides accurate information regarding the position of the thrombus and avoids the inevitable delays associated with CT angiography.

The use of thrombolytic therapy for intra-arterial occlusive disease has become well established over the past 30 years, primarily for lower limb arterial occlusions. The use of systemic thrombolytic agents for stroke is associated with relatively high rates of cerebral haemorrhage. Selective intra-arterial infusions improve haemorrhage rates and this are now a recognised management option for patients presenting with acute cerebral infarction. It is particularly effective if performed rapidly after the onset of symptoms. Logic therefore dictated that thromboembolic strokes after or during CEA should respond well to selective intra-arterial thrombolytic therapy. Despite this, evidence and experience of intraoperative thrombolysis for cerebral ischaemia following CEA remains scant. The first case of such management was described in 1995,[8] with various anecdotal case reports and a single case series since, demonstrating successful outcomes.

Despite the apparent success of this technique, it is clear that it carries a risk of thrombolysis-induced cerebral haemorrhage. Literature from non-iatrogenic thromboembolic stroke treated with thrombolysis suggests a cerebral haemorrhage rate of 4—11%.[9] Published literature indicates an increased risk of cerebral haemorrhage when thrombolysis is initiated more than 6 hours after the onset of cerebral ischaemia.[10] In the present case, therapeutic intervention was completed within 90 minutes. Recombinant tissue plasminogen activator (rt-PA) was used because of its short half-life and low immunogenic potential, and high dependency postoperative care is considered to be essential for early recognition of complications when it is used. There is no documented upper limit with regards to the volume of rt-PA that can be administered, although, in practice, the present authors would not exceed 50 mg because of the risk of causing haemorrhage.

The ability to perform immediate intraoperative angiography was central to providing immediate management. This was enabled in part by the availability of a hybrid operating theatre, which the authors advocate for this procedure. Clearly, for vascular centres without access to interventional neuroradiologists, vascular surgeons need to be either adequately skilled and equipped, or have the infrastructure in place for transfer of these patients to a tertiary centre.

CONCLUSION

This case demonstrated that intraoperative catheter directed thrombolysis can successfully treat post-CEA thromboembolic occlusion of the ICA/MCA with complete neurological recovery. Central to its success are prompt diagnosis, and the technical expertise and facilities to perform on-table angiography and intraoperative duplex scanning.

CONFLICT OF INTEREST

None.

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