

Internal Carotid Artery Aneurysm presenting as Hemiplegia

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ABSTRACT

A stroke / cerebro-vascular accident is defined as an abrupt onset of neurological deficit attributable to a focal vascular cause. Anterior circulation stroke is due to occlusion of the internal carotid artery and its branches. Congenital anomalies of the aortic arches and its branches presenting in geriatric age as focal neurological deficits are a rare presentation.¹

Most carotid aneurysms are considered benign lesions, most often asymptomatic and have a natural history with a low risk of life-threatening complications. However, several conditions may exist in which treatment of these aneurysms should be considered². Here we report a case of hemiplegia with a giant intracerebral internal carotid artery aneurysm.

Key-words : carotid artery, aneurysm, proptosis, hemiplegia



Fig. 1 : Patient with left eye ptosis, complete ophthalmoplegia and mature cataract

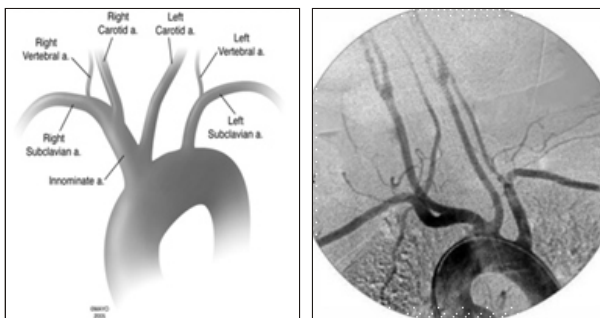


Fig. 2 : Bovine aortic arch anatomy

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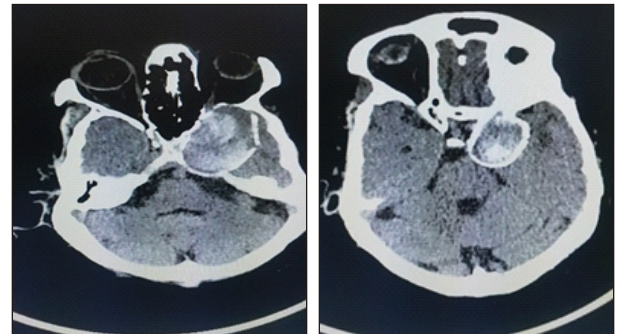


Fig. 3 : CT head (plain)
S/O Left ICA Thrombosed Aneurysm

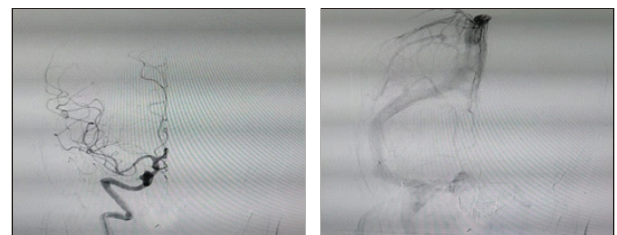


Fig. 4 : Digital Substraction Angiography :
Left vertebral artery is hypoplastic.
Right vertebral artery is dominant.
Left fetal origin of posterior cerebral artery

Introduction :

A 65 year old right handed, hypertensive female, presented with weakness of right upper and lower limb since 1 day. Weakness was acute in onset, with inability to move the right upper and lower limb and

it progressively evolved over six hours. She had past history of ptosis since 4 years with progressive and complete loss of vision in left eye over past two months.

On examination : Patient was conscious, alert and oriented to time, place and person.

BMI : 28 kg/m², Pulse : 90/min regular, RR : 20/min BP : 130/70 mmhg.

There was no pallor, icterus, clubbing, cyanosis, edema, lymphadenopathy.

CNS Examination : Higher mental functions were Normal, Speech Normal.

Cranial Nerve Examination : Proptosis of Left Eye with dilated pupil and mature cataract. She had total external and internal ophthalmoplegia involving cranial nerve III, IV, VI and V (Ophthalmic and Maxillary division).No chemosis.

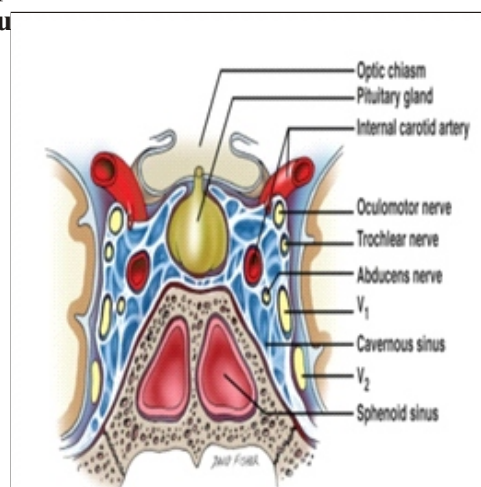
Seventh UMN Cranial Nerve Palsy on right side.

Sensory System WNL. Cerebellar signs and Gait could not be tested .

Respiratory System / Cardiovascular System / Per Abdomen WNL.

C T Head (plain) : Ill defined hypo-densities of 22HU noted in left frontal and left parietal lobe s/o

acute infarct. Well defined extra axial heterogenously iso-dense lesion with peripherally calcified walls of size 5 x 3.8 x 3.3 cm noted in left para-sellar and left temporal region arising from cavernous segment of left ICA. Hyper-dense component of 55 HU attenuation within s/o hemorrhagic component. Medially lesion is compressing the left cavernous sinus. Anteriorly lesion is causing erosion of lateral wall of left orbit and bulging into the apex. Optic nerve displaced medially and laterally causing mass effect on left temporal lobe. Features s/o left ICA **thrombosed aneu**



Pupillary Reflex	Right Eye	Left Eye
Direct	+	Absent
Indirect	Absent	Absent
Corneal Reflex		
Direct	+	Absent
Indirect	Absent	Absent
Conjunctival Reflex		
Direct	+	Absent
Indirect	Absent	Absent

	Right	Left
Tone	Hypotonia	Normal
Power	Grade 0/5	Grade 5/5

Reflex	Biceps	Triceps	Supinator	Knee	Ankle	Abdo	Plantar
Right	-	-	-	-	-	-	extensor
Left	2+	2+	2+	3+	2+	-	plantar

Digital subtraction angiography : Left vertebral artery is hypoplastic. Right vertebral artery is dominant. Left fetal origin of posterior cerebral artery noted. Bovine aortic arch present (origin of left common carotid artery is from right brachiocephalic trunk). Left common carotid artery could not be cannulated because of tortuous anatomy. Selective right common carotid artery angiography revealed two wide necked aneurysms in clinoid and supra-clinoid region measuring 4.2 × 4.3 mm and 2.5 × 2 mm respectively.

Discussion :

Intra-cavernous internal carotid artery aneurysms represent 3-5 % of all intracranial aneurysms. They account for 14 % of all internal carotid artery

aneurysms¹. Giant aneurysms are by definition more than 2.5 cm in diameter. The etiology of giant aneurysms is multifactorial and many structural and haemo-dynamic stress factors have been hypothesized. They can be idiopathic, iatrogenic, traumatic or infectious. The known risk factors are: female gender, hypertension and advancing age. The natural history of idiopathic cavernous aneurysms is not well known. They often remain asymptomatic and are detected incidentally.

These aneurysms produce a variety of neurological deficits : diplopia, oculomotor and abducent nerve palsies, corneal and facial anesthesia. The rupture rate of these aneurysms is relatively rare². The complication of rupture of intra-cavernous ICA aneurysm is sub-arachnoid hemorrhage, carotico-cavernous fistula or epistaxis. The giant aneurysm can cause local compression of surrounding structures and cause progressive neuropathy. Optimal management of giant carotid artery aneurysms remains controversial. The asymptomatic un-ruptured aneurysms are enclosed in a safe venous pouch, with tendency to grow from large to giant size. Intervention is justified when :

- Intradural extension with risk of sub arachnoid hemorrhage.
- Sphenoid bone erosion with risk of fatal epistaxis.
- Increasing size on serial angiograms.

The prevalence of aortic arch anomalies is 1-2%³. The incidence of bovine configuration of aortic arch is 9%³. The origin of left common carotid is from the innominate artery. It has been linked with development of thoracic aneurysms, increased risk of torsion and de-celeration injuries. This is due to presence of only two fixation points in comparison

to the normal three points.

The management of these large or giant intracranial aneurysms, with complex and refractory lesion, remains as a major challenge to cerebrovascular neurosurgeons.⁴ These giant aneurysms, most commonly found in the distal ICA segment, where velocity of blood flow is higher, require a multidisciplinary approach. Rapid advances in endovascular technology have significantly changed the options for treatment, but have not completely obviated the need for open treatment. ICA aneurysms are not a uniform group of lesions and require a thoughtful and individualized approach.

According to International Subarachnoid Aneurysm Trial (ISAT) and International Study of Unruptured Intracranial Aneurysms (ISUIA), endovascular treatment is relatively more successful in terms of clinical outcome.⁵

Balloon-and stent-assisted coiling techniques have been one of the milestones in the endovascular treatment of complex intracranial aneurysms and the flow-diversion devices represent the latest revolution in treatment of endovascular aneurysms.⁵

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