Case Report

Giant cavernous carotid aneurysm with spontaneous ipsilateral ICA occlusion: Report of 2 cases and review of literature

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ABSTRACT

Giant aneurysms of the cavernous carotid artery are rare entities which present predominantly with features of compression of the adjacent neural structures, most commonly the III, IV, VI and V cranial nerves. Historically, treatment options included occlusion of the feeding vessel, direct surgery on the aneurysm, bypass procedures and in recent times, the use of endovascular devices. While intramural thrombus formation is commonly seen in giant aneurysms, we present 2 cases of giant cavernous aneurysms which on evaluation were found to have spontaneous occlusion of the feeding internal carotid artery secondary to thrombus formation, and review the available literature regarding the same.

Key words: Balloon test occlusion, cavernous carotid artery, giant aneurysm, spontaneous occlusion

Introduction

Aneurysms of the cavernous carotid artery account for between 3-5% of all intracranial aneurysms.[1] Since they are extradural in location, they have a very small risk of bleeding. However, they can grow large in size, and can present with pressure symptoms due to mass effect on cranial nerves, such as ophthalmoplegia and facial pain.[2] Rarely there may also be involvement of the optic nerve and subsequent loss of vision.[3] Spontaneous intramural thrombosis of giant intracranial aneurysms occurs in between 13-20% of cases.[4] However, very few of these cases are also associated with ipsilateral carotid occlusion. We report 2 patients with spontaneous thrombosis of cavernous segment aneurysm, along with ipsilateral ICA occlusion.

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Case Reports

Case 1

A 65-year-old lady presented with 3-month history of facial numbness and occasional pain involving the left half of the forehead and left cheek and left sided ptosis. The symptoms were insidious in onset and not associated with any history suggestive of a subarachnoid hemorrhage. Clinical examination revealed complete ophthalmoplegia and hypoesthesia in the ophthalmic and maxillary divisions of the left trigeminal nerve.

She was evaluated with an MRI [Figure 1a and b] which showed a giant partially thrombosed aneurysm in the left ICA cavernous segment. She was planned for a DSA with possible balloon test occlusion. DSA revealed complete occlusion of left ICA, without any filling of the aneurysm [Figure 1c and d]. There was excellent cross-flow through anterior communicating artery, which was supplying the entire left hemisphere. There was no filling of the aneurysm through the right ICA or vertebral artery injection. MRA [Figure 2] revealed lack of flow through the left ICA and the cavernous aneurysm.

The patient was managed conservatively and was put on aspirin to prevent further progression of the thrombus. At 6 month follow-up, the patient was doing well, her

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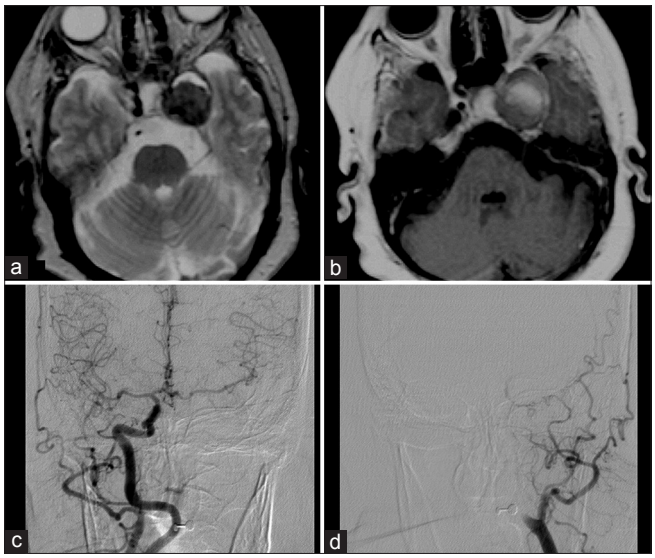

Figure 1: (a and b) shows a giant partially thrombosed aneurysm in the left ICA cavernous segment. DSA (c and d) shows complete occlusion of left ICA, without any filling of the aneurysm with cross-flow through anterior communicating artery

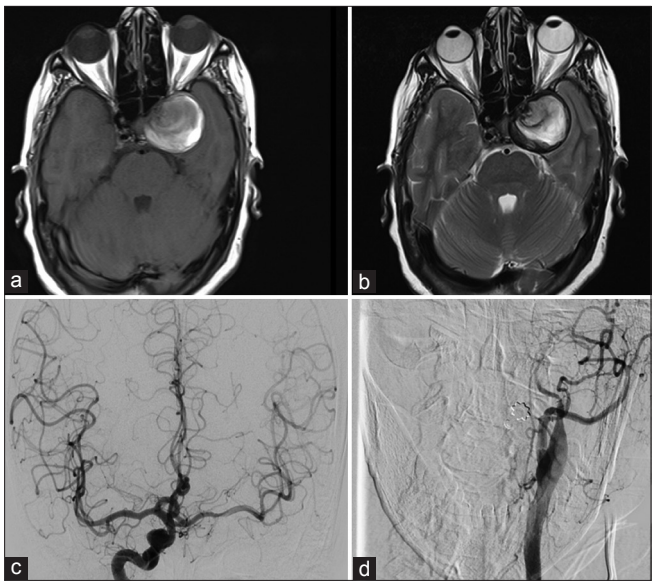

Figure 3: (a and b) shows a 2.8 × 2.6 × 2.1 cm lesion in the left cavernous sinus suggestive of giant ICA aneurysm. DSA (c and d) shows occlusion of the proximal ICA on the left side without any filling of the cavernous segment aneurysm with cross flow across the anterior communicating artery, with a small aneurysm in the right cavernous ICA

ophthalmoplegia had completely improved however, the pain and numbness over the left half of the face persisted.

Case 2

The second patient was a 55-year-old lady who presented with 2 episodes of generalized tonic clonic seizures 3 months prior to admission. She also developed visual deterioration in left eye, left ophthalmoparesis and left facial pain. Visual acuity in the left eye was perception of hand movements close to her face. She had paresis involving the left VI nerve, left relative afferent papillary defect and mild sensory loss in the left ophthalmic nerve distribution.

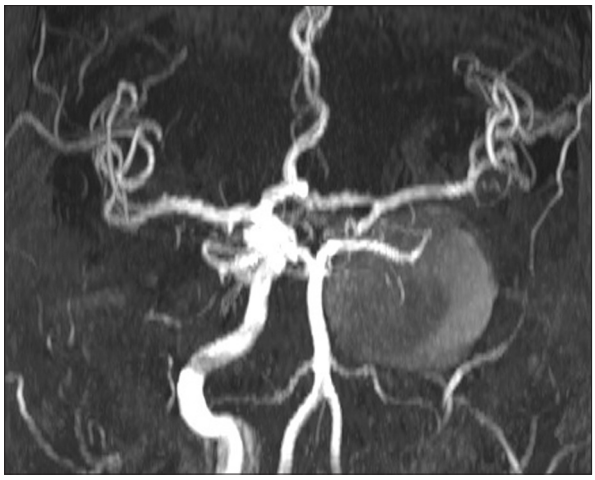

Figure 2: MR Angiogram showing absent flow in left ICA with a giant cavernous ICA Aneurysm

MRI brain showed a 2.8 × 2.6 × 2.1 cm lesion in the cavernous sinus suggestive of giant ICA aneurysm [Figure 3a and b]. DSA [Figure 3c and d] revealed occlusion of the proximal ICA on the left side without any filling of the cavernous segment aneur ysm. On the right ICA injection, there was excellent cross flow across the anterior communicating artery, with a small aneurysm in the contralateral cavernous ICA.

This patient was also put on aspirin for stroke prevention. At 3-month follow-up, her visual acuity remained the same however, her ophthalmoparesis improved.

Discussion

Giant aneurysms of the cavernous carotid artery with proximal occlusion of ICA are rarely reported in literature with few scattered case reports and a single series of 5 patients. Whittle, *et al.*[4] first reported a patient presenting with ophthalmoplegia and proptosis and unilateral headache. On imaging, he had ipsilateral ICA occlusion with thrombosed cavernous aneurysm. They treated their patient with open surgery and removal of intramural clot via an aneurysmotomy. While the headache and proptosis subsided there was no change in the ophthalmoplegia.

Since then a total of fifteen such cases have been reported in literature apart from the two cases outlined above. A brief summary of previously reported cases in given in the table below [Table 1].

Table 1: Reports of spontaneous occlusion of proximal ICA in cavernous ICA aneurysms

Author, Year	Age, Sex	Presentation	MRI/CT	DSA	Rx	Follow Up Findings, Additional
Mikabe *et al.*, 1980[1]	62, F	HA, III, IV	L MCF Mass	Occlusion of ICA	Sx	Improved
Whittle *et al.*, 1982[4]	27, M	HA, Prop, III, IV, VI	CT-R MCF Mass	Giant R CCA aneurysm with proximal occlusion	Sx	Asymptomatic at 3m
Gautier, 1986[5]	65, F	R Hemi, L III, IV, VI	L MCF Mass	L - ICA occl, Right 2 aneurysms	-	
Sato *et al.*, 1990[6]	49, M	HA/V, III, VI, V1	CT-L MCF Mass	Giant L CCA aneurysm with proximal occlusion	Cons	Improved
	21, M	II, L weakness, dysarthria	CT-R MCF Mass	Giant R CCA aneurysm with proximal occlusion	Cons	5 years – persistent ophthalmoplegia
Kurokawa *et al.*, 2001[7]	60, F	HA, VI	Giant R CCA aneurysm	R Giant CCA, L CCA, MCA, DACA	R EC-IC	BTO intolerant, at 1yr – DSA - complete occlusion
	50, F	V1	Giant R CCA aneurysm	Giant R CCA aneurysm with proximal occlusion	Correction of Sodium	BTO intolerant, 6m - Asymptomatic
Tsutsumi *et al.*, 2002[8]	75, M	R II, III, IV, VI, V1, V2	Giant R CCA aneurysm	Giant R CCA aneurysm with proximal occlusion	Nil	R circulation filling via ACoA
Dehdashti *et al.*, 2003[9]	31, F	Diplopia and HA	Giant R CCA aneurysm	-		18m – persistent III paresis MRI – dissection and occlusion of ICA, 24m - Asymptomatic
Perrini *et al.*, 2005[10]	47, M	Cav Sinus Syn	Giant R CCA aneurysm	Giant R CCA aneurysm with sluggish flow in ICA	Antiplatelet	6 weeks – extension of thrombus into cervical ICA. 6m - asymptomatic
Vasconcellos *et al.*, 2009[11]	47, F	HA, III, IV, VI, V1,2 R	-	Giant R CCA aneurysm with proximal block	Conservative	6m – DSA complete occlusion
	44, F	L HA, III, IV, VI, V1,2	-	Giant L CCA aneurysm with proximal occlusion		3m – DSA complete occlusion L Filling via Pcomm->MCA
	65, F	R HA dip, III, IV, VI	Giant R CCA aneurysm	R ICA with partial block		5y -> occl
	19, M	L HA, III	Giant L CCA aneurysm	L ICA with occl after 10 days		10 d -> occl, ptosis resolved
	84, F	L HA III, IV, V1,2	Giant L CCA aneurysm	L ICA with occl		3m - partial ptosis
Present study	65, F	III, V1,2	Giant L CCA aneurysm	L ICA with occl	Conservative	6m – ophthalmoparesis improved, residual pain
	55, F	GTCS, II, VI, V1	Giant L CCA aneurysm	L ICA with occl	Conservative	3m-visual acuity same, ophthalmoparesis improved

ICA - Internal carotid artery, DSA - Digital subtraction angiogram, MRI - Magnetic resonance imaging, MCF - Middle cranial fossa, CCA - Cavernous carotid artery, Pcomm - Posterior communicating artery, Occl - occlusion, d - days, m - months, Prop - proptosis, III - Occulomotor nerve, IV - Trochlear nerve, VI - Abducent nerve, V1,2 - Ophthalmic and maxillary divisions of trigeminal nerve, HA - Headache, Dip - Diplopia, EC-IC - Extracranial to intracranial bypass

Majority of the patients presented with features of mass effect with ophthalmoparesis, facial pain or hyposthesia. A more anterior extension through the supraorbital fissure leads to proptosis and optic nerve compression with resultant monocular visual loss.[4,8] In one case a more lateral extension of the giant aneurysm caused compression of the left perisylvian cortex leading to dysarthria.[6] In the present report, one patient presented with visual worsening, which might be due to medial extension and compression of the optic nerve by the expanding aneurysm.

Multiple theories have been proposed by authors explaining the simultaneous occurrence of ICA occlusion and cavernous aneurysm. This can occur at the presentation, as reported in our and most other cases, or on follow-up. Kurokawa *et al.*[7] reported 2 patients, who failed balloon occlusion test for cavernous segment aneurysm, and were followed up and developed spontaneous occlusion of ICA on follow-up. The theories proposed for this phenomenon include direct stretch and compression of the parent artery by the giant aneurysm,[4] proximal propagation of an intramural thrombus,[4,10] or compression of the ICA against the anterior clinoid process.[6,7] Baldawa *et al.* reported a patient with spontaneous thrombosis of giant cavernous aneurysm due to proximal ICA dissection and occlusion, This shows that ICA occlusion can be both a cause of, or result of the giant aneurysm.

The accepted methods of treatment for giant cavernous aneurysms is occlusion of the ICA, with or without bypass, depending on the cross-flow, as well as results of balloon test occlusion.[12] Endovascular interventions for both the feeding vessel and the aneurysm itself are also done with good results and less morbidity than an extensive open procedure. There have been reports of small case series of intracavernous aneurysms which

were managed conservatively and have shown no incidence of rupture over 10.5-13 years.[13,14] These series however, did not specifically look at the natural history of giant aneurysms of the cavernous carotid artery.

These reports suggest that there is definitely a subset of these patients with giant cavernous ICA aneurysms who undergo spontaneous occlusion of the feeding vessel [Table 1]. This phenomenon seems to occur over the course of time even if patients do not tolerate initial balloon test occlusion.[7] Therefore we believe there is a role for conservative management in patients who are not acutely and severely symptomatic and fail balloon test occlusion.[15] An option of conservative treatment can be given to such patients, with regular clinical and radiological follow-up.

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