Imaging Lesions of the Cavernous Sinus

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Authors: A. ARORA¹, R. BHUTANI², A. KAPOOR³, L. UPRETI⁴, S. K. PURI²; ¹NEW DELHI, DE/IN, ²NEW DELHI/IN, ³DELHI/IN, ⁴New Delhi/IN
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Learning objectives

To illustrate the diverse spectrum of pathologic conditions which commonly affects the cavernous sinus (CS) and elucidate their characteristic radiologic findings.

Background

The cavernous sinus (CS) is a small but complex structure containing vital neurovascular structures that may be affected by a wide spectrum of infective, non-infectious inflammatory, neoplastic, vascular, and infiltrative lesions. These lesions may arise in the cavernous sinus proper or may secondarily infiltrate the cavernous sinus.

Role of imaging is to throw light on the nature of lesion, its relationship to crucial neurovascular structures, and its extension into the adjoining structures. These details are imperative for choosing therapeutic modalities such as surgical intervention, radiation therapy, or medical treatment.

Since CT and MR imaging features of many of these lesions overlap and are often nonspecific, we will highlight those imaging features that allow one to formulate a reasonable differential diagnosis.

Imaging findings OR Procedure details

The imaging findings of relatively common lesions involving the cavernous sinus (CS) are discussed.

These lesions are divided into

(1) Neoplastic (Primary): Meningioma, Neurogenic Tumors, and Hemangioma

(2) Neoplastic (Secondary): Direct invasion of intracranial tumors such as Pituitary Adenoma, or invasion from Nasopharyngeal Carcinoma, Juvenile Nasopharyngeal Angiofibroma and Clival Chordoma.
(3) **Infectious:** Cavernous Sinus Thrombophlebitis, Sinonasal Fungal infiltration, Tuberculous Pachymeningitis

(4) **Inflammatory Non-infectious:** Tolosa-Hunt syndrome, Inflammatory Pseudotumor, Idiopathic Hypertrophic Pachymeningitis

(5) **Granulomatous:** Sarcoidosis

(6) **Vascular:** Internal Carotid Artery Aneurysm, Carotid-Cavernous Fistula, Thrombosis

(7) **Congenital Cysts:** Epidermoid or Dermoid Cyst

**NEOPLASMS:**

The most common neoplastic lesions in the cavernous sinus are caused by direct invasion of intracranial tumors such as pituitary adenoma, perineural spread of head and neck malignancy, or hematogenous spread from distant lesions [1,2]. However, primary tumors such as meningioma, neurogenic tumors, and hemangioma can also arise from the cavernous sinus itself.

**PRIMARY NEOPLASMS:**

**CAVERNOUS SINUS MENINGIOMA:** Meningiomas are the most common primary tumor involving the cavernous sinus [3]. Most cavernous sinus meningiomas arise from the lateral dural wall (Fig 1), but sometimes they may be exclusively inside the sinus (Fig 2) [1,3]. A meningioma is isointense with respect to gray matter at all MR imaging sequences and enhances intensely and homogeneously (Fig 2). A dural tail frequently is seen extending away from the edge of the tumor and often into the ipsilateral tentorium. Meningiomas characteristically constrict the lumen of the internal carotid artery.

**CAVERNOUS SINUS SCHWANNOMA:** A trigeminal nerve schwannoma commonly involves the CS and, in 50% of instances, has a typical dumbbell-shape (Fig 3) with bulky tumor in the Meckel cave and the prepontine cistern with a waist at the porous trigeminus [1,4]. Conversely, it may be found only involving the Meckel cave. It may be solid or have variable cystic or hemorrhagic components with occasional fluid levels. Small tumors tend to be homogeneous, whereas large ones are frequently heterogeneous in
appearance. Schwannomas are isointense-to-hypointense masses on T1 images, mostly T2 hyperintense, and show contrast enhancement (Fig 4). A clue to the diagnosis is that they follow the expected course of the nerves from which they arise [1,4].

CAVERNOUS SINUS HEMANGIOMA: Cavernous sinus hemangiomas are the third most common primary neoplasm of the cavernous sinus after meningioma and schwannoma [1,5]. Cavernous sinus hemangiomas (CSH) are frequently misdiagnosed as meningioma or schwannoma at pre-operative imaging. The need for accurate preoperative diagnosis of CSHs is important, because these lesions are notoriously difficult to surgically excise because of their location, propensity for profuse bleeding, and relationship to complex neurovascular structures. Surgical mortality rates of as much as 25% and rates of complete resection as low as 16% have been reported. At CT, CSHs are seen as a well-defined isoattenuated to hyperattenuated masses in the parasellar region with marked enhancement following contrast material administration (Fig 5). At MR imaging, CSHs are well-defined masses with low to isointense signal on T1-weighted images and markedly hyperintense signal on proton density-and T2-weighted images (Fig 5). CSHs should be carefully differentiated from meningiomas by the high signal that characterizes these lesions on T2-weighted MRI scans. The distinct T2-hyperintensity is unique for CSH, and helps in differentiating them from meningiomas which appear more isointense to the brain parenchyma on T2-weighted MR images. On contrast administration, CSHs show progressive "filling in" after contrast administration (Fig 6), similar to the centripetal 'filling in' seen with hepatic cavernous hemangiomas [1,2,5].

SECONDARY INVASION:

PITUITARY MACROADENOMA: The most common neoplastic lesions in the cavernous sinus are caused by direct invasion of intracranial tumors such as pituitary adenoma [1,6]. At CT or MR imaging, the tumor typically displays a 'figure-of-eight' appearance due to its suprasellar component. They frequently extend into the neighbouring areas such as the cavernous sinus, sphenoid sinus, and nasopharynx (Fig 7,8). A sign of cavernous sinus invasion is encasement of the intracavernous ICA by >30% of its diameter or tumor extension lateral to the top (12 o’clock) of the ICA. Interposition of abnormal soft tissue between the lateral wall of the cavernous sinus and the ICA is also a reliable indicator of cavernous sinus invasion. Unlike cavernous meningiomas, pituitary adenomas generally do not narrow the ICA [1,7].

CHORDOMA: Chordoma is a locally invasive midline primary clival tumor that may also originate slightly more laterally from the sphen-occipital synchondrosis and may extend into the CS [1]. The classic appearance at CT is that of a centrally located, well-circumscribed, expansile soft-tissue mass that arises from the clivus with associated
extensively lytic bone destruction. On T1-weighted MR images, chordomas display intermediate to low signal intensity and are easily recognized within the high signal intensity of the clival fat. They demonstrate high signal intensity on T2-weighted images and demonstrate moderate to marked enhancement. The enhancement pattern of the tumor sometimes has a "honeycomb" appearance created by intratumoral areas of low signal intensity [8,9] (Fig 9).

CHONDROSARCOMA: Chondrosarcomas are the lesions most often confused with intracranial chordomas. The majority of chondrosarcomas arise along the petro-occipital fissure i.e. away from midline. Linear, globular, or arclike calcifications when present in chondrosarcomas can help distinguish them from intracranial chordomas [8,9].

NASOPHARYNGEAL CARCINOMA: Nasopharyngeal carcinoma is the most common primary malignant extracranial neoplasm to invade the CS. Intracranial extension may occur directly via the skull base erosion or by perineural spread along branches of the trigeminal nerve (Fig 10). Tumor 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. Once the CS is invaded, bulky masses are present in the nasopharynx. The tumor is generally hypointense to iso-intense (relative to muscles) on T1-weighted images and T2 hypointense and shows moderate-to-intense contrast enhancement [1,10,11].

JUVENILE ANGIOFIBROMA: Juvenile angiofibroma is a highly vascular tumor that affects mostly adolescent boys. It can extend into the central skull base and to the anterior part of the CS through the foramen rotundum, vidian canal, or foramen lacerum. The tumor can invade the CS directly by erosion of the pterygoid bone (Fig 11). The characteristic signal-intensity voids on MR imaging, representing large vascular structures, are typical of this tumor [1].

METASTASES: Metastases to the CS can be hematogenous or perineural in nature. Distant tumors with hematogenous spread to the CS are generally renal, gastric, thyroid, lung, and breast cancers. MR imaging shows CS enlargement, outward bowing of its lateral wall, and replacement of the Meckel cave with soft tissue that homogeneously enhances (Fig 12). Perineural tumor spread is commonly seen along branches of fifth cranial nerve in patients with adenoid cystic or squamous cell carcinoma but may also be seen with lymphoma, melanoma, basal cell carcinoma, rhabdomyosarcoma, neurogenic tumors, and juvenile angiofibroma. MR imaging features of perineural tumor spread include nerve enlargement and enhancement and foraminal enlargement and destruction [1,12,13].
LYMPHOMA and LEUKEMIA: As with metastases, lymphoma and leukemia reach the CS by direct extension from a primary lesion or from hematogenous spread. MR imaging may show infiltrative lesions of the skull base invading the CS without arterial narrowing. Lymphoma and leukemia may also appear as diffuse enlargement and enhancement of the CS similar to the appearance of metastases however they tend to be T2 hypointense [1].

INFECTIOUS LESIONS:

FUNGAL INFECTION: Invasive aspergillosis may affect the sphenoid sinus in immunocompromised patients and may extend intracranially with invasion of the CS. This infection shows low signal intensity on both T1- and T2-weighted images (Fig 13), which is attributed to the presence of ferromagnetic elements and calcium in the fungal and mucous concretions [1,14]. It exhibits intense inhomogeneous contrast enhancement (Fig 14).

TUBERCULOUS PACHYMENINGITIS: Tuberculosis is a relatively common cause of pachymeningitis that produces diffuse or focal extra-axial masses along the surfaces of the CS (Fig 15). The lesions enhance homogeneously and intensely and may be nodular. The presence of dark signal on T2-weighted images, and the presence of basal meningitis elsewhere are helpful diagnostic clues. However, neurosarcoidosis may be identical in appearance. [1]

CAVERNOUS SINUS THROMBOPHLEBITIS: Thrombophlebitis of the cavernous sinus potentially is a lethal condition most commonly resulting from contiguous spread of infection from the nasal furuncle (50%), sphenoidal or ethmoidal sinuses (30%) and dental infections (10%). Less common primary sites of infection include tonsils, soft palate, middle ear, or orbit (orbital cellulitis). CT and MR imaging can provide diagnostic information with direct signs, including changes in signal intensity and in the size and contour of the cavernous sinus, presence of nonenhancing acute thrombus (Fig 16) and indirect signs, including dilatation of the tributary veins, exophthalmos, and increased dural enhancement along the lateral border of the cavernous sinus [2,15].

INFLAMMATORY NON-INFECTIOUS LESIONS:

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 [2,16]. The process is usually unilateral but may be bilateral in 5% of patients [1]. MR imaging findings include an enlarged CS containing abnormal soft tissues that are isointense to muscle on T1-weighted images and dark or bright on T2-weighted images (Fig 17) and display contrast enhancement with focal narrowing of the ICA (Fig 18) [1,2,16].

IDIOPATHIC HYPERTROPHIC PACHYMENINGITIS: Idiopathic hypertrophic cranial pachymeningitis is an uncommon inflammatory disease with diffuse dural involvement that may extend to the CS without any known etiology. MR imaging shows enhancement and dural thickening, which may be extensive or more localized (Fig 19). When the falx and tentorium are involved, extension into the CS is relatively common. This disease may lead to CS thrombosis, white matter edema, and hydrocephalus due to venous hypertension [1,17].

INFLAMMATORY PSEUDOTUMOR: These pseudotumors include a diverse group of nonspecific inflammatory lesions characterized by inflammatory cell infiltration and variable fibrotic responses according to the chronicity of the lesion. Typical MR findings include soft-tissue lesions infiltrating the skull base with intracranial dural involvement, bone destruction, iso- to hypointensity on T2-weighted images according to the fibrosis and high cellularity, and contrast enhancement (Fig 20, 21) [2,18].

GRANULOMATOUS LESIONS:

SARCOIDOSIS: The appearance of the lesions is nonspecific, but they tend to be hypointense on T2-weighted images and may be accompanied by neighbouring cerebral edema. Other findings which may raise the possibility of sarcoidosis include pachymeningitis or leptomeningeal enhancement, thickened cranial nerves, thickened hypothalamus, multiple scattered brain lesions, and periventricular multifocal white matter lesions [1].

VASCULAR LESIONS:

CAVERNOUS ICA ANEURYSM: Vascular ectasia and distal internal carotid artery aneurysms are the most common nonneoplastic parasellar masses in adults [2].
Cavernous carotid aneurysms compose 5% of giant aneurysms (>2.5 cm in diameter). A patent aneurysm shows signal-intensity void on spin-echo MR imaging sequences (Fig 22, 23). Partially thrombosed giant aneurysms show mixed signal intensities representing various stages of clot in their walls (due to chronic dissections) or within their lumen. Flowing blood through the patent portion of the lumen appears as a signal-intensity void on spin-echo images and high signal intensity on gradient technique (Fig 22) [1, 19-22].

**CAROTID CAVERNOUS FISTULA (CCF):** 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 [1]. 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 (Fig 24). 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 (Fig 25). Other supporting findings are a "dirty" appearance of the retro-orbital fat and enlargement of the extraocular muscles.

**CAVERNOUS SINUS THROMBOSIS:** CS thrombosis may be secondary to infection of the sinonasal cavities, orbits, and/or the middle third of the face. MR imaging signs of CS thrombosis include changes in signal intensity and/or in the size and contour of the CS. Enhancement of the peripheral margins of an enlarged CS may suggest a clot within it (Fig 26). Indirect signs that help to suggest the diagnosis are dilation of the superior ophthalmic veins, exophthalmos, and increased dural enhancement along the lateral border of CS and ipsilateral tentorium [1,23].

**CONGENITAL LESIONS:**

**EPIDERMOID/DERMOID CYST:** An epidermoid cyst may be of extracavernous origin and extend into the CS, originate in the lateral CS wall (interdural cyst), or be a true intracavernous lesion. The mass is T1-hypo and T2-hyperintense [1, 24]. On FLAIR images, it demonstrates heterogeneous signal intensity (Fig 27). These cysts do not enhance. Epidermoid cysts show restricted diffusion with higher signal intensity than that of CSF on diffusion-weighted imaging (Fig 28).

Images for this section:
An 18-year old girl with NF-2 shows a homogeneously enhancing MENINGIOMA from the LATERAL DURAL WALL of the right cavernous sinus (arrow). Also seen is an intra-orbital optic nerve sheath meningioma.

Fig. 1: Cavernous Sinus Meningioma
Fig. 2: Cavernous Sinus Meningioma

A 40-year old lady with CS meningioma. A HOMogeneously enhAnCing mass is seen on the righT side which shows isoIntense signal to the cortex on T1- and T2-weighted MRI and is causing mild constriction of the internal carotid artery.
A 50-year old male with diplopia. A DUMBBELL-SHAPE TUMOR is seen in the Meckel’s cave and the prepontine cistern on the right side with a waist at the porus trigeminus.

Fig. 3: Trigeminal Schwannoma
It shows a MIXED SOLID and CYSTIC appearance and a moderate heterogeneous contrast enhancement due to the presence of intralesional cystic areas.

Fig. 4: Trigeminal Schwannoma
A large well circumscribed T1-hypo and T2-HYPERINTENSE CS MASS is seen on the right side in a 48-years old lady with ptosis & ophthalmoplegia. It shows avid enhancement on the CT scan.

Fig. 5: Cavernous Sinus Hemangioma
CAVERNOUS SINUS HEMANGIOMA

It shows a progressive CENTRIPETAL contrast ‘FILLING IN’ on the dynamic contrast-enhanced MRI scan.

**Fig. 6:** Cavernous Sinus Hemangioma
A large sellar mass extending into the suprasellar cistern is noted. Indentation at the diaphragma sellae gives rise to a “FIGURE OF 8” configuration.

Fig. 7: Pituitary Macroadenoma
It shows homogeneous enhancement and is seen invading into the right cavernous sinus with encasement of the internal carotid artery.

**Fig. 8:** Pituitary Macroadenoma
A large solid-cystic locally invasive mass is seen in the midline skull base. It is invading the nasopharynx, sella turcica & the cavernous sinus.

**Fig. 9:** Clival Chordoma
A 66-year old male with nasopharyngeal carcinoma. The is an aggressive mass which is infiltrating the adjacent structures including the right cavernous sinus.

Fig. 10: Nasopharyngeal Carcinoma
A 15-year old boy with epistaxis. A large hypervascular nasopharyngeal mass is seen invading the right cavernous sinus.

Fig. 11: Nasopharyngeal Angiofibroma
45-year old lady with breast carcinoma complaining of headache and ptosis. Enlargement and lateral bowing of the right cavernous sinus suggests the diagnosis of metastases.

Fig. 12: Metastases
Aspergillosis in 60-year-old diabetic lady who presented with diplopia. Axial T2-weighted images show HYPOINTENSE lesion in right spheno-ethmoid sinus which extends into the cavernous sinus and the ipsilateral orbit.

Fig. 13: Invasive Aspergillosis
Aspergillosis in 60-year-old diabetic lady who presented with diplopia. Contrast enhanced T1-weighted scan shows a heterogeneous enhancement.

Fig. 14: Invasive Aspergillosis
A 32-year old male previously treated for Tuberculous meningitis presented with recurring headaches. Contrast enhanced T1-weighted scan shows a focal pachymeningeal thickening & enhancement along the left lateral CS wall.

**Fig. 15:** Tuberculous Pachymeningitis
A 16-year old boy with frank periodontal abscess presented with unilateral photophobia. Contrast enhanced T1-weighted scan shows a small nonenhancing hypointense focus within the anterior left CS presumed to be a clot representing CS thrombophlebitis.

Fig. 16: Cavernous Sinus Thrombophlebitis
A 25-year old male with ophthalmoplegia. Axial T2-weighted images show CS enlargement with MARKEDLY HYPOINTENSE soft tissue throughout the bilateral CS. Associated narrowing of the intracavernosal ICA is present.

Fig. 17: Tolosa-Hunt Syndrome
Post contrast T1-weighted images show that the abnormal soft tissue enhances prominently and diffusely. Extension into the superior orbital fissure and left orbital apex is also seen. Pachymeninges along the cavernous sinuses & tentorium are thickened. A remarkable response was seen following a course of steroids.

Fig. 18: Tolosa-Hunt Syndrome
Coronal postcontrast T1-w images exhibit NONSPECIFIC PACHYMENINGEAL THICKENING in the anterior cranial fossa & also the middle cranial fossa along the bilateral CS.

Fig. 19: Idiopathic Hypertrophic Pachymeningitis
Axial T2-w images show an infiltrative mass (arrows) in right orbit and right cavernous sinus causing narrowing of the ICA. Note MARKED HYPOINTENSITY on T2-weighted image.

Fig. 20: Inflammatory Pseudotumor
Fig. 21: Inflammatory Pseudotumor

It shows AVID ENHANCEMENT following contrast medium administration. Associated pachymeningeal thickening is also seen.
A 48-year old lady with ptosis. Axial T2-weighted image shows a LARGE SIGNAL VOID due to aneurysm of internal carotid artery in the left cavernous sinus.

Fig. 22: Cavernous Carotid Aneurysm
Fig. 23: Cavernous Carotid Aneurysm

Prominent FLOW ARTIFACTS are seen in the left cavernous ICA region on the coronal noncontrast enhanced MR angiogram image.
Contrast enhanced MR imaging shows DILATED LEFT SUPERIOR OPHTHALMIC VEIN and engorgement of the left cavernous sinus. Outward bulging of the lateral wall is also seen.

**Fig. 24:** Carotid Cavernous Fistula
Fig. 25: Carotid Cavernous Fistula

An ABNORMAL TUFT of vessels is seen in the left cavernous sinus (arrow) with faint visualization of the left superior ophthalmic vein (arrowhead) on the MR angiogram suggesting an abnormal arteriovenous communication.
Coronal postcontrast T1-weighted image shows an enlarged and inhomogeneous-appearing right CS that contains AREAS OF LOW SIGNAL INTENSITY (arrow) compatible with clot.

**Fig. 26:** Cavernous Sinus Thrombosis
A sharply demarcated NONENHANCING CSF-SIGNAL INTENSITY MASS is seen in the right CS. Intralesional HETEROGENEITY is seen on the FLAIR image.

Fig. 27: Congenital Epidermoid Cyst
Fig. 28: Congenital Epidermoid Cyst

This shows no significant contrast enhancement. RESTRICTED DIFFUSION is seen on the Diffusion Weighted imaging (DWI).
Conclusion

Cavernous sinus can be involved by various disease entities. Understanding the distinguishing radiologic features of different cavernous sinus lesions can assist in clinching the correct diagnosis and formulating a reasonable differential diagnosis.

Personal Information

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