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Advice and hints on imaging the lateral sellar compartments

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Abstract  The lateral sellar compartment is a complex anatomical structure containing many different elements, any of which can be at the root of a pathological condition. MRI is the examination of choice for this region, and requires the use of specific protocols and systematic examination of each of these elements to produce a suitable diagnosis.

The normal lateral sellar compartment

Anatomical overview [1–4]

The term ‘cavernous sinus’ is unsuitable and should now be replaced by ‘lateral sellar compartment’ or ‘parasellar compartment’ because it is a dura mater, extradural, interperistodural compartment which, according to Parkinson, is continuous with the orbital cavity anteriorly and with the epidural space of the spine, and contains nervous, arterial, venous and fatty components (Figs. 1–3). Venous sinuses are most often intradural, and only contain venous structures.

There are five walls to this compartment, which is in the shape of a quadrangular pyramid with its long axis running anteroposteriorly:
• the lateral wall is formed from two layers of dura mater (DM). The more lateral is the DM of the middle temporal fossa while the deeper is the meningeal sheath of cranial nerves (III, IV, V2 and VI). They are joined by connective tissue between them;

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• the superior wall or roof of the compartment is formed from two DM layers continuous medially with the diaphragma sellae. Its thickened lateral border is the interclinoid ligament. Its posterior part is crossed by CN III, which enters the compartment;
• the inferior wall, or floor, is the periosteum of the greater wing of the sphenoid and contains the opening of the carotid canal with a fibrous ring fixing the carotid at this point;
• the medial wall is formed in its inferior part by the periosteum of the lateral surface of the sphenoid and in its superior part by a thin, often dehiscent layer of DM limiting the sella turcica;
• the posterior wall or posterior border is formed by the dura mater between the lateral border of the quadrilateral lamina medially and the anterior end of the petros bone laterally, strengthened by the posterior petroclinoid ligament and crossed by Dorello’s canal taken by CN VI to penetrate the lateral sellar compartment;

The contents of the lateral sellar compartment consist essentially of a venous crossroads through which passes the internal carotid (IC), surrounded by sympathetic fibres, and certain cranial nerves:
• the veins: the lateral sellar compartments are composed of small calibre venous components separated by fibrous septa. Five groups are classically distinguished: the constant lateral veins, the inferolateral group below the IC opposite CN V1, the vein of the carotid sulcus between the IC and the sphenoid, the medial vein between the IC and the pituitary, and the pericarotid plexus. The venous afferents of the lateral sellar compartments are the inferior and superior ophthalmic veins, the central vein of the retina, the sphenoparietal sinus, the inter-cavernous sinus linking the two lateral sellar compartments. The efficients are the superior and inferior petrosal sinuses and the pterygoid plexuses via emissary veins;
• the internal carotid (IC): surrounded by sympathetic nerve networks, the IC crosses the lateral sellar compartment following a sinuous path (the carotid siphon) of five segments, only three of which are intracavernous (C5-4-3), segments C2 and C1 being supra-cavernous. It is fixed by fibrous adhesions only at its points of entry and exit. In the compartment it gives rise to the meningo hyphophageal veins and inferolateral trunks;
• the cranial nerves:
  o CN III enters the compartment in the posterior part of the roof then continues in the inner lamella of the lateral wall with CN IV, V1 and V2, surrounded by a meningeal sheath,
○ CN IV enters the compartment in the posterolateral part of the roof, posterior to CN III and then follows a path in the lateral wall,
○ CN VI enters the compartment though the posterior wall (Dorello’s canal), then crosses it, remaining in contact with the lateral surface of the internal carotid, and leaves via the superior orbital fissure accompanied by CN III, IV and V1,
○ CN V1, formed by the union of the lacrimal, frontal and nasociliary nerves in the superior orbital fissure, passes within the lateral wall of the compartment to reach the trigeminal ganglion posteriorly,
○ The intracavernous path of CN V2 is open to discussion: its intracranial penetration is via the foramen rotundum, then there is a short passage either in the posterior and inferior part of the lateral wall of the lateral sellar compartment or remaining extracavernous, before reaching the trigeminal ganglion.

Normal imaging: MRI ± CT

Technique for examining the lateral sellar compartments: thin slices, T1-T2-weighting with gadolinium injection, coronal incidence essential, time of flight MRA and/or MRA with injection of gadolinium, possibly dynamic. Diffusion. Possibly Ciss or Fiesta sequence ± gadolinium:
• the internal carotid appears hypointense with T1 and T2-weighting because of its rate of flow, hypointense on TOF MRAs and gadolinium enhanced MRAs;
• the vessels of the lateral sellar compartments are of variable intensity with T1-weighting due to differences in flow (hypointense if the flow is slow, hypointense if there is a rapid flow); with T2-weighting they appear rather hypointense and enhance uniformly following gadolinium injection;
• the cranial nerves are visible as ‘negatives’ (hypointense) within the T2-weighted venous hyperintensity [5] and the opacification of the lateral sellar compartments following gadolinium injection; CN III can be easily individualised [6];
• the dural walls of the lateral sellar compartment are clearly visible with T2-weighting, being hypointense and enhancing after contrast injection. The superior part of the medial wall is not constantly visible (T2-weighting, 3T) [7];
• a bone filter CT scan, without injection, shows the basal foramina. Their enlargement and the existence of osteolytic or osteoblastic lesions are diagnostic points to be considered when there is disease.

Anatomical variants of the lateral sellar compartment

Anatomical variants are:
• fat, normally present in the lateral sellar compartment in variable quantities;
• the internal carotids, sometimes protruding into the sella turcica;
• the trigeminal artery [8] (Fig. 4):
○ the most frequent form is transellar, arising from the posteromedial surface of the intracavernous carotid, passing into the sella, coming into contact with the sellar floor, perforating the dorsum sellae and re-joining the basilar trunk,
○ less often, it is laterosellar, arising on the posterolateral surface of the intracavernous carotid, crossing CN VI, going around the back of the sella on a path parallel to the trigeminal, then joining the basilar trunk. The trigeminal artery appears as a vascular structure, which is hypointense in T1 and T2-weighting.
• the sellar spine on the dorsum sellae may be very median but does not concern the cavernous sinus.

The pathological lateral sellar compartment [9–11]

Since a large number of structures, nerves, vessels, etc. pass through the lateral sellar compartment, it can be the site of various pathological conditions.

Locoregional pathologies

They are:
• tumours:
  ○ primary tumours = meningioma, schwannoma, haeman-
  gioma;
  ○ tumours in the vicinity extending to the lateral sel-
  lar compartment: adenoma, chordroma, chordoma,
  fibrous dysplasia, NP carcinoma, NP fibroma, etc.;
  ○ secondary tumours: metastases, lymphoma.
• vascular conditions: internal carotid aneurysm, carotid-
  cavernous fistula, thrombophlebitis.
• inflammatory/infectious conditions: Tolosa-Hunt syn-
  drome [12], tuberculosis, thrombosis of the compartment
  secondary to facial cellulitis.

General pathologies

They can be listed as:
• granulomatoses/infectious conditions: sarcoidosis,
  Wegener’s granulomatosis, tuberculosis, etc.;
• intracranial hypotension.

Clinical context

Before any imaging of the lateral sellar compartment, it is essential to know the clinical context to make best use of the imaging possibilities. Cavernous sinus lesions show as:
• oculomotor disorders through involvement of one or more cranial nerves which may extend to total ophthalmople-
  gia;
• sensory disorders in CN V1 territory;
• retro-orbital pain;
• Claude Bernard Horner syndrome by involvement of peri-
  carotid sympathetic fibres;
• possibly orbital signs, such as chemosis and/or exoph-
  thalma, and/or IC murmur;
• possibly disorders of visual acuity/visual field where there is an upper extension;
Figure 4. Anatomical variant. Laterosellar form of the trigeminal artery. Axial Fiesta (a), TOF MIP (b) TOF volume rendering (c).

- possibly hyperprolactinaemia where there is an endosellar extension.

The way the symptoms developed, triggering factors and other associated neurological and/or general symptoms suggest certain aetiologies; imaging often underpins or corrects these diagnoses.

Imaging practice

Systematic examination of the lateral sellar compartment is basically centred on its volume, followed by analysis of:

- the signal;
- the internal carotid;
- the lateral wall/posterior border;
- the basal foramina, the orbital fissures;
- the neighbouring regions (orbit, sella turcica, sphenoid sinus, clivus, Meckel’s cavum);
- the whole of the brain;
- the bone CT which may have been performed.

If the lateral sellar compartment is enlarged

Analysis of the signal

The analysis of the signal are presented as follows:

- T2-weighted hypointensity (Fig. 5): marked T2-weighted hypointensity of the whole compartment should, in the first instance, evoke vascular disease. The possibility of an aneurysm must be considered above all if there is also T1-weighted hypointensity, and a vascular sequence should be performed (3D TOF gadolinium enhanced MR angiography). Carotid aneurysms, intra or supra-cavernous, can be precisely located with thin T2-weighted volume sequences (CISS/Fiesta etc.), the risks being different depending whether the aneurysm is intra or extradural (SAH or CC fistula) [13]. A bone scan of the base of the skull may be useful for detecting possible lysis of the bony wall of the adjacent sphenoid (risk of very serious epistaxis if an aneurysm ruptures). A large lateral sellar compartment which is ‘too dark’ with T2-weighting may also indicate a lateral sellar compartment fistula. Direct carotid-cavernous fistulas are usually evident because of obvious clinical symptoms associated with dilatation of the superior ophthalmic vein, with exophthalmos etc. MRA confirms the diagnosis. On the other hand, dural fistulas of the lateral sellar compartment have often more discreet clinical symptoms (discreet chemosis, etc.) and the volume of the compartment is not greatly increased. Persistence is required to detect the following abnormal
little signs: the abnormal appearance of the veins of the lateral sellar compartment in 3D TOF, slight dilatation of the SOV. Dynamic MRA will provide the final diagnosis. Granulomatous lesions also appear as decided hypointensity in T2-weighting;

• marked T2-weighted hyperintensity (Fig. 6): most tumours give rather moderate hyperintensity in T2-weighting, but very marked hyperintensity should evoke a cystic tumour (a schwannoma) [14] or a haemangioma [15];

• abnormal hyperintensity in T1-weighting (Fig. 7): this should suggest a thrombosed aneurysm or a fatty tumour;

• enhancement: the absence of enhancement should bring to mind a thrombosed aneurysm, a cystic tumour (schwannoma, etc.). Moderate enhancement can indicate an invasive pituitary adenoma, more marked enhancement a meningioma, while intense enhancement indicates an aneurysm or haemangioma.
Figure 6. Enlarged lateral sellar compartment, hyperintense in T2-weighting: a, b: a meningioma of the lateral sellar compartment. Coronal T2-weighted image: a: meningioma developed in the lateral sellar compartment (asterisk) surrounding the internal carotid (arrow) reducing its calibre; the lateral border of the compartment has a slightly laminated appearance. Coronal slice after gadolinium injection; b: intense enhancement of the tumour.

Figure 7. Enlarged lateral sellar compartment, hyperintense in T1-weighting. Thrombosed aneurysm of the right intracavernous carotid with occlusion of the carotid before it: a: coronal T1-weighted image. T1-weighted hyperintense mass of the right lateral sellar compartment, extending into the sella turcica; b: coronal T2-weighted image. This mass is heterogeneously hypointense; c: 3D TOF MRA. The T1-weighted hyperintensity of the thrombosed aneurysm is visible but the right carotid before the aneurysm cannot be seen due to extensive thrombosis.
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**Analysis of the internal carotid**
If it is too big this should suggest an aneurysm, a fistula, etc. (Fig. 8).

If it is too small this may indicate compression by a solid tumour such as a meningioma, etc.

**Analysis of the lateral wall/posterior border**
A lateral wall of a large lateral sellar compartment which appears a little fuzzy, slightly 'laminated', suggests a meningioma, while a distinct wall may be a pituitary adenoma which has invaded the compartment, a schwannoma, etc. (Figs. 9 and 10).

A posterior border of a comet tail lesion in the first place suggests a meningioma, a rounded posterior border with an 'hourglass' appearance straddling the lateral sellar compartment and Meckel’s cavum suggests a schwannoma of CN V, and a filled oculomotor cistern is usually a schwannoma of CN III.

**Figure 8.** Enlarged lateral sellar compartment. Appearance and topography of the internal carotid: a, b: internal carotid too large. Aneurysm (arrow) of the right intracavernous IC. Coronal T2-weighted image, 3D TOF MRA: c, d: normal size carotid but pushed aside. 'Soft' tumour: pituitary adenoma (3), chordoma (4). T2-weighted coronal images; e: internal carotid too small. Solid tumour: meningioma of the left lateral sellar compartment which is compressing the lumen of the carotid; it also has a voluminous partially calcified temporal extension (arrow). T2-weighted coronal image.
**Figure 9.** Enlarged lateral sellar compartment. Appearance of the lateral wall: a, b: sharp border on T2-weighted coronal image. Invasive pituitary adenoma (a), CN V schwannoma (b); c: laminated border on T2-weighted coronal image. Meningioma.

**Figure 10.** Enlarged lateral sellar compartment. Appearance of the posterior border: a: T1-weighted axial slice after gadolinium injection. Comet tail appearance: meningioma; b: T1-weighted axial slice after gadolinium injection. Hourglass appearance, straddling Meckel’s c cavum and the lateral sellar compartment: schwannoma of CN V.

**Analysis of enlarged basal foramina and bony modifications of the base of the skull**

Meningeal tumours of the lateral sellar compartment, schwannomas, characteristically extend along the nerve pathways or meningeal sheaths, enlarging the foramina through which these nerves pass (Fig. 11). A bone scan of the base of the skull can pick this out complementing the MRI and, in cases of meningioma, also show osteoblastic lesions. Malignant ENT tumours also have a tendency to extend into the lateral sellar compartment via nerve structures.
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**Figure 11.** Enlarged lateral sellar compartment. Base of the skull: what is the appearance of the basal foramina? Are there bone modifications? Meningioma of the left cavernous sinus invading the sella turcica, extracranial extension via the foramen ovale which appears to be enlarged (arrow), bone invasion with osteoblastic activity at the base of the skull, the signal from which appears modified (asterisk): a: T1-weighted coronal image after gadolinium injection; b: T1-weighted axial slice after gadolinium injection; c: CT bone window scan: axial slice centred on the base of the skull.

**Analysis of neighbouring regions**

If the lateral sellar compartment is enlarged, the neighbouring regions (orbit, sella, clivus, sphenoid sinus, Meckel’s cavum, etc.) should be examined systematically, as they could be the site of tumours with secondary invasion of the lateral sellar compartment.

**Analysis of the whole brain**

The presence of other intracranial lesions as well as those of the lateral sellar compartment in many cases provides a pointer to the diagnosis.

If both lateral sellar compartments are enlarged

The main aetiologies are:
- neighbouring tumours invading the two lateral sellar compartments, so look at neighbouring regions (Fig. 12);
- dural fistulas draining bilaterally, so look at the signal on a 3D TOF MRA from the cavernous veins and the SOV, and if in doubt use a dynamic MRA sequence;
- inflammatory and infectious pathologies such as granulomatosis, Wegener’s granulomatosis, osteomyelitis of the base etc., which may infiltrate the two lateral sellar compartments;
- increased volume of the two lateral sellar compartments and possibly of any of the intracranial venous sinuses, due to intracranial hypotension. It is necessary to look for the other signs of intracranial hypotension (cerebellar tonsils in the foramen magnum, cerebral trunk flattened against the clivus, meningeal surfaces abnormally enhanced, etc.).

If a lateral sellar compartment is too small

The aetiology is essentially former thrombosis of the internal carotid.
Figure 12. Two enlarged lateral sellar compartments: a: Invasive pituitary macroadenoma in both cavernous sinuses. T2-weighted coronal slice: b, c: tuberculous pachymeningitis. T2 and T1-weighted coronal images after gadolinium injection: d–f: intracranial hypotension. Both lateral sellar compartments a little too voluminous, enhancement of all the intracranial meningeal surfaces.
Conclusion [16]

The lateral sellar compartment is an extremely rich anatomical structure, with the direct consequence that its pathology is very varied.

All imaging of the lateral sellar compartment must be performed taking into account the clinical context, to ensure that the best imaging technique is used.

The different sequences must be minutely examined and the anatomy and the signals from the different anatomical structures systematically analysed.

Only under these conditions can imaging of the lateral sellar compartment help make a diagnosis.

**TAKE-HOME MESSAGES**

- Lateral sellar compartment = a complex anatomical structure combining arteries, veins, nerves and meninges.
- Any of the anatomical components may be at the root of a pathological condition.
- There is a possibility of intra- and extracranial communication via the foramina and the fissures in the base and front of the skull.
- The MRI technique must be suitable for the suspected pathological condition. MRA should be used if there is the slightest doubt about the appearance of the vessels.
- Systematic analysis:
  - the volume of the lateral sellar compartment,
  - the signal,
  - the appearance of the internal carotid,
  - the appearance of the lateral wall/posterior border,
  - the foramina of the base, the fissures, the bony structures of the base of the skull,
  - the regions neighbouring the lateral sellar compartment,
  - the brain and possibly the bone CT.

Clinical case

This concerns a 36-year-old woman with right ophthalmoplegia, which has progressively appeared over several months associated with amenorrhoea with hyperprolactinaemia of 30 ng/mL. An MRI was performed (Fig. 13a–c).

Questions

1. What abnormalities can you see on these three images?
2. What diagnosis do you suggest?
3. What sequences would you use to advance the diagnostic process?

Answers

1. These three images show a mass which seems to be centred on the right lateral sellar compartment with heterogeneous intensity in T1 and T2-weighting, with alternating areas of hyper and hypointensity. The sellar floor has collapsed by the mass, there is discrete extension of the mass towards the sella turcica, the pituitary stalk is deflected to the left and the right hemichiasma upwards. The lumen of the internal carotid is visible below the lesion, but the supracloinoideal portion of the carotid seems to ‘project’ from the lesion.
2. Diagnostic hypothesis at this stage: a partially thrombosed vascular mass (heterogeneous signal) with a small amount of sellar extension responsible for disconnection hyperprolactinaemia (mass effect on the stalk).
3. The following sequences to be performed are MRA sequences: TOF (Fig. 13d) which will certainly be insufficient if it is a partially thrombosed vascular mass. 3D or 4D gadolinium enhanced MRA (Fig. 13e) is the sequence of choice. Systematic volume acquisition (Fig. 13f: coronal slice) will then be performed. A bone CT scan of the base of the skull (Fig. 13g: coronal slice) will reveal the state of the sellar floor by the aneurysm: there is no bone lysis (arrow), rupture of an aneurysm simply carries the risk of a direct carotid-cavernous fistula; on the other hand, if there were bone lysis, life-threatening epistaxis could occur if the aneurysm ruptured.
Figure 13. MRI: sagittal T1-weighted SE (a); coronal T1-weighted SE (b); coronal T2-weighted SE (c). 3D TOF MRA (d). Gadolinium enhanced MRA (e). Coronal slice, volume acquisition 3D SPGR after gadolinium injection (f). CT scan, coronal slice, bone filter (g).
Disclosure of interest

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

References


