Nonadenomatous Tumors of the Pituitary and Sella Turcica

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Abstract: While pituitary adenomas make up over 90% of all sellar masses, there are a number of less known tumors, both malignant and benign, which may arise within the sella turcica. These include relatively common tumors such as meningiomas and craniopharyngiomas, as well as extremely rare tumors such as pituitary astrocytomas and granular cell tumors. Unfortunately, many of these tumors lack characteristic imaging features, often making it extremely difficult to distinguish them by imaging alone from the more common pituitary adenoma. In this article, we review several nonadenomatous tumors of the sella, with a focus on their clinical features and typical MR imaging characteristics.

Key Words: nonadenomatous tumors, pituitary

Although pituitary adenomas are the most common sellar tumors, accounting for more than 90% of sellar masses and 10% to 15% of all intracranial neoplasms, there are other tumors that arise in the sella turcica. These tumors arise from normal pituitary elements (craniopharyngiomas, pituitary carcinomas, astrocytomas, and granular cell tumors) may be of nonpituitary origin (meningiomas, germ cell tumors, and lymphoma) or may be metastases.

Many of these nonadenomatous tumors lack specific imaging characteristics and are often indistinguishable from adenomas by imaging alone. In many instances, a definitive diagnosis is made only postoperatively, and the final histological diagnosis is often unexpected. Despite this, there are imaging findings that may be helpful in suggesting that a sellar lesion may not be an adenoma. In this article, we review several nonadenomatous sellar tumors and their imaging characteristics (Table 1).

CRANIOPHARYNGIOMAS

Craniopharyngiomas are common, benign neoplasms, accounting for 2% to 5% of all intracranial tumors. They have a bimodal age distribution with a primary peak between 5 and 14 years and a second smaller peak somewhere between the fifth and seventh decades. Approximately two thirds occur in individuals younger than 20 years. Craniopharyngiomas account for 5.6% to 13% of intracranial tumors in children and are 10 to 20 times more common than adenomas in this population. The incidence does not vary by race or sex.

Craniopharyngiomas are derived from squamous cell rests in the remnant of Rathke pouch between the adenohypophysis and neurohypophysis and can occur anywhere along the path of the craniopharyngeal duct, from the nasopharynx to the third ventricle. Most craniopharyngiomas have a suprasellar component, with only 4% to 25% being purely intrasellar. Two subtypes of craniopharyngioma have been described: the adamantinomatous type, which is predominantly a tumor of children and adolescents but which can be seen at any age, and the papillary type, which is seen almost exclusively in adults.

Patients commonly present with nonendocrine symptoms, including headache, nausea, vomiting, and symptoms related to compression of the optic chiasm. Endocrine dysfunction is less common and may be manifested in children as growth disturbances. Up to 80% of children with craniopharyngiomas have endocrine dysfunction at diagnosis, with 75% demonstrating growth hormone deficiency. Patients may present with hypopituitarism, hyperprolactinemia, or diabetes insipidus (DI). Treatment usually consists of surgical resection with or without adjuvant radiotherapy, based on whether gross total resection is achieved. Recurrence is rare after gross total resection. With subtotal resection, only 47% of patients are recurrence-free at 5 years, and only 38% are recurrence-free at 10 years.

On magnetic resonance imaging (MRI), craniopharyngiomas can appear cystic, solid, or both. Cysts are seen in 85% of craniopharyngiomas. Predominantly solid tumors are 2 times more likely to be seen in adults than in children, whereas predominantly cystic tumors are seen in children roughly 50% of the time and less frequently in adults. Cysts contain variable amounts of cholesterol, keratin, protein, methemoglobin, and necrotic debris, which accounts for their variable appearance on MRI. Cystic components are typically hyperintense, and less commonly isointense, to cerebrospinal fluid (CSF) on T1-weighted images. Fluid-debris levels can be seen within the cysts. Solid components have variable signal intensities, and they usually enhance after gadolinium administration.

Calcification is typical of craniopharyngiomas and is present in approximately two thirds of all cases; tumor calcification is seen in approximately 90% of childhood craniopharyngiomas and in 50% to 70% of adult craniopharyngiomas. Calcification is rare in papillary-type craniopharyngiomas. Computed tomography is the preferred modality for detecting calcification.
Solid craniopharyngiomas can be difficult to distinguish from adenomas. However, the apparent diffusion coefficient of craniopharyngiomas is higher than that of adenomas on average 49 (Fig. 1).

### RATHKE CLEFT CYSTS

Rathke cleft cysts, like solid craniopharyngiomas, occur along the craniopharyngeal duct and are felt to arise when there is incomplete obliteration of the central embryonic cleft separating the anterior lobe of the pituitary from the pars intermedia. Rathke cleft cysts are differentiated from craniopharyngiomas by having single-layered walls of columnar or cuboidal epithelium, often containing ciliated and goblet cells. 2,15 They are common lesions and usually asymptomatic, reported incidentally in up to 33% of autopsy cases. Rathke cleft cysts are 2 to 3 times more common in females than in males. 2,16 They can be seen at any age but, when symptomatic, usually present between 40 and 60 years of age. 2

Symptoms are more likely to be present when the cysts are large enough to cause mass effect on adjacent structures and are similar to symptoms observed with other sellar and suprasellar masses. 16,36 Rathke cleft cysts range in size from a few millimeters to very large, in excess of 4.5 cm. 15-17 When symptomatic, cysts can be treated with partial removal or cyst aspiration, with a low rate of recurrence.

The primary imaging differential diagnosis is a cystic craniopharyngioma. As with craniopharyngiomas, signal intensities of the fluid in Rathke cleft cysts can be variable on MRI, reflecting the heterogeneous composition of the fluid, which ranges from serous to mucinous. 2,15-17 The cysts are usually of higher T1 signal intensity than CSF, with the majority being isointense to hyperintense to white matter, probably reflecting the proteinaceous nature of the cyst fluid. 16,17 On T2-weighted imaging, Rathke cleft cysts demonstrate variable signal intensity; with high protein concentrations, there may be low signal intensity on T2-weighted images. In contradistinction to the cysts of craniopharyngiomas, Rathke cleft cysts usually demonstrate thin and uniform walls, and enhancement of the walls is uncommon. 2,16,17 Enhancement of the walls of a Rathke cleft cyst may reflect inflammation or infection. Wall calcification is uncommon. 2

One group reported the presence of T1 hyperintense, T2 hypointense, nonenhancing intracystic nodules in 10 of 13 patients with pathologically confirmed Rathke cleft cysts, corresponding to waxy, solid, intracystic masses intraoperatively, probably representing concretions of desquamated cellular debris. 18 They suggest that these nodules can be distinguished from soft tissue nodules seen in craniopharyngiomas by their signal intensities and lack of enhancement (Fig. 2).

### PITUITARY CARCINOMA

Pituitary carcinomas are extremely rare tumors, with fewer than 150 cases reported. 19 They are tumors of the adenohypophysis that undergo discontinuous craniospinal or systemic spread. 19,20 In patients with pituitary carcinoma,
47% have only systemic metastases, 40% have craniospinal metastases, and 13% have both. Common sites of hematogenous spread are the liver and bone; less common reported sites include the lungs, lymph nodes, ovaries, heart, pancreas, and myometrium. Craniospinal metastases usually involve the cortex, cerebellum, and cerebellopontine angles.

The pathogenesis of pituitary carcinomas is not understood. It is likely that most develop secondary to transformation of an existing pituitary adenoma rather than develop de novo. Most pituitary carcinomas are initially diagnosed as invasive macroadenomas, and there is generally a latency period of 5 to 10 years before finding metastases. Histologically, there are no reliable criteria for distinguishing adenomas from pituitary carcinomas. A diagnosis of pituitary carcinoma cannot be made without evidence of distant spread.

There is no sex predilection, and pituitary carcinomas can be seen in adults of any age (mean, 44 years). Approximately 75% of pituitary carcinomas are endocrinologically active; most are prolactin or adrenocorticotropic hormone-producing tumors.

Clinical features of pituitary carcinoma are similar to those of invasive adenomas, with symptoms due to mass effect on surrounding structures or to the excessive hormone production. A common clinical presentation is early recurrence of tumor after resection, followed by a prolonged course of repeated surgeries for local recurrences, before metastatic disease. Once metastases are detected, the mean survival time is 4 years; survival varies with endocrinologic...
tumor subtypes, with adrenocorticotropic hormone–producing tumors having the worst prognosis. Patients with only central nervous system (CNS) involvement seem to have longer survival than those with systemic metastases.

From a neuroimaging standpoint, distant spread is the only feature which distinguishes pituitary carcinomas from adenomas. With regard to the primary sellar tumor, carcinomas cannot be differentiated from invasive macroadenomas. Like macroadenomas, pituitary carcinomas appear as enhancing sellar masses with suprasellar/parasellar extension and invasion of the cavernous sinus or bone. The metastases of pituitary carcinoma are indistinguishable from metastases of other carcinomas (Figs. 3 and 4).

**PITUITARY ASTROCYTOMAS**

Astrocytomas arising from the pituitary gland are rare and arise from the neurohypophysis. Astrocytomas of the pilocytic type have been reported. In addition, there have been several reports of low-grade neurohypophyseal glial tumors, histologically distinct from pilocytic astrocytomas, which have been referred to as pituicytomas. Some confusion in terminology has arisen as some authors have used the term pituicytoma to encompass all astrocytomas arising from the gland, whereas others consider the pituicytoma to be a distinct entity.

The neurohypophysis contains specialized glial cells referred to as "pituicytes." Five different types of pituicytes have been described, with most pituicytomas arising from major and dark cell types. Pituicytomas are differentiated from pilocytic astrocytomas by having plump, spindle-shaped cells with a slightly fibrillar cytoplasm and by lack of Rosenthal fibers, microcysts, and granular bodies found commonly in pilocytic astrocytomas.

Pituicytomas are seen from the third to ninth decades in patients with a mean age of 40 years. There is a male predominance. Pituicytomas may be entirely intrasellar or suprasellar or involve both compartments. Clinical presentation is similar to that of other sellar and suprasellar masses, with the most common presenting complaint being hypopituitarism, followed by visual disturbances. Despite the neurohypophyseal origin of these tumors, DI is not a common presenting symptom. Total resection is usually curative.

Pituicytomas and pituitary pilocytic astrocytomas are indistinguishable from one another radiographically and, in general, cannot be distinguished from other neurohypophyseal tumors. Diagnosis of pituitary astrocytoma can only be made pathologically. For the sake of simplicity, we refer to pituitary tumors of glial origin as pituitary astrocytomas.

MRI features of pituitary astrocytomas are nonspecific and are those of a solid, circumscribed, enhancing sellar or suprasellar mass, usually isointense to gray matter on T1-weighted and hyperintense on T2-weighted sequences. Anterior displacement of the normally enhancing adenohypophysis by the tumor may suggest that it is of neurohypophyseal origin. A pituitary astrocytoma containing solid and cystic components has been reported, but the presence of cysts is an uncommon feature.

**GRANULAR CELL TUMORS**

Granular cell tumors are rare and benign tumors of the neurohypophysis and have also been referred to as choristomas, myoblastomas, and infundibulomas. They arise from granular cell–type pituicytes, but structurally identical...
tumors have been described elsewhere in the CNS. They are the most common primary tumor of the neurohypophysis and are seen in up to 17% of nonselected adult autopsies. The tumors are twice as common in females than in males. Granular cell tumors are usually asymptomatic but, when symptomatic, usually present in the fifth decade. As with other nonfunctional pituitary tumors, symptoms are primarily related to size and mass effect. Tumors are usually very large when discovered. Approximately 90% of symptomatic patients have visual complaints, and 50% have clinical or laboratory signs of hypopituitarism or hyperprolactinemia. Headache is a common complaint. Treatment for symptomatic granular cell tumors is surgical; postoperative radiation is controversial but appears to increase mean survival time and time to recurrence after surgery.

The imaging appearance of granular cell tumors is nonspecific, with an enhancing sellar or suprasellar mass seen on MRI. Eleven percent of symptomatic granular cell tumors are intrasellar, 42% are suprasellar, and 47% involve both compartments. The masses are isointense to gray matter on both T1- and T2-weighted sequences. Intense enhancement may be seen and reflects the high vascularity of these tumors. Calcifications may be present. Absence of the normal pituitary bright spot may be a clue that the tumor is of neurohypophyseal origin, but this finding is nonspecific, as the posterior pituitary bright spot may be absent in 10% to 20% of normal subjects (Fig. 5).

**GANGLIOCYTOMAS**

Although the pituitary gland does not contain neurons, a small number of reports of pituitary tumors composed at least partly of ganglion cells can be found in the literature. These tumors are referred to as gangliocytomas. The exact histogenesis of these tumors is debated, with some authors suggesting that they arise from embryonal pituitary cell rests that have features between neurons and adenohypophyseal cells and others suggesting a common hypothalamic origin. The tumors are found in adults and are more common in females. Although these tumors may consist exclusively of ganglion cells, the majority (65%–76%) of gangliocytomas are found in association with adenomas. Because of this, some suggest the name, mixed gangliocytoma-adenoma (MGA). Approximately 75% of patients with pituitary gangliocytomas demonstrate pituitary hormone hypersecretion, with oversecretion of growth hormone being the most common manifestation, followed by Cushing disease. Interestingly, the MGAs are more likely to be hormonally active than the pure gangliocytomas. In addition to the endocrine abnormalities seen with MGAs, visual changes and headaches can also occur.

On MRI, intrasellar gangliocytomas and MGAs may not be distinguishable from pituitary macroadenomas and appear as an enhancing sellar and suprasellar mass. Some have noted that the suprasellar portion of an MGA tends to be more spherical than macroadenomas and that they do not demonstrate the waist at the level of the diaphragma sella that macroadenomas do (Fig. 6).

**MENINGIOMAS**

Meningiomas can originate from any dural surface, including the tuberculum sella, olfactory groove, sphenoid wing, diaphragma sella, and sella turcica. They account for approximately 20% of all intracranial neoplasms; are tumors of adults, increasing in incidence with increasing age (peak incidence, 60–70 years); and are 2 times more common in females. Whereas 10% to 15% of meningiomas arise in the parasellar region, purely intrasellar meningiomas are rare. The majority of intrasellar meningiomas arise from the undersurface of the diaphragma sella, but meningiomas originating from the floor or walls of the sella have been reported.

FIGURE 5. Granular cell tumor. Unenhanced (A) and enhanced (B) coronal T1-weighted MR images through the pituitary demonstrate a well-circumscribed, enhancing nodule arising from the pituitary stalk.
Meningiomas are benign, slow-growing tumors. Intrasellar meningiomas may mimic nonfunctioning adenomas in their clinical presentation, with primary symptoms being headache, visual disturbances, visual field defects, and endocrinologic abnormalities (hypopituitarism or hyperprolactinemia).\textsuperscript{44-46} Cases of an intrasellar meningioma mimicking pituitary apoplexy have been reported.\textsuperscript{46,47} Although benign, meningiomas can be locally aggressive and recur after incomplete resection. Encasement and, ultimately, occlusion of an internal carotid artery can occur.\textsuperscript{2}

Preoperative diagnosis of an intrasellar meningioma is extremely useful in surgical planning and may sway surgeons away from the transsphenoidal approach used for most adenomas in favor of a transcranial approach.\textsuperscript{47-49} The high degree of vascularity of most meningiomas predisposes to excessive intraoperative bleeding during resection, which is generally more easily controlled from a transcranial approach. Because of the vascularity of meningiomas, preoperative embolization of tumors is advocated by some as being helpful in reducing intraoperative blood loss.\textsuperscript{44} Diaphragma sella meningiomas are typically supplied by arteries of the ophthalmic segment of the internal carotid arteries.\textsuperscript{45}

Purely intrasellar meningiomas can be extremely difficult to distinguish from adenomas by imaging.\textsuperscript{45,48} Intrasellar meningiomas typically appear as masses which are hypointense to isointense to gray matter on both T1- and T2-weighted sequences.\textsuperscript{2,7} Sellar enlargement is common.\textsuperscript{44,45,48} Enhancement after administration of gadolinium is marked and homogeneous, with homogeneous enhancement seen in more than 90%.\textsuperscript{7,50} The rate of enhancement is usually rapid.\textsuperscript{51} The enhancement profile of meningiomas may aid in distinguishing them from adenomas, as adenomas generally enhance less intensely and more heterogeneously than meningiomas and demonstrate a longer time-to-peak enhancement on dynamic imaging.\textsuperscript{7,50,51} The presence of an enhancing dural tail is not a feature of intrasellar meningiomas.\textsuperscript{45,52}

Identification of the pituitary gland as separate from a sellar mass is probably the single most useful finding in differentiating adenomas from sellar meningiomas. Visualization of a CSF cleft between a tumor and the gland, although uncommon, virtually excludes the diagnosis of adenoma.\textsuperscript{7,53} Another finding which may favor meningioma is hyperostosis of the floor of the sella or adjacent bony structures, seen in 34% of sellar meningiomas.\textsuperscript{7,51} Prominent vessels may be seen in approximately 65% of sellar meningiomas.\textsuperscript{7} Macroscopically detectable calcifications are uncommon and are seen in just over 10% of sellar meningiomas\textsuperscript{7,44} (Fig. 7).

**GERM CELL TUMORS**

Intracranial germ cell tumors are rare malignant tumors, representing only 0.1% to 2% of all primary brain neoplasms. They are believed to arise from totipotent germ cells that fail to migrate to the genital crest during embryonic life. Primarily midline tumors, germ cell tumors are subdivided into teratomas, germinomas, embryonal cell carcinoma, choriocarcinoma, endodermal sinus (yolk sac) tumors, and mixed germ cell tumors. Pure germinomas

![Image A](https://example.com/imageA.png)

![Image B](https://example.com/imageB.png)

**FIGURE 6.** Gangliocytoma. Coronal T2-weighted (A) and enhanced sagittal T1-weighted (B) MR images demonstrate a heterogeneous, enhancing sellar and suprasellar mass. Note the spherical configuration of the mass and the absence of a “waist” at the level of the diaphragma sella.

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MRI

A development of DI is due to the increase in number of immunocompromised patients. The majority of intracranial germ cell tumors are found in the pineal or suprasellar regions. Intracranial germ cell tumors occur twice as often in males than females, but this male predominance is limited to tumors arising in the pineal region. Suprasellar germ cell tumors occur slightly more frequently in females. Pure germinomas are radiosensitive and carry a favorable prognosis. More than 90% of germinomas can be treated effectively with radiation. Prognosis for patients with nongerminomatous tumors is less favorable, with only 40% to 60% demonstrating disease control with radiation therapy alone.

Primarily intrasellar germinomas are rare and have a female predominance. Most suprasellar germinomas originate either in the floor of the third ventricle or in the infundibulum. It is felt that intrasellar germinomas represent infundibular tumors with primarily intrasellar growth. Patients most commonly present with endocrine abnormalities, with DI being the most common symptom to prompt patients to seek medical attention. Development of DI is related to invasion or compression of the posterior lobe or infundibulum and may persist for years before a diagnosis is made. Other manifestations include hypopituitarism in children and adolescents and hypogonadism in adults. Hyperprolactinemia and precocious puberty have been observed. Large tumors and primarily suprasellar tumors may present with visual changes or oculomotor palsies.

Serum tumor markers can be extremely useful in establishing the diagnosis of a germ cell tumor. Production of either α-fetoprotein (AFP) or β-human chorionic gonadotropin (hCG) can be seen in a number of subtypes of germ cell tumors. Tumors with a yolk sac component secrete AFP. Choriocarcinomas produce high levels of hCG. Patients with pure germinomas can secrete hCG into the CSF at low levels, and this is considered an early indication of CSF dissemination. Because other intracranial tumors do not produce AFP or hCG, the presence of these markers in CSF are usually associated with intracranial tumors. Serum in the setting of an intracranial tumor is essentially diagnostic of a germ cell tumor.

Because intracranial germ cell tumors have a predilection to disseminate via CSF, MRI of the entire craniospinal axis and lumbar puncture for cytology are mandatory. MRI cannot reliably differentiate the different types of germ cell tumors. The earliest finding in cases of neurohypophyseal germ cell tumors is probably absence of the normal posterior lobe bright spot on T1-weighted images. This can be followed by swelling of the stalk and subsequent mass formation, which may displace the enhancing pituitary gland anteriorly. Loss of the normal pituitary bright spot and thickening of the stalk are also seen with idiopathic DI, granulomatous processes, and lymphocytic hypophysitis. Because of this, some have proposed serial MRI and endocrinologic evaluations in patients presenting with DI and loss of the posterior bright spot without a definite mass.

Larger sellar/suprasellar germ cell tumors have a nonspecific imaging appearance and usually cannot be distinguished from other tumors. The MR findings are those of an enhancing solid sellar mass, with or without suprasellar extension. Cysts and calcifications may be seen. Development of DI is related to invasion or compression of the posterior lobe or infundibulum and may persist for years before a diagnosis is made. Other manifestations include hypopituitarism in children and adolescents and hypogonadism in adults. Hyperprolactinemia and precocious puberty have been observed. Large tumors and primarily suprasellar tumors may present with visual changes or oculomotor palsies.

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Primary lymphoma arising in the sella is exceedingly rare, with only a handful of cases reported. Patients range
in age from 44 to 86 years. Presenting symptoms are not specific and include headaches, hypopituitarism, visual field deficits, and oculomotor palsies.

The imaging appearances of primary CNS lymphoma tend to differ based on whether patients have normal or suppressed immunity. CNS lymphoma in patients with normal immune systems usually appears as a solitary mass with intermediate to low signal intensity relative to gray matter on both T1- and T2-weighted sequences; enhancement with gadolinium is seen in nearly all cases and is homogenous in approximately three quarters of cases. The relative lack of T2 prolongation in CNS lymphoma is generally attributed to dense cellularity and to a high nuclear-cytoplasmic ratio and may be helpful in distinguishing lymphoma from other CNS tumors; unfortunately, this finding is relatively insensitive, as it is seen in just over 50% of cases. Calcification and hemorrhage in CNS lymphoma are rare.

In immunocompromised patients, lesions of CNS lymphoma are likely to be multiple and to demonstrate necrosis. In the setting of necrosis, the center of the lesion is T2 hyperintense, whereas the periphery remains of intermediate to low signal. Contrast enhancement is heterogeneous in this population, and rim enhancement is more common.

MRI characteristics of sellar lymphomas are largely nonspecific, with most cases appearing as homogeneously or heterogeneously enhancing sellar masses. Isointensity to hypointensity of masses relative to gray matter on T2-weighted imaging has been reported in sellar lymphoma and may be helpful in distinguishing it from pituitary adenoma.

**METASTASES**

Metastases to the pituitary gland and sella are uncommon. In surgical series examining patients undergoing transsphenoidal surgery for sellar or parasellar tumors, metastases are found in less than 1% of cases. In autopsy series, evidence of metastases to the pituitary is seen in approximately 5% of patients with known malignancy. In approximately two thirds of these cases, the pituitary is macroscopically normal. The most common primary malignancies to metastasize to the pituitary gland are breast and lung cancers which together account for more than 60% of all pituitary metastases. In 1 review, metastases to the pituitary were found in 17.6% of autopsies of patients with breast cancer. Although breast and lung cancers are the most common primaries to metastasize to the pituitary, metastases from tumors of nearly every tissue type have been reported.

Metastases can reach the sella turcica via hematogenous spread, spread from a hypothalamic or hypophyseal metastasis through portal vessels, direct extension from skull base tumors, or meningeal spread. Hematogenous metastases to the pituitary have a predilection to involve the posterior lobe. In 1 review of 201 cases of pituitary metastases, the posterior lobe alone was involved in 50.8% of cases, both lobes were involved in approximately 33.8% of cases, and the anterior lobe was involved in 15.4% of cases. The relative infrequency of metastases exclusive to the anterior lobe has been attributed to its lack of a direct arterial blood supply. It is hypothesized that the first capillary bed of the portal system may trap metastases before they can travel to the anterior lobe. Therefore, metastatic involvement of the adenohypophysis is more often due to contiguous...
spread from the posterior lobe than to direct hematogenous spread.78

Probably because of the predilection for metastases to involve the posterior lobe, the most common symptom observed in patients with pituitary metastases is DI, which occurs in 45% of cases.63 Other signs/symptoms include visual field deficits, anterior pituitary insufficiency, oculomotor palsies, and headache.63,76

Imaging features of pituitary metastases are variable, and in the absence of coexistent brain metastases, differentiation from an adenoma is difficult. On MRI, pituitary metastases appear as sellar or suprasellar masses which often are isointense or hypointense to gray matter on T1-weighted images and are usually of increased T2 signal. They enhance after gadolinium administration. Enhancement may be homogeneous or heterogeneous, and rim enhancement can also be seen.2,63 Like macroadenomas, metastases can appear as dumbbell-shaped intrasellar and suprasellar tumors. Invasion of the cavernous sinus and underlying sphenoid sinus may be seen.63 Loss of the normal posterior bright spot

FIGURE 9. Metastasis. Coronal T2-weighted (A) and unenhanced (B) and enhanced (C) coronal T1-weighted images through the sella demonstrate a predominantly rim enhancing sellar mass with suprasellar extension. Central areas of T1 hyperintensity in B and T2 hypointensity in A probably reflect hemorrhage. The mass displaces the optic chiasm superiorly. This was a metastasis from breast cancer.

FIGURE 10. Melanoma metastasis. Unenhanced sagittal T1-weighted (A) and coronal T2-weighted (B) MR images demonstrate a sellar mass which is markedly hyperintense on the T1-weighted image and hypointense on the T2-weighted image relative to adjacent brain parenchyma. The signal characteristics presumably are due to the presence of melanin.
has been reported. Rapid growth of tumor, infundibular enlargement, and apparent destruction rather than remodeling of the bone of the sella turcica have also been reported and may favor metastasis over adenoma.79,80 (Figs. 9 and 10).

CONCLUSION

Differentiating pituitary adenomas from any of the above entities can be extremely difficult and, at times, even impossible. In some patients, however, there are specific imaging findings which, when present and when correlated with appropriate clinical information, can lead the radiologist to make the diagnosis or at least to suggest the actual diagnosis.

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