Making Sense of Sellar Region Pathology: Image-Based Diagnostic Algorithm

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After participating in this activity, the radiologist should be better able to evaluate the regional anatomy, clinical presentation, pathophysiology, and imaging characteristics of common sellar region pathology.

Key Words: Imaging of Sellar Region, Pituitary Adenomas, Pituitary apoplexy, Meningioma, Craniopharyngioma, Empty Sella

Lesions in the sellar and parasellar regions are encountered frequently in clinical radiology. Complex anatomy of this region contains numerous key structures, which can be affected by a variety of pathologic processes. Although “SATCHMO,” a commonly used mnemonic, may help the radiologist to remember differential diagnoses when imaging findings overlap, it has limited diagnostic utility. (See page 7 for description of SATCHMO.) A systematic approach to imaging findings in the sellar region can provide valuable information regarding the extent of pathology, aid in treatment planning, and help detect complications. This article discusses regional anatomy, clinical presentation, pathophysiology, and imaging characteristics of common sellar region pathology.

Sellar Anatomy

The sella turcica is a saddle-shape, concave, midline osseous structure in the basi-cranium formed by the sphenoid bone. It is bounded anteriorly by the tuberculum sella and posteriorly by the dorsum sella. The lesser wings of the sphenoid bone form anterior clinoid processes, which are superior and lateral to the tuberculum sella. The roof of the sphenoid sinus and the clivus form the floor of the sella turcica. The superior border is marked by a dural reflection, termed diaphragma sella, which encases the pituitary gland.1

The cavernous sinuses are located on either side of the sella turcica, and they can be involved secondarily by pituitary lesions. Several cranial nerves traverse the lateral wall of the cavernous sinuses, which include oculomotor (CN III), trochlear (CN IV), and the ophthalmic and maxillary branches of trigeminal (CN V1, CN V2, respectively) nerves. In addition, the abducens cranial nerves (CN VI) are located within the cavernous sinuses, adjacent to the cavernous portion of the internal carotid artery.1,2 Structures superior to the pituitary gland in the suprasellar cistern also can be affected by pituitary pathology and include optic nerves, optic chiasm, hypothalamus, pituitary infundibulum, circle of Willis, and the third ventricle.1

The abducens cranial nerve (CN VI) is located within the cavernous sinus, adjacent to the cavernous portion of the internal carotid artery.

The pituitary gland is composed of the neurohypophysis posteriorly and the adenohypophysis anteriorly, and these divisions are separated by the pars intermedia.1,2 Embryologically, the adenohypophysis develops from oral ectoderm or Rathke pouch, and it is divided into pars tuberalis, pars...
intermedia, and pars distalis. The adenohypophysis involves production of adrenocorticotrophic hormone (ACTH), follicle-stimulating hormone (FSH), growth hormone (GH), luteinizing hormone, prolactin, and thyroid-stimulating hormone (TSH). The blood supply to the adenohypophysis is unique because it has little or no direct arterial supply. Instead, the adenohypophysis has a primary and secondary portal venous system formed by capillaries arising from bilateral superior hypophyseal arteries. The neurohypophysis develops embryologically from neuroectoderm in the floor of the forebrain, and the infundibular stalk develops from ventromedial hypophal- thalamus. The neurohypophysis comprises the pars nervosa, infundibular stalk, and infundibula proper. It contains modified glial cells: pituicyte and tancyte. The blood supply to the neurohypophysis arises from paired inferior hypophyseal arteries.

The size of a normal pituitary gland differs with age and sex. It is largest in pubertal girls and during pregnancy, measuring up to 12 mm in the latter. The size of the gland decreases with age. The pituitary gland is connected with the hypothalamus via a neurovascular connection (infundibular stalk) that allows axons of paraventricular and supraoptic nuclei from the hypothalamus to reach the neurohypophysis, where vasopressin and oxytocin are stored. The neurohypophysis appears hyperintense on T1-weighted MR sequences. In addition, various regulatory signals are transmitted from the hypothalamus to the anterior pituitary gland via this neurovascular connection (tuberohypophyseal neural tract) and hypophyseal portal system.

Indication for Pituitary Imaging

Imaging of the pituitary gland is performed in patients with clinical signs and symptoms suggestive of central or pituitary endocrinopathies. Imaging also is performed in patients with visual field deficits or palsies of the aforementioned cranial nerves.

Imaging Protocol

CT is occasionally performed in emergent situations such as trauma, subacute visual disturbance, worsening headaches, or in cases where patients have contraindications to MRI. Evaluation of osseous structures is better assessed with CT, which also is used in presurgical planning.

MRI is the imaging modality of choice for evaluation of sellar and suprasellar pathology because of its unparalleled tissue resolution providing soft tissue detail while minimizing artifact from adjacent osseous structures. MRI helps to demonstrate the relationship of pathology to the optic chiasm and cavernous sinuses, and to distinguish solid, cystic, or hemorrhagic components of a lesion. Pituitary imaging commonly is performed using thin slice thickness (≤3.0 mm) without interslice gap, and both pre- and postcontrast images should be obtained. Dynamic postcontrast imaging is essential in evaluation of pituitary microadenomas.

The normal adenohypophysis is isointense to gray matter on both T1- and T2-weighted MR images, whereas the neurohypophysis appears hyperintense on T1-weighted and hypointense relative to gray matter on T2-weighted MR images. On T1-weighted post-gadolinium-enhanced sequences, normal pituitary gland enhances homogeneously.

Pituitary Adenoma

Pituitary adenomas are the most common neoplasm in the sella turcica and represent approximately 10% to 15% of all intracranial neoplasms. Pituitary adenomas are benign lesions and are classified according to their size, with microadenomas less than 10 mm in size and macroadenomas larger than 10 mm. In addition, pituitary adenomas can be characterized on the basis of their endocrine activity. The most common functional pituitary adenoma (40%) is a prolactinoma (Figure 1), which results in serum prolactin levels higher than 150 ng/mL. Prolactinomas clinically present with galactorrhea and dysmenorrhea in females and gynecomastia and hypogonadism in males. Growth hormone-releasing adenoma is the second most common functional pituitary adenoma, resulting in acromegaly, diabetes mellitus, hypertension, and osteoarthritis. ACTH-releasing adenoma is the third most common functional pituitary adenoma, resulting in Cushing syndrome.

Pituitary adenomas are the most common neoplasm in the sella turcica, representing approximately 10% to 15% of all intracranial neoplasms.

On unenhanced T1-weighted MR images, pituitary microadenomas may appear hypointense relative to the adjacent normal pituitary gland. A dynamic post-gadolinium-enhanced MR sequence is essential to confirm the diagnosis,
and it improves sensitivity of detecting small pituitary microadenomas. Pituitary adenomas are hypovascular and demonstrate slight delayed or decreased enhancement as compared with normal pituitary tissue, and, therefore, they appear as a focal hypoenhancing region within the pituitary during the first 60 to 90 seconds after gadolinium administration.2-4,7 Although findings on T2-weighted MR images are variable, most pituitary adenomas exhibit hyperintense signal relative to normal adenohypophysis, the most common being prolactinomas (80%). A smaller percentage of pituitary adenomas is hypointense on T2-weighted images, mostly growth hormone-secreting adenomas (40%–60%).3

CT greatly lacks sensitivity for detection of pituitary adenomas, especially microadenomas, with a high false-negative rate.7 On post-contrast CT, pituitary adenomas appear hypointense relative to the normally enhancing pituitary gland.7 However, quantum mottle, image noise, and artifacts from adjacent osseous structures may degrade image quality and obscure lesions.7

Macroadenomas have similar imaging characteristics as microadenomas except for size and potential for hemorrhage or a cystic component. Although dynamic imaging can be used for macroadenomas, it is not as crucial for detection of microadenomas. Most patients with macroadenomas (60%–70%) present with abnormal serum hormone levels, but some may present only with symptoms associated with mass effect.2 Normal pituitary tissue usually is not identifiable in a patient with a macroadenoma. Macroadenomas secondarily can involve parasellar structures, usually through either invasion or mass effect leading to compression syndrome(s). For example, superior growth of a macroadenoma leads to compression of an optic nerve and/or optic chiasm, which in turn leads to bitemporal hemianopsia (Figure 2); and lateral growth leads to invasion of the cavernous sinuses, and when large enough, compression of the cranial nerves. Macroadenomas can encase the cavernous portion of the internal carotid artery, usually without narrowing, however, as opposed to meningiomas occurring in this region. Occasionally, macroadenomas can behave more aggressively and invade adjacent structures including supralaterally (frontal and temporal lobes), anteriorly (ethmoid sinus, orbits), posteri- orly (interpeduncular cisterns), and inferi-orly (sphenoid sinus, nasal cavity, and clivus).2 These pituitary lesions are referred to as invasive macroadenomas (Figure 3).

**Pituitary Apoplexy**

Pituitary apoplexy refers to a clinical syndrome presenting with acute onset of headache, ophthalmoplegia, visual disturbance, and/or mental status changes related to hemorrhage and infarction of the pituitary gland. Pituitary adenomas commonly are associated with apoplexy. Imaging findings are variable depending on the clinical onset of apoplexy, presence or absence of a pituitary adenoma, degree of pituitary infarction, and presence of hemorrhage.

**Pituitary apoplexy is related to hemorrhage and infarction of the pituitary gland; pituitary adenoma often is associated with the apoplexy.**

Acute hemorrhage appears hyperdense on unenhanced CT and variable signal on MRI, depending on the age of the blood products (Figure 4). In postpartum patients, a pituitary gland infarction is referred to eponymously as Sheehan syndrome when infarction occurs in the absence of a pituitary adenoma. The infarction occurs in the setting of significant postpartum hypovolemia (usually secondary to postpartum hemorrhage) resulting in hypoperfusion of the peripartum hypertrophied pituitary gland. Occasionally, Sheehan syndrome remains subclinical until the patient notices amenorrhea in the months postpartum.3

**“Empty” Sella**

The empty sella refers to cerebrospinal fluid (CSF) filling the sella turcica with markedly “flattened” or nonvisualized pituitary tissue with or without expansion of the sella (Figure 5).2 It results from incompeptence of the diaphragma sella. Chronic CSF pulsation leads to eventual expansion of the
sella. Empty sella should neither be confused nor equated with pituitary hypoplasia, as patients with empty sella still have a functional pituitary gland. Occasionally, empty sella is seen in cases of idiopathic intracranial hypertension (e.g., pseudotumor cerebri) where patients present with dizziness and headaches.  

Patients with an empty sella still have a functional pituitary gland.

Rathke Cleft Cyst

Rathke cleft cyst occurs in an embryological remnant Rathke pouch that normally gives rise to the adenohypophysis. Rathke cleft cyst often is identified incidentally in the midline sellar or suprasellar region presenting as a well-circumscribed cyst and is usually asymptomatic. Imaging characteristics vary depending on the intracystic protein content; Rathke cleft cyst appears hypo- to hyperdense on CT scans and hypo- to hyperintense on T1- or T2-weighted images (Figure 6). Occasionally, Rathke cleft cyst may contain an intra-cystic nodule that appears hypointense on T2-weighted images and does not enhance on postcontrast images. Rathke cleft cyst rarely calcifies and does not enhance on postcontrast images, which helps to distinguish it from a craniopharyngioma. If Rathke cleft cyst is entirely contained within the sella turcica, it may be difficult to discriminate from pituitary cysts or cystic/hemorrhagic pituitary adenomas on imaging alone, thereby, requiring biopsy for confirmation.

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**Figure 2.** Coronal, postcontrast T1-weighted MR image in a 55-year-old woman demonstrates a large pituitary macroadenoma with a cystic component (arrows) extending into the suprasellar cistern and compressing the optic chiasm (arrowhead).

**Figure 3.** Sagittal, postcontrast T1-weighted MR image demonstrates a heterogeneously enhancing mass lesion involving the sella turcica and clivus. A normal pituitary gland was not visualized. Histopathologic evaluation was consistent with an invasive pituitary macroadenoma.

**Figure 4.** Axial, T2-weighted MR image demonstrates a fluid-fluid level in the sella turcica with layering T2 hypointense signal that represents a hematocrit level in a patient with sudden onset of severe headache and visual loss. This finding is most consistent with pituitary apoplexy.
Imaging characteristics of a Rathke cleft cyst vary depending on the intracystic protein content.

Craniopharyngioma

A craniopharyngioma usually presents as a midline suprasellar mass, although up to 10% of craniopharyngiomas can be confined within the sella turcica. Craniopharyngiomas have a bimodal age distribution presenting in late childhood to early adolescence and again in the fifth to seventh decades of life. There are two craniopharyngioma histologic subtypes: papillary and adamantinomatous. Adamantinomatous craniopharyngioma is the more common of the two histologic subtypes and is seen mostly in late childhood to early adulthood. Adamantinomatous craniopharyngiomas usually demonstrate an enhancing solid component with calcification and a cystic component. The cystic component may contain proteinaceous or hemorrhagic fluid, and hence varies on imaging (Figure 7). Papillary craniopharyngioma subtype is seen more commonly in the fifth to seventh decades of life. Papillary craniopharyngiomas generally present as a solid mass with more uniform appearance. Papillary craniopharyngioma tends to have variable T1- and T2-signal, but the presence of postcontrast enhancement in the solid component of a suspected craniopharyngioma suggests the papillary craniopharyngioma subtype. The North American Skull Base Society has classified craniopharyngiomas on the basis of location relative to the infundibulum. Preinfundibular craniopharyngioma is type I, infundibular involvement is type II, retroinfundibular is type III, and isolated involvement of the third ventricle or optic recess, or any location not accessible with endonasal technique, is type IV. Craniopharyngiomas usually present as a midline suprasellar mass, but 10% can be confined within the sella turcica.

Meningioma

Parasellar meningiomas can occur arising from the tuberculum sella, diaphragma sellae, clinoid process, olfactory...
pituitary gland and/or infundibular stalk with variable T1 and T2 appearance. Sellar involvement of sarcoidosis, Wegner granulomatosis, and other granulomatous processes (including tuberculosis and fungal infections) are difficult to discern on the basis of imaging alone because of significant overlap, thus they require clinicopathologic correlation. Extrasellar involvement, including leptomeningeal or cranial nerve involvement (as evidenced by abnormal T2 hyperintensity and postcontrast enhancement), can suggest a more systemic process.

**Dermoids, Epidermoids, Arachnoid Cysts, and Lipomas**

Epidermoids, dermoids, arachnoid cysts, and lipomas generally are found along the midline or paramedian region, mostly in the suprasellar and occasionally in the sellar region. Imaging findings in these lesions are typical and correlate with their histopathologic findings. Dermoids are fat containing; therefore, they appear hyperintense on T1-weighted MR images, but because of variable concentration of proteinaceous content, they have variable appearance on T2-weighted MR images. Rarely, dermoids can rupture and present with chemical meningitis. MRI signal characteristics of epidermoids are similar to CSF on T1- and T2-weighted MR sequences; however, they have a somewhat mixed signal on fluid-attenuated inversion recovery (FLAIR) sequences, with increased diffusion restriction on diffusion-weighted imaging. Arachnoid cysts follow normal CSF signal on all MRI sequences. Lipomas present as well-circumscribed, homogeneously hypodense masses measuring fat attenuation on CT and hyperintense on T1-weighted MRI with loss of internal signal on fat-suppressed sequences. Rarely, dermoids can rupture and present with chemical meningitis.

**Metastasis**

Metastasis to the sellar and parasellar region can occur hematogenously or through CSF seeding and frequently involves the pituitary stalk and/or the neurohypophysis. Metastasis to this region most commonly occurs from lung cancer; breast cancer; and high-grade primary central nervous system (CNS) neoplasms (CNS lymphoma, glioblastoma multiforme, and germinoma). The imaging findings of metastasis are variable depending on the histologic characteristics of the primary neoplasm. Therefore, clinical history, imaging findings (additional intra- or extracranial masses/malignancies and leptomeningeal disease), and histopathologic correlation are required to confirm the diagnosis.

**Aneurysm**

Given the close proximity of the internal carotid arteries and circle of Willis to the sellar region, aneurysms can
Table 1. Location-Based Diagnostic Algorithm

<table>
<thead>
<tr>
<th>Sellar</th>
<th>Parasellar</th>
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<tr>
<td>• Expanding sella turcica</td>
<td>• Suprasellar</td>
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<td>o Pituitary macroadenoma</td>
<td>o Enhancing</td>
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<td>• Nonexpansile</td>
<td>&gt; Craniopharyngioma</td>
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<td>o Intensity</td>
<td>&gt; Germ cell tumor</td>
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<tr>
<td>Hyperintense T1</td>
<td>&gt; Meningioma</td>
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<tr>
<td>&gt; Rathke cleft cyst (if protein)</td>
<td>&gt; Aneurysm</td>
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<tr>
<td>&gt; Dermoid cyst (children)</td>
<td>o Nonenhancing</td>
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<tr>
<td>Hyperintense T2</td>
<td>&gt; Hamartoma</td>
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<tr>
<td>&gt; Meningioma</td>
<td>• Pituitary stalk</td>
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<td>&gt; Rathke cleft cyst</td>
<td>&gt; Lymphocytic hypophysitis</td>
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<td>Hypointense T1</td>
<td>&gt; Langerhans histocytosis</td>
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<td>&gt; Pituitary microadenoma</td>
<td>&gt; Germ cell tumor</td>
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<td>Hypointense T2</td>
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<td>&gt; Pituitary apoplexy</td>
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<td>&gt; Glioma</td>
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SATCHMO is an acronym that stands for lesions in sellar and parasellar regions:

- S: Sellar adenoma (pituitary adenoma) or sarcoidosis
- A: Aneurysm
- T: Teratoma or tuberculosis
- C: Craniopharyngioma
- H: Hypothalamic glioma
- M: Meningioma/metastasis
- O: Optic nerve glioma

mimic a sellar neoplasm, especially those involving the supraclinoid and/or cavernous internal carotid artery.²,³,⁷ These aneurysms have variable imaging appearance depending on size and intra-aneurysm thrombosis. A thrombosed aneurysm appears hyperdense on unenhancement CT, hyperintense on T1-weighted and FLAIR sequences, and hypointense on susceptibility-weighted MRI. On the contrary, a patent aneurysm appears isointense on unenhanced CT with preserved flow voids on MRI. Also, aneurysms can have eggshell pattern calcification or can be near-completely calcified.²,³,⁷,⁹ Angiographic imaging studies can confirm aneurysms and allow for Interventional treatment planning.²,⁷

Conclusion

Lesions in the sellar and parasellar region are encountered frequently in clinical neuroradiology. The complex anatomy of this region contains numerous key structures, which can be affected by various pathology. Although “SATCHMO,” a commonly used mnemonic, may help the radiologist to remember differential diagnoses because of overlap in imaging findings, it has limited diagnostic utility. This CME activity emphasizes that a systematic approach is required to narrow the differential diagnosis. We propose using an algorithmic approach on the basis of lesion anatomic location, sellar size, MRI signal intensities, and enhancement patterns to formulate a more precise working differential diagnosis (Table 1).

References

CME QUIZ: VOLUME 38, NUMBER 22

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1. Which one of the following intracranial lesions rarely can present as chemical meningitis?
   A. Pituitary macroadenoma
   B. Arachnoid cyst
   C. Lipoma
   D. Craniopharyngioma
   E. Dermoid

2. Which one of the following pituitary lesions is most likely to be complicated by hemorrhage?
   A. Metastasis
   B. Macroadenoma
   C. Dermoid
   D. Lipoma
   E. Epidermoid

3. Which one of the following statements concerning craniopharyngiomas is false?
   A. The majority are suprasellar.
   B. They have a bimodal age distribution.
   C. They do not calcify.
   D. They may be cystic.
   E. They occasionally are confined within the sella turcica.

4. A parasellar mass demonstrates associated hyperostosis of the clivus and narrowing of an encased internal carotid artery on head CT and a dural tail on head MRI. The most likely diagnosis is
   A. lipoma
   B. internal carotid artery aneurysm
   C. meningioma
   D. Rathke cleft cyst
   E. pituitary macroadenoma

5. Which one of the following conditions may be seen with an “empty” sella?
   A. Galactorrhea
   B. Idiopathic intracranial hypertension (pseudotumor cerebri)
   C. Cushing syndrome
   D. Sheehan syndrome
   E. Acromegaly

6. Which one of the following cranial nerves is located within the cavernous sinuses?
   A. Oculomotor (CN III)
   B. Trochlear (CN IV)
   C. Ophthalmic branch of trigeminal (CN V₁)
   D. Abducens (CN VI)
   E. Maxillary branch of trigeminal (CN V₂)

7. All of the following structures lie superior to the pituitary gland in the suprasellar cistern, except
   A. fourth ventricle
   B. circle of Willis
   C. optic chiasm
   D. hypothalamus
   E. pituitary infundibulum

8. All of the following hormones are produced by the adenohypophysis of the pituitary gland, except
   A. follicle-stimulating hormone
   B. adrenocorticotropic hormone
   C. growth hormone
   D. thyroid-stimulating hormone
   E. parathormone

9. Which one of the following intracranial lesions commonly is associated with pituitary apoplexy?
   A. Craniopharyngioma
   B. Pituitary adenoma
   C. Meningioma
   D. Internal carotid artery aneurysm
   E. Tuberculoma

10. All of the following osseous structures help to form the sella turcica, except
    A. tuberculum sella
    B. anterior clinoid processes
    C. planum sphenoidale
    D. clivus
    E. roof of sphenoid sinus