



A CASE OF CAROTID CAVERNOUS FISTULA DIRECT TYPE A IMAGING PRESENTATION

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Abstract : Carotid Cavernous Fistula is a type of fistulous communication between internal carotid artery and cavernous sinus that occurs within the cavernous sinus. Two types of carotid cavernous fistula are direct and indirect. Direct fistula results from traumatic or non-traumatic cause. Here we are presenting a case of traumatic direct carotid cavernous fistula, discussing the imaging features and differential diagnosis.

Keyword : Trauma, Carotid Cavernous Fistula, Computed Tomography, Magnetic resonance image.

Introduction:

Direct carotid cavernous fistula usually occur following head injury due to central skull base fracture and stretch injury to internal carotid artery. As the typical clinical features occur days to weeks following trauma imaging determines the presence of arterio venous communication within the cavernous sinus. Here now we will discuss a case report of traumatic direct carotid cavernous fistula.

CASE REPORT:

35 years old male had h/o. fall from bus and sustained trauma to head. On the 3rd day he developed swelling of the left eyelid, restricted eye movements. No h/o. headache/ vomiting/ seizure. No h/o. focal neurological deficit. Bulk, Tone, Power, Reflexes in all 4 limbs normal. No sensory deficit, No cerebellar signs. Examination of left eye: Chemosis present, Proptosis present, Restriction of eye movements are noted with no perception of light, Bruit-Heard. CT BRAIN Imaging revealed: Bilateral undisplaced fractures involving squamous part of temporal bone. Mild displacement of petrosphenoid synchondrosis (fig. 1).

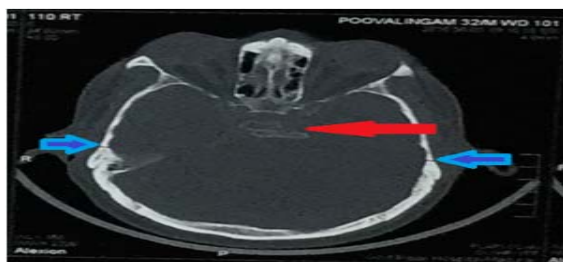


fig. 1

Sub arachnoid haemorrhage noted along occipital convexity on right side, left sylvian fissure (fig.2)

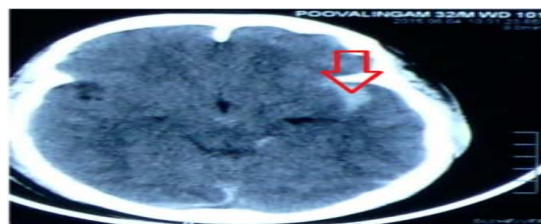


fig. 2
and suprasellar cistern region (fig.3).



fig. 3

Magnetic Resonance Imaging revealed: preseptal edema with proptosis of left eye . Dilated left cavernous sinus with flow voids within noted (fig.4) .

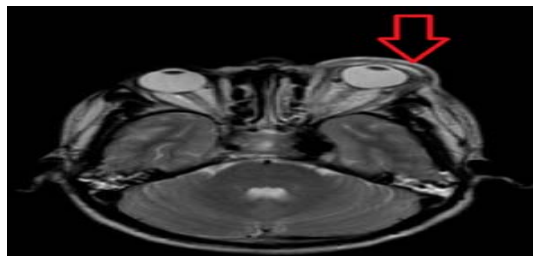


Fig. 4

Lateral wall convexity of left cavernous sinus noted. Dilated and tortuous left superior ophthalmic vein noted (fig. 5).

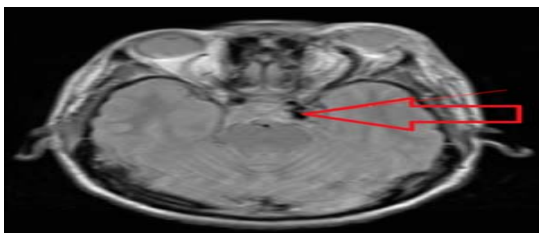


fig. 5

Magnetic Resonance Angiography after administration of i.v gadolinium contrast done revealed increased flow related signal in left cavernous sinus, flow noted in left superior ophthalmic vein (fig. 6)

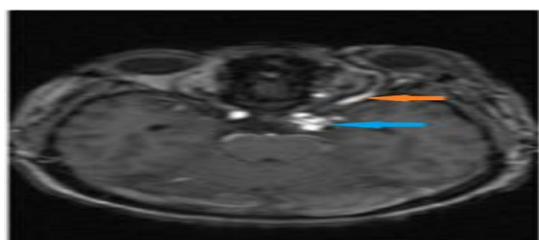


fig. 6

DSA: High flow fistulous AV shunting between posterior genu of left cavernous ICA and cavernous sinus. Venous drainage from left cavernous sinus is noted in retrograde manner to left superior ophthalmic vein. (fig. 7 and fig. 8). An imaging diagnosis of carotid cavernous fistula direct Type A was given.

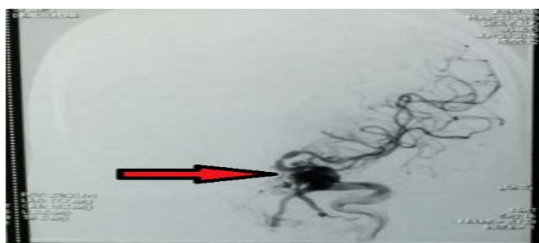


fig. 7

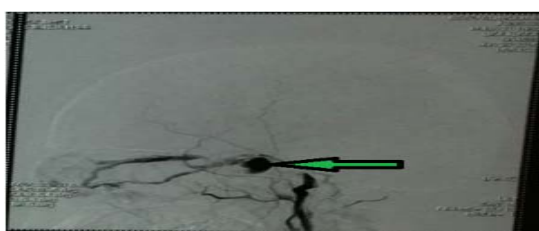


fig. 8

TREATMENT :

Intervention - coiling of the sac done achieving significant reduction of shunt. POST INTERVENTIONAL DSA: Fig 9, Fig 10 shows Coil mass noted insitu within the left cavernous sinus. No evidence of fistula or posterior shunting from cavernous sinus noted.

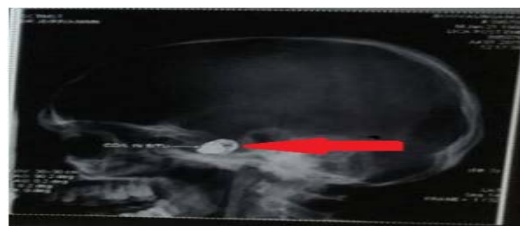


fig. 9

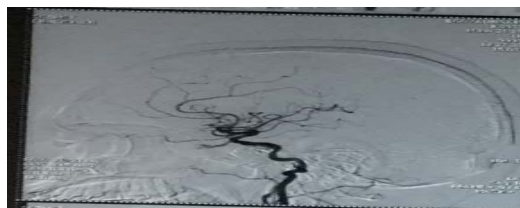


fig. 10

Discussion:

Carotid-Cavernous fistula results from abnormal communication between previous normal carotid artery and cavernous sinus. 70 to 90 % etiology of direct CCF is trauma. Patient present with proptosis, conjunctival chemosis, pulsating exophthalmos, diplopia and blindness. **BARROW CLASSIFICATION OF CAROTID-CAVERNOUS FISTULAS**

Type A: Direct ICA-cavernous sinus high-flow shunt

Type B: Dural ICA branches-cavernous sinus shunt

Type C: Dural ECA-cavernous sinus shunt

Type D: Both ICA/ECA dural branches shunt to CS

In a direct CCF, arterialized flow causes dilatation of the CS with venous hypertension and retrograde flow into the superior and inferior ophthalmic veins. Indirect CCFs demonstrate enlarged crack-like vessels within the CS.

CT FINDINGS: NECT scans may demonstrate mild or striking proptosis, a prominent CS with enlarged superior ophthalmic vein (SOV), and enlarged Extraocular muscles. "Dirty" fat secondary to edema and venous engorgement may be present. Occasionally, subarachnoid hemorrhage from trauma or ruptured cortical veins can be identified. CECT scans often nicely demonstrate an enlarged SOV and CS. Inferior drainage into a prominent pterygoid venous plexus and posterior drainage into the clival venous plexus are sometimes present.

MR FINDINGS: T1 scans may show a prominent "bulging" CS and SOV as well as "dirty" orbital fat. T2-weighted images may show asymmetric flow-related signal loss in the affected veins. Too many "flow voids" in the CS is a common finding with CCFs. Strong, uniform enhancement of the CS and SOV is typical. Enlarged, tortuous intracranial veins may occur with high-flow, high-pressure shunts. Rare cases of high-flow, aggressive direct CCFs with prominent pontomesencephalic and perimedullary venous drainage causing progressive myelopathy have been reported.

ANGIOGRAPHY: DSA is required for definitive diagnosis and treatment. Complete delineation of the arterial supply and venous drainage pattern is the goal. *Direct* CCFs typically demonstrate rapid flow with very early opacification of the CS. Selective ICA injection with very rapid image acquisition is often necessary to localize the fistula site precisely. A single-hole fistula is usually present, typically between the C4 and C5 ICA segments.

TREATMENT: The primary goal in treating a direct CCF is fistula closure, typically by transarterialtransfistula detachable balloon embolization. This technique is successful in over 90% of cases. Transvenous embolization via the internal jugular vein and inferior petrosal sinus is another option. If the ICA is torn,

covered stent placement may be effective.

DIFFERENTIAL DIAGNOSIS: Cavernous sinus thrombosis, arteriovenous malformation, cavernous sinus tumors, orbital tumors, skull base tumors and mucocoele.

SUMMARY:

Imaging features play important role in the early diagnosis and early intervention before extensive collateral formation. MR Angiography are especially important to rule out cavernous sinus thrombosis.

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