Differential Diagnosis of Sellar Masses

Pamela U. Freda, MD, and Kalmon D. Post, MD

Pituitary adenomas are the most common cause of a mass in the sella. In as many as 9% of cases, other etiologies are responsible for mass lesions in the sellar region (Table 1). The differential diagnosis of nonpituitary sellar masses is broad and includes cell rest tumors, germ cell tumors, gliomas, meningiomas, metastatic tumors, vascular lesions, and granulomatous, infectious, and inflammatory processes (Table 2). Differentiating among these potential etiologies may not always be straightforward because many of these lesions, tumorous and nontumorous, may mimic the clinical, endocrinologic, and radiographic presentations of pituitary adenomas. In some cases, there are no features that clearly distinguish the unusual etiologies from the clinically nonfunctioning pituitary adenoma. In others, certain endocrine, neurologic, and radiographic findings that are more characteristic of patients with a nonpituitary sellar mass may be present and can help in their differentiation. Correct preoperative diagnosis is clinically important because the treatment of choice for many of these nonpituitary sellar masses differs from that of a pituitary tumor. This article provides an overview of the clinical and radiographic characteristics of both pituitary tumors and the nonpituitary lesions found in the sellar/parasellar region and discusses in detail the specific nonpituitary etiologies of the sellar mass.

Signs and Symptoms of Pituitary Tumors

Pituitary tumors vary in presentation. Clinical findings depend largely on whether the tumor is hormone secreting or clinically nonfunctioning, on the size and pattern of tumor growth, and on whether normal pituitary gland function is disrupted. Hormone-secreting tumors most commonly present with a clinical...
Table 1. DIAGNOSES IN 1120 PATIENTS UNDERGOING TRANSSPHENOIDAL SURGERY FOR SELLAR MASSES FROM JANUARY 1981 THROUGH MAY 1998 BY KDP

<table>
<thead>
<tr>
<th>Diagnosis</th>
<th>No. of Patients (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Pituitary tumors</strong></td>
<td></td>
</tr>
<tr>
<td>Hormone-secreting tumors</td>
<td>533 (48)</td>
</tr>
<tr>
<td>Nonsecreting tumors</td>
<td>483 (43)</td>
</tr>
<tr>
<td><strong>Nonpituitary sellar/parasellar lesions</strong></td>
<td></td>
</tr>
<tr>
<td>Cell rest tumors</td>
<td></td>
</tr>
<tr>
<td>Craniopharyngioma</td>
<td>17 (16)</td>
</tr>
<tr>
<td>Rathke's cleft cyst</td>
<td>35 (33)</td>
</tr>
<tr>
<td>Epidermoid</td>
<td>1 (1)</td>
</tr>
<tr>
<td>Chordoma</td>
<td>10 (10)</td>
</tr>
<tr>
<td>Other cyst</td>
<td>5 (5)</td>
</tr>
<tr>
<td><strong>Benign lesions</strong></td>
<td></td>
</tr>
<tr>
<td>Meningioma</td>
<td>8 (8)</td>
</tr>
<tr>
<td><strong>Metastatic tumors</strong></td>
<td></td>
</tr>
<tr>
<td>Breast</td>
<td>11 (10)</td>
</tr>
<tr>
<td>Prostate</td>
<td></td>
</tr>
<tr>
<td>Lung</td>
<td></td>
</tr>
<tr>
<td>Renal cell</td>
<td></td>
</tr>
<tr>
<td>Parotid</td>
<td></td>
</tr>
<tr>
<td>SNUC*</td>
<td></td>
</tr>
<tr>
<td>Unknown primary</td>
<td></td>
</tr>
<tr>
<td><strong>Lymphoma</strong></td>
<td></td>
</tr>
<tr>
<td><strong>Vascular lesions</strong></td>
<td></td>
</tr>
<tr>
<td>Aneurysm</td>
<td></td>
</tr>
<tr>
<td>Granulomatous, infectious, and inflammatory</td>
<td></td>
</tr>
<tr>
<td>Sarroid</td>
<td></td>
</tr>
<tr>
<td>Granulomatous hypophysitis</td>
<td></td>
</tr>
<tr>
<td>Pituitary abscess</td>
<td></td>
</tr>
<tr>
<td>Mucocoele</td>
<td></td>
</tr>
<tr>
<td>Lymphocytic hypophysitis</td>
<td></td>
</tr>
<tr>
<td>Miscellaneous (CSF-related)</td>
<td></td>
</tr>
<tr>
<td>Arachnoid cyst</td>
<td></td>
</tr>
<tr>
<td><strong>TOTAL</strong></td>
<td>1120</td>
</tr>
</tbody>
</table>

*SNUC = sino-nasal undifferentiated carcinoma.

syndrome reflecting the hormone in excess; however, in some patients with these tumors, particularly, some growth hormone– or prolactin-secreting tumors, the clinical syndrome may go unrecognized until the tumor is large, producing clinical findings characteristic of nonsecreting adenomas. Nonsecreting pituitary tumors typically present with visual compromise, including impairment of the visual fields and decreased acuity, signs of hypopituitarism, and nonspecific symptoms such as headache. Modest hyperprolactinemia secondary to hypothalamic-pituitary stalk compression is common. Diabetes insipidus is extremely rare at presentation of a pituitary adenoma. A cranial neuropathy may occasionally be found at presentation of a pituitary tumor. Not infrequently, even large tumors may be asymptomatic and discovered incidentally after an imaging study of the region is performed for an unrelated reason. If hemorrhage into a pituitary tumor occurs, that is, pituitary apoplexy, patients may present with varying combinations of sudden onset of severe headache, cranial neuropathy, visual impairment, and hypopituitarism. If very large, pituitary tumors may cause hydrocephalus and signs of increased intracranial pressure.
Table 2. CLASSIFICATION OF PARASELLAR AND INTRASELLAR NONPITUITARY LESIONS

<table>
<thead>
<tr>
<th>Cell rest tumors</th>
<th>Benign lesions</th>
</tr>
</thead>
<tbody>
<tr>
<td>Craniopharyngioma</td>
<td>Meningioma (olfactory, tuberculum, diaphragms, sphenoid wing)</td>
</tr>
<tr>
<td>Rathke’s cleft cyst</td>
<td>Enchondroma</td>
</tr>
<tr>
<td>Epidermoid (cholesteatoma)</td>
<td>Metastatic tumors</td>
</tr>
<tr>
<td>Infundibuloma</td>
<td>Vascular lesions</td>
</tr>
<tr>
<td>Chordoma</td>
<td>Granulomatous, infectious, and inflammatory</td>
</tr>
<tr>
<td>Lipoma</td>
<td>Pituitary abscess, bacterial and fungal</td>
</tr>
<tr>
<td>Colloid cyst</td>
<td>Sarcoid</td>
</tr>
</tbody>
</table>

| Primitive germ cell tumors|                                |
|---------------------------|                                |
| Germoblastoma             | Tuberculosis                    |
| Germinoma                 | Giant cell granuloma            |
| Teratoma                  | Echinococcal cyst               |
| Atypical teratoma (dyserminoma) | Mucocele (sphenoid)         |
| Ectopic pinealoma         | Histiocytosis X                 |
| Gliomas                   | Lymphocytic hypophysitis        |
| Chiasmatic-optic glioma   | Miscellaneous (CSF-related)     |
| (astrocytoma, hypothalamic glioma) | Benign intracranial hypertension (pseudotumor cerebri) |
| Oligodendroglioma         | Empty sella syndrome            |
| Ependymoma                | Arachnoid cyt                   |
| Infundibuloma             | Suprasellar-chiasmatic arachnoiditis |
| Astrocytoma               |                                     |
| Microglioma               |                                     |

SIGNS AND SYMPTOMS OF NONPITUITARY SELLAR LESIONS

Many nonpituitary sellar and parasellar masses, similarly to pituitary adenomas, may present with symptoms of anterior pituitary hormone dysfunction. These symptoms include gonadal dysfunction, secondary hypothyroidism, and, less often, clinical adrenal cortical insufficiency. Children may present with growth failure and lack of secondary sexual development with lesions compressing the pituitary, hypothalamus, or infundibulum. The onset of symptoms of a sellar mass and hypopituitarism in late pregnancy or the early postpartum period should raise suspicion for lymphocytic hypophysitis.

Hyperprolactinemia, which may be asymptomatic or accompanied by hypogonadism, is often found at presentation of lesions of the sellar region such as meningiomas, craniopharyngiomas, and cystic lesions. These lesions can compress the hypothalamic-pituitary stalk, elevating the prolactin level, which typically is in the range of 20 to 100 ng/mL but may be as high as 150 to 200 ng/mL. An elevation of prolactin to a level greater than 200 ng per mL is consistent with a prolactinoma. A prolactinoma should be considered and excluded by measurement of the prolactin level in all patients with sellar or parasellar masses. Samples should always be diluted to guard against the reporting of very high prolactin levels as spuriously normal because of the high dose hook effect found in certain immunoradiometric assays.

Clinical diabetes insipidus at presentation is highly suggestive of a nonpituitary etiology of a sellar or parasellar mass. Sarcoidosis and metastatic disease to the sellar region are especially likely to lead to diabetes insipidus. Diabetes insipidus may result from involvement or compression of the pituitary...
stalk, hypothalamus, or paraventricular region of the third ventricle by the lesion. Vasopressin deficiency may be partial or transient in some patients because regeneration of the vasopressin-containing neurohypophyseal fibers may occur. In addition, the apparent spontaneous improvement of diabetes insipidus in some patients may coincide with the development of hypopituitarism. The syndrome of inappropriate antidiuretic hormone secretion leading to potentially severe hyponatremia may also occur in patients with nonpituitary sellar and parasellar lesions.

Visual loss is a common presenting complaint with sellar/parasellar nonpituitary lesions because of the proximity of the optic nerves, chiasm, and optic tracts to the sella turcica. Because visual loss may be insidious in onset and progress slowly, severe deficits frequently are present before the patient seeks medical attention. In children, in particular, severe visual loss as a result of optic nerve compression by lesions such as craniopharyngiomas may occur before a vision problem is noticed. The particular visual field loss may provide some clue as to the nature of the lesion. Lesions anterior to the chiasm, such as meningiomas of the optic nerve sheath, can produce unilateral visual loss, whereas lesions compressing the visual system more posteriorly along the optic tract, such as meningiomas or aneurysms, can produce homonymous hemianopias that are characteristically incongruous. Lesions within the sella, such as Rathke's cleft cysts, can produce the typical bitemporal field deficit associated with chiasmal compression from below, whereas lesions in the suprasellar area, such as meningiomas, can present with bitemporal field cuts of the classic superior chiasmal compression variety. Lesions involving the chiasm, such as gliomas, may present with more unusual visual deficits. Tumors causing obstruction of cerebrospinal fluid (CSF) flow may lead to an increase in intracranial pressure, which may manifest as transient visual obscurations. In contrast to adenomas, nonpituitary masses more commonly originate from or infiltrate parasellar structures that are within close proximity of the cranial nerves that traverse through the cavernous sinuses. As a result, there is an increased incidence of cranial neuropathy with these lesions; as many as 25% of patients with nonpituitary sellar or parasellar masses will present with cranial nerve abnormalities, with cranial nerves II, III, IV, or VI being the most commonly affected. Although the acute onset of cranial neuropathy often accompanies apoplexy into an adenoma, the presence of a cranial neuropathy at presentation is still suggestive of a nonpituitary etiology for a sellar/parasellar mass.

Signs and symptoms of increased intracranial pressure may be the presenting sign of tumors such as craniopharyngioma, meningioma, and germinoma. Headache is often a prominent symptom in patients with large tumors that produce ventricular dilatation. Headache may also be present in patients with other parasellar tumors, including intrasellar or suprasellar cysts and inflammatory disorders that are too small to raise intracranial pressure. Headache in these patients may be caused by distortion of the diaphragms or irritation of the parasellar dura.

Patients with masses compressing the deep subfrontal region, including the septal portion of the anterior third ventricle, may present with personality changes and dementia. Tumors in this location may compress the olfactory tracts, which are in close proximity to the suprasellar region, leading to anosmia. Hypothalamic tumors in children may produce the diencephalic syndrome manifest as wasting, poor development, and sexual immaturity. Hypothalamic dysfunction in adults may lead to disruption of the control of appetite and cause syndromes of polyphagia and massive obesity or severe starvation.
OVERVIEW OF RADIOGRAPHIC FEATURES OF PITUITARY TUMORS AND NONPITUITARY INTRASELLAR AND PARASELLAR LESIONS

Magnetic resonance imaging has become the imaging modality of choice for the evaluation of the sellar and parasellar regions. The normal anterior pituitary gland demonstrates a signal intensity similar to white matter, whereas the posterior pituitary gland appears bright in most patients owing to the presence of phospholipid within the neurosecretory granules. This bright spot may not be seen in 10% to 20% of normal individuals. With the administration of gadolinium DTPA (paramagnetic contrast agent), the pituitary gland and stalk enhance or become brighter in signal intensity. Pituitary adenomas, by contrast, are most often hypointense relative to the normal gland on T1-weighted images, and approximately one third to one half are hyperintense on T2-weighted images. Following contrast administration, the normal pituitary gland typically enhances earlier and more intensely than an adenoma. On delayed images, this pattern may be reversed. Pituitary adenomas may be confined to the sella, extend suprasellarly toward the optic chiasm, laterally into the cavernous sinuses, and inferiorly into the sphenoid sinus. In 94% to 100% of cases, adenomas will enlarge the sella. As many as 50% of nonadenomatous masses such as meningiomas, craniopharyngiomas, and Rathke’s cleft cysts can enlarge the sella. Therefore, only the lack of sellar enlargement is helpful in diagnosis, which is suggestive for a nonpituitary lesion. Adenomas may contain cystic degeneration and may invade and erode bone in more than 50% of cases as do many nonpituitary masses. Therefore, this finding is not particularly helpful in the differential diagnosis. Although some nonpituitary lesions are indistinguishable from adenomas on MR imaging, many lesions do have characteristic findings that are helpful in the differential diagnosis.

In general, MR imaging has supplanted the use of CT for the evaluation of a known sellar region mass. CT still provides some advantage over MR imaging in detecting the presence or absence of tumoral calcification and in the evaluation of bony anatomy. Calcification suggests craniopharyngiomas, meningiomas, chordomas, teratomas, gliomas, or an aneurysm, but pituitary adenomas may also contain calcifications. Occasionally, skull films incidentally lead to the diagnosis of a sellar region mass, which is suggested by bony destruction, enlargement of the sella turcica, or the presence of abnormal calcification. Erosion of the floor of the sella can be seen with adenomas, intracavernous aneurysms, meningiomas of the middle fossa, Rathke’s cleft cyst, arachnoid diverticula, and elevated intracranial pressure from any source.

Some recently developed imaging techniques may become clinically useful in the differential diagnosis of sellar masses. In some cases, positron emission tomography scanning may distinguish viable tumor tissue from fibrous tissue, cysts, and necrosis. Magnetic resonance spectroscopy or somatostatin receptor imaging with 111In-octreotide may become helpful in the evaluation of certain brain and parasellar tumors.

SPECIFIC LESIONS OF THE SELLAR AND PARASELLAR REGIONS

Cysts of the Sellar and Parasellar Regions

The most common nonpituitary sellar and parasellar masses are primarily cystic lesions. Most of these cysts, including craniopharyngiomas, Rathke’s cleft
cysts, and epidermoid and dermoid cysts, are derived from cystic epithelium. Others, such as arachnoid cysts, have fluid-secreting walls and contain CSF. In general, both intrasellar and suprasellar cysts can produce signs and symptoms similar to those of adenomas, such as visual impairment or hypopituitarism, and therefore are important considerations in the differential diagnosis of an adenoma. The typical clinical presentations and radiographic appearances of the more common types of cysts found in the sellar and parasellar regions are discussed in the following sections.

**Craniopharyngiomas**

Craniopharyngiomas are derived from embryonic squamous cell rests of Rathke’s cleft that remain after upward migration of stomodeal epithelium to the upper portion of the anterior lobe of the pituitary gland. They exhibit a spectrum of cytologic patterns ranging from the well-differentiated papillary type to the more primitive and invasive adamantinomatous subtype. Craniopharyngiomas have a bimodal peak of incidence, occurring predominantly in children between the ages of 5 and 10 years, in whom they account for 5% to 10% of brain tumors. A second smaller peak in incidence occurs in the sixth decade. There is a female preponderance. Most craniopharyngiomas present as a calcific, cystic suprasellar mass; others present as a sellar/suprasellar mass and, in rare cases, as a completely intrasellar mass.

The presenting signs and symptoms of craniopharyngiomas vary with the age of the patient, the size of the tumor, and whether the lesion is primarily intrasellar or suprasellar in location. Therefore, the clinical presentation of some craniopharyngiomas may more closely resemble that of an adenoma. Children typically present with headache, nausea and vomiting, and signs of increased intracranial pressure, including papilledema. Visual compromise, with loss of acuity, visual field deficits, or both, is also common in children with craniopharyngiomas. Endocrinologic dysfunction in children is most often manifest as either growth failure, seen in as many as 93% of patients at presentation, or delayed sexual development, seen in approximately 20%. The visual and endocrinologic abnormalities frequently are initially unrecognized, and as a result, these tumors can become large and cause CSF obstruction and signs of increased intracranial pressure before the diagnosis is made.

As many as 80% of adults with craniopharyngiomas present because of visual difficulties. On examination, additional patients are found to have previously unnoticed visual abnormalities, including optic atrophy, papilledema, and visual field deficits. The most commonly seen visual field deficit is a bitemporal hemianopsia, usually asymmetrical, but the deficit may vary depending on the position of the optic nerves and chiasm relative to the tumor. Abnormalities of the third, fourth, and sixth cranial nerves and the first division of the trigeminal nerve may occur owing to tumoral involvement in the cavernous sinus. Many adults present with a variety of anterior pituitary hormone deficiencies. The most common is gonadotropin deficiency, although growth hormone, pituitary-adrenal, and thyroid axes can also be impaired. Prolactin levels may be moderately elevated in as many as half the patients owing to compression on the hypothalamic-pituitary stalk. Diabetes insipidus is present in 23% of patients preoperatively and provides an important clue to the nonpituitary nature of the lesion. Other hypothalamic symptoms can also occur in adults, such as obesity, somnolence, and hypothermia or hyperthermia.

The appearance of craniopharyngiomas on MR imaging varies because they differ in the proportions of solid versus cystic components, in the amount of
calcifications, and in the content of the cyst fluid. The solid portions typically appear isointense or hypointense on T1-weighted images and hyperintense on T2-weighted images\textsuperscript{61} but can also have a mottled appearance owing to calcific regions on MR imaging.\textsuperscript{155} Cystic components demonstrate a high signal on T1-weighted images owing to their high protein content or hemorrhagic components. Tumoral calcification, which may be best appreciated on CT scan,\textsuperscript{81} is particularly common and is seen in 70\% to 90\% of childhood craniopharyngiomas and 40\% to 60\% of adulthood tumors.\textsuperscript{163} Suprasellar calcification in a child is highly suggestive of the diagnosis of craniopharyngioma.\textsuperscript{124} Although the presence of calcifications may be helpful in the differential diagnosis, it is not specific. An abnormal sella may be seen in 50\% to 70\% of patients of all ages (Figs. 1 to 5).\textsuperscript{124}

\textit{Rathke's Cleft Cysts}

Rathke's cleft cysts form from remnants of the squamous epithelium of Rathke's pouch. The cyst wall consists of a single layer of epithelial cells, with mucoid, cellular, or serous contents in the cyst fluid. These tumors generally

\begin{figure}[h]
\centering
\includegraphics[width=\textwidth]{image}
\caption{A and B, T1-weighted nonenhanced MR images of a 15-year-old girl with amenorrhea demonstrating high signal within an intrasellar mass. No suprasellar extension was seen. Pathology was a craniopharyngioma.}
\end{figure}
Figure 2. A and B, MR images of a 34-year-old woman presenting with headache. Endocrinologic function and neurologic examination were normal. Enhanced MR image shows a round mass above and separate from the pituitary gland and the optic chiasm. The pituitary stalk is posterior to the lesion. Pathology was a craniopharyngioma.

occur in adults, with a mean age at presentation of 38 years and a female-to-male ratio of 2:1.\textsuperscript{157}

Although Rathke's cleft cysts typically remain small, intrasellar, and asymptomatic, approximately one third can extend suprasellarly.\textsuperscript{111} On occasion, they can be entirely suprasellar in location.\textsuperscript{76} Rarely, but more commonly than previously believed,\textsuperscript{47} these cysts may become symptomatic and compress adjacent structures, leading to symptoms similar to those associated with adenomas, such as headache and abnormalities of vision, typically visual field deficits, and anterior pituitary hormone deficiency.\textsuperscript{47, 54, 111, 157} Some patients may also present with hyperprolactinemia owing to stalk compression and others with diabetes insipidus.\textsuperscript{54, 111, 157}

Rathke's cleft cysts typically are seen as discrete cystic lesions on MR imaging, but MR signal intensity varies because of differences in cyst fluid composition.\textsuperscript{7, 81, 136} Rathke's cleft cysts can appear to have the density of CSF, with low signal intensity on T1-weighted images and high signal intensity on T2-weighted images, which on occasion has led to a mistaken diagnosis of arachnoid cyst.\textsuperscript{118} Other Rathke's cleft cysts can appear isointense to hyperintense
Figure 3. A and B, MR images of a 30-year-old woman presenting with visual loss. The enhanced study shows a mixed signal with solid and cystic components. The mass is completely separate from the pituitary gland as seen on the sagittal image.

on T1-weighted images.\textsuperscript{141, 157} Only the cyst wall enhances in a thin rim with contrast\textsuperscript{139, 81} which may help in the differentiation from craniopharyngiomas.\textsuperscript{36} Rarely, Rathke's cleft cysts may be associated with pituitary adenomas.\textsuperscript{111, 115} Although calcification of the cyst has been reported in isolated cases,\textsuperscript{4, 113} lack of calcification is thought to be characteristic of these cysts and useful in differentiating them from craniopharyngioma.\textsuperscript{111}

Preoperative diagnosis, if possible, is helpful surgically because, in many cases, drainage of the cyst without full resection is sufficient.\textsuperscript{127, 157} Although these lesions usually do not recur, as many as one third of patients may require repeat resection\textsuperscript{54, 111} (Figs. 6 and 7).

**Arachnoid Cysts**

Arachnoid cysts are a rare cause of cystic lesions in the sella. They may be the result of congenital maldevelopment of the subarachnoid space or secondary to a postinfectious adhesive arachnoiditis.\textsuperscript{36, 124} Arachnoid cysts may be
Figure 4. A, Coronal enhanced T1 MR image of a 15-year-old boy presenting with a blind right eye and temporal visual field deficit in the left eye. A cystic rim enhancing mass is seen above the pituitary gland. B, Sagittal-enhanced T1 MR image demonstrating the enlarged sella with the mass filling the sphenoid sinus and suprasellar regions. C, Axial T2 MR image demonstrating the cystic content and irregular enhancing solid component. Pathology is a craniopharyngioma.

intranasal or suprasellar in location. They may present with visual symptoms from compression of the optic nerves, chiasm, or tracts or signs of increased intracranial pressure. Hydrocephalus secondary to obstruction of the foramina of Monro can occur.

On MR imaging, arachnoid cysts appear as smooth, contoured, well-marginated masses that are isointense to CSF on all sequences. The differential diagnosis of such a signal pattern includes a Rathke’s cleft cyst, ependymal cyst, or a parasitic cyst. Calcifications are absent, and these cysts do not exhibit central or rim enhancement with contrast (Fig. 8).

Epidermoid Cysts

Epidermoid cysts are rare, slow-growing epithelial inclusion cysts that may present, typically in the fourth and fifth decades, as masses of the sellar and parasellar regions. Epidermoid cysts can present with visual disturbance, hypothalamic, diabetes insipidus, and cranial nerve abnormalities. These cysts generally appear isointense to CSF on T1-weighted images and hypointense to hyperintense in signal on T2-weighted images and do not enhance with contrast.
Figure 5. Plain skull radiograph (A) and CT scan (B) demonstrating suprasellar calcification in a craniopharyngioma.

OTHER PARASELLAR LESIONS

Chordomas

Chordomas are rare tumors that arise from notochordal remnants within the clivus in approximately 39% of cases. In other instances, these tumors arise from elsewhere within the sellar or parasellar region.\textsuperscript{93} Chordomas typically produce bony destruction with local infiltration. They tend to recur.\textsuperscript{101, 158} Although chordomas occur in adults of all ages, the majority present between the ages of 30 and 50 years.\textsuperscript{93, 101, 158} These tumors are more common in men.\textsuperscript{81}
Figure 6. Coronal (A) and sagittal (B) T1 MR images of a 28-year-old woman presenting with headache and amenorrhea. A cystic thin rim enhancing intra- and suprasellar lesion is seen. The normal gland is displaced around the cyst. Pathology is a Rathke's Cleft cyst.

Symptoms depend on the direction of growth of the tumor. Cranial neuropathy and diplopia are the most common presenting symptoms. Approximately one third of patients have involvement of the sixth, third, or fourth cranial nerves, which is typically asymmetric, and most patients have more than one nerve affected. Field defects, when they occur, are similar to those seen with pituitary adenoma. Headaches occur early, and neck pain and symptoms of nasopharyngeal congestion may be present. Endocrinologic dysfunction is unusual, but anterior pituitary insufficiency or mild hyperprolactinemia may occur in some cases.

Magnetic resonance imaging is the procedure of choice for detecting a clival chordoma and determining the extent of the tumor and involvement of adjacent

Figure 7. Sagittal-enhanced MR image of a 50-year-old woman presenting with headache and visual loss shows a large cystic lesion, with layering of fluid, reaching well into the third ventricle. Preoperative diagnosis was pituitary apoplexy. Pathology was a Rathke's Cleft cyst.
Figure 8. Axial (A) and sagittal (B) MR images of a sellar and suprasellar cyst without enhancement. Pathology was an arachnoid cyst.

structures. On MR imaging, chordomas most commonly appear as destructive invasive lesions in the clivus. Most lesions are isointense to gray matter on T1-weighted images, heterogeneously hyperintense on T2-weighted images, and may enhance after contrast administration. Calcification is seen in as many as 50% of cases. In some cases, normal pituitary gland can be seen as distinct from the tumor on MR imaging, a finding that is helpful in differentiating this tumor from invasive pituitary adenomas, which can often similarly lead to extensive bony destruction. CT may compliment MR imaging because of its better visualization of bony lysis. The sella is rarely ballooned as seen in patients with pituitary tumors, but the sellar floor may be thinned or completely destroyed in many cases (Fig. 9).

Primitive Germ Cell Tumors

As many as 40% of central nervous system germ cell tumors can present in the suprasellar region. These tumors include germinomas, teratomas, and ectopic or metastatic pinealomas and present most commonly in the second to third decades of life. Germinoma is the most common germ cell tumor to arise in the sellar/suprasellar regions. Germinomas may arise in the anterior third ventricle and involve the hypothalamus. These tumors can also extend inferiorly and posteriorly to involve the chiasm and optic nerves or may extend into the sella, disrupting pituitary function. Germinomas of the ventral hypothalamus may occur in association with germinoma in the pineal region. It is likely that the former are metastatic lesions, but they may also be part of multifocal disease. Rarely, pure intrasellar germ cell tumor with hypopituitarism has been reported. Primary suprasellar germinomas have no sexual predilection, but germinomas originating in the pineal gland that are ectopic or metastatic to the suprasellar region show a male predominance.

Germinomas often present with endocrinologic abnormalities, including hypopituitarism or pituitary hyperfunction, manifest in some cases as precocious puberty and diabetes insipidus. Diabetes insipidus may be a prominent and
early finding in patients with suprasellar germinomas. As is true for other causes of diabetes insipidus, the condition may appear only after the administration of steroids to a patient with associated anterior pituitary insufficiency. Visual disturbances, including visual field deficits or optic atrophy, hypothalamic symptoms or hydrocephalus, and signs of increased intracranial pressure may also be found.124

On MR imaging, a germinoma can appear as a suprasellar often infiltrative mass that is usually isointense to brain on T1-weighted images and isointense or hyperintense on T2-weighted images.36, 81 Germinomas tend to be homogeneous in signal intensity, rarely are cystic, and markedly enhance with contrast administration. Loss of the posterior pituitary bright spot is typical. Teratomas show a mixed signal intensity with fat and calcifications on MR imaging (Fig. 10).

Germinomas metastasize within the central nervous system in approximately 10% of cases, and a majority have abnormal CSF. The tumor may elaborate beta-human chorionic gonadotropin or alpha-fetoprotein, and this may
Figure 10. Sagittal-enhanced T1 MR image of a 28-year-old man presenting with diabetes insipidus and mild bitemporal visual field defects. Endocrine studies showed mild hyperprolactinemia to 38 ng/mL, and low testosterone and thyroid hormone levels. An irregular mass is seen in the suprasellar postchiasmatic third ventricular region and a second mass is seen in the pineal region. Serum levels of alpha fetoprotein and human chorionic gonadotropin were normal. Pathology was a germinoma.

be detected in the CSF or blood, helping to confirm the diagnosis. Extracranial metastases to the lung have been reported. In the appropriate clinical setting and presentation, biopsy may be all that is needed to secure the diagnosis before instituting radiotherapy and chemotherapy.

Dermoid Tumors

Dermoid tumors are rare developmental tumors that present in childhood. A recurrent meningitis from leakage of tumor contents may be a clue to the diagnosis. On MR imaging, dermoid tumors are typically midline lesions that appear heterogeneous and bright on T1-weighted images owing to fat signal within the tumor.

Optic Nerve Gliomas

Optic nerve gliomas are rare tumors that comprise approximately 2% of orbital tumors at all ages. The patterns of disease differ somewhat between children and adults. The childhood variety is typically benign and slow-growing, but the tumors can infiltrate along the chiasm and optic nerves, with occasional malignant degeneration. Most children present at less than 10 years of age. Children with neurofibromatosis have an increased incidence of these tumors, and in these cases, the tumors tend to be multicentric. Overall, less than 20% of childhood optic gliomas are found in children with neurofibromatosis. The most frequent presenting symptoms in children are loss of vision, headaches, and proptosis. Often, the visual loss remains unrecognized until the tumor is advanced.

The adult type of optic glioma tends to be more malignant and often presents initially with monocular blurring of vision and retrobulbar pain which may progress rapidly to blindness. The patterns of field defects are extremely
variable and nonspecific. Rarely, optic nerve glioma has presented as an intrasellar cystic mass.\textsuperscript{166} On MR imaging, these lesions can usually be localized to the optic chiasm. Typically, they are isointense to hypointense on T1-weighted images and hyperintense on T2-weighted images, with characteristic demonstration of infiltration along the optic nerves. Optic nerve gliomas enhance with contrast homogeneously in most instances.\textsuperscript{81} Important in the differential diagnosis are benign hamartomas, which occur in association with neurofibromatosis but do not enhance with contrast and do not grow rapidly.\textsuperscript{21} The sella is normal in most patients. In children, the young age, marked visual change, intact pituitary function, and radiographic presentation are usually distinct for optic nerve glioma\textsuperscript{124} (Fig. 11).

**Hypothalamic Glioma**

Hypothalamic glioma is a rare tumor, occurring almost always in early life. These lesions usually present with disruption of hypothalamic function, diabetes

\begin{figure}[h]
\centering
\includegraphics[width=\textwidth]{figure11.png}
\caption{A, An enhanced coronal T1-MR image in a 38-year-old woman demonstrating a suprasellar mass to the right of the pituitary stalk. It appears to blend with the right side of the optic chiasm. The pituitary stalk is displaced to the left. B, Sagittal T1 nonenhanced MR image demonstrating a mass indistinguishable from the optic chiasm. Pathology was an optic glioma. C, Coronal enhanced T1 MR image in another patient showing a mass in and about the right side of the optic chiasm. The gland is clearly separate from the mass. Pathology was also an optic glioma.}
\end{figure}
insipidus, and visual loss with optic atrophy. On MR imaging, these tumors do not spread along the optic nerves but rather are more invasive into the hypothalamus. Hypothalamic gliomas are typically isointense on T1-weighted images and isointense to hyperintense on T2-weighted images. These lesions usually enhance homogeneously with contrast. The tumor may extend into the suprasellar cistern or the surrounding brain parenchyma and, when large, may mimic a suprasellar adenoma radiographically.

Parasellar Meningioma

Meningiomas are benign lesions that can present as sellar/suprasellar masses. They can arise from the tuberculum sella, planum sphenoidal, or diaphragma sellae. Laterally, they may also grow from the medial sphenoid ridge and cavernous sinus and project into the sella, simulating an adenoma. Intrasellar meningiomas are rare but have been reported. Meningiomas are more common in women and usually present in adult life, with the peak age at diagnosis between 40 and 50 years. The clinical presentation of a patient with a meningioma may provide clues to differentiate these lesions from pituitary adenomas. Meningiomas often present with severe visual disturbance but lack of endocrine dysfunction. The visual loss, which is the most common presenting complaint, may begin with blurring in part of a monocular field or a decrease of central visual acuity and then progress to bilateral loss of vision. The visual field deficits produced by meningiomas are varied. Although an asymmetric variant of a bitemporal hemianopsia is common, various deficits of both central and peripheral visual fields can occur. Visual loss, in contrast to retrobulbar neuritis, is not associated with pain on eye movement and is progressive rather than sudden. Optic atrophy is often seen on examination. Extraocular muscle palsies may occur. Both frontal and orbital headaches are common. Meningiomas uncommonly present with endocrine dysfunction, but mild-to-moderate hyperprolactinemia may be found at presentation in as many as half of patients. Meningiomas have been reported to increase in size during menses or pregnancy and to become symptomatic.

Certain characteristics on MR imaging favor the diagnosis of meningioma over other sellar masses. Meningiomas typically appear isointense on T1-weighted as well as T2-weighted images, helping to differentiate them from craniopharyngiomas and Rathke’s cleft cysts that typically appear hyperintense on T2-weighted images. Dense or bright homogeneous enhancement is characteristic of meningiomas. Although not specific, flow voids and prominent vascularity are typically associated with meningiomas. A tapered extension of an intracranial dural base or “tail” is suggestive of a meningioma but is not specific and can be seen in association with an adenoma, metastases to the pituitary, lymphoma, and lymphocytic hypophysitis. A suprasellar rather than sellar epicenter of the tumor and obtuse margins at the edge of the meningioma are also characteristic. These lesions can often be seen as distinct from the pituitary gland on MR imaging. Typical CT findings in patients with meningiomas include a smooth well-defined outline, high baseline density on noncontrast scan, and uniform intense contrast enhancement. Hyperostosis of the contiguous bones or dense calcification of associated dural structures is also suggestive of meningiomas and, in particular, tuberculum sella meningiomas. The sella is typically normal in size. With cavernous sinus invasion, the carotid
Figure 12. Coronal (A) and sagittal (B) enhanced T1-weighted MR images showing a small diaphragma sella meningioma. The pituitary gland can be seen below the mass. Often only a fine line of separation exists between the tumor and the normal gland.

Figure 13. A, Sagittal MR image of a sellar and suprasellar tumor creeping along the planum sphenoidale typical of a meningioma. Pathology was a pituitary adenoma. B, Coronal T1 MR image (same patient as in A). Note the slight waist effect from the diaphragma sella. C, Similar MR image of a different patient with more extensive spread along the planum. Pathology was a meningioma.
artery may be narrowed. This is most unusual with a pituitary adenoma (Figs. 12 to 14).

The preoperative diagnosis of a meningioma, if possible, is important because, in most cases, craniotomy rather than transsphenoidal surgery is the approach of choice for resection.143

**Pituitary Metastases**

Metastatic carcinoma to the pituitary is another important diagnostic consideration in the patient presenting with a sellar mass. Although metastases to the pituitary gland frequently are not clinically significant, when they become symptomatic, they are usually associated with known metastatic carcinoma elsewhere.96, 134 In some cases, the pituitary lesion may be the only metastatic focus in a patient with a known malignancy. Not infrequently, the pituitary lesion is the presenting sign of a carcinoma.24, 54, 128 Typically, patients are aged more than 50 years. The most common metastatic lesions to the pituitary are from breast carcinoma in women and from lung carcinoma in men. Tumors of the gastrointestinal tract, kidney, and prostate and melanoma are some of the other tumors commonly reported to metastasize to the pituitary gland.24, 54, 128

The clinical manifestations of metastatic carcinoma to the sellar region may be helpful in differentiating these lesions from an adenoma. In addition to having anterior pituitary dysfunction and visual field abnormalities, both often seen with adenomas, these patients frequently present with diabetes insipidus and cranial nerve palsies, which are atypical for adenomas. Involvement of the posterior pituitary gland occurs in approximately 85% of cases of metastatic disease to the pituitary,96 and diabetes insipidus may develop in 28% to 70% of patients with these lesions.24, 104, 130 Diabetes insipidus may be the initial manifestation of the malignancy and, in some cases, may be transient.85 Cranial nerve palsies are found in 12% to 43% of patients.24, 108, 138 A rapidly enlarging mass is highly suggestive of a metastatic lesion.134 The combination of age greater than 50 years, diabetes insipidus, cranial nerve abnormalities, and a rapidly enlarging
mass should increase the clinical suspicion for a metastatic lesion to the pituitary.\textsuperscript{1, 24}

On MR imaging, metastatic lesions to the pituitary can appear as enhancing sellar/suprasellar masses. These masses may on occasion be dumbbell-shaped, indenting the diaphragma sellae in contrast to adenomas, which usually expand the diaphragma.\textsuperscript{134} Loss of the posterior pituitary bright spot and thickening of the stalk may also be seen. These findings may correlate with infiltration of the posterior pituitary and stalk with metastatic tumor.\textsuperscript{88} Extensive erosion of bone and the sella can also occur with metastases.\textsuperscript{1} This is nonspecific, and erosion can be seen with pituitary adenomas and meningiomas (Figs. 15 and 16).

**Miscellaneous Tumors**

Several other unusual tumors are rare causes of sellar or parasellar masses. Hypothalamic hamartomas\textsuperscript{67} can occur in the sellar and parasellar areas. These tumors are often associated with precocious puberty, probably owing to secretion luteinizing hormone-releasing hormone. On MR imaging, hypothalamic hamartomas appear as nonenhancing sessile or pedunculated masses in the region of the tuber cinereum that are isointense to gray matter on T1-weighted images and hyperintense on T2-weighted images.\textsuperscript{23, 26, 64} Typically, they do not grow or invade. MR imaging should reliably identify these lesions as distinct from the pituitary gland.

Ganglion cell tumors or gangliocytomas are rare benign tumors that may originate in the pituitary or elsewhere in the sellar and suprasellar regions.\textsuperscript{16} They may consist of purely neuronal or mixed adenomatous and neuronal

![Figure 15. Coronal CT scan in a 30-year-old woman with known history of breast carcinoma. A large enhancing mass is seen enlarging the sella and extending suprasellarly. It is completely compatible with an adenoma. Pathology was metastatic breast carcinoma.](image-url)
Figure 16. A–C, MR image demonstrating a large mass in the left side of the sphenoid sinus and cavernous sinus. The pituitary gland is seen above the mass and is pushed slightly from left to right. Pathology was metastatic prostate carcinoma.

tissue. Typically, they have been found in association with a hormonally active adenoma, producing Cushing’s disease, acromegaly, or hyperprolactinemia. On MR imaging, these lesions are not different from macroadenomas.

Parasellar granular cell tumors, including myoblastomas and choristomas, and infundibulomas are rare tumors originating from the neurohypophysis or infundibulum. Although these tumors are rarely large enough to produce symptoms, patients can present with visual loss and visual field deficits, and approximately half will present with some degree of hypopituitarism or hyperprolactinemia. These lesions are not typically associated with diabetes insipidus. In later stages, hydrocephalus and signs of increased intracranial pressure may occur. On MR imaging, the tumors appear as masses of the posterior pituitary or stalk that are low or isointense in signal on T1-weighted images and show strong inhomogeneous enhancement with contrast. Rarely, an intrasellar solitary plasmacytoma may mimic a pituitary adenoma. Isolated cases have been reported of sellar/suprasellar hemangiopericytoma and rhabdomyosarcoma of the sphenoid sinus presenting similarly as an adenoma with headache and visual loss. A primary intrasellar melanoma has also been reported that appeared bright on T1-weighted images and dark on T2-weighted images. Lymphoma originating in the parasellar region is a very unusual cause of a sellar mass but can have a presentation similar to that of other lesions described here. A patient seen by the authors presented with cranial neuropathy (Fig. 17). Schwannomas typically involve the vestibular
branch of the acoustic nerve in the cerebellopontine angle, but the trigeminal nerve may be involved. In those cases, schwannomas may appear as a parasellar mass that is hypointense to gray matter on T1-weighted images and usually hyperintense on T2-weighted images with homogeneous contrast enhancement.\textsuperscript{81}

**ANEURYSMS**

An aneurysm arising from the cavernous, infraclinoid, or supraclinoid internal carotid arteries can mimic a mass in the sella.\textsuperscript{159} Aneurysms in the parasellar and suprasellar region may compress the optic nerve, chiasm, or both and produce signs of visual loss, which vary depending on the location of the aneurysm.\textsuperscript{124} Bitemporal deficits that begin as inferior quadrant loss, the sudden development of symptoms, and the accompanying findings of ocular motor palsies, supraorbital pain, and intense headache are suggestive of aneurysm.\textsuperscript{124} Sellar/parasellar aneurysms may also extend into the sella and cause direct pituitary compression and thus may be associated with modest hyperprolactinemia and hypopituitarism.\textsuperscript{84, 106, 159} Hypopituitarism ranges from symptoms of isolated hormone deficiency to panhypopituitarism and diabetes insipidus.

The introduction of MR imaging has greatly facilitated the differentiation of an aneurysm from other mass lesions. On precontrast T1-weighted MR images, aneurysms have the density of CSF. On spin echo MR imaging, a distinctive flow void caused by rapid flow through the aneurysm lumen is diagnostic.\textsuperscript{81}
Heterogeneously increased signal caused by deoxyhemoglobin or methemoglobin and calcification within the aneurysm are also characteristic. CT cannot reliably distinguish an adenoma from an aneurysm or other pituitary lesion, but intense homogeneous blush with contrast may suggest an aneurysm. This blush is similar to that of a highly vascular meningioma. Asymmetric enlargement and destruction of the sella turcica may occur in association with an aneurysm. A rim of calcification in the wall is characteristic but may resemble a craniopharyngioma. Aneurysms may be found incidentally in conjunction with pituitary tumors, but there does not seem to be an association between the entities.

Before transsphenoidal exploration, MR imaging should be performed to exclude an aneurysm (Fig. 18).

GRANULOMATOUS, INFLAMMATORY, AND INFECTIOUS PROCESSES

Several granulomatous and infectious processes can appear as sellar or parasellar masses and mimic an adenoma.

**Tuberculosis**

Tuberculosis of the central nervous system can involve the sellar and parasellar regions in numerous ways. In tuberculous meningitis, a dense plaquelike exudate at the base of the brain can involve the sellar and parasellar region. Tuberculosis may also present as a tuberculoma with all of the signs of a mass lesion, which can compromise hypothalamic or pituitary function. A tuberculoma may be sellar, suprasellar, or intrasellar. Hypopituitarism is frequently associated with the various presentations of tuberculosis of the sellar/parasellar region. Visual field defects and diabetes insipidus are common. Isolated tuberculoma of the sella has been reported to present with hypopituitarism, headache, and visual compromise. Although most patients with hypothalamic-pituitary tuberculosis have signs of active tuberculosis elsewhere, this is not invariably true. As a result, the diagnosis of a tuberculoma in the region may, in some cases, be made only histologically after transsphenoidal surgery and biopsy.

On MR imaging, tuberculous lesions are usually isointense on T1-weighted images and isointense to hyperintense on T2-weighted images. Both intrasellar and suprasellar tuberculomas show strong enhancement with contrast and are often accompanied by thickening and enhancement of the stalk and dura. Some lesions may extend into the hypothalamus.

**Sarcoid**

Sarcoidosis involves the central nervous system in 5% to 15% of patients with this disease. When neurosarcoidosis occurs, it seems to have a predilection for the hypothalamic-pituitary region. Typically, neurosarcoidosis is associated with systemic sarcoidosis, and only 5% of cases have no disease elsewhere. The pituitary as well as the hypothalamus can be infiltrated with the lymphocytic and granulomatous infiltrates characteristic of sarcoidosis, causing varying degrees of hypopituitarism with or without associated symptoms of an intrasellar mass. Because of the predilection for the hypothalamus, posterior pitu-
Figure 18. A, Axial bone window CT scan showing bony erosion of the left anterior clinoid region suggestive of an aneurysm. B, Axial-enhanced CT scan showing a right sellar and parasellar mass about the right carotid artery. C, Arteriogram (same patient as in B) showing an internal carotid artery aneurysm. Coronal (D) and sagittal (E) T1 MR images showing a large intrasellar flow void adjacent to the left internal carotid artery. F, Arteriogram (same patient as in D and E) showing a giant left internal carotid aneurysm filling the sella.

ity, and stalk, diabetes insipidus is common. Hyperprolactinemia has been described with hypothalamic-pituitary sarcoid. Cranial neuropathy may be present.\textsuperscript{53} Although hypothalamic-pituitary sarcoid is usually accompanied by other signs of neurosarcoid or systemic sarcoid that help make the diagnosis evident, signs of a sellar mass or hypothalamic-pituitary dysfunction may in rare cases be the initial or only sign of the disease.\textsuperscript{53}
On MR imaging, the intraparenchymal, meningeal, or sellar lesions of sarcoidosis appear isointense on T1-weighted images and variable on T2-weighted images. These lesions enhance and are typically accompanied by leptomeningeal enhancement after contrast administration. The stalk may be thickened and also enhance. The lesions of sarcoid may mimic an adenoma with an appearance similar to a sellar tumor and very rarely have been reported to be cystic (Fig. 19).

Giant Cell Granuloma

Giant cell granuloma or granulomatous hypophysitis is a rare disease that can present with a sellar/suprasellar mass and hypopituitarism. It is characterized by the presence of noncaseating giant cell granulomata that either partially or completely replace the pituitary gland in the absence of granulomatous involvement of other organ systems. It is most common in middle-aged to older women but can occur in men and younger women. The cause is unknown. The clinical presentation usually reflects varying degrees of hypopituitarism and hyperprolactinemia. Interestingly, hypopituitarism may be more profound with smaller inflammatory or granulomatous processes of the pituitary than would be expected with similarly sized adenomas. The granulomatous involvement in granulomatous hypophysitis is usually confined to the anterior lobe of the pituitary in contrast to sarcoidosis, which demonstrates early and preferential involvement of the posterior pituitary, infundibulum, and hypothalamus. Therefore, diabetes insipidus, although it has been reported to occur with giant cell granuloma, is usually not manifested early in the course of the disease.

Granulomatous hypophysitis may appear on MR imaging as a sellar/suprasellar mass similar to a pituitary adenoma, but unlike an adenoma, the mass demonstrates hyperintense enhancement with contrast. A tongue-like extension of the enhancing lesion may be seen along the basal hypothalamus accompanied by thickening of the pituitary stalk. Thickening of the stalk, although it can be seen in pituitary adenomas that compress the stalk from below, is also suggestive of this inflammatory process.

Histiocytosis X

Histiocytosis X encompasses a group of diseases characterized by proliferation and infiltration of histiocytes in affected sites. There is a spectrum of disease ranging from unifocal to multifocal eosinophilic granuloma, also called Hand-Schüller-Christian disease, to the more malignant form Letterer-Siwe disease. Central nervous system histiocytosis X seems to have a predilection for the hypothalamus. As a result, as many as one half of all patients with this disease have diabetes insipidus, and it is often a presenting sign. Other endocrinopathy can result from the hypothalamic or stalk involvement. Growth retardation is a common finding in children with histiocytosis X. Although the classic description of Hand-Schüller-Christian disease includes the triad of diabetes insipidus, exophthalmos, and lytic bone disease, only 25% of patients present with these findings. Bone disease is most commonly manifest as punched out skull, mandible, or long bones. Occasionally, these classic bone findings are seen as part of the clinical presentation and in combination with abnormal findings on MR imaging will yield the correct diagnosis. If there is no evidence
Figure 19. Sagittal (A) and coronal (B) gadolinium-enhanced T1 MR images of a 50-year-old woman presenting with diabetes insipidus and memory loss. Note the irregular enhancing mass in the hypothalamic area extending along the diencephalon. It is indistinguishable from a hypothalamic glioma. Pathology is sarcoidosis.
of histiocytosis X outside the central nervous system, correct preoperative diagnosis may be difficult.

On MR imaging, a suprasellar mass, hypothalamic lesions, and a thickened stalk may be seen, which appear hypointense on T1-weighted images and hyperintense on T2-weighted images. Lesions enhance brightly with contrast. There may also be loss of the posterior pituitary bright spot.

**Lymphocytic Hypophysitis**

Lymphocytic hypophysitis is a rare but increasingly recognized cause of sellar mass lesions; at least 100 cases have been reported since its first description. Lymphocytic hypophysitis preferentially affects women and in 60% to 70% of cases presents in late pregnancy or during the postpartum period. The diagnosis should be highly suspected in this setting. Approximately 20% to 25% of patients have a history of other autoimmune diseases, most commonly autoimmune thyroiditis, which can be a clue to the diagnosis. Lymphocytic hypophysitis is characterized by diffuse lymphocytic and plasma cell infiltration of primarily the pituitary and can present with an enlarging intrasellar and suprasellar mass and varying degrees of pituitary insufficiency. Hypopituitarism is present in 60% to 70% of patients, cranial neuropathy in 6%, hyperprolactinemia in as many as 38%, and diabetes insipidus in as many as 19%. Symptoms of the sellar mass with headache and visual impairment occur in 56% to 70%. In some cases, partial recovery of pituitary function, resolution of the sellar mass, or both in patients clinically suspected of having lymphocytic hypophysitis has occurred spontaneously or with conservative corticosteroid and hormone replacement therapy. A woman who has a presentation consistent with lymphocytic hypophysitis but who does not have significant visual compromise can be observed clinically and radiographically. The diagnosis will be confirmed by resolution of the mass over time. Surgery is needed if severe or worsening neurologic, visual, or other compressive symptoms develop.

Magnetic resonance imaging may show an enhancing pituitary mass in as many as 95% of cases, often with suprasellar extension and dural enhancement. There may also be loss of the posterior pituitary bright spot, thickening of the pituitary stalk, and, in some cases, extension of the lesion to the hypothalamus (Fig. 20).

**Pituitary Abscess**

Pituitary abscesses are rare but occur in all age groups. In most cases, they develop from direct extension of adjacent infection in the sphenoid sinuses and in other cases from CSF infection. Infection may also be spread by bacteremia. Very rarely, abscesses of the pituitary may be associated with cavernous sinus thrombosis. Most abscesses arise in previously healthy pituitary glands, but approximately one third have been found to be associated with other sellar lesions such as pituitary adenomas, craniopharyngiomas, or Rathke’s cleft cysts that undergo secondary infection. Pituitary abscess formation infrequently occurs following surgery to the region. The presence of CSF rhinorrhea and recurrent meningitis may also predispose to abscess formation. Although meningitis is present along with the abscess in approximately 60% of patients, symptoms suggestive of meningitis and fever may or may not be seen. Pituitary abscesses are often reported to be sterile; isolated organ-
isms are typically but not exclusively gram-positive cocci.\textsuperscript{18, 77, 136} Primary pituitary abscesses caused by fungi such as \textit{Aspergillus} or \textit{Candida albicans} and others caused by \textit{Mycobacterium tuberculosis} have been reported.\textsuperscript{77, 161, 162}

Patients with pituitary abscesses commonly present with symptoms indistinguishable from those found in patients with pituitary tumors, including headache, visual disturbances, and endocrinopathy.\textsuperscript{18, 77, 102, 161} As many as 75\% of patients present with signs of visual compromise and chiasmal compression.\textsuperscript{102} Visual defects in a patient with meningitis, especially when other suggestive clinical findings are present, should raise the possibility of an infectious pituitary lesion. Hypopituitarism is also frequently present at diagnosis of a pituitary abscess and may range from mild hyperprolactinemia to panhypopituitarism.\textsuperscript{45, 77, 124, 161} In addition, symptoms of diabetes insipidus are reported to be present in 50\% of patients,\textsuperscript{161} which may help differentiate patients with pituitary abscesses from patients with adenomas in whom diabetes insipidus at presentation is extremely rare.

On precontrast MR images, abscesses appear similar to adenomas with a signal intensity that is isointense or moderately hypointense on T1-weighted images and hyperintense on T2-weighted images.\textsuperscript{102, 161} Abscesses with the opposite pattern have also been reported.\textsuperscript{3, 82} In contrast to adenomas, abscesses characteristically demonstrate ring enhancement and a central cavity that is isointense to brain with contrast administration.\textsuperscript{36} Therefore, contrast-enhanced images should be obtained, if possible, in patients in whom the diagnosis of pituitary abscess is suspected.\textsuperscript{161} Absence of the posterior pituitary bright spot has also been reported on MR imaging, consistent with the frequently reported diagnosis of diabetes insipidus in these patients.\textsuperscript{161} The sella may be enlarged and, occasionally, extensively eroded\textsuperscript{46} (Fig. 21).

\textbf{Other Infectious Diseases}

Pituitary involvement has been reported with a number of fungal infections such as histoplasmosis, coccidioidomycosis,\textsuperscript{132} cryptococcus, candida, and \textit{aspergillus}.\textsuperscript{18} Cryptococcus infection has been reported to present as a sellar/suprasellar mass.\textsuperscript{164} Aspergillus infection of the sphenoidal sinus may develop into a

\begin{figure}[h]
\centering
\includegraphics[width=\textwidth]{image.png}
\caption{A and B, Two examples of women with hypopituitarism following pregnancy with sellar/suprasellar relatively homogeneous masses typical of adenoma or lymphocytic hypophysitis. They can be quite large. Pathology was lymphocytic hypophysitis.}
\end{figure}
sellar mass in some cases simulating a pituitary tumor. Intracellular coccidiodomycosis simulating a pituitary adenoma has been reported. Cerebral cysticercosis has been described as an intracellular or suprasellar cystic mass. Pneumocystis carinii infection involving the central nervous system and pituitary gland can occur.

Hemochromatosis can result in pituitary infiltration, most commonly affecting the gonadotropin-secreting cells owing to excess iron (hemosiderin and ferritin) deposition in the gland. The gland appears abnormally dark on T2-weighted images owing to the iron deposition despite no apparent abnormality on other imaging modalities.

**SPHENOID SINUS MUCOCELES**

Although the cause of sphenoid sinus mucoceles is not known, they may be initiated by an inflammatory occlusion of the ostium draining the sinus. Inflammatory conditions, tumors, trauma, and previous surgery may predispose to mucocele development. A cystic accumulation of secretions expands and erodes the sinus, eventually compressing surrounding structures such as the cavernous sinus, pituitary gland, cranial nerves I through VI, and the carotid arteries. There is no specific age preponderance.

Sphenoid mucoceles usually evolve over a long period, often years, with nonspecific headaches and the relatively late onset of visual loss. The headache is usually severe. Atypical facial pain with paresthesias secondary to trigeminal nerve irritation may be seen.

Visual loss owing to direct nerve compression by the mass or from scarring caused by an inflammatory reaction is usually slowly progressive but may be suddenly worsened by vascular compromise of the optic nerve. Optic neuropathy is most often unilateral but occasionally is bilateral, and bitemporal field deficits are typically lacking in contrast to patients with adenoma. Exophthalmos is present in about half of patients. Dysfunction of the third and, less often, the fourth cranial nerves leading to diplopia is common. Endocrine dysfunction is unusual, but patients may present with or without hormone deficiency or hyperprolactinemia.

On MR imaging, mucoceles have a variable appearance but most often show a high signal on T2-weighted images. Somewhat characteristic is a thin
regular rim of enhancement with contrast administration. Prominent opacification of the sphenoid sinus is present. Expansion of the sphenoid sinus, usually with intact walls, is found in most patients (Fig. 22).

HYPERPLASIA OF THE PITUITARY GLAND

Physiologic Enlargement of the Pituitary Gland

The normal increase in pituitary gland height seen during puberty may create confusion with an adenoma. In pubertal girls, the pituitary gland may project above the sella to a height of 10 mm and demonstrate a marked convexity and spherical appearance on coronal MR images. Although boys may also have some enlargement, typically, this is only to 7 to 8 mm, and the gland does not take on the spherical shape seen in girls. Physiologic enlargement of the gland may be seen during pregnancy, at which time it may increase in size and weight up to 100%. The pituitary gland may reach a height of 10 mm in pregnancy and has been reported to be as high as 12 mm with a convex upper border in the first postpartum week. After the first postpartum week, the gland rapidly returns to normal regardless of whether the woman is breast-feeding.

Pathologic Pituitary Hyperplasia

Primary hypothyroidism may be complicated by an enlarged pituitary gland, particularly in children. This should be clinically apparent and resolve within a few months of treatment of the hypothyroidism with thyroid hormone (Fig. 23). Pituitary hyperplasia has also been reported in central precocious puberty and in association with production of hypothalamic-releasing factors such as corticotropin-releasing hormone or growth hormone–releasing hormone by extrapituitary neoplasms.

Figure 22. Coronal (A) and sagittal (B) T1 MR images demonstrating a homogeneous mass filling the entire sphenoid sinus. On coronal image, the pituitary gland is suggested as a separate structure just above the sinus. Pathology was a mucocele.
DIFFERENTIAL DIAGNOSIS OF SELLAR MASSES

Figure 23. A and B, Coronal (A) and sagittal (B) T1-enhanced MR images in a 24-year-old woman with hypothyroidism. TSH level was greater than 1000 mlU/mL. Coronal (C) and sagittal (D) T1-enhanced MR images after 6 months of thyroid hormone replacement. The pituitary gland now appears completely normal.

References


116  FREDA & POST


parasellar tumors. In Tindall GT, Cooper PR, Barrow DL (eds): The Practice of Neurosurgery, vol I. Baltimore, Williams & Wilkins, 1996, p 1069


Address reprint requests to
Pamela Freda, MD
Department of Medicine
Columbia College of Physicians & Surgeons
630 W. 168th Street
New York, NY 10032