



## Carotid Cavernous Aneurysms - A Report of Two Cases With Review of Literature

### KEYWORDS

External ophthalmoplegia, internal carotid artery aneurysm

#### Dr.C.Sreenivasulu

MD,D.M.,(Neurology) I/C Professor of Neurology,Kurnool Medical College,Kurnool

#### Dr.M.Ramanjuyulu

MS,Mch (Neurosurgery), I/C professor of neurosurgery, Kurnool Medical College,Kurnool

#### Dr.G. Shaul Hameed

M.S.,(ENT) Tutor in Neurology, Kurnool Medical College,Kurnool.

#### Dr. T.Vinil

Resident in general Medicine, Kurnool Medical College,Kurnool.

**ABSTRACT** Incidence of internal carotid aneurysms in cavernous segment represent only 3-5% of all intracranial aneurysms and 15% of those originated in internal carotid artery. Most commonly seen in females at age group of 5th and 6th decades. Most commonly present as ophthalmoplegia. Management is medical most of the time as defined by Linskey et al . We report two cases of internal carotid artery aneurysm presenting as painful external ophthalmoplegia.

In the first case,a 50 years female presented with ptosis of left eyelid with pain in left forehead and back of head on left side with diminished vision in left eye for 6 months. Examination revealed complete ptosis on left side with loss of pupillary reflexes and restriction of all ocular movements with ,diminished sensation on left side of face in maxillary division of trigeminal nerve. MRI brain with orbit revealed large saccular aneurysm arising from internal carotid artery . CT Brain angiogram showed giant aneurysm from cavernous segment of left carotid artery. In case two a 65years female presented with history of ptosis with intermittent periorbital pain on right side. Examination showed complete ptosis of right side with loss of pupillary reflexes and restricted ocular movements. MRI brain revealed saccular aneurysm arising from internal carotid artery . CT brain -Angiogram revealed saccular aneurysm arising from cavernous part of internal carotid artery.

#### Case 1:

A 50 years female presented with ptosis of left eyelid with pain in left forehead and back of head on left side with diminished vision in left eye for 6 months. On examination we noticed complete ptosis on left side with loss of pupillary reflexes and restricted ocular movements,diminished sensation on left side of face in maxillary division of trigeminal nerve. HB – 9.8 gms/dL TLC – 8,500/Cu.mm Platelets – 2.08L RBS – 87 mg/Dl Blood urea – 32 mg/dL Serum creatinine – 0.7 mg/dL Serum bilirubin – 0.7 mg/dL

Funduscopy revealed occlusion of superonasal branch of retinal artery. MRI brain with orbit revealed large saccular aneurysm with flow voids arising from internal carotid artery . CT Brain angiogram showed giant aneurysm arising from cavernous segment of left carotid artery

Fig 1. File photo of the patient showing Left Ophthalmoplegia



**Fig 2. CT Angio showing Giant Aneurysm arising from Cavernous segment of Left Internal Carotid**



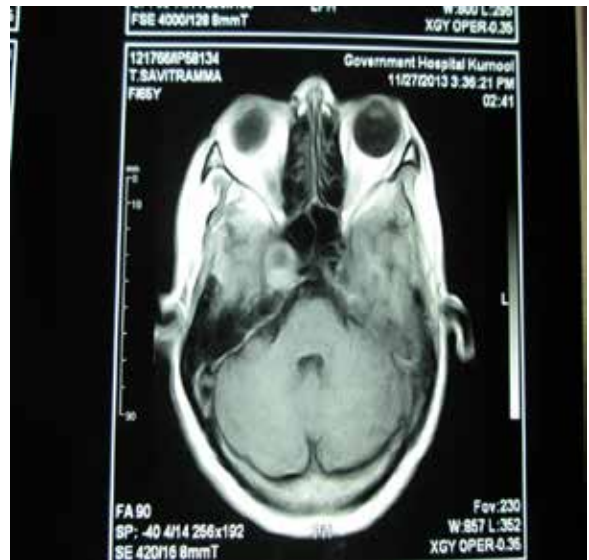
**CASE 2:-**

A 65years female presented with history of drooping of right eyelid with intermittent periorbital pain for 1 year duration .H/O Hypertension present.On examination we noticed complete ptosis of right side with loss of pupillary reflexes and restricted ocular movements. Decreased sensation on the right side of the face in maxillary division of trigeminal nerve . routine investigations are within normal limits MRI brain showed right saccular aneurysm arising from internal carotid artery. CT brain -Angiogram showed saccular aneurysm arising from cavernous part of internal carotid artery.

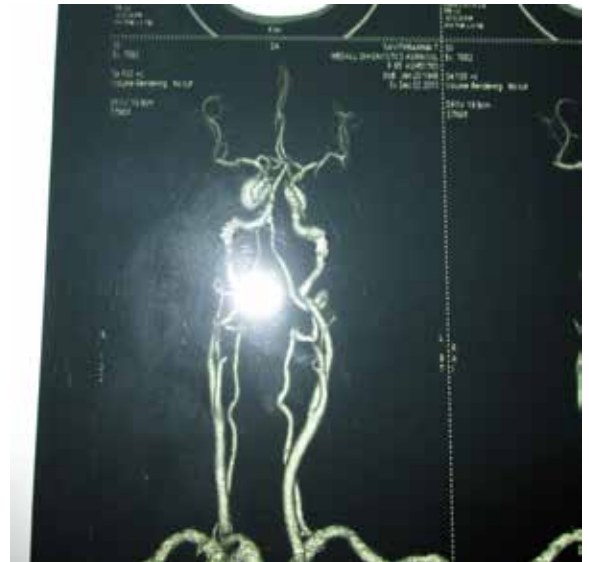
**Fig 3. File photo of the patient showing Right Ophthalmoplegia**



**Fig 4. MRI showing Right Carotid Cavernous Aneurysm**



**Fig 5. CT Angiogram showing Right Carotid Cavernous Aneurysm**



**Discussion:**

Internal carotid aneurysms in its intracavernous segment represent approximately 3-5% of all intracranial aneurysms<sup>1</sup> and 15% of those originated in the internal carotid<sup>2</sup>. Carotid cavernous aneurysms (CCA) can arise from any segment of cavernous carotid artery, but most commonly are originated in the horizontal segment, being projected forwardly and laterally, with the superior orbital fissure and below the anterior clinoid process<sup>3</sup>. This preferential site is related with the three most common branches of this segment (McConnell’s capsular artery, inferolateral trunk and meningohypofisary trunk)<sup>4</sup>. This suggests that the hemodynamic stress verified in these bifurcations can contribute to the aneurysms’ genesis<sup>3,5</sup>. Other aneurysmatic sites within the intracavernous segment are also common, what can interrogate the existence of other pathogenic mechanisms as atherosclerosis and dissection, spontaneous or traumatic<sup>6</sup>.

CCA morbidity and mortality indices are low<sup>7-10</sup>, however,

pain and neuro-ophthalmologic deficits due to neurovascular compression are frequent, what highlights the possibility of surgical treatment<sup>6,11</sup>. The vast majority of intracranial intradural aneurysms can be micro-surgically treated, commonly through aneurysmatic isolation without vascular occlusion, while CCA, when operated, frequently are through occlusion of ipsilateral internal carotid artery (ICA), with cerebral ischemia and amaurosis risks<sup>12-14</sup>. ICA endovascular occlusion has apparently a better outcome than ICA ligation<sup>13,15,16</sup>, although there is still much controversy around this matter, with authors in favor of surgical treatment of CCA patients with or without symptoms<sup>15,17</sup>, and others which are contrary to surgical treatment in both groups<sup>18</sup>. The reason for this controversy is in the lack of data on the natural history and long term outcome of CCA surgical patients<sup>16,19,20</sup>.

As to measure, according to Liskey et al<sup>6</sup>, pain symptoms and neurological deficits, the pain was graduated in severe, moderate, weak or absent, while neurological deficits were classified as severe, in the presence of cavernous sinus syndrome including trigeminal neuropathies; moderate, if there were complete involvement of III, IV and VI cranial nerves; weak, if there were deficits in one or two cranial nerves; and absent. Aneurysms are classified as Giant (> 2.5 cms), Large (1-2.5 cms) and small (<1cm).

Intracranial aneurysms seem to be more common in females. All 2 cases in this series were females. Thirty three of Jefferson's (4) 36 cases were females too. Angiography clinches the diagnosis but it may not show the entire aneurysm filling only partially. Treatment consists of ligating the common carotid artery and this was sufficient in most of the cases. Where this is not sufficient further ligation of internal carotid is advisable.

Using Pubmed database there were identified only three studies with large series of cavernous cave aneurysms(CCA) including one with natural history of 79 aneurysms with a follow up of 16 years. CCA's are more frequent in white females, with the beginning of symptoms around 5th and 6th decades. Similarly, systemic arterial hypertension is a risk factor for the management of intracranial aneurysms.

Rarely do CCA's suffer rupture and subarachnoid haemorrhage by the time of diagnostics, due to the fact that cavernous sinus are composed by dural slices, which lay over the body of sphenoid and are infrequently projected towards the subarachnoid space.

Aneurysms that arise within the cavernous sinus initially may be quite small, but that gradually enlarge to fill the sinus and thus acquire an added dural coat (the dural lining of the sinus). Once aneurysms fill the sinus, rupture is extremely unusual. Anterior expansion erodes the optic foramen and superior orbital fissure, resulting in compressive optic neuropathy, ocular motor nerve paresis, and proptosis. Expansion posteriorly may erode the petrous portion of the temporal bone. Inferior expansion is associated with erosion of the aneurysm into the sphenoid sinus (or) rarely into the nasopharynx. Rupture of such aneurysm causes dramatic, often fatal epistaxis. Medial expansion may cause destruction of the sella turcica and the pituitary gland, producing hypopituitarism with hyperprolactinemia.

Most of the cavernous sinus aneurysms did not require neurological intervention, as defined by Linsky et al., Therapeutic internal carotid artery occlusion is a common treatment in the management of symptomatic intracavernous carotid aneurysms, giant ICA aneurysms, and certain skull base neoplasms. Most of the centers now manage these lesions with endovascular carotid occlusion with or without cerebral revascularisation. The most widely accepted endovascular technique is the clinical balloon test occlusion introduced by Serbinenko in 1974. By incorporating clinical BTO, the stroke rate after carotid occlusion has markedly improved.

#### Conclusion:

In conclusion, cavernous carotid artery aneurysms present a satisfactory long-term neurological outcome with low complication rates if submitted to interventionist treatment, what determines improvement or resolution of pain symptoms in comparison with patients conservatively treated, as well as stabilization or partial improvement of neuro-ophthalmological deficits.

#### REFERENCE

1. Bars HW, Blackwood W, Meadows SP. Intracavernous carotid aneurysms. A clinical-pathological report. *Brain* 1971;94:607-622. [ Links ]
2. Bavinzki G, Killer M, Ferraz-Leite H, et al. Endovascular therapy of idiopathic cavernous aneurysms over 11 years. *Am J Neuroradiol* 1998;19:559-565. [ Links ]
3. Derdeyn CP, Cross DT, Moran CJ, et al. Postprocedure ischemic events after treatment of intracranial aneurysms with Guglielmi detachable coils. *J Neurosurg* 2002;96:837-843. [ Links ]
4. German WJ, Black SP. Cervical ligation for internal carotid aneurysms: an extended follow-up. *J Neurosurg* 1965;23:572-577. [ Links ]
5. Goddard AJ, Annesley-Williams D, Gholkar A. Endovascular management of unruptured intracranial aneurysms: does outcome justify treatment? *J Neurol Neurosurg Psychiatry* 2002;72:485-490. [ Links ]
6. Goldenberg-Cohen N, Curry C, Miller NR, et al. Long term visual and neurological prognosis in patients with treated and untreated cavernous sinus aneurysms. *J Neurosurg* 2004;75:863-867. [ Links ]
7. Higashida RT, Halbach VV, Dowd C, et al. Endovascular detachable balloon embolization therapy of cavernous carotid artery aneurysms: results in 87 cases. *J Neurosurg* 1990;72:857-863. [ Links ]
8. Inagawa T. Follow-up study of unruptured aneurysms arising from C3 and C4 segments of the internal carotid artery. *Surg Neurol* 1991;36:99-105. [ Links ]
9. Inoue T, Rhoton AL Jr, Theele D, et al. Surgical approaches to cavernous sinus: a microsurgical study. *Neurosurgery* 1990;26:903-932. [ Links ]
10. Johnston SC, Wilson CB, Halbach VV, et al. Endovascular and surgical treatment of unruptured cerebral aneurysms: comparison of risks. *Ann Neurol* 2000;48:11-19. [ Links ]
11. Kupersmith MJ, Berenstein A, Choi IS, et al. Percutaneous transvascular treatment of giant carotid aneurysms: neuro-ophthalmologic findings. *Neurology* 1984;34:328-335. [ Links ]
12. Kupersmith MJ, Hurst R, Berenstein A, et al. The benign course of cavernous carotid artery aneurysms. *J Neurosurg* 1992;77:690-693. [ Links ]
13. Kupersmith MJ, Stiebel-Kalish H, Huna-Baron R, et al. Cavernous carotid aneurysms rarely cause subarachnoid hemorrhage or major neurologic morbidity. *J Stroke Cerebrovasc Dis* 2002;11:9-14. [ Links ]
14. Kupersmith MJ. Aneurysms involving the motor and sensory visual pathways. In: *Neuro-Vascular Neuro-Ophthalmology*. Heidelberg: Springer-Verlag, 1993:254-261. [ Links ]
15. Linsky ME, Sekhar LN, Hirsch WL Jr, et al. Aneurysms of the intracavernous carotid artery: natural history and indications for treatment. *Neurosurgery* 1990;26:933-937. [ Links ]
16. Locksley HB. Natural history of subarachnoid hemorrhage, intracranial aneurysms and arteriovenous malformations. *J Neurosurg* 1966;25:321-368. [ Links ]
17. Miller NR. Carotid-cavernous sinus fistulas. In: Miller NR, Newman NJ (Eds). *Welsh & Hoyt's clinical neuro-ophthalmology*. Baltimore: Williams and Wilkins, 1997:3263-3322. [ Links ]
18. Newman SA. Aneurysms. In Miller NR, Newman NJ (Eds). *Walsh & Hoyt's clinical neuro-ophthalmology*. Baltimore: Williams and Wilkins, 1997:2975-3261. [ Links ]
19. Stiebel-Kalish H, Kalish Y, et al. Presentation, natural history, and management of carotid cavernous aneurysms. *Neurosurgery* 2005;57:850-857. [ Links ]
20. van der Schaaf EH, Buskens E, Rinkel GJ. Endovascular treatment of aneurysms in the cavernous sinus: a systematic review on balloon occlusion of the parent vessel and embolization with coils. *Stroke* 2002;33: 313-318. [ Links ]
21. Yasuda A, Rhoton AL Jr, de Oliveira E, et al. Microsurgical anatomy and approaches to the cavernous sinus. *J Neurosurg* 2005;56:4