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The Pituitary Mass: Diagnosis and Management

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Several types of lesions may present within the sella turcica and in parasellar areas, of which the most common are pituitary adenomas. Less commonly, sellar lesions are of non-pituitary origin with a broad range of potential etiologies including neoplastic, non-neoplastic cystic lesions, inflammatory and infectious lesions, vascular lesions and pituitary hyperplasia [1]. Differentiating among these various causes may be difficult, as they share similar clinical and radiographic features and easily mimic each other and pituitary adenomas. Making the correct preoperative diagnosis is of clinical relevance since the treatment of choice for many of these non-pituitary sellar masses differs from that of a pituitary adenoma. An overview of the clinical and radiographic features of the more common lesions as well as their management is provided in this article.

Types of Pituitary Mass Lesions

Table 1 lists the possible lesions in the sella turcia and parasellar areas. Pituitary adenomas account for over 90% of these lesions [1–3]. In many lesions of non-pituitary origin, the patient may seem to have a nonfunctioning pituitary adenoma and the diagnosis is only made histologically. There are distinguishing features which may aid in the differential of sellar masses and these will be discussed in the sections below.

Signs and Symptoms of Sellar Masses

Sellar masses typically present in one or more of three ways: (1) hormonal abnormalities either related to hormonal excess from a hormone secreting pituitary adenoma or related to pituitary hormone deficiencies from mass effects; (2) neurologic symptoms related to the mass effect of the lesion; and (3) as an incidental finding on magnetic resonance imaging (MRI) or computed tomography (CT) performed for some other reasons. The clinical presentation varies depending on size and pattern of tumor growth, whether the tumor is hormone secreting or clinically nonfunctioning, and whether normal pituitary function is disrupted.

A few characteristics may aid to distinguish pituitary adenomas from non-pituitary lesions. The age of subject at time of presentation with a sellar mass may provide helpful in determining the nature of the lesion. For example, craniopharyngiomas and Rathke's cleft cysts may occur in children while pituitary macroadenomas are rare in this age group [4]. However, when prolactinomas occur in children and adolescents, they are more commonly macroadenomas than microadenomas, in contradistinction to when they present in adults [5]. Metastatic carcinoma to the pituitary gland is rare and for the most part has been reported in individuals over the age of 50 years [6]. Metastatic lesions to the pituitary gland usually appear as part of a widely metastatic carcinoma but rarely they may be the initial presentation of a carcinoma [1,7]. Lymphocytic hypophysitis primarily occurs in the last trimester of pregnancy or postpartum so should be a strong consideration in a woman who presents with a sellar mass at the end of her pregnancy or postpartum [8,9]. Pituitary abscess is more common in the immunocompromised, in individuals with concurrent pituitary lesions or prior pituitary surgery or irradiation [10]. Inflammatory or infectious processes involving the pituitary gland are often associated with a history of prior or concurrent systemic involvement [2]. However, rarely cases of isolated tuberculoma [11] or neurosarcoidosis [12,13] without any systemic manifestations have been reported.

Both hormone deficiencies and excesses may occur. Diabetes insipidus is an extremely rare finding at presentation in patients with pituitary adenomas and its presence is highly suggestive of a nonpituitary etiology [1,7,14]. Hypopituitarism may occur for three reasons: (1) compression of the hypothalamic-pituitary stalk from a mass lesion in this area; (2) direct compression of the pituitary may sometimes occur with a large macroadenoma; and (3) direct hypothalamic damage, often indicating a lesion originating in the hypothalamus such as a craniopharyngioma. The prolactin (PRL) level is often helpful in distinguishing

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Table 1. *Differential diagnosis of sellar masses*

I.	Pituitary adenomas
II.	Cystic lesions
	Rathke's cleft cyst
	Craniopharyngioma
	Arachnoid cyst
	Other cystic lesions
III.	Neoplasms
	Meningioma
	Germ cell tumor
	Chordoma
	Granular cell tumor
	Glioma
	Metastatic lesions
	lymphoma/leukemia
IV.	Inflammatory/Infectious lesions
	Sarcoidosis
	Tuberculosis
	Pituitary abcess
	Langerhan's histiocytosis
	Lymphocytic hypophysitis
V.	Vascular lesions
	Aneurysm

the 2nd possibility from the other two, as low levels usually indicate direct pituitary damage [15] and lesions which compress the hypothalamic-pituitary stalk or cause direct hypothalamic damage can lead to elevations of PRL levels. Intrasellar craniopharyngiomas and Rathke's cleft cysts can sometimes cause mild hyperprolactinemia in this way, mimicking a prolactin-secreting microadenoma [16]. At times PRL levels can reach as high as 150-200 ng/ml from stalk compression [1]. Large prolactinomas (lesions greater than 2 cm) generally have PRL levels ranging from several hundred to several thousand nanograms per milliliter [17]. Accordingly, if PRL levels are less than 200 ng/ml in the presence of a large sellar mass, other diagnoses than prolactinoma should be considered [3,17]. An additional caveat in the diagnosis of prolactinomas is the hook effect related to the use of immunometric assay for measurements of serum PRL levels [18]. This effect is mainly observed for large prolactinomas when the PRL level is erroneously reported to be in the normal to 200 ng/ml range but on serial dilution the actual level is found to be in the thousands. Thus, for a pituitary mass of greater than 3 cm, measurement of PRL on a 1:100 dilution of the serum should be performed to avoid missing the diagnosis of a prolactin secreting adenoma [15]. A correct diagnosis in this case is essential, as prolactin-secreting adenomas are best treated by medical management. With any large sellar mass, a detailed evaluation for hypopituitarism is warranted so that any deficits can be treated. Excessive secretion of other hormone would only come from a specific hormone-secreting adenoma.

Cranial nerve palsies may occur with non-pituitary lesions originating from or infiltrating parasellar structures or from macroadenomas that invade the cavernous sinus [1,19]. Psychiatric and cognitive deficits, such as reduced short term memory and personality changes, have been reported more frequently for craniopharyngeomas compared to other cystic lesions [20]. Signs and symptoms related to hormone excess are only present with functioning pituitary adenomas compared to other causes of sellar mass; however, these tumors (especially growth hormone and prolactin secreting tumors) may grow very slowly leading to subtle and insidious symptoms that may go unrecognized by the patient for a long time [3].

Visual compromise is common with both pituitary and non-pituitary lesions, especially if the lesion is large [1]. However, the progression of symptoms may be very slow and not be recognized by the patient for years. More commonly, a large sellar mass is discovered unrelated to visual compromise and significant visual field defects are detected at the time of presentation. The pattern of visual loss maybe of help in diagnosing the type of sellar lesion. In a retrospective study of 149 patients with sellar mass who had chiasmal syndrome at presentation, the following features were predictive of a non-pituitary mass: symptomatic visual loss, younger age, unilateral optic disc pallor, a relative afferent papillary defect, and an absolute or a complete visual field defect [21].

Overview of Radiographic Features of Sellar Lesions

MRI is the imaging modality of choice for the evaluation of the sellar and parasellar regions, as it offers more information on pituitary morphology and surrounding structures [22–24]. However, there are specific situations in which a CT scan may be of use, such as for detection of calcifications [23,24] or for obtaining detailed information regarding bone anatomy [24]. The standard protocol for evaluation of pituitary gland with MRI consists of obtaining T1-weighted images and then repeating the T-1 images after administration of an intravenous contrast agent containing Gadolinium-based media [24]. On T1 precontrast images, the normal anterior pituitary gland is similar to the gray matter in terms of signal intensity, while the posterior pituitary appears as focal area of high signal (bright spot). After administration of contrast, there is homogenous enhancement of both anterior and posterior lobes of the pituitary gland [24]. Pituitary adenomas appear as focal areas of low signal within the pituitary gland on T1 precontrast images. Following contrast administration, the majority of pituitary microadenomas demonstrate slow enhancement relative to the normal pituitary gland and so appear less intense; however, in about 30% of cases the adenoma may have higher signal intensity compared to the normal

pituitary gland [24,25]. Pituitary macroadenomas are easier to identify because of their larger size and frequent extrasellar extension.

One cardinal finding that we often use in differentiating pituitary adenomas from other lesions is that pituitary adenomas that invade the cavernous sinus do not compress the internal carotid arteries. In our experience, such carotid artery narrowing is only found with lesions other than pituitary adenomas, such as meningiomas.

Sellar enlargement is present in 94–100% of macroadenomas [23] and as many as 50% of nonpituitary sellar masses so absence of sellar enlargement favors a nonpituitary mass [26]. The pattern of displacement of the pituitary gland by the lesion may also prove useful in determining its etiology. Pituitary adenomas usually displace the normal pituitary gland superiorly, although in 40% of cases the normal pituitary may not be detectable [23]. Suprasellar lesions typically are centered superior to the sella and may also flatten and displace the pituitary gland inferiorly or anteriorly [27].

Certain lesions have specific signal characteristics or constellations of features on MRI which may aid in their diagnosis. The presence of calcification in a large lesion with increased T2-weighted signal and cystic components is suggestive of craniopharyngioma [20]. Intense homogeneous enhancement following contrast administration on MRI, presence of linear extension of enhancement at the periphery of the tumor along the dura (dural tail), as well localized bone thickening (hyperostosis) are characteristics of meningioma [24].

Pituitary Adenoma

Pituitary adenomas constitute over 90% of pituitary lesions and are common findings at autopsy and as incidental findings on imaging studies of the brain [3,28,29]. The vast majority of these incidental adenomas are microadenomas as defined by size less than 1 cm in diameter [3,28,29]. Almost all pituitary adenomas are benign, although there may be localized areas of invasion. True pituitary carcinomas are so rare as to be reportable and are diagnosed based on the presence of distant metastasis [3].

Pituitary adenomas appear to be monoclonal in nature and originate from a single cell mutation followed by clonal expansion [30–32]. The pathogenic mechanisms leading to formation of these tumors remain enigmatic [32]. Pituitary hormones and growth factors involved in normal pituitary gland development have been implicated in this process. The initial event is most likely a genetic alteration in cell proliferation or survival which then allows cells to become more responsive to hormones or growth factors and promotes clonal expansion [32]. An example of such an initial event is the activating mutation in the stimulatory G-protein α-subunit gene leading to persistent activation of adenyl cyclase that has been described in a subset of pituitary somatotroph adenomas as well as in the McCune-Albright syndrome [32].

Current classification of pituitary adenomas relies on immunocytochemical staining. In general this classification is in agreement with the clinical presentation of the individual patient but there are clinically silent adenomas that stain positively for pituitary hormones. Rarely, clinically nonfunctioning tumors have positive staining for adrenocorticotropic hormone [33,34] or growth hormone [35]. The natural history of these tumors is not known although adrenocorticotropic staining nonfunctioning tumors appear to be more aggressive than non-secreting tumors and have a recurrence rate similar to that of corticotroph secreting adenomas that cause clinical Cushing's disease [34,36].

Based on the current classification, prolactinomas are the most common type of pituitary adenomas [29,37]. Table 2 provides a list of the various types of adenomas and the most commonly used diagnostic tests. Gonadotroph adenomas are the second most common pituitary adenomas based on immunocytochemical staining. Clinically most of these tumors appear to be nonfunctioning, even

Table 2. *Hormone secreting pituitary adenomas: Diagnostic testing*

I.	*Prolactin secreting adenoma*:
	Elevated basal levels
	Exclusion of other causes by history, physical examination and thyroid function test
	Measurments of serum prolactin on serial dilution to rule out hook effect for large
	adenomas
II.	*Gonadotroph pituitary adenoma*:
	Elevated α-subunit, LH, FSH levels
	Elevated ratio of α-subunit to LH and FSH
III.	*Corticotropin-secreting adenoma*:
	Elevated 24 hour urine cortisol level (level 3$\times$ normal is diagnostic) Failure to suppress 8 AM serum cortisol levels $<3\mu$g/dl after 1 mg dexamethasone given orally at midnight Elevated midnight cortisol level >7.5 μg/dl Best confirmatory test is combined CRH-dexamethasone test: Dexamethasone 0.5 mg is administered orally every 6 hours for 24 hours followed by an intravenous bolus of CRH. Serum cortisol is measured 15 mins later and a level >1.4 μg/dl is diagnostic. Inferior petrosal sinus sampling may be necessary in some cases to differentiate between a pituitary adenoma and ectopic ACTH secretion
IV.	*Growth hormone-secreting adenoma*:
	Elevated insulinlike growth factor 1 (IGF-1)
	Failure to suppress GH levels to <1 ng/ml with oral glucose load
V.	*Thyrotropin-secreting adenoma*:
	Elevated basal T4, T3 with nonsuppressed or elevated levels of TSH
	Elevated levels of α-subunit and a-subunit:TSH ratio

though frequently they produce intact gonadotropins or their glycoprotein subunits *in vivo* or *in vitro* [38–40]. Since these tumors are very inefficient in hormone secretion, symptoms related to hormonal excess are rare and frequently presentation is related to the mechanical effects of a large tumor mass [15].

Growth hormone (GH) secreting adenomas account for the majority of cases of acromegaly. Rarely, acromegaly is caused by oversecretion of growth hormone releasing factor from ectopic tumors or hypothalamic lesions [41]. The signs and symptoms of acromegaly are usually insidious in nature and may go unrecognized by the patient for years before coming into medical attention. The majority of patients with acromegaly have a macroadenoma at the time of the diagnosis. In addition to disfiguring features such as bony overgrowth, soft tissue swelling, and skin changes, acromegaly is also associated with serious and life threatening complications such as increased prevalence of diabetes, hypertension, cardiovascular disease and respiratory disorders [41]. Accordingly, early diagnosis and treatment of this disorder is crucial to avoid long term complications.

Corticotropin (ACTH) secreting pituitary adenomas account for 65–70% of all causes of Cushing's syndrome. At presentation, the majority of these tumors are microadenomas and even though most are benign, they are usually more invasive than other types of pituitary adenomas. A brief description of screening tests for Cushing's syndrome is provided in Table 2. At times, it may be very difficult to differentiate an ectopic ACTH producing tumor from an ACTH-producing pituitary adenoma by testing and inferior petrosal sinus sampling comparing ACTH levels to peripheral venous levels may be necessary.

Thyrotropin (TSH)-secreting pituitary adenomas are very uncommon and account for only 1% of all pituitary adenomas. The clinical manifestations include mild hyperthyroidism, a goiter, and inappropriately high levels of TSH for the peripheral thyroid hormone levels. This disorder may be confused with thyroid hormone resistance which is characterized by high or mildly increased thyroid hormone concentrations associated with normal or mildly increased TSH concentration. However, patients with thyroid hormone resistance are usually clinically hypothyroid or euthyroid and do not have a goiter. The majority of TSH-secreting pituitary adenomas are large at the time of diagnosis and patients may have signs of hypopituitarism as well as other symptoms related to mass effect.

Nonfunctioning pituitary adenomas often present with symptoms related to tumor mass, various degrees of hypopituitarism, or as incidental findings on imaging studies of the brain. Even though the majority of these incidental adenomas are microadenomas, macroadenomas may also be discovered as incidental findings [3,42]. An endocrine evaluation is recommended in case of any incidental adenoma regardless of its size, as at times the signs and symptoms of hormone oversecretion are very subtle and may go undetected by the patient or the physician [3]. As noted above, when large lesions are present an evaluation for hypopituitarism is also warranted.

Management of Pituitary Adenomas

Management of pituitary adenomas depends on tumor size and type as well as the individual patient's presentation and treatment goals. Overall, treatment should aim at normalization of hormonal excess if present, recovery of normal pituitary tissue and function, replacement of hormonal deficiencies, and treatment of issues related to mass effects caused by the tumor. For prolactinomas, medical therapy with dopamine agonists is very effective in reducing prolactin levels as well as in decreasing tumor size and should constitute the primary mode of treatment [17].

Surgical resection by transsphenoidal technique is the primary treatment modality for other types of pituitary adenomas. The outcome of surgery is determined by the surgeon's experience, the size of the adenoma and the degree of tumor extension [43]. Extension into the cavernous sinus is almost always associated with incomplete surgical removal [43]. Transsphenoidal surgery is usually well tolerated with minimal complications. The most frequent complication is diabetes insipidus which is usually of transient nature [43]. If, after surgical resection, residual tumor mass or persistent hormonal overproduction is detected, adjuvant radio- or medical therapy should be employed (for details see ref. [41]). For asymptomatic non-functioning adenomas, careful observation and monitoring as opposed to surgical resection is a reasonable option, especially if the tumor is small and intrasellar. If this option is chosen, periodic monitoring of tumor size using imaging studies is recommended, as up to 20% of these lesions either progress or lead to complications such as apoplexy [44]. In all cases, anterior pituitary hormone deficiencies should be appropriately evaluated and treated.

Cystic Lesions

In a large surgical series, cystic lesions were the most common type of non-pituitary sellar mass [1]. Rathke's cleft cysts, craniopharygiomas, and arachnoid cysts account for the majority of these lesions with the first two being more common The three lesions share many similarities in clinical presentation as well as biochemical and radiographic findings, making it difficult to distinguish them from each other. As management and follow up for

these lesions varies, preoperative diagnosis is important. As an example, in the case of Rathke's cleft cyst, drainage of the cyst without full surgical resection may be sufficient treatment.

Rathke's cleft cysts and craniopharyngiomas are derived from cystic epithelium [45,46]. Arachnoid cysts arise from herniation of the arachnoid membrane into the sella and are filled with cerebrospinal fluid [47]. Craniopharyngiomas are the most frequent sellar tumor of childhood and comprise 10% of brain tumors in children; however, they can occur at any age [4]. Rathke's cleft cysts are predominantly seen in adults, presenting at mean age of 38 years [20]. Individuals with arachnoid cysts are usually older at presentation [20]. Craniopharyngiomas are the most aggressive of the three and cause the most symptoms. However, hyperprolactinemia and neurologic symptoms such as headaches and visual field defects are frequently reported with all 3 lesions [20]. Psychiatric and cognitive dysfunction, primarily reduced short term memory, have been reported in one third of cases of craniopharyngiomas but not in patients with the other two cystic lesions [20]. Anterior pituitary hormone deficiencies have also been reported with all three lesions but occur most frequently with craniopharyngiomas and least frequently with arachnoid cysts [1,20,48]. The most common anterior pituitary hormone deficiencies involve the gonadrotropin and growth hormone axes [2,20,49]. Diabetes insipidus has been reported frequently in association with craniopharyngiomas but rarely with the other two lesions [2,20].

Certain radiographic findings may aid in differentiating the three lesions from each other. Craniopharyngiomas rarely are entirely intrasellar, in contrast to Rathke's cleft cysts and arachnoid cysts, which are commonly completely intrasellar [2,20,23,24]. However, suprasellar Rathke's cleft cysts and arachnoid cysts have also been reported [20,24]. The presence of solid components favors the diagnosis of craniopharyngioma [20,23,24]. Calcifications are much more common in craniopharyngiomas compared to Rathke's cleft cysts. Arachnoid cysts usually have a smooth contour, a signal intensity similar to cerebrospinal fluid, and lack calcifications [23,24]. Rim enhancement is a common finding in Rathke's cleft cysts but is not present in arachnoid cysts [23]. In summary, differentiating the three lesions from each other is best achieved by a careful examination of a combination of clinical, biochemical, and radiographic features.

Craniopharyngiomas are aggressive tumors and, if incompletely removed, have a high recurrence rate. To insure complete resection, subfrontal surgical resection is favored unless the tumor is small and intrasellar, in which case transsphenoidal surgery is an alternate option [50]. Many patients require postoperative radiation to reduce disease recurrence. Symptomatic Rathke's cleft cysts as well as arachnoid cysts are best treated with surgical drainage and/or excision and have low recurrence rates [4].

Other Neoplasms

Meningiomas, primitive germ cell tumors, and chordomas are a few of the more common lesions belonging to this category. Meningiomas are benign lesions that arise from the diaphragma sella, tuberculum sella, or planum sphenoidale. In a large surgical series, they accounted for 10% of sellar masses of non-pituitary origin [1]. Visual disturbance, most commonly bitemporal hemianopsia, is a frequent symptom in patients with meningiomas [2]. While endocrine dysfunction is less common, mild to moderate elevation of prolactin levels may be present in 50% of patients [1]. Two frequently observed radiographic features of sellar meningiomas include a dural tail and hyperostosis (localized bone thickening) [24]. These tumors are best managed surgically and, if completely resected, have a low recurrence rate [51].

Germ cell tumors can present in the suprasellar region and include germinomas, teratomas, and ectopic or metastatic pinealomas of which the most common are germinomas. These lesions account for 1% of intracranial lesions, are derived from residual germ cells along the midline and are identical to germ cell tumors of the gonads and mediastinum. Endocrine abnormalities, including diabetes insipidus and hyperprolactinemia, are common with these tumors. In many cases, they secrete beta-human chorionic gonadotropin subunit or alpha-fetoprotein, thus aiding in their diagnosis when found in the cerebrospinal fluid [2]. On MRI, there is strong and homogeneous enhancement of these tumors [23]. Germinomas are highly radiosensitive [52].

Chordomas are another parasellar lesion that typically produce bony destruction and lead to cranial neuropathy and neurologic symptoms. These tumors arise from the notochordal remnants of the clivus. On MRI imaging, these tumors commonly appear as destructive and invasive lesions and in 50% appear to have calcifications [2]. Treatment includes surgical resection followed by radiotherapy if the tumor is not completely removed.

Certain malignancies such as lung and breast carcinoma and, less frequently, tumors of gastrointestinal tract, kidney, prostate, and melanoma may metastasize to the sellar area and should be considered in the differential diagnosis of sellar or suprasellar lesions [6,7,53]. In autopsy series, latent metastatic disease to pituitary gland has been detected in 5% of patients with known malignancies, although in one-third the pituitary is macroscopically

normal [7]. Pituitary disease may be the presenting sign of an unrecognized malignancy in occasional cases [1,54] and in rare cases, the primary tumor remains undetectable [7]. Metastatic disease to the pituitary usually is reported in individuals older than 50 years but occasionally it may also occur in early adulthood. The most common clinical symptom is diabetes insipidus, which has been reported in greater than 70% of patients and at times may be transient [6]. Cranial nerve involvement is also frequent [6]. The rapidity of symptoms is also suggestive of a metastatic lesion. On MRI, distinguishing metastatic lesions from the more common pituitary adenomas or other lesions is not easy, but metastatic lesions often involve the stalk and hypothalamus and there may be evidence for other metastatic foci [24].

Inflammatory and Infectious Lesions

A variety of inflammatory and infectious disorders can also involve the sellar region, tuberculosis and sarcoidosis being the most common of such disorders. In both cases, often there is evidence for systemic disease but at times sellar involvement may be the first and only manifestation [13,55]. In sarcoidosis, diabetes insipidus is common due to predilection for hypothalamic and posterior pituitary involvement [12].

Langerhans cell histiocytosis (histiocytosis X) is a rare disorder that may involve the hypothalamic-pituitary axis. This disorder occurs more frequently in children but occurs in adults as well and the clinical phenotype varies between the two age groups [56]. Diabetes insipidus is a common finding and has been reported in 15–50% of adult patients with this disorder. In a long-term study of patients with this disease, anterior pituitary hormone deficiencies also developed frequently over time [56]. In this study, radiotherapy was helpful in achieving local control of tumor but did not influence the hormonal abnormalities [56]. Although histiocytosis usually presents as an infiltrative disorder [54], it may rarely present as a mass lesion [57].

Lymphocytic hypophysitis is an unusual disorder of the pituitary gland that presents with a sellar mass and/or hypopituitarism, most often associated with pregnancy and the postpartum period. The true incidence of lymphocytic hypophysitis is unknown, but it is likely to be underestimated due to confusion with Sheehan's syndrome and due to the spontaneous recovery of undiagnosed cases. Symptoms resulting from partial or panhypopituitarism occur in approximately 80% of cases [8,9]. Lymphocytic hypophysitis has an inexplicable unique predilection for the corticotrophs and thyrotrophs while sparing the gonadotrophs [9]. Diabetes insipidus has been reported in one third of patients with lymphocytic hypophysitis [9]. MRI imaging may show an enhancing pituitary mass in as many as 95% of cases, often with suprasellar extension [8,9]. 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 [23,24]. The natural history of the disease is variable and spontaneous remissions have been reported. However, in most cases there is progression of the disease and development of multiple anterior pituitary hormone deficits requiring replacement of appropriate hormones. In some but not all cases, use of large doses of corticosteroids has been associated with clinical improvement and reduction in size of the mass but the overall benefit of such use has not been clearly established. Transsphenoidal resection is recommended for compressive symptoms due to an enlarging mass [4,9].

Hyperplasia of the Pituitary Gland

The normal pituitary gland undergoes an increase in size during puberty, especially in girls. Pregnancy is also associated with physiologic enlargement of the pituitary gland that may be as much as a 100% increase. After the first postpartum week, the gland rapidly returns to normal. Certain pathological conditions also predispose to pituitary hyperplasia such as primary hypothyroidism and central precocious puberty. On imaging studies, pituitary hyperplasia is manifest by symmetrical pituitary enlargement that may mimic a pituitary adenoma or hypophysitis [23]. The signal and enhancement characteristics are of normal pituitary tissue and there is no extrapituitary pathological enhancement or any focal lesions [23].

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