Differential diagnosis of sellar, suprasellar, and parasellar cystic lesions

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Authors: R. Calandrelli, S. Gaudino, G. M. Di Lella, T. Tartaglione, A. M. Costantini, M. Rollo, C. Colosimo; Rome/IT
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Learning objectives

- To summarize the most common Sellar/Parasellar and Suprasellar cystic lesions
- To define precise anatomical location and origin
- To assess the correct imaging approaches and to recognize imaging findings
- To propose most likely differential diagnoses

Background

Sellar, suprasellar, and parasellar cystic lesions are quite commonly encountered (FIG. 1) on page .

- **Primary intrasellar cystic lesions** arise from the intrasellar structures, may extend or not into the supra/para-sellar region, and can be non-neoplastic (e.g. arachnoid cyst, pituitary cyst, Rathke’s cleft, colloid and parasitic cyst) or neoplastic (Adenoma, Craniopharyngioma).

- **Primary extrasellar cystic lesions**, on the other hand, arise outside the sella turcica (supra- or para- sellar), may subsequently involve it, and can be both non-neoplastic (sphenoid sinus mucocele, arachnoid cyst) and neoplastic (craniopharyngioma, epidermoid cyst, glioma, meningioma, hemangioblastoma), as well.

Imaging findings OR Procedure details

A step-by-step assessment of the imaging findings should be performed to correctly derive a differential diagnosis:

- **Step 1 - Site/Origin**

First, establish the lesions anatomical origin (intra-or extra-axial, intra- or extra-pituitary, adeno/neuropituitary):
- **Primary intrasellar** lesions usually enlarge the sellar cavity, displace the infundibular stalk, and may displace upward the optic chiasm.

Moreover, cysts derived from Rathke pouch remnants and adenopituitary cysts lie *anterior to the infundibular stalk*, unlike neurohypophysis cysts (choristomas), that stay behind.

(Fig 1) on page 5

- **Suprasellar** lesions may enlarge the sellar aditus and deform the optic groove

- **Parasellar** lesions may grow and reach the sellar/supra-sellar regions, eventually displacing the chiasm and pituitary gland downward.

• **Step 2- Signal/Density:**

Then, recognize changes in tissue MR signal intensity and CT tissue density:

- **T1 signal hyperintensity** is a common finding at MR imaging, deriving from different sources: it may be related to bleeding (e.g. in hemorrhagic suprasellar pituitary adenoma, pituitary apoplexy), to a high concentration of protein (RCC, craniopharingioma or mucocele), to fat (lipoma, dermoid cyst, lipomatous meningioma), to calcifications (craniopharingioma), or to a paramagnetic substance (metastatic melanoma). However, after surgery, T1 signal hyperintensity may also result from the presence of materials used for surgical packing (such as gelatine sponge or fat). (Fig 2) on page 6

- **T2-signal hypointensity (FSE)**, is related to hypercellularity, melanine, fibrous tissue, hyperproteinaceous de-hydrated content and calcium (Fig 3) on page 7.

- **T2-signal hyperintensity** can show peritumoral oedema (meningioma), parenchymal infiltration (e.g. cystic glioma), fluid-fluid levels within the mass from sedimentation of blood products (more likely observed in pituitary adenomas than in craniopharyngiomas or RCCs) and cystic content (isointense to the CSF or not) (Figs 4, 5).

- **T2*-signal hypointensity (GRE)** may result from calcified components or hemosiderin, *although CT scan remains the gold standard for calcium detenction* (Fig 6) on page 10

• **STEP 3 - Post-Contrast behaviour**

- Contrast-enhanced MR imaging above all plays an essential role in differentiating non-neoplastic cysts from cystic neoplasms (unlike non-neoplastic cysts, cystic tumors usually show wall enhancement) (Fig 7) on page 11, but it is also useful to evaluate tumors that have both cystic and solid components (hemangioblastoma, cystic macroadenoma, craniopharyngioma, meningioma,) (Fig 8) on page 12 or to suggest inflammatory
reactions (e.g. abscess). Notably, fat suppression techniques help in differentiating solid, lipidic and cystic components.

- Additional information may be obtained from **post-Gd delayed images**, to differentiate cystic from solid adenomas: in fact, a solid lesion, in an early post-contrast phase, can be hypovascular relative to the richly vascular pituitary gland and cavernous sinus, thus mimicking a cystic lesion. (Fig 9) on page 13

• **Step 4 - Recognition of mass effect, edema-like changes and sellar bone change**

-Sellar and suprasellar lesions may induce mass effect on the optic chiasm, the pituitary gland and the infundibular stalk, on the brain parenchyma and on the third ventricle, and may also invade the cavernous sinus (Figs 10-12). Moreover, in expansive cystic lesions, the pituitary stalk is flattened and displaced according to the growing direction of the mass, unlike in the empty sella in which the stalk is constantly flattened towards the dorsum, and elongated (Fig 13) on page 17.

-All sellar, suprasellar and parasellar lesions may cause bone change, although in different ways, depending on their growth pattern and primary origin. **CT** can help to restrict the differential diagnosis, as it is superior in demonstrating bone sequestra, hyperostotic reaction adjacent to the tumor (meningioma), remodelling of the bone (arachnoid cyst), dural "tail" sign (meningioma), bone destruction (chordoma, mucocele, metastases, macroadenoma) and mass effect with sellar enlargement (macro-adenoma). (Figs 14,15).

• **Step 5 - Contribution from Additional MRI Techniques**

-**Diffusion-Weighted Imaging (DWI + ADC)** may contribute to differentiate epidermoid cysts from arachnoid cysts, since epidermoid cysts show restricted diffusion and higher signal intensity (Fig 16) on page 20.

-**MR spectroscopy**, in selected instances, can help identifying neoplastic solid tissues and differentiating neoplastic and non-neoplastic cystic walls, by showing increase or decrease of different metabolites peaks:
Fig.: Essential metabolites peaks for differentiating neoplastic and non-neoplastic cystic walls

References: R. Calandrelli; Bioimaging and Radiological Sciences, Institute of Radiology, Pol. A. Gemelli, Catholic University of Rome, Rome, ITALY

- **Step 6- Post-operative assessment**

During surgery, the sellar cavity may be filled with packing/haemostatic material (usually fat or gelatine sponge).

These surgical "patches" may be used as sort of "landmarks" in follow-up controls when they are found to be modified/displaced by recurrent disease. (Figs 17,18)

Images for this section:
Step 1 – Location/Origin
(intra- or extra-pituitary, adeno/neuropituitary)

Adenopituitary cysts lie anterior to the infundibular stalk...

...unlike neuropituitary cysts (choristoma)

Primary sellar and suprasellar cystic lesions can displace the infundibular stalk/optic chiasm
**Step 2 – Signal**

**What’s bright \(\bullet\) on T1-WI in the sella and around?**

<table>
<thead>
<tr>
<th>Normal</th>
<th>Pathology</th>
</tr>
</thead>
<tbody>
<tr>
<td>- Posterior lobe (vasopressin)</td>
<td>- Hemorrhage (methemoglobin)</td>
</tr>
<tr>
<td>- Gadolinium enhancement</td>
<td>- Concentrated protein (Rathke cleft cyst, craniopharyngioma)</td>
</tr>
<tr>
<td>- Pituitary hyperactivity: pregnancy newborn…</td>
<td>- Fat tissue</td>
</tr>
<tr>
<td>- Bone</td>
<td>- Melanine</td>
</tr>
<tr>
<td>- Artifacts: magnetic susceptibility</td>
<td>- Manganese deposit</td>
</tr>
<tr>
<td></td>
<td>- After treatment, surgical implant (gelatine sponge, fat)</td>
</tr>
</tbody>
</table>
### Step 2 - Signal

**What induces signal loss on T2 WI?**

<table>
<thead>
<tr>
<th>Normal</th>
<th>Pathology</th>
</tr>
</thead>
<tbody>
<tr>
<td>■ Diploe</td>
<td>■ Hypercellularity</td>
</tr>
<tr>
<td>■ Artifacts (turbulent flow)</td>
<td>■ Hemorrhage (intracellular methemoglobin, hemosiderin, deoxyHb)</td>
</tr>
<tr>
<td>■ Highly concentrated c.m.</td>
<td>■ Ferritine</td>
</tr>
<tr>
<td></td>
<td>■ Melanine</td>
</tr>
<tr>
<td></td>
<td>■ Fibrous tissue</td>
</tr>
<tr>
<td></td>
<td>■ Hyperproteinaceous de-hydrated content</td>
</tr>
<tr>
<td></td>
<td>■ Calcium</td>
</tr>
</tbody>
</table>

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**Fig. 3**
Step 2 - Signal / Density

CT

Cystic content

Iperproteinaceous content produces a signal intensity higher than CSF

craniopharyngioma

mucocoele

Rathke cleft cyst

Suprasellar cyst

Fig. 4
Step 2 - Signal / Density

Fluid-fluid levels within the mass (from sedimentation of blood products or from a different protein concentration) is more likely observed in pituitary adenomas than in craniopharyngiomas or Rathke cleft cyst.

macro-adenoma

craniopharyngioma

DIFFERENTIAL DIAGNOSIS

Fig. 5
**Step 2 - Signal / Density**

**MR:** detection and characterization

**CT:** specificity (calcifications in 3.5%)

- **suprasellar**

- **Sellar/suprasellar/parasellar**

**NODULAR or CURVILINEAR calcifications** (intra or peri-lesional) strongly suggest

**craniopharyngioma**

Fig. 6
Step 3 - Post-Contrast behaviour

Cystic wall features

Contrast-enhanced MR imaging plays an essential role in differentiating non neoplastic from neoplastic cysts: cystic tumors usually show CE of the wall, unlike non neoplastic cysts.

Fig. 7
Step 3 - Post-Contrast behaviour

Contrast-enhanced MR imaging plays an essential role in differentiating non neoplastic cysts from cystic neoplasms: tumors may have both cystic and solid components.

Fig. 8
Step 3 - Post-Contrast behaviour (*delayed*)

Dynamic study

Cystic or solid adenoma?

Delayed post-contrast images help to differentiate cystic from solid adenoma (solid adenoma, in early acquired images, can mimic a cystic lesion because it may be hypovascular relative to the richly vascular pituitary gland and cavernous sinus.)

Delayed image

Fig. 9
Step 4 - Recognition of Mass effect

Suprasellar cyst

Cystic adenoma

Suprasellar craniopharyngioma

Sellar/suprasellar craniopharyngioma

Sellar, suprasellar lesions may have mass effect on the optic chiasm, pituitary gland and the infundibular stalk

Fig. 10
Step 4 - Recognition of Mass effect

Fig. 11

Sellar/suprasellar/parasellar lesions may have mass effect on brain parenchyma, on third ventricle and ........
Step 4 - Recognition of Mass effect

Condrosarcoma

Macroadenoma

.....and can invade cavernous sinus

Fig. 12
Step 4 - Recognition of Mass effect

In expansive cystic lesions, the pituitary stalk is flattened and displaced according to the growing direction of the mass, unlike in the empty sella, in which the stalk is constantly flattened towards the dorsum and elongated.

Cystic macro-adenoma

Arachnoid cyst

Empty sella

Fig. 13
Step 4 - Recognition of Bone change

In macroadenoma and intrasellar craniopharyngioma the sella is **usually** enlarged…….

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cystic macro-adenoma

craniopharyngioma

...*But* in suprasellar craniopharyngioma the sella is **not** enlarged

---

Suprasellar craniopharyngioma

Fig. 14
Step 4 - Recognition of Bone change

In parasellar lesions the sellar walls may be eroded

condrosarcoma

Sphenoidal sinus may be invaded and the clivus may be expanded

mucocle

craniopharyngioma

Fig. 15
Fig. 16

Step 5 - Additional MRI Techniques
Diffusion imaging

Epidermoid cysts show higher signal intensity on DWT unlike arachnoid cysts / adamantinomatous craniopharyngiomas and meningocele

Fig. 16
Step 6 - Post-operative assessment

Pre-operative

1 year Post-operative

Fat sat sequences help to recognize surgical fat implant

Fig. 17
Step 6 - Post-operative assessment

Fig. 18

Recurrent arachnoid cyst:
the fat implant appears displaced upward due to cystic re-filling
Conclusion

- MRI is the 1st-choice diagnostic tool in patients with sellar, supra-sellar and para-sellar cystic lesions; CT scans are performed in selected instances.
- MRI and CT techniques must be tailored on the basis of previous imaging findings, laboratory data and clinical scenarios.
- Neuroimaging findings must be integrated with laboratory and clinical data to correctly address the proper surgical treatment.

Personal Information

Affiliations:

Institute of Radiology
Dept. of Bio-imaging and Radiological Sciences
Catholic University and School of Medicine
Rome - ITALY

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References