The cavernous sinus is one of the most important dural venous sinuses located between the endosteal dura and meninges. The roof of the sinus is formed by the inner layer of the meningeal dura, which is continuous with the diaphragma sellae covering the pituitary gland.

Overview

The roof of the sinus is attached anteriorly to the anterior and middle clinoid processes and posteriorly to the tentorium as it connects to the posterior clinoid process.

The floor of the sinus is formed partly by the periosteum of the greater wing of the sphenoid and endosteal dura mater.
The lateral and medial wall is formed by the **meningeal dura**.

The interior of the sinus is separated into caverns or spaces by **trabeculae**. These trabeculae are less prominent in the living organism than in cadavers. The sinus is called cavernous due to its **cavernous appearance in cadavers** when its cavity collapses and the nerves and arachnoid granulations in its walls encroach into the cavity.

The cavernous sinus is a **true dural venous sinus** and not a venous plexus. It is clinically important because of its location, its close relationship to several **cranial nerves** and the **internal carotid artery**, and the complex of veins without valves which drain from and to the paired cavernous sinuses.
Location of the Cavernous Sinus

It is located in the middle cranial fossa, on either side of the sella turcica or pituitary fossa and the body of the sphenoid at the base of the skull. Usually, the paired cavernous sinuses are situated superolateral to the sphenoid or the posterior ethmoid sinuses and posterior to the optic chiasma.

The sinuses have an irregular shape, and each sinus is formed within layers of the dura. Normally, its lateral wall can be either concave or straight and is formed by the visceral dural mater.

Anatomy of the Cavernous Sinus

Relations

Anterior: superior orbital fissure
Posterior: apex of the petrous temporal bone
Superior: internal carotid artery, optic tract and optic chiasma
Lateral: uncus of the temporal lobe
Medial: the body of the sphenoid and sella turcica

Contents

The internal carotid artery (ICA) enters the posterior inferior aspect of the cavernous sinus forming the cavernous part of the artery. Then it travels horizontally anteriorly within the sinus.

Once it reaches the anterior wall of the sinus, it traverses vertically upwards towards the roof of the sinus and exits to form the cerebral part. It gives rise to two branches within
the sinus: the meningohypophyseal branch and the inferolateral branch. It is clinically significant that this part of the internal carotid artery is the only artery which is surrounded by a network of veins.

**Sympathetic nerves** around the carotid plexus: these nerves arise from the superior cervical ganglion and surround the cavernous part of the ICA.

The abducent nerve (CN VI) traverses the sinus inferolateral to the ICA and exits by entering the superior orbital fissure anteriorly. It innervates the lateral rectus muscle once it reaches the orbit.

The oculomotor nerve (CN III) is the most superior nerve in the lateral wall of the sinus. As it reaches the anterior wall of the sinus, it divides into superior and inferior branches, which pass through the superior orbital fissure. CN III and the sympathetic plexus around the ICA innervate the levator palpebrae superioris, the inferior oblique, and superior, medial and inferior recti muscles of the orbit.

The trochlear nerve (CN IV) lies in the lateral wall of the sinus, below CN III. It leaves through the anterior wall of the sinus and enters the superior orbital fissure. It supplies the superior oblique muscle in the orbit.

The ophthalmic nerve and maxillary nerve are branches of the trigeminal nerve (CN V). They lie below CN IV in the lateral wall of the cavernous sinus. The ophthalmic branch exits via the superior orbital fissure, while the maxillary nerve exits the sinus via the foramen rotundum.

**Fat:** in obese patients, and in patients taking corticosteroids, fatty deposits may occasionally be seen within the cavernous sinus.

**Connections**

![Image: "Anatomical preparation. Cavernous sinus." by Anatomist90 – Own work. License: CC BY-SA 3.0](Image)

**Tributaries or incoming veins:**

- Superficial middle cerebral vein
- Inferior cerebral vein from the temporal lobe
- Intercavernous sinus, which connects the cavernous sinuses on either side
- Inferior and superior ophthalmic veins (which drain the facial veins) from the orbit
- Sphenoparietal sinus from the meninges
Central retinal vein, which may drain into the superior ophthalmic vein.
- Tributary of the middle meningeal vein, which may drain into the pterygoid plexus or sphenoparietal or cavernous sinus.

**The cavernous sinus drains via:**
- Venous plexus around the ICA to the basilar venous plexus
- Superior petrosal sinus to the sigmoid sinus
- Inferior petrosal sinus to the internal jugular veins
- Emissary veins passing through various foramina in the skull base, e.g., foramen ovale, foramen lacerum, and sphenoidal foramen

Since the superior ophthalmic veins and the other complex of veins do not have valves, the blood flow is bidirectional depending on the pressure gradients. The cavernous sinus receives blood via this bidirectional route and hence infections from the mid-face, nose, paranasal sinuses, orbits, tonsils, and even the middle ear can spread to it easily.

**Clinical Relevance of the Cavernous Sinus**

**Cavernous sinus thrombosis (CST)**

This condition was first described in 1831 by Bright. It is a late complication of infection in the dangerous (central) area of the face (e.g., furuncle on the nose, dental caries, etc.) or paranasal sinuses and a medical emergency requiring urgent management with a high incidence of morbidity and mortality.

With the advent of broad-spectrum antimicrobial drugs, the incidence of CST has decreased markedly. The infection may cause thrombosis within the facial veins and when the clot breaks off and travels to the cavernous sinus, it can cause CST.

Also, due to the close relationship of the cavernous sinus to the paranasal sinuses, the orbit, the complex of veins, cranial nerves, internal carotid artery and meninges, and the absence of valves in the veins draining to and from the cavernous sinus, infection from draining tissues can result in cavernous sinus thrombosis.

This is characterized by edema of the eyelids, conjunctiva, and paralysis of the cranial nerves intimately related to the cavernous sinuses.

The common organisms involved in CST include Staph aureus, Strep pneumococcus, gram-negative bacteria, anaerobes as well as fungi like Rhizopus and Aspergillus.

**Diagnosis of CST** is done clinically and confirmed with either CT scan or MRI with MR venogram, which is the study of choice.

Treatment consists of empirical broad-spectrum antibiotics with corticosteroids to reduce edema. Surgery may be required to drain/clean infected material from the paranasal sinuses. Delay in diagnosis and treatment is associated with high morbidity and mortality. Patients who survive may have a visual impairment or cranial nerve deficits.

**Cavernous sinus syndrome (CSS)**

The pituitary gland is located in a fossa between the two cavernous sinuses. As Pituitary tumors grow, they can expand towards and later compress the cavernous sinus. This can lead to cavernous sinus syndrome, which is characterized by
ophthalmoplegia (paralysis of CN III, IV, VI), loss of sensation in the region of the ophthalmic and maxillary nerves as well as Horner’s syndrome due to compression of the sympathetic plexus around the internal carotid artery.

CSS can also be caused by tumors extending from the nasopharynx, pituitary or metastasis or even following CST.

Carotico-cavernous fistula (CCF)

CCF is formed due to an unnatural direct communication between the cavernous sinus and the ICA traversing through it. This direct fistula is formed due to either trauma or rupture of an aneurysm.

Arterial dissection, collagen vascular diseases like Ehler Danlos and fibromuscular dysplasia could also be causes of CCF.

Patients may present, among other symptoms, with pulsatile proptosis, orbital congestion, chemosis, corneal exposure, diplopia, paralysis of CN III, IV, VI, and retinopathy.

CT angiography is the test of choice.

Usually, these fistulae resolve spontaneously. Persistent symptomatic fistulae require treatment, which consists of steroids in the acute phase to reduce edema, followed by definitive surgery.

Endovascular approaches with obliteration of the fistula and restoration of arterial and venous flow lead to resolution of the fistula.

Triangular space near the cavernous sinus

Parkinson, in 1965, described a triangular space between the ophthalmic and trochlear nerves, to approach lesions around the cavernous part of the ICA. With the advent of radiosurgery and endovascular surgery, this direct approach through the triangular space is rarely required.

If endovascular surgery or occlusion of carotico-cavernous fistula fails, then direct surgery through this triangular space may be necessary. Also, in the case of certain tumors like meningiomas, schwannomas, pituitary adenomas, and chondromas, neurosurgeons may have to gain access to the tumors through this triangle.

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