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Pictorial Review

# The imaging of conditions affecting the cavernous sinus

Y. Tang*, T. Booth, M. Steward, T. Solbach, T. Wilhelm

*Royal Free Hospital, Pond Street London NW32QG, UK*

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The cavernous sinus can be affected by a wide range of conditions including tumours, infection, inflammation, and trauma. Disease in the cavernous sinus can produce characteristic signs and symptoms, which relate to the numerous crucial structures traversing and surrounding the cavernous sinus. Imaging, with the use of different techniques, plays a crucial role in diagnosis and management. The anatomy and imaging of the different disease entities in the cavernous sinus will be reviewed.



## Introduction

The cavernous sinus (CS) is a complex, paired structure found on either side of the sella turcica. It was first described by Wepfer when describing the intracavernous internal carotid artery (ICA) as passing through a "deep and conspicuous space."[1] Within the CS are multiple crucial neurovascular structures. When affected by disease, they give rise to CS syndrome. Patients with CS syndromes may present with ophthalmoplegia, chemosis, proptosis, and Horner's syndrome. Conditions affecting the CS are wide ranging and include congenital epithelial cysts, acquired infections, inflammatory disorders, vascular lesions, and neoplasms. Imaging can often provide a diagnosis,[2] which helps determine the need and approach for surgical or endovascular intervention. In this review, pathological conditions that can affect the CS and their imaging findings are reviewed.

## Anatomy

The CS is a complex and loculated venous space, which lies between periosteal and meningeal layers of dura adjacent to the sphenoid bone. It is a paired structure lying on either side of the sella turcica. The two halves communicate freely via the two intercavernous sinuses, which pass anterior and posterior to the pituitary gland. The CS extends anteriorly from the superior orbital fissure to the apices of the petrous temporal bones. Superiorly is the diaphragm sellae and inferiorly is the greater wing of sphenoid. The sinus is surrounded laterally by dura. As there are no lateral bone boundaries, all diseases of the CS cause volume expansion and thus lateral bulging of its walls,[3] which is best seen on coronal examinations.

The symptoms of CS syndrome correlate with the involved structures of the venous space. Medially within the CS is the ICA with its periarterial sympathetic plexus, involvement of which may cause Horner's syndrome. Inferolateral to the ICA is the abducens nerve. Within the lateral dural border of the CS lie the ophthalmic and maxillary division of the trigeminal nerve, oculomotor nerve, and trochlear nerve. (Figs. 1 and 2) Involvement of these cranial nerves can cause ophthalmoplegia or facial sensory loss. Compromise of venous drainage may give rise to chemosis and proptosis. The CS has complex, valveless, venous communications. It communicates directly or indirectly with almost every important venous structure in the head and neck. Draining into the CS are the superior and inferior ophthalmic veins, sphenoparietal sinuses, and middle meningeal vein. The CS communicates with the facial vein and pterygoid venous plexus via the superior and

* Guarantor and correspondent: Y. Tang, Royal Free Hospital, Pond Street London NW32QG, UK.
*E-mail address:* yenzhitang@doctors.net.uk (Y. Tang).

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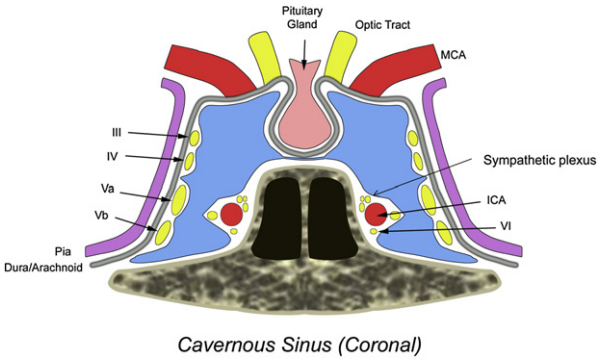


**Figure 1** Anatomic diagram of the CS in a coronal view demonstrating the intracavernous structures.

inferior ophthalmic veins. This communication explains how CS thrombosis may result from facial infections. Draining from the CS are the superior and inferior petrosal sinuses, which eventually drain into the sigmoid sinus and internal jugular vein. The CS are connected via two intercavernous sinuses.

## Neoplasms

### *Meningiomas*

Meningiomas are the most frequent tumour in the CS.[4] They have a broad base attachment, are isohypointense on T1-weighted images, and demonstrate early, intense, homogeneous enhancement (Fig. 3). They are isohyperintense on T2-weighted imaging. They may demonstrate mass effect by constricting the lumen of the ICA.[5] Adjacent hyperostosis is characteristic, and a dural tail is seen in 60%. Meningiomas, although resectable, are more difficult to excise from the CS compared to other benign tumours as they invade the adventitia of the intracavernous ICA and nerves.[6–8] There may be heterogeneous signal within the meningioma due to calcification, cystic components, or haemorrhage.[9]

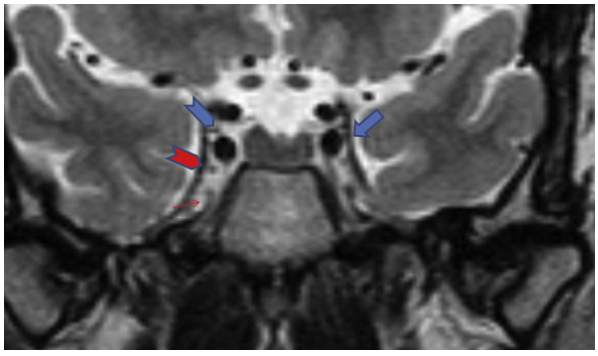

**Figure 2** Coronal, T2-weighted sequence through the CS. The oculomotor nerve (arrowhead), trocholear nerve (blue arrow), and abducens nerve (red arrowhead) are seen within the CS. The exiting mandibular nerve (red arrow) is also seen although it is not within the CS.

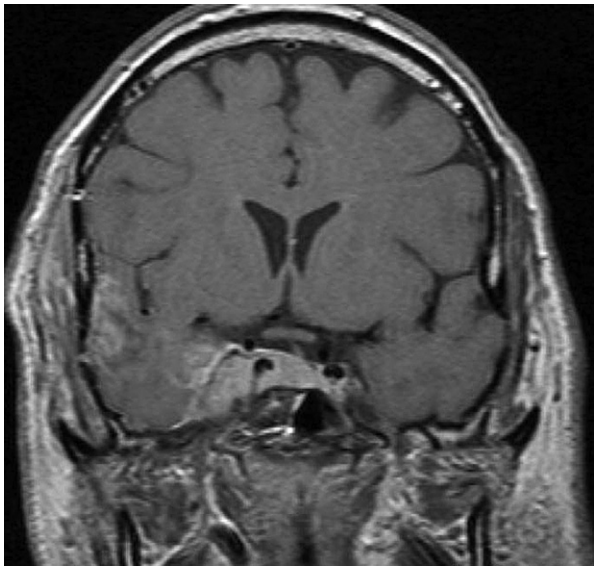

**Figure 3** Coronal, T1-weighted, contrast-enhanced image demonstrating a homogeneously enhancing meningioma encasing the right internal carotid artery which is of normal calibre.

### *Schwannomas*

Schwannomas have a propensity to involve sensory nerves, and if involving the CS, commonly arise from the trigeminal nerve. They demonstrate contrast enhancement, and when straddling a narrow passage, e.g., a foramen, they have a characteristic dumbbell or saddle-shaped configuration. Schwannomas are positioned posteriorly within the CS and may show extension into the posterior fossa, or erosion of the foramen ovale, helping to differentiate them from other tumours.[2] Signal intensity is usually dependant on tumour size, with smaller tumours being homogeneous and larger tumours being heterogeneous. They are isohyperintense on T1-weighted images, and enhance with contrast media. They are generally hyperintense on T2-weighted imaging (Fig. 4).

### *Adenomas*

Pituitary adenomas can laterally expand to compress or invade the CS. Here, 6–10% of pituitary adenomas involve the CS[10] often unilaterally.[11] They are hypointense on T1-weighted imaging and hyperintense on T2-weighted imaging. (Fig. 5) Some heterogeneity may be seen on T2-weighted images reflecting cystic and necrotic parts within the tumour. The adenoma enhances slightly post-contrast medium administration, but less than surrounding normal pituitary tissue.[12] Diagnosis of CS invasion with imaging is important as clinical signs are delayed due to the lateral position of the cranial nerves within the CS. Invasion of the CS complicates surgical resection, and additional treatment may be required. Total encasement of the intracavernous ICA reliably diagnoses CS invasion, but is a late sign.[13] Reliable signs for earlier invasion, as well as non-invasion, are listed in Table 1.[11,14]