CASE REPORT

Transient Blindness after Endovascular Parent Artery Occlusion to Treat Giant Aneurysm of Internal Carotid Artery: a Case Report

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INTRODUCTION

Giant aneurysm of the cerebral circulation is an arterial outpouching ≥ 25 mm. Cavernous internal carotid artery (ICA) aneurysms account for approximately 15% of all aneurysms arising from the ICA.¹ Owing to their large size, they may present with symptoms due to compression of adjoining nerves but are less likely to rupture.²

We present a patient who underwent endovascular proximal parent artery occlusion for management of a giant ICA aneurysm. Aneurysmal thrombosis, which is an expected outcome, caused an unexpected complication of delayed-onset blindness. We describe the mechanism of complication, and its management and follow-up. Use of steroid as a preventive and therapeutic option in similar cases is proposed.

CASE REPORT

A 45-year-old woman with no previous co-morbidities presented with a 2-month history of headache and vomiting. Her headaches were dull and aching, and centred towards the left hemicranium. The intermittent episodes of vomiting were non-projectile. She had no history of loss of consciousness, seizures or other motor deficits. Non-contrast computed tomography (CT) scan and magnetic resonance imaging performed prior to her visit to our institution had revealed a giant aneurysm involving the cavernous segment of the left ICA.

General examination was unremarkable. Power was 5/5 in bilateral upper and lower limbs. Cranial nerve examination detected visual acuity of 6/12 bilaterally. Perimetry evaluation revealed deficit in the temporal field of the left eye.

Digital subtraction cerebral angiography revealed a giant saccular aneurysm involving the cavernous segment of the left ICA measuring 30 mm \times 35 mm \times 30 mm (Figure 1). The ICA distal to the aneurysm was faintly filling. Left cerebral perfusion was maintained from the right ICA across the anterior communicating artery. The ophthalmic artery on the left side was not opacified.

The patient gave consent. Balloon occlusion test was performed using a 7-Fr Swan-Ganz balloon. Occlusion was applied for 10 minutes at baseline blood pressure and 10 minutes of hypotensive challenge was given, reducing the mean blood pressure by 20 mm Hg. Adequacy of

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Ethics Approval: Ethical committee approval was not required for this case report as standard care was provided for the clinical scenario. The patient was treated in accordance with the tenets of the Declaration of Helsinki. The patient provided written informed consent for all treatments and procedures.

Transient Blindness after ICA Occlusion

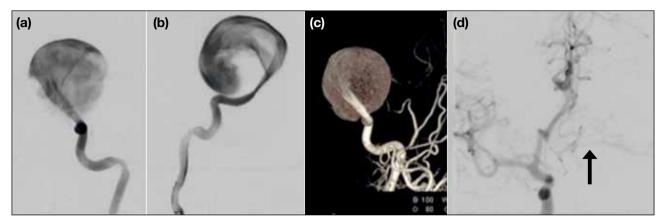


Figure 1. (a) Anteroposterior and (b) lateral views of left carotid angiogram showing giant aneurysm filling from left internal carotid artery (ICA) with contrast whirling. (c) Three-dimensional reconstruction of the left ICA angiogram showing giant aneurysm. (d) Right ICA angiogram showing cross-flow to left ICA (black arrow).

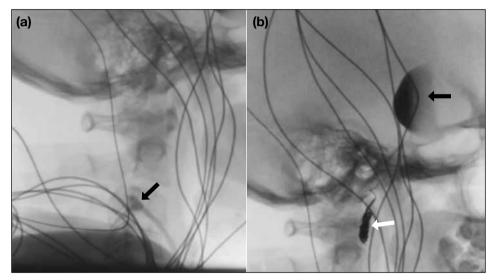


Figure 2. (a) Single spot fluoroscopic image showing compliant Swan-Ganz balloon is placed in the internal cerebral artery (black arrow). (b) Single spot fluoroscopic image after coil embolisation (white arrow) showing stagnation of contrast in the aneurysm (black arrow). Wires noted in both images are that of leads to monitor somatosensory evoked potential.

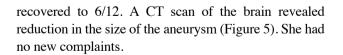
collaterals was evaluated by periodic clinical testing approximately every 2 minutes. Somatosensory evoked potential monitoring was done. The venous delay in the contralateral ICA angiogram was <2 seconds. After successful completion of the test, a 2.7-Fr Progreat microcatheter (Terumo) was passed along the side of the balloon and then pushable coils (0.018-inch) were used to tightly pack a short segment of petrous ICA maintaining the balloon inflated in the cervical ICA (Figure 2). Her immediate postoperative recovery was uneventful and she was discharged home on the fourth day.

One-week post-procedure she presented to the emergency department with rapidly worsening vision in the left eye. Objective acuity testing revealed only perception of light in the left eye. On examination, the left pupil was larger with sluggish reaction to light. Contrast enhanced CT scan revealed increased thrombosis within the aneurysm causing a marginal increase in diameter of 5 mm (Figure 3). Magnetic resonance imaging showed that because of its close proximity, the aneurysm was compressing the optic chiasma (Figure 4). It was assumed that this increase in volume was due to thrombus formation with consequent compression of the adjoining left optic nerve. The patient was immediately started on oral steroids (dexamethasone 4 mg every 6 hours). After 1 week she showed minimal improvement in visual acuity and was discharged from the hospital on tapering dose of steroids. At a follow-up examination 4 months after surgery, the patient's visual acuity had

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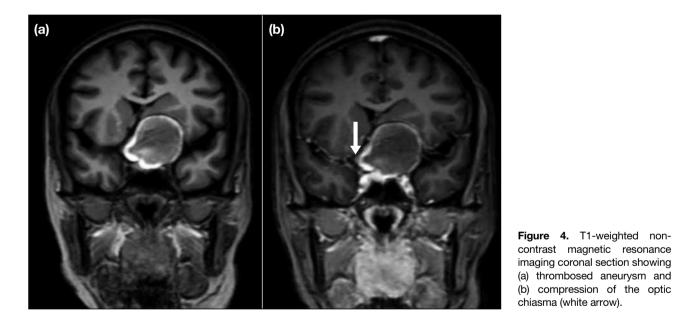
Figure 3. Axial plain computed tomography scans on (a) day 4 and (b) day 7 after surgery showing marginal increase in dimension of the aneurysm



DISCUSSION

Owing to their interdural location, giant cavernous ICA aneurysms are less likely to cause life-threatening subarachnoid haemorrhage, unlike aneurysms in an intradural location (distal to the cavernous segment).

Treatment of cavernous segment aneurysms is usually restricted to symptomatic patients who present with chronic headaches or compressive cranial nerve palsies around the cavernous sinuses. Rarely, they may rupture into the cavernous sinus leading to formation of type I carotid-cavernous fistula.3 Spontaneous intra-aneurysmal eccentric thrombus sometimes can be seen and maybe a source of embolism causing transient ischaemic attacks or strokes.4



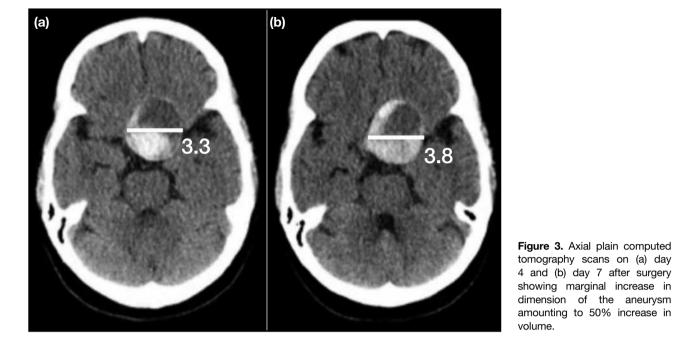




Figure 5. Axial computed tomography scan at 4 months after surgery showing significant reduction in aneurysm size.

Two options were considered for the treatment of aneurysm in this patient: parent artery occlusion following balloon occlusion test or flow diversion. Flow diversion involves placement of a braided stent across the aneurysm neck in the parent artery to regulate the flow into the aneurysm, thus causing gradual thrombosis.⁵ Flow diversion comes with a significant additional cost and requires the patient to take anti-aggregatory medications for a prolonged period.⁶

Parent artery occlusion of the vessel harbouring the giant aneurysm is a less expensive but effective way noted in early work by Drake et al.⁷ Occlusion of the parent artery alters the flow dynamics within the aneurysm causing it to thrombose. Balloon occlusion test is a technique used to temporarily occlude the parent artery using a compliant balloon intended for permanent sacrifice. This testing provides valuable information about the efficiency of alternative circulatory pathways to maintain the brain supply.

Expansion of a blood vessel following acute thrombus formation is a known phenomenon. Evidence of this may be more prominent in veins owing to their non-muscular walls. Such examples are obvious in cases of peripheral deep venous thrombosis or renal vein thrombosis where increase in size of the vein is a common finding. Similar changes may occur in arteries leading to a marginal increase in size. This size increase is due to clot formation that involves cytokines and clotting factors and expands the volume. The size increase sometimes results in compression on adjoining vital structures. Steroids help to reduce this response of inflammation during clot formation. They also play a role in reducing inflammation of the compressed structure, which was the left optic nerve in this case.⁸

A quick volumetric analysis with comparison on CT was useful in our case. The marginal increase in diameter caused an approximate 50% increase in overall volume. This hypothesis correlates with the onset of visual symptoms 1 week after the procedure. Improvement was seen with steroid treatment with gradual recovery over a few months.

CONCLUSION

Giant ICA aneurysm causing compressive symptoms warrants treatment. Endovascular options include parent artery occlusion or flow diversion. Parent artery occlusion may cause thrombosis within the aneurysm causing expansion and more compression. In our patient, this presented as delayed loss of vision, subsequently managed by steroids. Steroids may be useful in such cases and may also be considered as prophylaxis in similar cases.

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