Radiology Section

Imaging Findings with 3D CT Angiographic Reconstruction of Carotico-cavernous Fistula-A Case Report

SHIVU JAYADEV, KARTHIK ADIRAJU, BHUSHITA LAKHKAR, PARTH VAISHNAV

### ABSTRACT

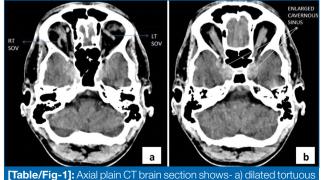
Carotico Cavernous Fistula (CCF) is an abnormal communication between the carotid arteries and the cavernous sinus. They are divided into direct or indirect types based on the etiology. Direct CCFs present with acute and rapid onset of symptoms, are often secondary to trauma most commonly seen in the head injuries and young male patients. Indirect CCFs have insidious onset with predilection for the postmenopausal female patients. We report a case of 30 years old male with history of trauma four months back, presented with inability to move right eye laterally and diplopia since two months, contrast enhanced CT brain showed findings suggestive of CCF. Rare incidence with gradual onset of CCF post trauma and 3D CT reconstruction imaging makes this case unique.

Keywords: Cavernous sinus, Dilated ophthalmic veins, Diplopia internal carotid artery, Imaging of CCF

#### **CASE REPORT**

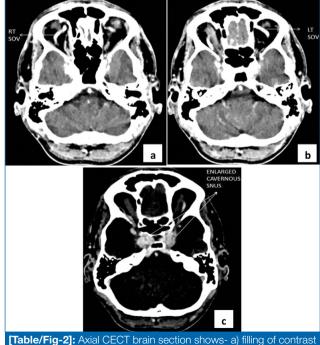
A 30-year-old male presented to our department with headache, right orbital pain, inability to move right eye laterally and diplopia since 2 months. Patient had history of trauma four months back. Patient was non diabetic and non hypertensive. On examination patient was stable with vitals in normal range.

On plain CT brain (Somatom Scope 32 slice configuration, Siemens) brain plain [Table/Fig-1] bilateral Superior Ophthalmic Veins (SOVs) were dilated and tortuous, right SOV was measuring about 3.5 mm and left SOV was measuring about 3.2 mm, Cavernous Sinus (CS) was enlarged with bulging of bilateral lateral walls noted. Post contrast scans [Table/ Fig-2] in arterial phase showed filling of contrast in dilated and tortuous bilateral SOV and irregular intense enhancement of CS indicating formation of a communication between Internal



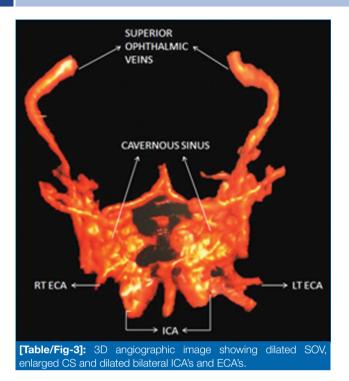
bilateral superior ophthalmic veins: b) enlarged cavernous sinus.

Carotid Artery (ICA) and CS. Further, 3D CT Angiographic reconstruction [Table/Fig-3] shows dilated, tortuous bilateral ICAs and CS, helping in better visualisation of dilated bilateral SOVs, enlarged CS, bilateral internal and external carotid arteries and suggesting direct type of CCF.



[Table/Fig-2]: Axial CEC1 brain section shows- a) filling of contrast in dilated and tortuous right; b) left superior ophthalmic veins; c) intense enhancement of cavernous sinus.

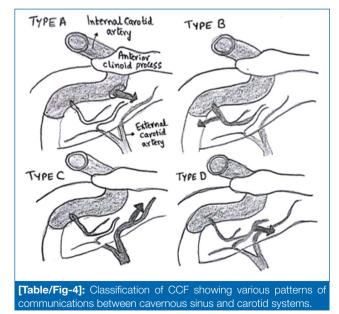
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Hence, on imaging a diagnosis of direct type CCF was made with the help of 3D reconstruction imaging. Patient was referred to higher center.

# DISCUSSION

CCF is an abnormal communication between the carotid arteries and the cavernous sinus. CCF's are classified into several types, based on its anatomy (direct or indirect),



etiology (traumatic or spontaneous) and flow dynamics (high or low flow).

Based on arterial supply, CCF's are classified as follows: [1] [Table/Fig-4].

Type A fistulas have direct connections between the intracavernous ICA and CS, usually associated with high flow rates, and are mostly secondary to trauma (facial and skull base fractures) [2].

Type B, C and D are low flow indirect fistulae: Type B fistulas have dural branches of ICA to CS, and are relatively uncommon; Type C fistulas have arterial supply from dural branches of ECA; Type D fistulas have dural ICA and ECA branches to the CS. It is the most prevalent indirect fistula.

**Direct CCF:** Proximal horizontal intracavernous segment of the ICA near the inferolateral trunk is the most frequent location of direct CCF. Traumatic disruption of the vessel wall is the most common cause although 20% of cases are regarded as spontaneous (conditions that predisposes weakness of the ICA wall, e.g., Ehlers-Danlos syndrome) [3].

**Indirect CCF:** Indirect CCF's are generally insidious with predisposing factors like hypertension, diabetes, pregnancy, collagen vascular disease and occur frequently in post-menopausal women. Sometimes it can resolve spontaneously without treatment [4,5].

**Clinical Presentation:** It is variable and is due to the elevated intracavernous pressure, dominant pattern of venous drainage, the size and location of CCF and the presence of collateral vessels (arterial or venous). In direct CCF, symptoms appear suddenly and present with pulsatile exophthalmos, loss of eye movements, diplopia and pain. Indirect CCF's present with late onset of symptoms showing progressive glaucoma, proptosis and red eye.

Complications of CCF include visual loss, intracranial haemorrhage (both intraparenchymal and subarachnoid), venous infarct, epistaxis, increased intracranial pressure with venous hypertension and cranial nerve palsies (III,IV,V and VI).

The clinical history of the patient is fundamental and plays a crucial role in diagnosis. Patients presenting with subtle signs and insidious symptoms, the hypothesis of DCSF should be considered [6].

**Imaging of CCF:** Various modalities available to diagnose CCF include orbital Doppler ultrasonography, CT-scan, MRI and Digital Subtraction Angiography (DSA).

On orbital Doppler ultrasonography, reversal of flow is seen in SOV [6].

CT findings include dilatation of SOVs and enlargement of cavernous sinus with bulging of lateral walls. On contrast administration, filling of contrast in dilated and tortuous bilateral SOV and enhancement of bilateral cavernous sinuses is seen, which helps in differentiating direct from indirect CCF. Faster and intense contrast uptake by the cavernous sinus is seen in cases of direct CCF, whereas in cases of indirect CCF slower uptake of contrast by CS is seen. Intracranial haemorrhage as a complication of CCF is better evaluated in CT [7].

MRI plain and contrast study shows, enhancing enlarged CS and SOV with adjacent dural enhancement.

DSA is considered the gold standard for the diagnosis of CCF. It shows rapid filling of enlarged CS and venous drainage into the SOV after contrast injection into ICA. It is the best modality for anatomical evaluation, classification and to decide therapeutic approach for CCF [8].

However, recent developments in noninvasive techniques, the volume rendering method in CT angiography provides a more realistic 3D appearance and depicts anatomic relationships more accurately and has now become new tool for the early and safe diagnosis of CCF [7-9].

Differentials include CS thrombosis, thyroid ophtalmopathy, masses in orbital apex and anomalous intracranial venous drainage.

**Treatment and Prognosis:** CCF has a varied natural history, which includes spontaneous closure to rapidly progressive symptoms. Poor prognostic factors involve the aneurysm of feeding vessel (indirect CCF) and retrograde filling of cortical veins resulting in increased risk of haemorrhage.

Several therapeutic options has been developed in management of CCF, endovascular approach has become the first line option in clinical emergencies, being used in symptomatic direct CCF's and indirect fistulas with progressive symptoms [10-12].

The available treatment options include carotid compression therapy, most useful in indirect fistulas resulting in spontaneous closure (30%) than direct fistulas (due to the higher flow) only successful in 17% of cases [11]. Transarterial balloon embolisation is the treatment of choice for symptomatic direct CCF wherein both arterial and venous access (including superior ophthalmic vein) can be employed. A combined transarterial (closure of feeders) and transvenous (closure of cavernous sinus) approach is needed in indirect fistulas with higher rate of spontaneous closure. Surgical treatment with ligation or trapping of involved segments [11].

# CONCLUSION

Post traumatic direct CCF can have insidious onset, CECT with 3D angiography reconstruction can be considered as a reliable first line diagnostic tool in evaluating and classifying CCF in a suggestive clinical context, an accurate and safe method for noninvasive and reproducible follow-ups. However, due to lower spatial resolution, CT angiography is unable to depict the topography of the shunt in few CCFs or the feeding branches in dural arteriovenous malformations. Therefore, it cannot replace conventional angiography, which is still considered the gold standard modality for diagnostic and endovascular treatment planning in CCF

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#### AUTHOR(S):

- 1. Dr. Shivu Jayadev
- 2. Dr. Karthik Adiraju
- 3. Dr. Bhushita Lakhkar
- 4. Dr. Parth Vaishnav

#### PARTICULARS OF CONTRIBUTORS:

- Junior Resident, Department of Radiodiagnosis and Imaging, Shri BM Patil Medical College, Hospital and Research Centre, BLDE University, Vijayapur, Karnataka, India.
- Junior Resident, Department of Radiodiagnosis and Imaging, Shri BM Patil Medical College, Hospital and Research Centre, BLDE University, Vijayapur, Karnataka, India.
- Assistant Professor, Department of Radiodiagnosis and Imaging, Shri BM Patil Medical College, Hospital and Research Centre, BLDE University, Vijayapur, Karnataka, India.

4. Junior Resident, Department of Radiodiagnosis and Imaging, Shri BM Patil Medical College, Hospital and Research Centre, BLDE University, Vijayapur, Karnataka, India.

# NAME, ADDRESS, E-MAIL ID OF THE CORRESPONDING AUTHOR:

Dr. Shivu Jayadev, Junior Resident, Department of Radiodiagnosis and Imaging, Shri BM Patil Medical College, Hospital and Research Centre, BLDE University, Vijayapur-586103, Karnataka, India. E-mail: shivujaydev@gmail.com

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