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Neuroendocrine Imaging

Pathology, Clinical Algorithms, Imaging Appropriateness, and Management of Incidental Findings

Judah Burns, MD*, Kevin Hsu, MD, Keivan Shifteh, MD, Amichai J. Erdfarb, MD
Department of Radiology, Montefiore Medical Center, 111 East 210 Street, Bronx, NY 10467, USA

KEYWORDS
• Pituitary • Neuroendocrine • Microadenoma • Macroadenoma • Rathke cleft cyst • Incidental finding

KEY POINTS

- Lesions of the sella turcica and suprasellar cistern may present clinically with headache, visual changes, and hormonal abnormalities.
- In most cases, MRI is the preferred imaging modality to evaluate the pituitary gland and parasellar regions.
- Incidental findings of the sella are common. Follow-up recommendations should relate to the likelihood of solid malignancy or lesion growth as well as the risk for intralesion hemorrhage.
- The differential diagnosis of suprasellar lesions includes primary and secondary lesions, which may be either benign or malignant.

INTRODUCTION

The spectrum of neuroendocrine disorders includes abnormalities of both hypersecretion and hyposecretion of pituitary hormones of central cause. These clinical syndromes may be caused by intrinsic masses of the pituitary gland, suprasellar cistern, and other disease processes, which affect the hypothalamic-pituitary axis. This article reviews common and uncommon lesions of the sella and parasellar regions, with a focus on imaging appropriateness in the workup of various clinical syndromes and follow-up of incidental findings.

NORMAL ANATOMY AND IMAGING TECHNIQUE

The sella turcica is located centrally in the skull base, a semicircular depression in the clivus, within the body of the sphenoid bone [1]. The tuberculum sella forms the anterior wall, and the dorsum sella forms the posterior wall. Variations of sphenoid sinus pneumatization affect the size and morphology of the clival interface as well as the appearance of the sphenoid bones, and are important to recognize when planning for transspheniodal pituitary surgery [2,3] (Fig. 1).

The pituitary gland borders the medial dural wall of the cavernous sinuses, which lie just lateral to the sella turcica [4]. Morphologic variations in pituitary anatomy include lateral extension of the gland, which can mimic cavernous sinus invasion by an adenoma; strict criteria-based analysis is needed to determine cavernous sinus invasion [5]. Conversely, medial bowing of the cavernous carotid artery segments may be due to a weak dural margin and can narrow normal pituitary morphology, complicating evaluation for small pituitary adenomas [6] (Fig. 2).

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*Corresponding author, *E-mail address:* jburns@montefiore.org

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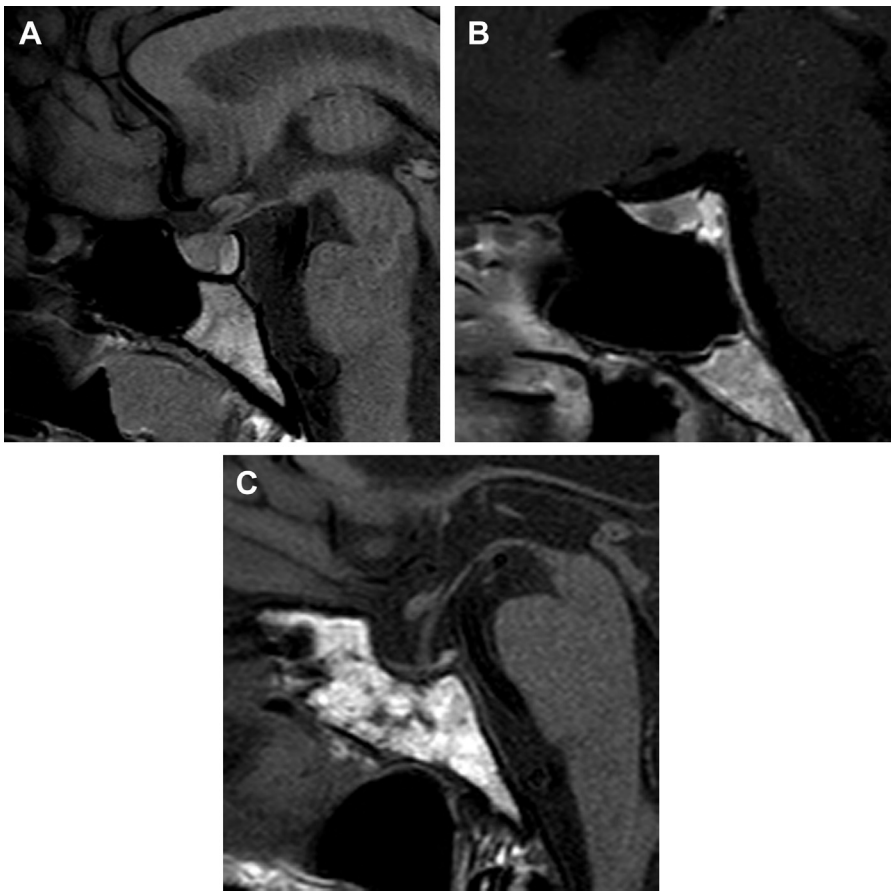

FIG. 1 Normal pituitary gland with variations of sphenoid pneumatization. (**A**) Sagittal T1WI MRI demonstrates a normal size sella within the upper clivus, with normal anterior gland and hyperintense posterior pituitary lobes. The sphenoid sinus is inferior and anterior to the sella. (**B**) An expanded, hyperpneumatized sphenoid sinus. (**C**) Arrested pneumatization of the sphenoid sinuses.

The anterior pituitary gland is made up of many tissue types, including the adenohypophysis (anterior gland) and neurohypophysis (posterior gland) [7,8]. The anterior gland derives from Rathke pouch and typically has a concave upper surface [9]. The gland enlarges after puberty but rarely exceeds 10 mm in height, and is larger in women, particularly during pregnancy and the peripartum period [10,11]. The adenohypophysis secretes numerous hormones [12], including the following:

- Prolactin
- Growth hormone (GH)
- Thyroid-stimulating hormone (TSH)
- Follicle-stimulating and leutinizing hormone
- Corticoctropin (ACTH) precursor hormones

The posterior gland derives from neuroectoderm along the base of the brain, stores oxytocin and antidiuretic hormone, and is typically of high signal intensity on T1-weighted (T1WI) MRI (Fig. 3). The pituitary stalk, or infundibulum, extends from the inferior projection of the hypothalamus (the tuber cinereum, along the floor of the third ventricle) and has a tubular shape, tapering as it reaches the pituitary surface. The diaphragm sella is a dural sheet that separates the contents of the sella from the suprasellar cistern, which contains optic chiasm and the circle of Willis.

Dedicated pituitary imaging is performed using high-resolution MRI, including small field of view (FOV) and 2.5-mm slice thickness (Table 1). Sellar

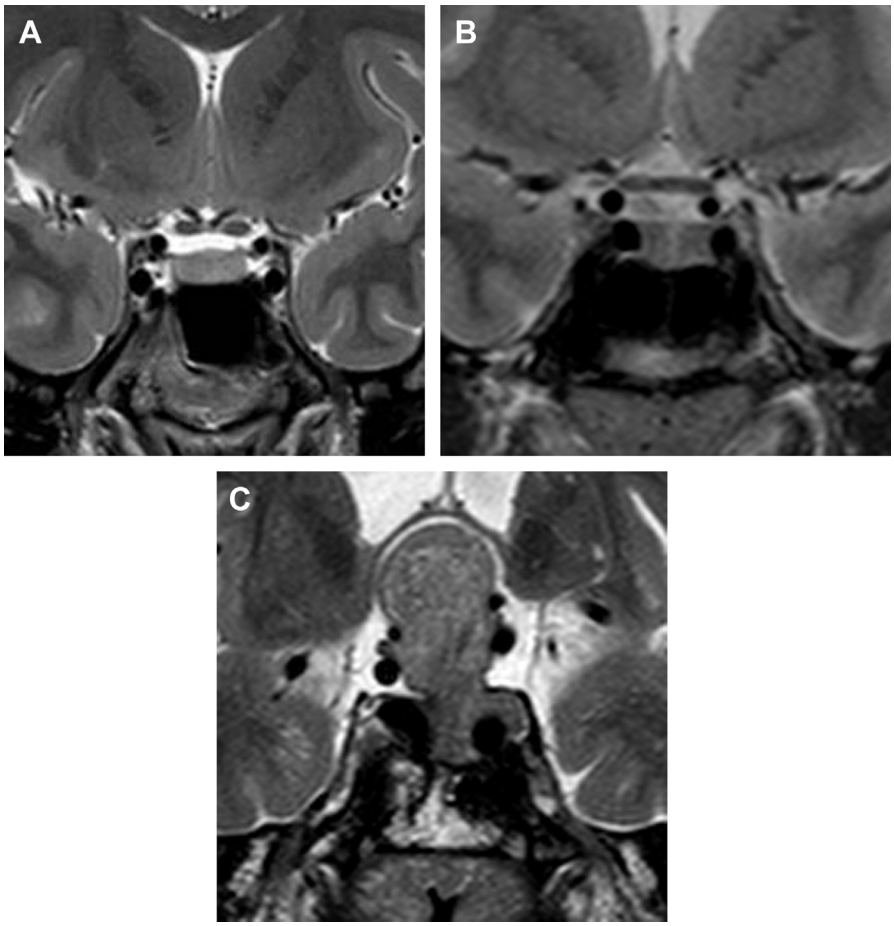

FIG. 2 Normal sella and suprasellar cistern with variations of cavernous carotid artery. (**A**) Normal. (**B**) The left cavernous carotid bows slightly medially. (**C**) Large macroadenoma with suprasellar extension. There is medial position of cavernous carotids within the sella, with encasement by tumor. The vessels are at risk during transsphenoidal decompression if not recognized preoperatively.

morphology is described, and lesions are classified as originating within either the sella or the suprasellar/parasellar regions. T2-weighted (T2WI) and contrast-enhanced sequences help classify lesions as solid, cystic, or mixed-type lesions. The presence of cysts, blood products, enhancement, and invasion may help characterize lesions. Anterior pituitary lobe enhancement is slightly delayed because of indirect portal vascular supply, in contrast to the direct arterial supply to the posterior pituitary lobe. Rapidly acquired dynamic T1WI imaging obtained immediately following intravenous contrast administration is used to help identify small anterior pituitary adenomas [13] (Fig. 4).

Computed tomography (CT) is not routinely used for pituitary assessment, but can help characterize some masses. CT is useful for evaluation of the sella tucica and sphenoid sinus bony anatomy and anatomic variations [14]. CT is often used to obtain stereotactic guidance for presurgical planning [15]. Dual-energy CT has been used to differentiate between pituitary adenoma and suprasellar meningioma [16]. PET-CT may reveal hypermetabolic activity in the sella or parasellar region, often as an incidental finding, but is not commonly used to characterize known lesions.

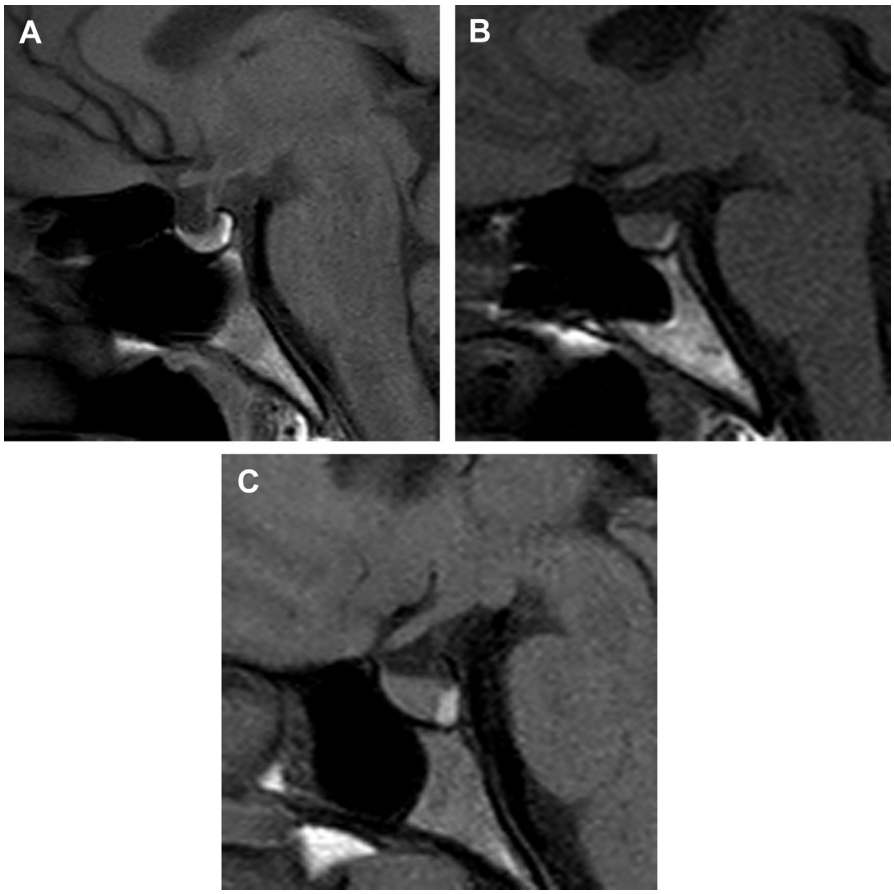

FIG. 3 (**A–C**) Varying degrees of normal posterior pituitary lobe. The posterior pituitary may be thin or globular, located both high and low in position along the dorsum sella.

CLINICAL SYNDROMES

Syndromes of pituitary dysfunction may result from intrinsic pituitary abnormality related to congenital or developmental abnormalities or masses [17]. Extrinsic mass effect may also cause abnormal regulation of hormone release and can be caused by primary sellar/suprasellar lesions or lesion of remote origin. Small secreting pituitary adenomas may inhibit further hormonal release via an inhibitory feedback cycle. Intrinsic pituitary dysfunction may be present without imaging abnormalities. Syndromes of pituitary hypofunction include panhypopituitarism, GH deficiency, growth deceleration, thyroid dysfunction, and hypogonadotropic hypogonadism.

Secondary causes of decreased pituitary secretion include extrinsic compression of either the gland or the pituitary stalk by solid or cystic lesions. Examples include craniopharyngioma, Rathke cleft cyst, suprasellar meningioma, or germ cell tumor. Inflammatory lesions (ie, sarcoidosis, tuberculosis, Langerhans cell histiocytosis [LCH], lymphocytic hypophysitis [LH]) and cerebrospinal fluid (CSF) -disseminated or metastatic tumors may also inhibit normal pituitary hormone release. In particular, because these lesions may affect the pituitary stalk and hypothalamic-pituitary axis, they may also be a cause of centrally mediated diabetes insipidus.

Pituitary hyperfunction typically results from a hormone-secreting adenoma. In children, precocious puberty may be caused by tumor, infection, or hydrocephalus, in addition to gonadotropin-releasing hormone production from tumor or extrinsic disease. In adults, clinical syndromes include hyperthyroidism

TABLE 1
Pituitary Imaging Protocol

MRI Sequence	Imaging Assessment
Routine brain C- (ie, DWI, 3D FLAIR, 3D spoiled gradient echo)	Routine brain evaluation
Sagittal T1WI C-	Sellar morphology Sphenoid pneumatization Posterior pituitary bright spot Suprasellar cistern
Coronal T2WI C-	Sellar morphology Cavernous sinuses Suprasellar cistern Optic chiasm Cyst formation
Axial T2WI or GRE C- (optional)	Intrapituitary hemorrhage Cyst formation Cavernous sinuses
Coronal dynamic C + T1WI	Pituitary enhancement Microadenoma evaluation
Coronal/sagittal C + T1WI	Pituitary enhancement Sellar/suprasellar masses Cavernous sinus enhancement

High-resolution, 3-mm slices, small FOV.

(TSH), Cushing syndrome (ACTH), amenorrhea or galactorrhea (prolactin), and acromegaly or gigantism (GH). Hormone-secreting tumors are typically smaller microadenomas (measuring <10 mm). Larger macroadenomas (>10 mm) are often nonsecreting and cause symptoms primarily due to mass effect, such as chiasmatic compression. As such, they may be initially asymptomatic and present as large lesions [18].

IMAGING APPROPRIATENESS

The American College of Radiology (ACR) Appropriateness Criteria help to define typical clinical scenarios and describe the appropriateness of various diagnostic tests [19]. In almost all clinical scenarios, MRI of the sella (noncontrast or with and without contrast) is described as the most appropriate imaging examination, giving the ability to achieve high tissue contrast with small FOV imaging. It is controversial whether precocious puberty is a clinical indication that should spur neuroimaging to evaluate the pituitary and parasellar regions [20–24].

CT scanning (with or without contrast) may be appropriate in most clinical scenarios but is not recommended as a first-line test. It can help characterize some suprasellar masses (ie, calcification, hemorrhage)

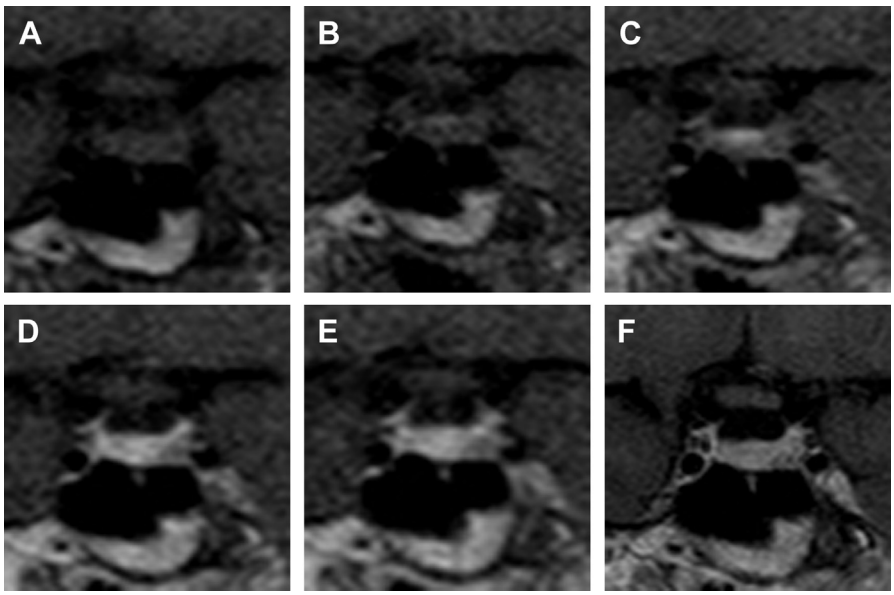

FIG. 4 Dynamic contrast enhancement. (**A**–**E**) Serial small FOV coronal C + T1WI pituitary images, showing progressive pituitary enhancement. A small adenoma is present in the left aspect of the pituitary. (**F**) Delayed coronal C + T1WI image shows the adenoma.

and is useful to assess for bone invasion in aggressive tumors. In cases where transsphenoidal surgical approach is warranted, CT is especially helpful to characterize developmental variants that increase the surgical risk and is often used for stereotactic operating room guidance.

Petrosal sinus venous sampling may be used to localize hormonal hypersecretion as a problem-solving tool in complicated cases. Vascular imaging (ie, CT angiography, MR angiography) is rarely indicated as a first-line test, but may be useful in complicated or postoperative cases.

IMAGING FINDINGS

Classical differential diagnosis distinguishes between intrasellar and suprasellar lesions, recognizing that there is often overlap between these categories (Tables 2 and 3). The authors discuss common and uncommon primary lesions of the sella and suprasellar region, with focus on their specific imaging characteristics. Nonprimary cystic and solid lesions of the sella and suprasellar are also included and are summarized in Tables 4 and 5.

Ectopic or Absent Posterior Pituitary

Congenital anomalies of the pituitary and posterior pituitary may be due to abnormal neuronal migration, often mediated by genetic factors [25], and should spur evaluation for other midline structural anomalies of the brain and spine. Short stature and central diabetes insipidus are the most commonly associated clinical conditions. The pituitary infundibulum may be small or absent, and the posterior pituitary bright spot may be completely absent in both symptomatic and asymptomatic individuals. An ectopic T1WI bright spot can be found along the stalk or at the level of the tuber cinereum [26] (Fig. 5).

TABLE 2
Lesions of the Sella Turcica

Common intrasellar lesions	Uncommon intrasellar lesions
Pituitary adenoma	Craniopharyngioma
Empty sella	Pituitary abscess
Pars intermedia cyst	Arachnoid cyst
Rathke cleft cyst	Granulomatous disease
Pituitary hemorrhage	Metastasis

TABLE 3
Lesions of the suprasellar cistern

Common suprasellar lesions	Uncommon suprasellar lesions
Pituitary macroadenoma (sella + suprasellar)	Germinoma
Rathke cleft cyst	Epidermoid tumor
Craniopharyngioma	Tuberculosis (TB)/sarcoidosis
Meningioma	Metastasis/lymphoma/LCH
Arachnoid cyst	Lipoma
Aneurysm	Hamartoma
Hypothalamic/ chiasmatic glioma	

TABLE 4
Imaging Features of Sellar/Suprasellar Cysts

Sella/Suprasellar Cyst	Characteristic Imaging Features
Rathke cleft cyst	Intrasellar/suprasellar T2WI hyperintensity Variable T1WI Nonenhancing Noncalcified
Pars intermedia cyst	Intrasellar Variable T2WI Nonenhancing
Craniopharyngioma	Suprasellar/intrasellar Mixed solid-cystic lesion Enhancing nodule or cyst walls Calcifications common
Empty sella	Sellar expansion Compressed pituitary gland Stalk insert along floor of sella Isointense to CSF on all sequences
Arachnoid cyst	Suprasellar/intrasellar Isointense to CSF on all sequences Noncalcified No DWI restriction
Epidermoid cyst	Suprasellar Low T1WI/high T2WI DWI restriction (characteristic)

TABLE 5
Other Suprasellar Lesions of Nonpituitary Origin

Suprasellar Lesions	Characteristic Imaging Features
Craniopharyngioma	Suprasellar/intrasellar Mixed solid-cystic lesion Enhancing nodule or cyst walls Calcifications common
Metastasis	Solid or heterogeneous enhancement Nodular leptomeningeal enhancement Stalk enhancement
Granulomatous disease (ie, sarcoid, LCH, TB)	Nodular basilar meningeal enhancement Stalk enhancement
Meningioma	Follows gray matter on T1WI/T2WI Solid enhancement Dural tail
Suprasellar germinoma	Hypodense on CT Intense enhancement Leptomeningeal dissemination ± Calcification
Chiasmatic glioma	Extends along optic tracts Occasional cysts Enhancement of high-grade lesions
Clival chondrosarcoma/ chordoma	Calcifications/bony matrix Bone destruction Central skull base location

The posterior pituitary enhances avidly; a small, ectopic or "dim" posterior pituitary, which is not recognized on standard T1WI images, may be identifiable only on contrast-enhanced sequences [27].

Pituitary Microadenoma

Microadenomas are small intrapituitary lesions that can present clinically due to hormonal hypersecretion or as incidental findings. Prolactin and GH are the most commonly secreted hormones [28] and are commonly treated medically rather than surgically; close to 20% of microadenomas are nonsecreting [29]. These lesions are increasingly recognized and may compromise greater than 15% of intracranial tumors. Because they are small, they may be confused with small intrapituitary cysts (ie, pars intermedia, Rathke cleft cysts).

Pituitary microadenomas are best seen on contrast-enhanced sequences. In most cases, microadenomas enhance more slowly than normal pituitary tissue and can be identified as well-defined hypoenhancing lesions (Fig. 6). Occasionally, the pituitary gland may enhance heterogeneously and simulate a small adenoma. Some lesions may be isoenhancing and not seen without dynamic contrast-enhanced scans [30,31]. Serial thin-section coronal T1WIs are acquired through the pituitary gland every 15 seconds following contrast administration. CT is rarely useful in the detection and characterization of microadenomas.

Pituitary Macroadenoma

Macroadenomas are pathologically similar to microadenomas, yet larger (>10 mm) [32]. They are genetically associated with genetic abnormalities of the *MEN1* tumor suppressor gene. These tumors are generally benign but can have aggressive growth features and may present due to hormonal secretion or mass effect. These tumors have a capsule of compressed pituitary tissue and most commonly bulge upward into the suprasellar cistern, causing mass effect on the optic chiasm. Rarely, pituitary adenomas can arise from "rest cells" and present as primary masses of the clivus.

On imaging, the normal gland is often obscured. The pituitary stalk should be identified on contrast-enhanced MRI sequences as deviated when planning surgical decompression. A "snowman" (figure-of-8) configuration is classic, although the larger lesions may have lobular and irregular margins. CT is used to delineate bony anatomy, such as remodeling of the sella or other bony erosion [33]. MRI features of macroadenomas include the following:

- Variable signal intensity, although typically moderately hyperintense on T2WI
- Intralesion hemorrhage (fluid levels and T1WI signal hyperintensity) [34]
- Heterogeneous enhancement
- Necrosis and cyst formation (Fig. 7)

Cavernous sinus invasion may be identified, although the specificity of this imaging finding must be carefully measured. Criteria with high specificity include cavernous carotid encasement, lateral extension beyond the lateral intercarotid line, and involvement of the inferolateral sulcus [35]. Skull base invasion may also be present, mimicking a more aggressive mass.

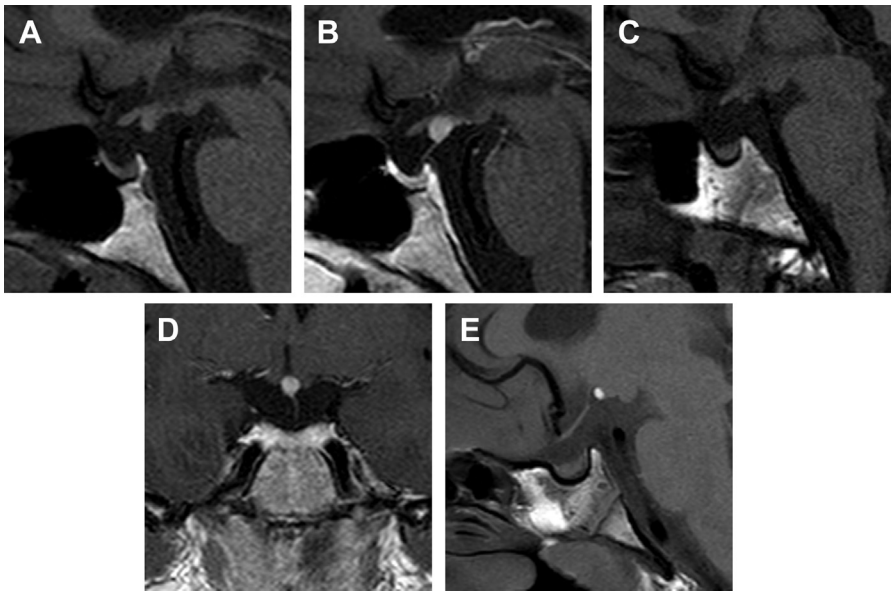

FIG. 5 Ectopic posterior gland; 3 examples on precontrast and postcontrast T1WI. (**A**, **B**) A 57-year-old man with hypogonadotropic hypogonadism; globular posterior pituitary located ectopically at the base of the pituitary stalk. (**C**, **D**) A 19-year-old man with hypopituitarism; posterior pituitary is "dim" precontrast, seen best with contrast at the base of the hypothalamus. (**E**) A 5-year-old girl with panhypoituitarism; a small posterior pituitary "bright spot" is located ectopically along the tuber cinereum. Note the underpneumatized sphenoid bone and developmentally small sella turcica.

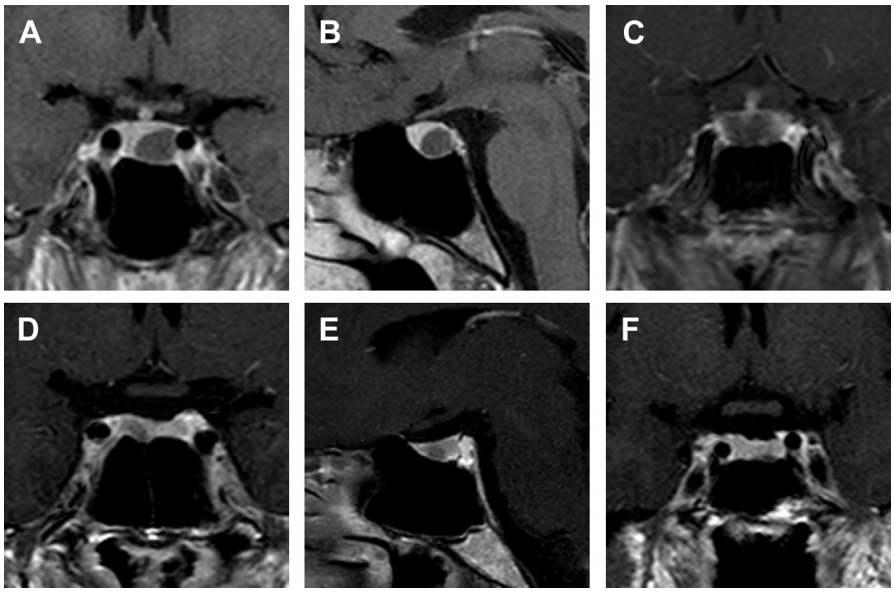

FIG. 6 Pituitary microadenoma; 4 examples on coronal and sagittal C + T1WI. (**A**, **B**) A 19-year-old woman with hyperprolactinemia; well-defined, rounded hypoenhancing intrapituitary lesion. (**C**) A 13-year-old man with pituitary gigantism; central microadenoma. (**D**, **E**) A 54-year-old man with hyperprolactinemia; small lateral well-defined hypoenhancing lesion. (**F**) A 47-year-old woman with hyperprolactinemia, and a tiny hypoenhancing lesion along the floor of the sella.

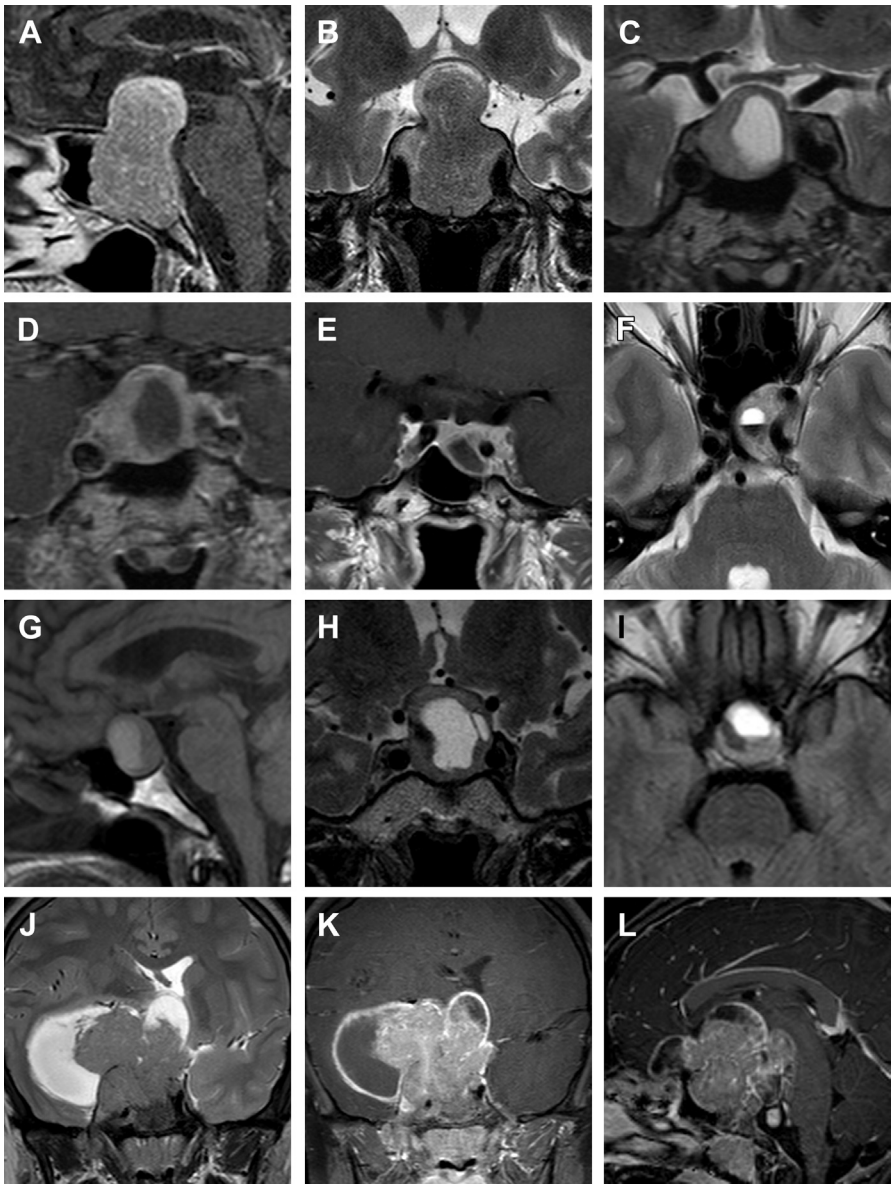

FIG. 7 Pituitary macroadenoma; 5 examples on coronal T2WI and C + T1WI. (**A**, **B**) An 84-year-old man with classic "snowman" or "figure-of-8" configuration; there is sellar expansion and suprasellar extension. (**C**, **D**) A 57-year-old man with hyperprolactinemia, and cystic sellar/suprasellar lesion that had enlarged from a prior examination. (**E**, **F**) A 39-year-old man with incidentally found macroadenoma involving the left cavernous sinus. Fluid levels seen on axial imaging due to internal hemorrhage. (**G–I**) A 35-year-old woman with visual disturbance; suprasellar extending mass with cystic degeneration and internal layering hemorrhage. (**J–L**) A 9-year-old boy with altered mental status; giant solid and cystic mass arising from the sella.

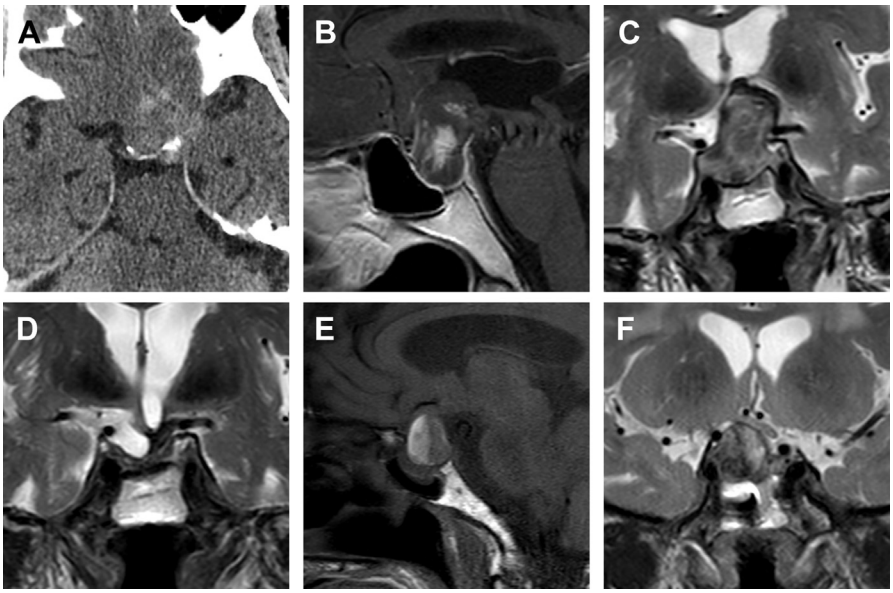

FIG. 8 Pituitary apoplexy. (**A**) An 86-year-old man with acute onset headache. Noncontrast CT shows sellar enlargement with a hyperdense lesion and adjacent subarachnoid hemorrhage. (**B**) Sagittal T1WI and (**C**) coronal T2WI show a sellar mass bulging upward into the suprasellar cistern with heterogeneous internal signal due to diffuse intralesion hemorrhage. (**D**) Following transsphenoidal decompression, there is decreased mass effect, chronic hemosiderin, and tethering of the optic chiasm. (**E**, **F**) A 74-year-old man with acute endocrine collapse. Sagittal T1WI and coronal T2WI show an enlarged pituitary with heterogeneous signal intensity and a hemorrhagic fluid level.

Pituitary Apoplexy and Abscess

Apoplexy is an acute clinical syndrome caused by hemorrhagic or ischemic infarction of the pituitary gland. The indirect vascular supply of the adenohypophysis makes it more susceptible to ischemia, particularly in the setting of an enlarging pituitary adenoma [36]. Clinical symptoms of pituitary apoplexy include the following:

- Headache
- Visual defects
- Cranial neuropathy
- Acute endocrine collapse

The clinical syndrome of pituitary apoplexy should be distinguished from benign hemorrhage into a smaller adenoma, often incidentally seen [37], requiring direct communication with referring physicians.

On imaging, the pituitary gland is enlarged, with heterogeneous signal characteristics based on the degree and stage of hemorrhage (Fig. 8). Diffusion abnormality may be present in ischemic infarction but can be obscured due to artifact from the bony skull base. Heterogeneous enhancement is common, and often adjacent dural enhancement or sphenoid sinus mucosal enhancement is also present [38].

A pituitary abscess is a rare complication of systemic illness that may present clinically with features similar to pituitary apoplexy. The pituitary is generally enlarged, bowing upward into the suprasellar cistern. T2WI signal hyperintensity is most common, with variable enhancement. Diffusion-weighted imaging (DWI) may show signal hyperintensity, but is insensitive. Prompt clinical recognition is essential, necessitating surgical treatment.

Rathke Cleft Cyst

Rathke cleft cysts are nonneoplastic and arise from developmental remnants of Rathke pouch [39]. The cysts are typically small, less than 1.5 cm, and may be purely intrasellar or extend upward into the suprasellar cistern. The cysts are typically well defined and rarely clinically symptomatic.

On CT, Rathke cleft cysts are typically hypodense and rarely calcified. Varying cyst content (mucoid vs serous) may account for varying T1WI signal on MRI, although the lesions are typically T2WI

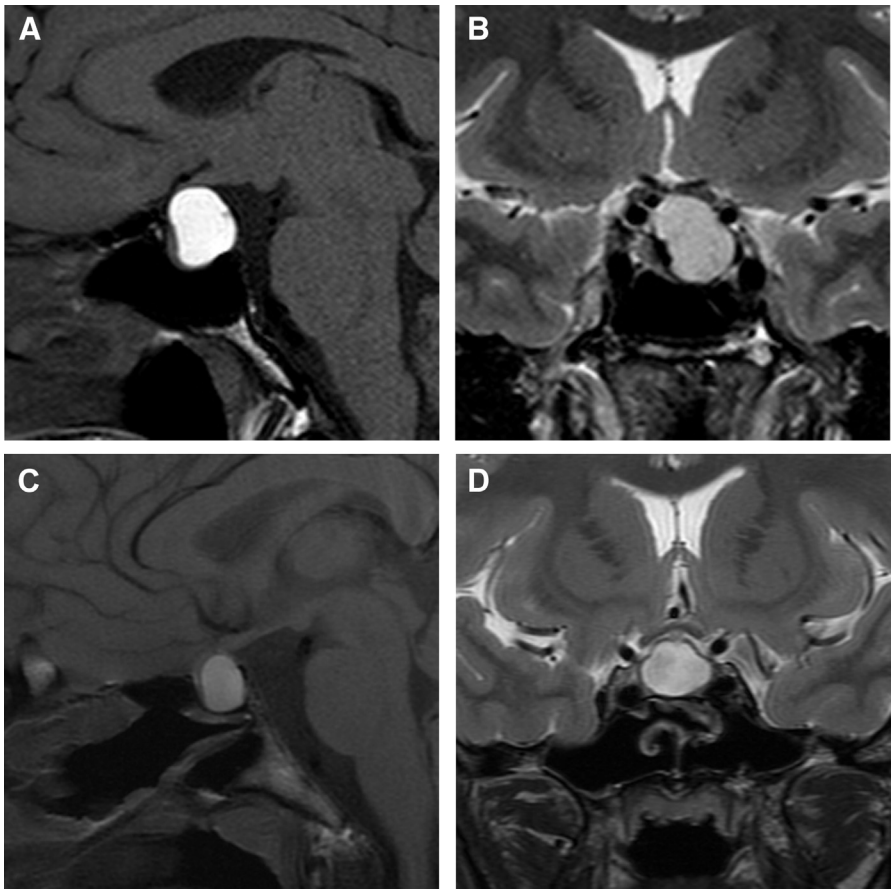

FIG. 9 Rathke cleft cyst; 2 examples. (**A**, **C**) Sagittal T1WI and (**B**, **D**) coronal T2WI. Intrasellar lesions with suprasellar extension, demonstrating high intrinsic T1WI signal hyperintensity and cystic appearance on T2WI.

hyperintense (Fig. 9). They are characteristically nonenhancing, in contrast to craniopharyngiomas, although a small (nonenhancing) intracyst nodule may be present.

Craniopharyngioma

Craniopharyngioma is a low-grade tumor derived from Rathke pouch epithelium, which presents with a bimodal age distribution and varying imaging features. The adamantinomatous subtype is more common, occurs in children (5–15 years), and is characteristically a large, lobulated, cystic lesion [40]. Multicompartment involvement beyond the sella and suprasellar cisterns may involve the anterior and middle cranial and/or posterior fossae. As such, headache, visual symptoms, and endocrine abnormalities are the commonest clinical manifestations.

Imaging features of craniopharyngioma include the following (Fig. 10):

- Enhancing solid tumor tissue
- Variable T2WI signal intensity
- Enhancing cyst walls
- Variable cyst protein content (TIWI cyst hyperintensity)
- Mural and chunky solid calcification (seen on CT)

The papillary subtype of craniopharyngioma is more common in adults older than 50 years. In contrast to the pediatric subtype, these lesions are more commonly solid, enhancing, and rarely calcified.

Lymphocytic Hypophysitis

LH is an idiopathic inflammatory condition affecting the pituitary stalk [41]. The differential diagnosis of lesions of the pituitary stalk is shown in Box 1. LH

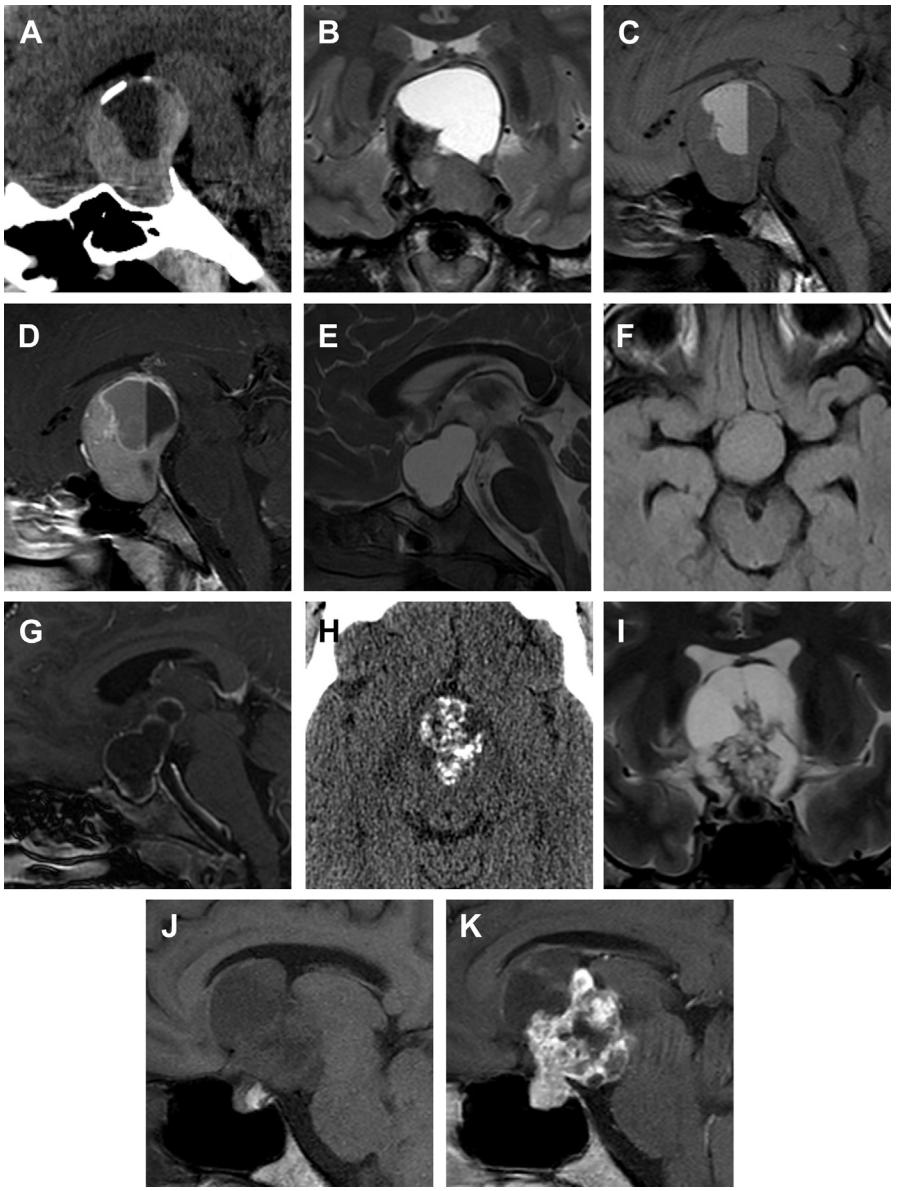

FIG. 10 Craniopharyngioma; 3 examples. (**A–D**) A 24-year-old man with cystic craniopharyngioma, including proteinaceous fluid levels, mural calcification, and solid enhancing portions. (**E–G**) An 11-year-old boy with proteinaceous sella/suprasellar cystic lesion and mildly thickened mural enhancement. (**H–K**) A 34-year-old woman with heavily calcified, heterogeneously enhancing, partially cystic sella/suprasellar mass.

presents most commonly in the peripartum period with headache and endocrine abnormalities, but has also been seen in diabetic men. Treatment is conservative, and the condition is often self-limited. Imaging features of LH include the following (Fig. 11):

- Stalk thickening greater than 2 mm
- Nontapering or rounded stalk morphology
- Adenohypophysis enlargement and hyper enhancement
- Absent posterior pituitary bright spot

BOX 1
Lesions of the Pituitary Stalk

Abnormalities of the pituitary stalk

Ectopic posterior pituitary
Metastasis
Lymphoma
Sarcoidosis/TB
Prolactinoma
Lymphocytic hypophysitis
Langerhans cell histiocytosis
Stalk injury/transection

Tuber Cinereum Hamartoma

The tuber cinereum of the hypothalamus forms the floor of the third ventricle and is contiguous with the infundibular stalk of the pituitary. Congenital gray matter heterotopia as a migrational anomaly can occur at this level, is usually small (<5 mm) and nonenhancing, and has nearly similar imaging characteristics to gray matter (Fig. 12) [42]. Seizures (classically gelastic or laughing seizures) are the most commonly associated clinical symptom and often present early, although the lesion can be associated with central precocious puberty [43].

Other Cystic Lesions of The Sella and Suprasellar Cistern

Other cystic abnormalities of the sella and suprasellar region require special mention, either because they represent "normal" or near-normal variants or because the pathologic conditions are not specific to this region and are seen elsewhere in the head. Imaging characteristics of these lesions are summarized in Table 4.

A pars intermedia cyst is a small, commonly incidental intrapituitary cyst that arises along the junction of the anterior and posterior pituitary lobes. It is indistinguishable from a small intrapituitary Rathke cleft cyst on imaging.

A primary empty sella is a herniation of the subarachnoid space through a widened diaphragm sella, remodeling and expanding the sella, causing flattening of the pituitary gland along the sellar floor, often without deviation of the pituitary stalk [44] (Fig. 13). Secondary empty sella can be seen following surgery or medical treatment of pituitary adenoma and may distort the pituitary stalk [45] (Fig. 14).

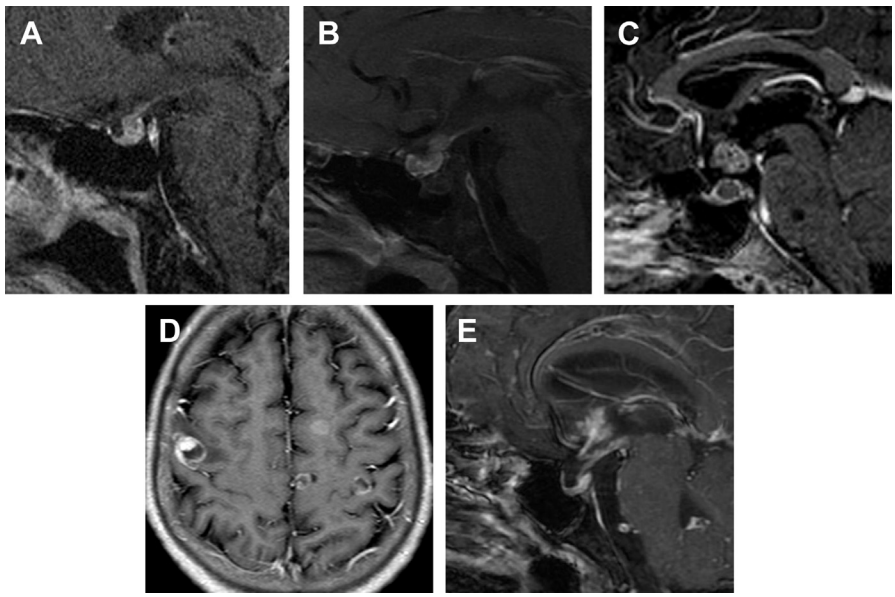

FIG. 11 LH and other stalk lesions. (**A**) A 33-year-old woman with postpartum hypopituitarism. (**B**) A 72-year-old man with a history of metastatic lung cancer and hypopituitarism, and presumed stalk metastasis. (**C**, **D**) A 64-year-old man with metastatic lung cancer. Nodular stalk mass is seen, with numerous additional enhancing brain lesions. (**E**) A 44-year-old man with neurosarcoidosis and stalk involvement.

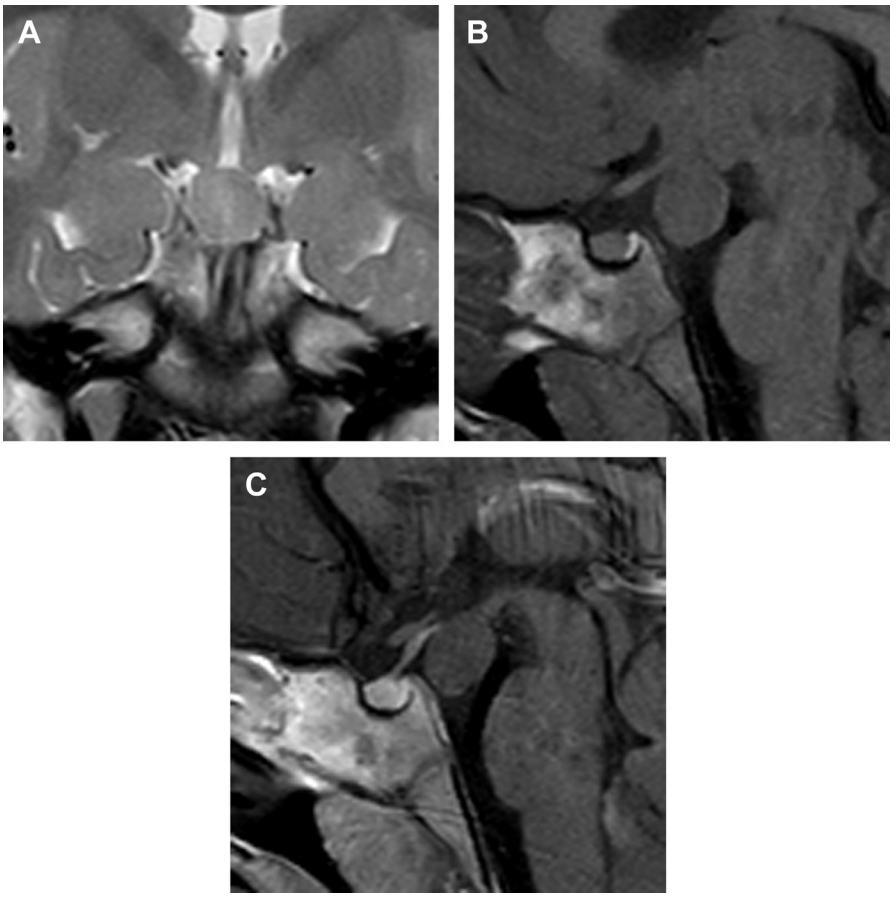

FIG. 12 Tuber cinereum hamartoma. (**A**–**C**) A 3-year-old girl with premature thelarche. Nonenhancing lesion is identified as exophytic from the hypothalamus. The lesion projects posteriorly into the interpeduncular cistern.

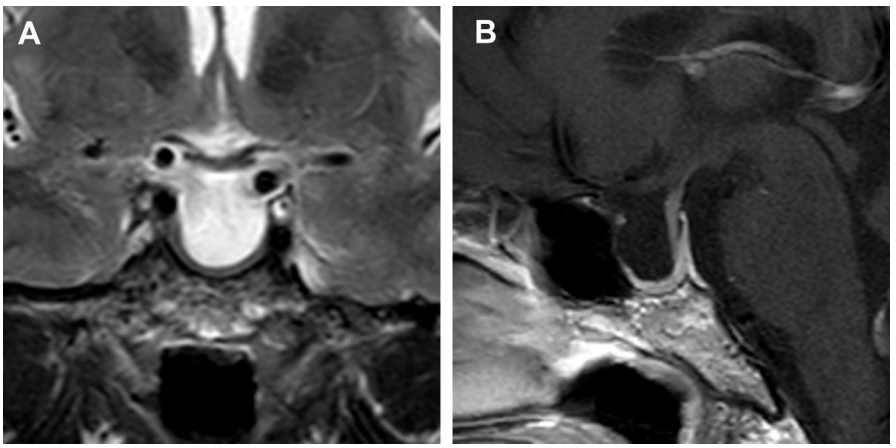

FIG. 13 Empty sella. A 47-year-old woman with dizziness. (**A**) Coronal T2WI shows an expanded, fluid-filled sella. (**B**) Sagittal C + T1WI shows the pituitary stalk inserting into a flattened pituitary along the floor of the sella.

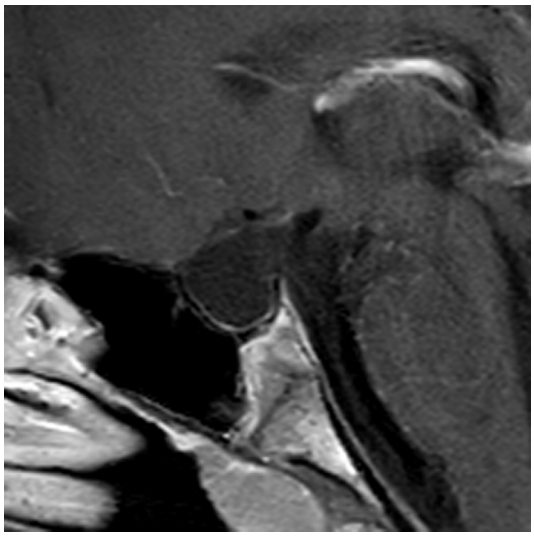

FIG. 14 Secondary empty sella. Postoperative, fluid-filled sella following decompression of cystic craniopharyngioma (same patient as Fig. 10E).

Arachnoid cysts are common extra-axial lesions that arise within the arachnoid membrane and may occur in the suprasellar cistern or herniate into the sella turcica. Epidermoid cysts are intracranial ectodermal inclusions that have more masslike properties, insinuate between tissues, and show characteristic signal hyperintensity on fluid-attenuated inversion recovery (FLAIR) and DWI.

Other Suprasellar Lesions of Nonpituitary Origin

Solid suprasellar masses may also include regional presentation of other systemic diseases. Solid metastatic masses may overlap with a variety of primary suprasellar lesions. Leptomeningeal metastasis within the subarachnoid space may implant on the brain surface and mimic infectious and noninfectious granulomatous processes, such as tuberculous meningitis, sarcoidosis, and Langerhans cell histiocytosis.

A meningioma may arise from the floor of the anterior cranial fossa and extend into the suprasellar cistern, sella tucica, or prepontine cistern. Imaging features mimic meningioma elsewhere, including gray matter signal intensity and avid contrast enhancement (Fig. 15). Intrasellar extension rarely causes sellar expansion with meningioma, distinguishing these lesions from large macroadenomas.

Suprasellar germinoma is an aggressive midline lesion of germ cell origin and accounts for nearly one-third of germinomas. Diabetes insipidus is the most common presenting clinical scenario. Masses enhance avidly, insinuate into CSF spaces, extend along the pituitary stalk, and are prone to CSF dissemination, necessitating imaging of the entire spinal axis [4]. Cyst formation and necrosis are common in larger masses (Fig. 16).

Hypothalamic and chiasmatic astrocytomas have overlapping and often indistinguishable features. Extension along the optic tracts is

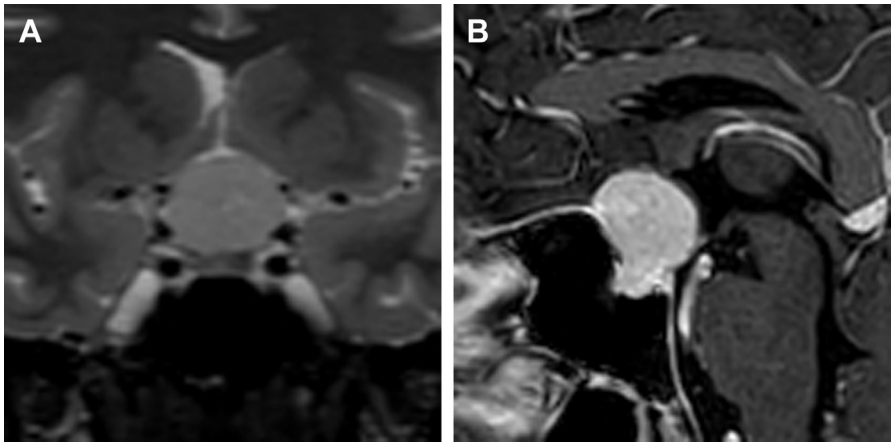

FIG. 15 Suprasellar meningioma. (**A**) Coronal T2WI shows a suprasellar lesion with signal intensity similar to gray matter. (**B**) Sagittal C + T1WI shows avid postcontrast enhancement and broad dural attachment extending posteriorly from the planum sphenoidale.

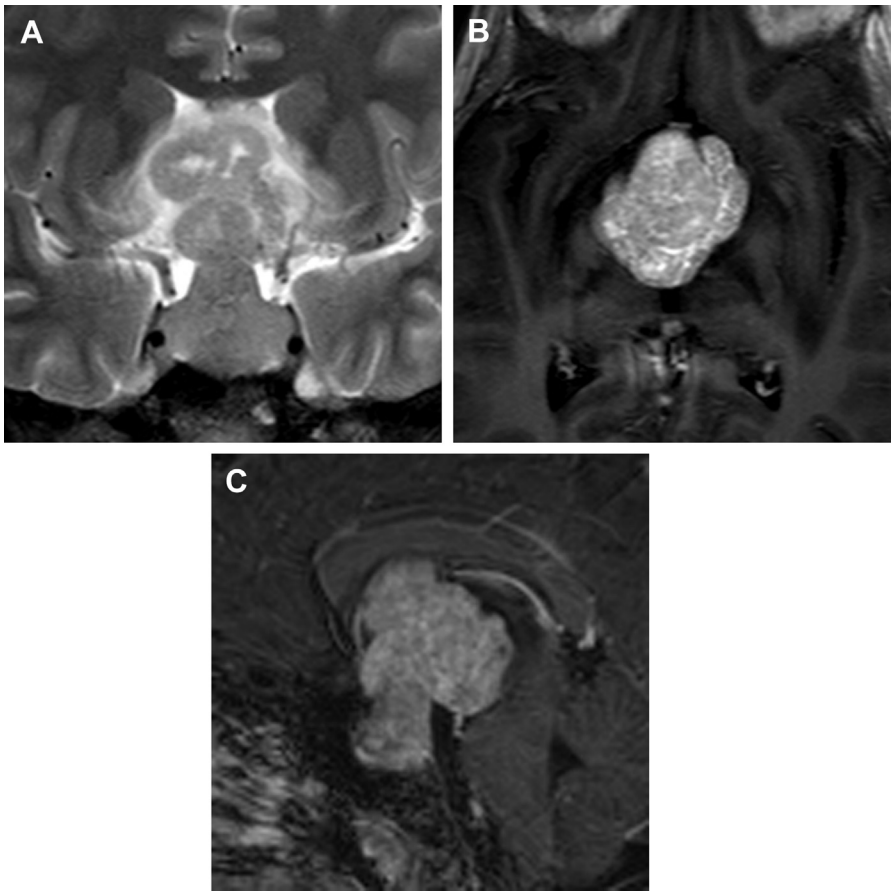

FIG. 16 Suprasellar germinoma. A 19-year-old woman with visual disturbance. (**A**) Coronal T1WI, (**B**) axial, and (**C**) sagittal C + T1WI show a heterogeneous mass with avid enhancement centered in the suprasellar cistern with significant mass effect on the undersurface of the brain.

characteristic of chiasmatic gliomas. Low-grade pilocytic lesions are common in neurofibromatosis type I in both children and adults (Fig. 17) and may be nonenhancing (low grade). Intermediate- to high-grade lesions have a more aggressive clinical course and imaging profile, including avid enhancement, necrosis, and cyst formation [46].

INCIDENTAL FINDINGS

Incidentally detected pituitary and parasellar lesions are common, with reported prevalence ranging from 1 to greater than 10% of routine brain MRI examinations [47–49]. Incidental PET with fludeoxyglucose uptake is more uncommon [50,51]. Most incidentally detected lesions are small, measuring less than 10 mm. As most incidental pituitary findings are detected on routine examinations performed by non-pituitary specialist referrers, management guidelines are helpful to provide a framework to referring physicians [52].

Pituitary microadenomas and small Rathke cleft cysts are the most common incidental lesions [53]. An empty sella may be seen incidentally in 5% to 10%, with herniation of the subarachnoid space into the sella turcica. Although this is usually an incidental finding, close to 30% of patients may demonstrate some hypopituitarism upon testing [44]. Larger macroadenomas and other suprasellar lesions are easily detected as incidental findings, but are less common [54].

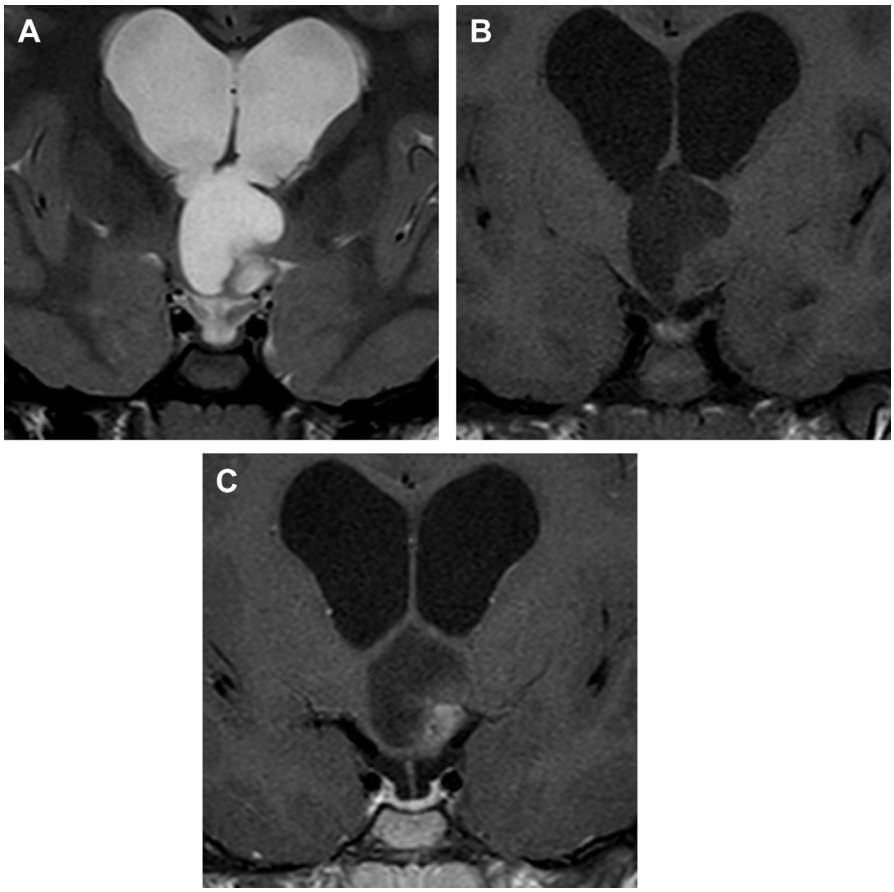

FIG. 17 Hypothalamic astrocytoma. A 5-year-old girl with altered mental status due to hydrocephalus. An obstructing suprasellar mass (juvenile pilocytic astrocytoma) arises exophytically from the hypothalamus. (**A**) Coronal T2W, (**B**) precontrast, and (**C**) postcontrast coronal T1WI.

The likelihood of small adenomas to grow or hemorrhage is small [55–57]. Screening for hypersecretion in incidentally detected lesions is clinically advocated [58]. Hypersecretion may be mild or occult in preclinical stages [59], although the incidence of hypersecretion itself is less common than the detection of small tumors. Incidentally detected larger lesions can continue to grow, are at greater risk for hemorrhage, and may cause hormonal insufficiency due to compression of normal gland.

An ACR White Paper on the management of incidental pituitary findings outlines a paradigm for reporting and guiding the management of incidentally detected pituitary findings in asymptomatic adults. Five principles of management described by Hoang and colleagues [52] are summarized in Table 6. By their classification, simple cysts are presumed Rathke cleft cysts and require no follow-up. Solid or mixed solid-cystic lesions less than 5 mm may be true or pseudolesions, such as due to nodular hyperplasia or artifact related to technical aspects of small FOV imaging, but are unlikely to grow or hemorrhage, and therefore, do not require imaging follow-up. Solid or mixed solid-cystic lesions measuring 5 to 10 mm should be correlated for pituitary dysfunction, but rarely require imaging follow-up due to the low rate of growth. Lesions greater than 10 mm, however, should be followed for interval growth in 6 to 12 months. The presence of mass effect or tumor invasion should trigger early referral for medical or surgical management.

TABLE 6
Principles of Management for Incidental Pituitary Lesions

Lesion Characteristics	Management Recommendation
Simple cysts	No additional workup
Incidental lesions <5 mm	No follow-up imaging
Solid (or mixed solid) lesion 5–10 mm	Correlate for endocrine dysfunction No imaging follow-up if normal hormone levels
Solid (or mixed solid) lesion >10 mm	Correlate for endocrine dysfunction 6–12 mo follow-up to monitor growth
Any lesion with mass effect or invasion	Surgical consultation

Data from Hoang JK, Hoffman AR, González RG, et al. Management of incidental pituitary findings on CT, MRI, and 18F-Fluorodeoxyglucose PET: a white paper of the ACR incidental findings committee. J Am Coll Radiol 2018;15(7):966–72.

SUMMARY

The range of pituitary and sellar abnormalities includes both symptomatic and incidental lesions. Understanding the relevant clinical presentations provides context for distinguishing between lesions, which may have overlapping imaging manifestations. Knowledge of the natural history of pituitary, sellar, and suprasellar masses allows for reasoned recommendations for follow-up and clinical assessment.

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