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IMAGING OF CAVERNOUS SINUS AND ITS PATHOLOGIES



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ABSTRACT

This article reviews the normal anatomy of the Cavernous sinus (CS) and the imaging findings of different lesions involving it. CS lesions may arise from different components of the CS or from adjacent structures and spaces. They can be classified as neoplastic, inflammatory, infectious, vascular and congenital. MRI is the imaging modality of choice and helps in differentiating different pathologies and come to a single best diagnosis.CT helps in identifying aneurysms and bone involvement better in selected cases. These lesions are more commonly seen in the older age group and in the female gender. Meningiomas,Schwannomas,meningitis and pituitary macroadenomas are the common lesions encountered in this study.Cavernous angioma, perineural spread from nasopharyngeal carcinoma are the few rare cases seen.

KEYWORDS

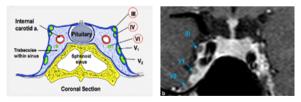
Cavernous Sinus(cs), Meningioma, Schwannoma, Craniopharyngioma, Epidermoid, Cavernous Angioma, Chordoma, Sphenoid Sinus Carcinoma, Perineural Spread, Tolosa Hunt Syndrome, Internal Carotid Artery(ica)aneurysm.

I. INTRODUCTION

Cavernous sinus (CS) is a venous plexus located laterally at both sides of the sella turcica and contains vital neurovascular structures. It can be a potential location of a wide range of pathologies as multiple structures can be involved. Here in this article, the normal anatomy of the cavernous sinus, a wide range of CS pathologies and their imaging features are described.

ANATOMY:

The CS is composed of 2 layers of the dura. Each dural wall contains an outer layer apposed to the bone and an inner layer in contact with blood or CSF. The CS extends from the orbital apex and superior orbital fissure anteriorly to the Meckel cave and farther posteriorly to the dura and the pores that allow nerves to enter it. The internal carotid artery (ICA) is the most medial structure inside the CS, called carotid trigone. Cranial nerves III and IV and the first and second divisions of the cranial nerve V (from superior to inferior) are located in the lateral dural wall of the CS (called the oculomotor trigone). Cranial nerve VI courses in the central part of the CS inferolateral to the ICA. Inside, the CS is a multiseptate space, which shows intense contrast enhancement of the slower flowing venous blood. The main venous influx into the CS is the superior and inferior ophthalmic veins, pterygoid plexus, and Sylvian vein. The outflow of the CS occurs via the superior and inferior petrosal sinuses. Its transverse diameter is 5-7 mm, its vertical diameter is 5-8 mm, and its anteroposterior diameter is 10-15 mm.



Cavernous sinus pathologies can have various clinical manifestations, ranging from subtle to devastating, and including headache, ophthalmoplegia, exophthalmos, chemosis, vision loss, trigeminal neuralgia, and, rarely, cerebral infarction owing to ICA involvement.⁽²⁾

MRI is the imaging modality of choice for assessment of the cavernous sinus. CT and DSA are useful in certain cases. Imaging plays a crucial role in confirming the disease, assessing its extent and planning therapy.⁽³⁾

II. MATERIALS AND METHODS STUDY DESIGN: Prospective descriptive study

STUDY LOCATION: NRIAS, Chinakakani Guntur.

SAMPLE SIZE : 40 cases

INCLUSION CRITERIA:

Any age and gender with symptoms related to cavernous sinus

EXCLSION CRITERIA: Claustrophobics and patients with ferromagnetic implants

METHODOLOGY:

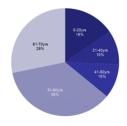
MRI imaging of the CS was performed on 1.5 T GE Healthcare Signa Excita. The imaging protocol included Sagittal T1, Axial T2, fluidattenuated inversion recovery (FLAIR), DWI, GRE and precontrast T1-weighted fat-saturated images of the entire brain. Post-contrast T1weighted 2-3mm thick images were obtained in the axial and coronal planes with a fat-saturation technique. Thin-section 3D heavily T2weighted images may allow visualization of individual cranial nerves in the CS and adjacent cisterns. Further sequences (MRA TOF, dynamic MRA) carried out depending on the clinical context and the signal of the lesion. CT is performed on GE 16slice multidetector scanner after intravenous administration of iodinated contrast medium. Acquisitions in axial or coronal planes by using <1 mm thick sections are obtained and then reformatted in other planes.

III. RESULTS

In our study during the period of three years, 40 cases were diagnosed with Cavernous sinus pathologies. Females are affected most commonly. More than half of the cases were of more than 50years of age. There were wide ranges of pathologies, most of them being meningiomas (16cases), followed by Schwannomas (3cases).Other pathologies included meningitis(3cases), Tolosa hunt synd rome(2cases), ICA aneurysm(2cases), Cavernous fistula (1case), perineural spread (2cases), metastasis from carcinoma thyroid(1case), Sphenoid carcinoma(1case), Chordoma(1case), cavernous hemangioma(1case), arach noid cyst(2cases), craniopharyngioma(1case)..Pituitary adenoma (2cases), Schwannoma(3cases).

AGE DISTRIBUTION:

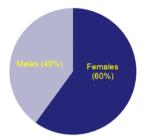
Cavernous sinus pathologies were seen in all age groups, most commonly in the age group of 50-60yrs(14cases).there were 6 cases in less than 20years age group,4 cases each in 21-40yr and 41-50yr age group,11 cases in the 61-70yrs age group.



Age distribution

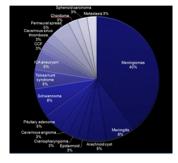
GENDER DISTRIBUTION

Out of 40 cases, 24 were females, and 16 were males. So, cavernous sinus lesions are most commonly seen in females, but this is not of much significance.



Gender distribution

Distribution Of Various Pathologies:



IV. DISCUSSION

CS lesions may arise from different structures of the CS or from adjacent structures and spaces. They can be classified as neoplastic, inflammatory/infectious, granulomatous, vascular and congenital.

- (1) Neoplastic (Primary and secondary): Primary arise from the cavernous sinus itself such as Meningioma, Neurogenic Tumors, and Hemangioma. Secondary are caused by direct invasion of the sinus by Pituitary Adenoma, or invasion from nasopharyngeal Carcinoma, Juvenile Nasopharyngeal Angiofibroma and Clival Chordoma.
- (2) Infectious: Cavernous Sinus Thrombophlebitis, tuberculous Pachymeningitis, Sinonasal Fungal infiltration,
- (3) Inflammatory : Tolosa-Hunt syndrome, Idiopathic Hypertrophic Pachymeningitis Inflammatory Pseudotumor,
- (4) Vascular: ICA Aneurysm, Carotid-Cavernous Fistula, ICA Thrombosis
- (5) Congenital: Epidermoid or Dermoid Cyst.

MENINGIOMA

Meningiomas are the most common primary tumour involving the Cavernous sinus. It arises from the arachnoid cap cells attached to the dural wall of the CS, most commonly the lateral dural wall. A meningioma is isointense to the grey matter at all MR imaging sequences and shows homogenous and intense enhancement. A dural tail frequently is seen extending away from the edge of the tumour. Meningiomas characteristically constrict the lumen of the internal carotid artery which is a distinguishing feature from a pituitary adenoma⁽⁵⁾.(Fig 1)

It may contain calcifications, induces adjacent hyperostosis and enhances strongly after contrast administration. Meningiomas may extend inside the CS and the Meckel cave and via the porous trige minus into the preportine cistern.^(3,4)

PITUITARY ADENOMA

They are primary tumours of adults. These tumours can be functional or clinically and biochemically silent. Around 6–10% of all pituitary adenomas invade the cavernous sinus ⁽⁴⁾. Preoperative identification of invasion of the cavernous sinus is an important prognostic factor with regard to surgery for pituitary adenomas, with a higher incidence of both intraoperative ICA injury and postoperative leakage of (CSF) when cavernous sinus invasion is present. Cottier et al. used the percentage of ICA circumference contacting the tumour and the invasion of five venous compartments around the ICA. Greater than 67% circumferential contact with the ICA indicates invasion, and less

than 25% contact and the tumour not crossing the medial intercarotid line have high negative predictive values for invasion.(Fig 2)

MENINGITIS

Tuberculosis is a common cause of pachymeningitis that produces diffuse or focal extra-axial masses along the surfaces of the CS. The lesions enhance homogeneously and intensely and maybe nodular. The presence of a dark signal on T2-weighted images and the presence of basal meningitis elsewhere are helpful diagnostic clues. However, neurosarcoidosis may be identical in appearance⁽⁵⁾

EPIDERMOID CYST.

An epidermoid cyst may be of extracavernous origin and extend into the CS, originate in the lateral CS wall, or be a true intracavernous lesion. The mass is hypointense or isointense and T2 hyperintense. On FLAIR and T2-weighted images, it demonstrates a heterogeneous signal intensity. These cysts do not enhance. Epidermoid cysts show restricted diffusion with higher signal intensity than that of CSF on $DWI^{(i)}(Fig 3)$

CAVERNOUS SINUS HEMANGIOMA

More commonly seen during the fifth decade of life in female patients. This tumour is formed by sinusoidal spaces with an endothelial lining that contains slow-flowing or stagnant blood. A preoperative diagnosis is important because of its propensity to bleed at the time of resection. These tumors are nearly hyperintense on T1- and T2-weighted images and are attached to the outer wall of the CS, and their diagnosis may be suggested when they show progressive "filling in" after contrast administration. Angiography shows 80% show a vascular blush, due to the high degree of hypervascularity.(Fig 4)

SCHWANNOMA

Intracranial schwannomas are usually developed mainly from cranial nerves III and V. Schwannoma is a well-delineated mass which appears iso- to hypointense compared to the grey matter on T1- weighted images, hyperintense on T2 and enhances after contrast. The trigeminal schwannoma frequently has a typical dumbbell-shape within the Meckel's cave and cavernous sinus, along the cisternal trigeminal course.⁽³⁾

TOLOSA HUNT SYNDROME

Tolosa-Hunt syndrome is a recurrent painful ophthalmoplegia due to nonspecific granulomatous inflammation in the anterior cavernous sinus, superior orbital fissure, or orbital apex. The diagnosis is based on findings of painful ophthalmoplegia accompanied by variable deficits of the oculomotor through the abducens nerves, excellent response to corticosteroid therapy, and exclusion of other lesions. On MRI, the CS is hypointense on T1 and T2-weighted images, with homogeneous enhancement and focal narrowing of ICA.⁽⁶⁾

CHORDOMA

Intracranial chordoma is a rare malignant tumour usually developed from the notochordal remnants within the clivus and may invade the CS. It is a soft tissue mass, hypo- to isointense on T1 weighted images and hyperintense on T2- weighted images. It shows important and heterogeneous enhancement. On CT, bone destruction and intratumoral calcifications are often seen. Small spots of T1 hyperintensities can be noted within the tumour and are related to intratumoral haemorrhage or mucus.^{(8:9}(Fig 5)

CAROTICO-CAVERNOUS FISTULA

Direct CCF (type A) is a high-flow communication between the ICA and the CS that occurs after trauma or secondary to a ruptured aneurysm of the cavernous ICA. These lesions present acutely with pulsating exophthalmos, chemosis, and CS syndrome. Dural CCFs (types B-D) are low-flow fistulas occurring between meningeal branches of the carotid artery and CS, which tend to have milder symptoms than direct fistulas.^[10]On CT or MR imaging, the diagnosis depends on morphologic changes such as exophthalmos and enlargement of the superior ophthalmic veins, cavernous sinus, or extraocular muscles. MR imaging is able to depict flow voids in the involved cavernous sinus. The presence of flow-related enhancement in the CS on MR angiography suggests the diagnosis in the right clinical setting. Other supporting findings are a "dirty" appearance of the retro-orbital fat and enlargement of the extraocular muscles.

The reference standard for the diagnosis of CCF is digital subtraction angiography. Features of CCF at routine MRI include a bulky

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ipsilateral cavernous sinus, prominent adjacent flow voids, a dilated superior ophthalmic vein, stranding of the retro-orbital fat, and bulky extraocular muscles.⁽⁵⁾

PERINEURAL SPREAD FROM NASOPHARYNGEAL CARC INOMA:

The intracranial extension may occur directly via the skull base erosion or by perineural spread along branches of the trigeminal nerve. Tumour can extend through the petro-occipital synchondrosis and foramen lacerum into the inferior CS or via the carotid canal to gain access to the CS without destroying bone. The tumour is generally hypointense to iso-intense on T1-weighted images and T2 hypointense and shows moderate-to-intense contrast enhancement. ⁽⁵⁾(Fig 6).

CAVERNOUS SINUS THROMBOSIS

Cavernous sinus thrombosis most often occurs as a complication of bacterial or fungal sepsis in the paranasal sinuses, the face, the orbits, and the skull base. Imaging signs of cavernous sinus thrombosis include a bulky cavernous sinus, a convex configuration of the lateral wall, and filling defects within the sinus. Indirect signs that support the diagnosis of cavernous sinus thrombosis are backpressure changes such as an engorged or thrombosed superior ophthalmic vein manifest as a loss of its flow void, ipsilateral retro-orbital fat stranding, bulky extraocular muscles, and exophthalmos. T1 and T2 signal intensity is not reliable for the diagnosis of cavernous sinus thrombosis in isolation, because the signal intensity of a normal cavernous sinus is variable. Diffusion-weighted MRI can be especially useful in the acute stages of cavernous sinus thrombosis and can demonstrate diffusion restriction within the sinus itself. ⁽⁷⁾(Fig 7)

ICA ANEURYSM

At CT, cavernous ICA aneurysms are hyperattenuating and can show peripheral calcifications. The signal intensity within the aneurysm on MR images can be highly variable and depends on the patency and flow rate through the aneurysm. On T2-weighted MR images, the aneurysm can be seen as a flow void or can be heterogeneously hyperintense owing to slow flow or partial thrombosis. Digital subtraction angiography plays an important role in planning management of cavernous ICA aneurysms. It is important to note that the signal intensity of the lesion on MR images can be misleading, and the clue to the diagnosis is the round shape of the lesion and its continuity with the ICA⁽⁷⁾(Fig 8)

SPHENOID SINUS CARCINOMA:

Malignant tumours of the sphenoid sinus include squamous cell carcinoma and adenocarcinoma. They tend to destroy bone and directly spread to the CS. Sphenoid sinus carcinomas typically have low-to-intermediate T1 signal intensity and low T2 signal intensity and show contrast enhancement.⁽¹⁾

METASTASIS:

Metastasis to the cavernous sinus can occur by way of hematogenous, perineural, or direct vascular spread. Head and neck malignancies are the most common ones to metastasize to the cavernous sinus. Distant tumours that metastasize to the cavernous sinus are lung, breast, renal, and gastric malignancies. Diagnosis of a metastatic lesion should be given consideration especially when a lesion with aggressive features, such as bone destruction, is encountered(7). MR imaging shows CS enlargement, the convexity of its lateral wall, and replacement of the Meckel cave with soft tissue that homogeneously enhances. Perineural tumour spread is commonly seen along branches of V nerve in patients with adenoid cystic or squamous cell carcinoma, lymphoma, melanoma, basal cell carcinoma, rhabdomyosarcoma, neurogenic tumours, and juvenile angiofibroma. MR imaging features of perineural tumour spread include nerve enlargement and enhancement and foraminal enlargement and destruction.^(5,10)

CASES

Fig1: En plaque Meningioma of left cavernous sinus appears isointense to grey matter on T1 and T2 and shows intense homogenous enhancement in post contrast study

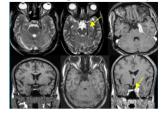


Fig 2: Isointense Pituitary macroadenoma showing moderate enhancement with suprasellar extension and invasion of right cavernous sinus with right ICA encasement.

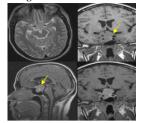


Fig 3:Epidermoid cyst of left cavernous sinus with suprasellar extension - Hypointense on T1 and hyperintense on T2 and shows diffusion restriction on DWI

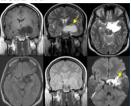


Fig 4:Cavernous angioma appearing homogenously hyperintense on T2 and FLAIR and hypointense on T1.Much variable presentation from a typical intraparenchymal cavernous angioma

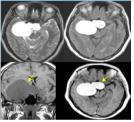


Fig 5:Chordoma:Hyperintense on T2 and hypointense on T1 with Cavernous invasion

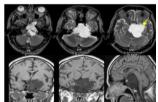


Fig 6: Heterogenously enhancing nasopharyngeal carcinoma on left with perineural spread to Cavernous sinus through Foramen ovale.

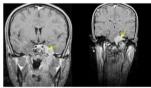


Fig 7:Left Cavernous ICA thrombosis causing fronto-parietal infarct.

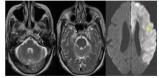
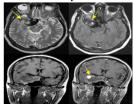


Fig 8:Right cavernous ICA aneurysm



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V. CONCLUSION

Cavernous sinus has a complex anatomy with wide spectrum of pathologies .MRI is the imaging modality of choice and helps in differentiating various pathologies.

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