25th Annual Conference of INDIAN SOCIETY OF NEURORADIOLOGY

CASE REPORT OF COMPLICATED CAROTID CAVERNOUS FISTULA MANAGED BY ENDOVASCULAR INTERVENTION

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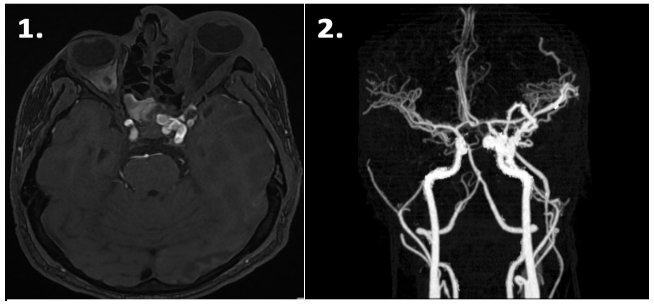
INTRODUCTION

A carotid cavernous fistula is a type of dural AV fistula which is a result of abnormal vascular communication between the ICA / ECA and the venous channels of the cavernous sinus [1]. The clinical symptoms of CCFs mostly arise due to effects on important neural and vascular structures in the cavernous sinus which include cranial nerves III, IV, V1, V2, and VI.
Direct CCFs result from a traumatic tear in the artery from a skull base fracture, from an acceleration-deceleration force, or an iatrogenic injury after an endovascular intervention or spontaneously after rupture of an intracranial aneurysm.[4]
Indirect CCFs result from a dural branch rupture of the carotid artery caused by a genetic condition or a comorbidity such as hypertension. [3]This case report reviews the cause, pathophysiology, and presentation of CCF and highlights the role of interventional radiology in its management.

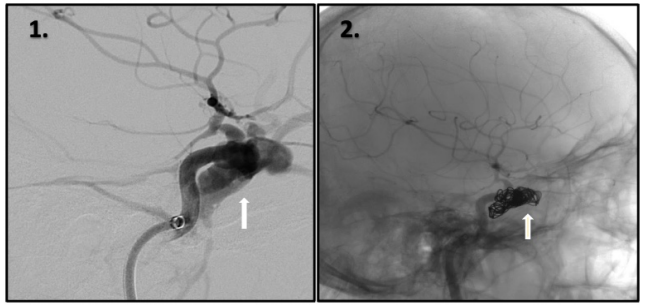
CASE REPORT

A 22-year-old male presented with complaints of drowsiness, pain, progressive swelling in the left eye and vision loss, and right upper limb weakness for 2 weeks.
His past history revealed a history of head injury two months back.
On examination, the patient had an altered sensorium, aphasia, and right upper limb weakness.
On ophthalmological examination, the patient had periorbital edema with proptosis, mild discharge, and chemosis of conjunctiva. The cornea was clear but the pupil was dilated and fixed with restricted eye movements in all directions. Vision in left eye was light perception.
On MRI Brain, a large left MCA infarct with hemorrhagic transformation was seen in the left frontopareitotemporal lobes, insular cortex, and basal ganglia with moderate mass effect, uncal herniation, and midline shift.
Also, a diagnosis of complicated left orbital cellulitis with multiple orbitofacial fractures was made.
MRA brain suggested a post-traumatic left carotid-cavernous fistula.



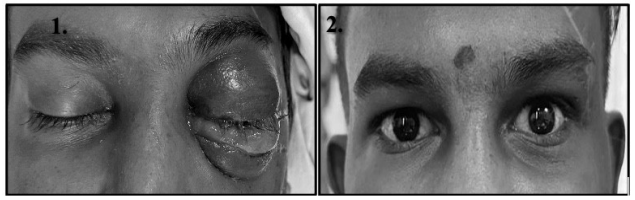
1. MRA contrast axial image showing site of carotid cavernous fistula.
2. 3D reconstructed CT angiogram image showing the site of carotid cavernous fistula.

CT angiogram revealed a complicated post-traumatic carotid-cavernous fistula with prominent left inferior ophthalmic vein with a dilated and tortuous left superficial Middle cerebral vein – implying a retrograde fistulous flow.
Neuro DSA also revealed a large left carotid-cavernous fistula. Plan for endovascular coiling was made.
Elective intubation and balloon-assisted coil embolization of the carotid-cavernous fistula was done under GA and fluoroscopic guidance. Check angiogram, immediately post-procedure revealed good distal flow with complete occlusion of the CCF. The patient was then extubated.



1. DSA image showing fistulous opening from the internal carotid artery to the cavernous sinus – Type A CCF.
2. Post coiling, check angiogram showing complete occlusion at the fistula site with free flow into the intracranial branches of ICA.

Post-procedure, the patient was under observation for a few days. His mass effect and midline shift decreased on subsequent CTs. His sensorium and right upper and lower limb weakness improved. His orbital cellulitis also improved and he slowly regained his vision over a period of 4 weeks.



1. Image showing the patient on presentation. Note the proptosis of left eye with periorbital edema and chemosis of conjunctiva
2. Image showing the same patient on one month follow up post coil embolization. (consent for the reference images taken)

DISCUSSION

Normal venous flow to the cavernous sinus is impeded in CCF as high pressure arterial blood enters the cavernous sinus.
The mechanism of vision loss from a CCF is due to generation of venous hypertension and the consequent reduction of perfusion. The stagnant flow anoxia leads to ischemia of optic nerve which is a mechanical and conduction block due to increased IOP or compression of optic chiasma or optic nerve die to distended cavernous sinus. This vision loss is usually reversible. However, if immediate intervention is not done, irreversible optic neuropathies – optic atrophy/ infarction and trauma will occur.

CONCLUSION

The classical triad of symptoms of CCF are pulsating exophthalmos, ocular bruit and conjunctival chemosis. However, Incidence of vision loss is also reported in many cases of CCF.
In cases of CCF with visual impairment, fistula obliteration is urgently required to prevent irreversible vision loss. Recovery from blindness is not possible even after successful CCF embolization if timely interventional management is not done.

REFERENCES

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