CONTINUING EDUCATION PROGRAM: FOCUS...

No more fear of the cavernous sinuses!

F. Charbonneau, M. Williams, F. Lafitte, F. Héran*

* Corresponding author.
E-mail address: fraheran@gmail.com (F. Héran).

Imaging Department, A. de Rothschild Ophthalmological Foundation, 25-29, rue Manin, 75940 Paris cedex 19, France

Abstract  After a review of the anatomy of the cavernous sinuses (CS), this work presents the clinical picture and imaging protocol of lesions which occur in this area. It outlines extension and imaging features of these lesions. It emphasises MRI appearance, such as T1, T2 and diffusion signal, type of contrast medium uptake. A complementary CT scan is performed if an associated abnormality of the base of the skull is suspected on MRI (lysis, condensation). This paper proposes a straightforward classification system depending on imaging and sets out the principal symptoms of the main aetiologies of CS lesions which are represented by various diseases such as tumours, inflammations, vascular abnormalities. Complementary to imaging, their diagnosis is based on clinical data i.e. known cancer, signs suggesting inflammation. Its rich iconography allows this article to be used as a reference in current clinical practice.

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The cavernous sinuses are located on both side of the sellar area. Pathways of communication between orbit, face and brain, they contain many neurovascular elements.

The purpose of this article is to show how anatomical understanding and systematic analysis of the cavernous sinuses are helpful to make the diagnosis of the main lesions found in this area (vascular, inflammatory, neoplastic) and to explain their diversities and functional effects.

Anatomical review and correlation between anatomy and MRI [1—3]

The cavernous sinuses are osteo-duromeningeal compartments located on both side of the sellar area (Fig. 1). They communicate via the superior orbital fissure with the orbit, via the foramen rotundum and the inferior orbital fissure with the pterygopalatine fossa and via the oval foramen with the infratemporal fossa and the masticator space.
Their inferior and lateral walls and their roof are extensions of the dura mater. The medial wall is either nonexistent or incomplete and consists then of a thin layer of collagen. They contain vascular structures: the carotid siphon, surrounded by sympathetic fibres and venous plexuses receiving the superior ophthalmic vein, cerebral veins (median, inferior) and the spheno-parietal sinus. These veins drain backwards via the inferior and superior petrous sinus in the transverse sinus and the jugular bulb. They also contain cranial nerves (CN) III (oculomotor nerve), IV (trochlear nerve), branches of the V (trigeminal nerve): V1 (ophthalmic) and V2 (maxillary), which run from top to bottom in the lateral wall of the cavernous sinus and V1 (abducens nerve) which follows the length of the lower and lateral sides of the intracavernous portion of the carotid siphon. Due to rapid flux, the internal carotid artery appears as a hypointense (flow void) structure on spin echo T1 (with or without injection) and T2 sequences and as a hyperintense structure on MRA (TOF without or with injection) sequences. The venous plexuses have a signal that varies as a function of their flux (slow flux shows as hyperintense, fast flux shows as hypointense). They are homogeneously enhanced after injection. The cranial nerves (CN) appear as structures with an isosignal relative to the white matter in T1 and T2 spin echo. They are not enhanced after injection. CN IV and VI are more difficult to visualise with MRI than CN III due to their smaller size and their location within the cavernous sinuses.

**When should impairment of the cavernous sinuses be suspected? [3]**

**Ophthalmological impairment**

Due to the elements that go through the cavernous sinuses and surrounding structures, cavernous sinus pathology should be considered when an ophthalmological symptomatology is discovered (Fig. 2). The clinical symptoms combine various types of signs:

- oculomotor disorders (binocular diplopia) due to impairment of one or more ocular nerves affecting, in descending order, III, VI and IV and reaching in severe cases a total ophthalmoplegia (in 30% of cases), sometimes painful, especially if inflammatory [4]. Notice that isolated impairment of III, appearing suddenly and associated with headache, should systematically and in emergency lead to investigate a ruptured aneurysm.
inducing III impairment (particularly if this aneurysm is developed on the posterior side of the carotid siphon, at the origin of the posterior communicating artery);
• visual acuity loss, related to compression of the anterior visual pathways (optic nerve or optic chiasm), due to either the superior development of the lesion or to the compression of the optic nerve within the orbital apex by the anterior extension of the lesion via the superior orbital fissure, found in 40% of cases. Recent alteration of visual acuity due to a compression requires in emergency a neurosurgical consultation;
• Claude Bernard-Horner Syndrome due to peri-carotid sympathetic plexus impairment and associating ptosis and myosis, much more rare (4% of cases);
• exophthalmos painful or painless, pulsatile or non-pulsatile, depending on its etiology;
• chemosis due to venous swelling or inflammation.

Other clinical symptoms
Other clinical symptoms suggestive of a lesion of the cavernous sinuses may develop: paraesthesia and neuralgia by impairment of CN V, retro-orbital pain and headache, disturbances of the hypothalamic-pituitary axis, neurological deficiencies due to brainstem or acoustic-facial bundle compression, seizures complicating temporal irritation caused by posterior or lateral extension of the lesion.

Cerebral ischemia by compression of the internal carotid artery is exceptional.

How to investigate a lesion of the cavernous sinuses? [5]
The purpose of imaging
The purpose of imaging is to specify the nature of the lesion, to assess its extensions following various spatial planes (Fig. 3) and its effects (direct or indirect) on orbital components [6].

Investigation of the cavernous sinuses by MRI
The MRI protocol associates coronal thin slices (2—3 mm) on T2 spin echo and T1 spin echo after contrast medium injection covering the cavernous sinuses and orbits, and axial thin slices on T1 spin echo with injection and fat saturation, in some cases completed by axial thin slices on T2 spin echo.

The T2 spin echo coronal study covering the cavernous sinuses and orbits allows depiction, whether they exist, of:
• effects of the lesion on the anterior visual pathways: compression of optic chiasm, compression, signal abnormality and atrophy of the optic nerves, dilation of the optic nerve sheath (Fig. 4);
Figure 3. Extension of lesions of the cavernous sinus: a: at the top near the chiasma (yellow arrows) and optic nerves; b: laterally (blue arrows) towards the temporal lobe outwardly, the sellar region inside; c: at the bottom towards the infratemporal fossa via the foramen ovale (red arrow); d: to the front via the superior orbital fissure (green arrow); e: at the bottom and to the front towards the pterygopalatine fossa via the foramen rotundum and the inferior orbital fissure; f: at the back (pink arrows) towards Meckel’s cavum and the meanings of Pacchioni’s foramen ovale.
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Figure 4. Right supracavernous carotid aneurysm responsible for compression of the optic nerve in its cisternal portion (a, red arrow). Atrophy and T2 hypersignal of the right optic nerve in its orbital portion (b, blue arrow). MRA TOF (c).

- direct effects (compression, invasion) or indirect compression (venous swelling, denervation) on the oculomotor muscles (hypertrophy or atrophy with abnormal high T2 signal of muscle);
- dilation of the superior ophthalmic vein, due to impaired drainage (Fig. 5).

T1 axial study with injection and fat saturation sequence provides an excellent assessment of orbital extension of the lesion.

Investigation of an inferior extension into the pterygopalatine fossa via the foramen rotundum (and inferior orbital fissure) and exophthalmos is performed in the axial plane.

Thin T2 spin echo axial sequence may be very useful in identifying minor vascular impairment of the cavernous sinuses.

Vascular investigation by MRA TOF should be performed in case of a sudden onset of CN III impairment and acute headache (suspicion of ruptured aneurysm), if a carotid-cavernous, a dural fistula or compression of the internal carotid artery are evocated (Fig. 6). Complementary dynamic MRA or arterial angioscan offer a precise depiction of carotid-cavernous or dural fistula (Fig. 7).

Orbital echo-Doppler

This examination proves to be very useful to explore vascular abnormalities of the cavernous sinuses as it detects indirect signs of fistula consisting of inversion and arterialisation of the flux in a dilated superior ophthalmic vein (Fig. 8) [7].

CT scan

CT scan should be carried out to study the skull base. It provides arguments to differentiate identical tissular lesions, due to their specific bone involvement i.e.; hyperostosis in case of meningioma, lysis caused by metastases and other malignant lesions, It depicts microcalcifications (chondrosarcoma), It is mandatory to evaluate facial bones or base of the skull impairment due to the lesion, and in presurgical phase in order to better identify bone and lesion relationships.
Figure 5. Dural fistula of the right cavernous. Hypertrophy and discreet T2 signal of the right medial, inferior and lateral muscles (a, green arrows) and dilation of the superior ophthalmic vein (a, blue circle). Abnormal vessels in the right cavernous sinus (b). Dynamic MRA (c): opacification in the arterial phase of the cavernous sinuses and superior ophthalmic vein (c, red arrows).

Figure 6. Meningioma of the right cavernous sinus, shortening the calibre of the internal carotid artery, which appears to be medially suppressed (a, red circle). Confirmation on MRA 3D TOF of Circle of Willis (b). Right median cerebral artery suppressed to the top.
Figure 7. Angioscan in the arterial phase. Post-traumatic right carotid-cavernous fistula: increase of the right cavernous volume, dilation of the arterialised superior ophthalmic vein, grade I exophthalmia, hypertrophy of the oculomotor muscles.

Figure 8. Orbital echo-Doppler in the investigation of a dural fistula (follow on from the case in Fig. 4). Indirect signs of a dural fistula with to the right a dilated superior ophthalmic vein, and with arterialised flux. To the left, physiological direction and normal flux of the superior ophthalmic vein.

Cavernous sinus pathology: how to get clues?

Lesions are often difficult to distinguish from each other.

There is no correlation between lesion size and clinical symptoms.

The first step is given by the clinical data

In case of sudden and acute onset of the symptoms, rupture of an aneurysm of the posterior aspect of the carotid artery or carotid-cavernous fistula have to be ruled out.

The carotid-cavernous fistula complicates the rupture of an intracavernous aneurysm or a trauma of the skull base and is responsible for pulsatile exophthalmos.

If the onset is subacute and painful, inflammatory condition in young patients or lymphoma in older patients are the main diagnostic hypothesis.

A progressive non-painful onset indicates slowly progressive disease, such as meningioma. If these clinical features are associated to chemosis, conjunctival hyperhaemia in a woman over the age of 60, a CS dural fistula has to be ruled out.

The second step depends on radiological findings

When a lesion is found within a cavernous sinus, the following questions need to be answered:
Figure 9. Bi-temporal hemianopsia and left diplopia. Mass syndrome centred on the sellar and suprasellar region (a, pink cross) with invasion of the left cavernous sinus (b, c, d: green arrows) in favour of hypophyseal adenoma responsible for compression of the optic chiasma pushed upwards (b, pink arrows).

- Where is the initial location of the lesion? In the cavernous sinus or nearby, as this lesion can be an extension of an extra cavernous lesion, especially hypophyseal (Fig. 9), of the skull base (Fig. 10) or the facial bones?
- Which T2 signal does it show? Intense T2 hyposignal or hypersignal?
- How does it enhance after contrast injection?
- What are its extensions in the three spatial planes?
- What are its effects on the main structures involved (optic nerves, optic chiasm, etc.)?

Notice that thin T2 spin echo sequences (coronal ± axial) are the first sequences to perform.

Practical classification as a function of imaging results [8,9]

Vascular origin

This origin is suspected if the cavernous sinus is enlarged and shows a strong hyposignal on T2.

Therefore, further vascular sequences need to be carried out: MRA TOF, dynamic MRA, rather than “standard” sequences with injection. Several pathologies may be found, such as aneurysm, carotid-cavernous fistula, dural fistula (Fig. 11).

Comments: The size of the cavernous sinus can be normal in case of dural fistulas. The signal in
macro-aneurysms or thrombosed aneurisms is often heterogeneous (global hyposignal with components in isohypersignal on T2 and hypersignal on T1). MRA TOF with injection may be used to differentiate circulating and thrombosed portions.

**Figure 10.** Spheno-orbital osteomeningioma: left exophthalmos and ophthalmoplegia, non-painful, progressive for several years in a 55-year-old patient. Tissue lesion centred on the main wing of the sphenoid (a, red circle) invading the left cavernous sinus (b) associated with hypertrophy and condensation of the large wing of the sphenoid (c, red star), orbital infiltration pushing into the right lateral muscle and right superior complex — eyelid elevator (c).

Characteristics of contrast enhancement after injection

Added to cavernous sinus increased volume, and analysis of T2 and T1 signal of the lesion, the characteristic of enhancement after injection bring supplementary etiological arguments:

- if the contrast medium uptake is intense and extends along the tentorial and temporal meninges, a meningioma is strongly suspected (Fig. 12) [10];
- a moderate contrast medium uptake is less specific. The lesion could be a metastasis (should always be kept in mind) (Fig. 13) or specific or non-specific inflammation, the most frequent being sarcoidosis (Fig. 14) [8]. It can be due to infiltration of the cavernous sinus by an haemopathy such as lymphoma (Fig. 15). The clinical context and a thoraco-abdomino-pelvic CT scan provide arguments for the most likely aetiology.

**Characteristic appearance of certain lesions [9]**

Very heterogeneous lesions, lesions globally hyperintense on T2 with areas strongly hypointense (CT scan shows calcification), demonstrating heterogeneous and intense contrast medium uptake, are generally of cartilaginous origin. Final diagnosis disclosed are chondroma either chondroid or chondromyxoid (Fig. 16), chondrosarcoma (Fig. 17) and more rarely chordoma [11].

Lesions with a center on hypersignal T2, an hypointense border rim on T2 sequence and which enhance after contrast medium injection in a speckled gradual manner, are suggestive of cavernous haemangioma (Fig. 18) [12,13].

CN V intracavernous schwannoma may cause facial neuralgia. The lesion is centred on Meckel’s cave, appears globally hyperintense on T2 sequence with cystic portions and is intensely enhanced after injection. It may be responsible for erosion of the orbital apex on the CT scan [14].
Figure 11. Right exophthalmos and chemosis, painless, progressive for several years in a 70-year-old patient. Hypertrophied right cavernous sinus, clear T2 hyposignal (a), dilation of the superior ophthalmic vein (b and d, red arrow), hypertrophy of the right oculomotor muscles and discreet T2 hypersignal (b, yellow arrows), arciform formation with clear T2 hyposignal behind the internal carotid artery (c, red arrow) corresponding to the dilated veins. MRA TOF: rapid flux within the right cavernous sinus, peripheral to the internal carotid artery (e, red circle) (arciform image visible on native cuts), reflecting arterialisation of the cavernous sinus (e), dynamic MRA; opacification from the arterial phase of the right cavernous sinus and the dilated right superior ophthalmic vein (f).

Figure 12. Meningioma of the left cavernous sinus: non-painful progressive left ophthalmoplegia in a 58-year-old patient. Tissue lesion located on the left cavernous sinus (a) whose volume is increased in T2 isosignal (b) and strongly enhanced after injection (c). Extension upwards towards the tentorium cerebelli (a, red arrow) and supero-internally, invading the sellar region, compressing the left portion of the optic chiasma (b, red arrows) and laterally pushing back the temporal lobe (without abnormality of the parenchymatous signal).
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Figure 13. Painless left ophthalmoplegia, rapidly progressive, in a 75-year-old patient with a history of mammary neoplasm. Cerebral and left cavernous sinus metastases. Several nodular formations with T2 hyposignal (a, blue arrow) with little contrast uptake (b and c; blue arrows) of the roof and lateral wall of the left cavernous sinus. Signs of denervation of the oculomotor muscles: atrophy, T2 hypersignal (d). Presence of other lesions: pituitary stem (a, pink arrow), right external temporal subcortical and left temporal insular (e, yellow arrows).

Figure 14. Painful ophthalmoplegia in a 35-year-old woman: sarcoidosis of recent appearance with increased volume of the left cavernous sinus by nodular formations with a T2 isosignal (yellow arrows) moderately enhanced after injection.
Figure 15. Painful ophthalmoplegia of recent onset with increased volume of the left cavernous sinus by nodular formations with a T2 isosignal (yellow arrows) moderately enhanced after injection. In a 70-year-old man: lymphoma.

Figure 16. Right cavernous sinus syndrome. MRI shows multilobulated mass, T2 hypersignal (a), T2 hyposignal (b), heterogeneously enhanced (c). Chondromyxoid chordoma.

Figure 17. Progressive right VI. Heterogeneous lesion presenting many zone of T2 hyposignal (b). Intervention stopped due to haemorrhagic complications. Heterogeneous calcification in the residual lesion on the postoperative scan (a).
Lesions hyperintense on T1 and T2, vanishing on fat suppression sequences are fatty tumours (dermoid cyst, lipoma).

Conclusion

There are numerous cavernous sinus pathologies. MRI analysis needs specific protocols. Correlations between imaging findings and clinical data are mandatory to reach the narrowest diagnostic range and in a large amount of cases the correct diagnosis.

**TAKE-HOME MESSAGES**

- If a cavernous sinus lesion is suspected, we propose the following protocol Brain examination:
  - sagittal T1;  
  - axial FLAIR or T2 ± diffusion;  
  - coronal T1 with injection of gadolinium;  
  - cavernous sinuses and orbit examination using thin slices (2–3 mm);  
  - coronal T2 ± T1;  
  - axial T1 with injection and fat saturation.
- Further sequences (MRA TOF, dynamic MRA) will be carried out depending on the clinical context and the signal of the lesion.
- There is no correlation between the size of the lesion and the severity of clinical symptoms, cavernous sinus therefore exploration needs both a systematic analysis and the use of thin focused slices.
- Analysis of MRI is specially focused on the epicentre of the lesion, its signal on T2 sequences, the type of enhancement it shows.
- Complementary CT scan of the base of the skull, in order to study bone changes and thoraco-abdomino-pelvic CT scan investigating a general pathology: neoplastic, inflammatory, infectious… may be helpful.

**Clinical case**

Mr A.B., 52 years old, monitored in ENT, consults for the gradual onset of diplopia. On examination, impaired elevation and adduction of the left eye and mild left myosis are found associated to a left cheek hypoesthesia. There is no exophthalmia, no visual disturbance. Neurological examination is otherwise normal. The patient did not bring back his previous images but had probably underwent a brain MRI and CT scan. A new MRI is proposed (Figs. 19—21).

**Questions**

1. Which probable location fits these clinical signs?
2. Look at the MR images and describe the lesion (Figs. 19—21)
3. Would you ask for another imaging examination? (Fig. 22)
4. What is the most likely aetiological diagnosis?

**Answers**

1. The association of isolated oculomotor cranial nerve, of Horner syndrome (myosis) and trigeminal cranial nerve impairment without any other neurological signs is very suggestive of a cavernous sinus lesion.
2. The lesion is a tissular mass involving both the face and the middle fossa, invading the left cavernous sinus, the infratemporal fossa and the lower part of the left parapharyngeal space, the pituitary area and extending towards the upper temporal lobe. It is hypointense on T2 sequence and enhanced after injection. It is aggressive, as it destroys the base of the skull (sphenoid, clivus) and is responsible for a temporal lobe oedema.

3. As in all cases of lesions that can damage the skull base, a plain high resolution CT scan is necessary to precise bone abnormalities.

4. The epicentre of this destructive lesion is located not in the cavernous sinus but in the left parapharyngeal space. This is very suggestive of an ENT lesion. The diagnosis hypothesis to suggest for this CS lesion is an extension of the neoplasm and, in particular, a tumour of the cavum.

**Diagnosis**

This is a case of intracranial extension of UCNT of the nasopharynx, complicating a treatment failure.

**Disclosure of interest**

The authors declare that they have no conflicts of interest concerning this article.

**References**


