Pituitary adenomas represent 10% of all intracranial neoplasms and make up more than 90% of all sellar masses [1]. The differential diagnosis of nonpituitary sellar masses is broad and includes inflammatory and infectious diseases, cell rest tumors, germ cell tumors, gliomas, meningiomas, metastatic tumors, and vascular lesions [2]. Nonadenomatous pituitary lesions can be classified as shown in Table 1 and as described later [3]. Craniopharyngioma is described in the article by Karavitaki and Wass elsewhere in this issue.

**Clinical presentation**

Nonadenomatous sellar lesions do not present with any hypersecretory syndrome but rather with neurologic or hypopituitary symptoms as a result of the mass-effect mechanism.

Neurologic symptoms include headache, visual disturbance, cranial neuropathy, hydrocephalus, and mental changes. Hypopituitarism most often is characterized by growth hormone deficiency and gonadal dysfunction, followed by secondary hypothyroidism and adrenal insufficiency. Hyperprolactinemia secondary to stalk compression is a common cause of hypogonadism. Diabetes insipidus is highly suggestive of nonadenomatous sellar lesions, especially in sarcoidosis and in metastatic sellar involvement.
Specific lesions

Neoplastic/developmental lesions

Cell rest tumors

Rathke’s cleft cyst. Cysts derived from Rathke’s pouch are found between the pars anterior and the infundibular process in 13% to 22% of the pituitary glands [4]. Typically, Rathke’s cleft cysts are small and asymptomatic. Symptomatic cysts can occur at any age but more frequently between ages 40 and 60. This lesion usually is intrasellar and may present with impaired vision, hypopituitarism, and headache.

On CT scanning, cysts usually are hypodense and not enhanced by contrast. The lack of calcification is important to differentiate them from craniopharyngioma. In MRI, the cyst signal often is similar to cerebrospinal fluid on T₁- and T₂-weighted images [2].

Surgical approach is recommended when a cyst is large enough to cause symptoms. Transsphenoidal approach can drain a cyst effectively but if it recurs, a transcranial approach may be needed to resect the cyst wall. Cyst content may be clear and colorless, oily, or milky [4].

Epidermoid and dermoid cysts. Epidermoid and dermoid cysts result from the inclusion of epithelial elements during neural tube closure. The contents of dermoid lesions are desquamated epithelium, sebaceous material, and, sometimes, dermal appendages, whereas epidermoid cysts contain a white cheesy material (keratin) within a thin capsule. They appear as hypodense cysts with no enhancement in CT or MRI [4].
Chordomas. Chordomas are rare, slow-growing tumors of midline, representing 1% of all malignant bone tumors and 0.1% to 0.2% of all intracranial neoplasms. They arise from notochordal remnants in the clivus, usually producing sphenoid basis destruction. Chordomas involving the sellar region are rare and fewer than 30 cases are described in the literature [5]. The most common symptoms are headaches, visual deficits, neck pain, diplopia, and nasopharyngeal obstruction.

Bone destruction and calcification are common, occurring in more than 50% of cases and are seen better on CT than on MRI. Chordomas may extend along the entire skull base and the sella usually is destroyed instead of ballooned as often seen in pituitary adenomas. The location, bone destruction, and calcification usually make the differential diagnosis with pituitary adenomas easier. Fig. 1 shows an MRI of a parasellar chordoma.

Surgery is the treatment of choice; however, because of bone invasiveness, total excision generally is not possible. Radiotherapy can be recommended as adjunctive therapy [2,4].

Primitive germ cell tumors
Germinomas. Germinomas are malignant intracranial tumors with peak incidence in children and adolescents. Their usual localizations are the midline central nervous system structures, most frequently the pineal gland. Three patterns are described: germinomas of the ventral hypothalamus associated with germinoma in the pineal region; germinomas in the anterior third ventricle that can involve the pituitary fossa as extension; and intrasellar germinoma mimicking an intrasellar adenoma. The most common symptom is diabetes insipidus, seen in more than 80% of cases, followed by visual disturbances and obesity. CT scanning depicts isodense or hyperdense mass, sometimes multicentric, that can be enhanced markedly. Although only 5% of pure germinomas present with high values of β-chorionic gonadotropin

Fig. 1. Sellar chordoma: sagittal T1-weighted image (A) and coronal T2-weighted image (B) depicting a mass invading sphenoid sinus and eroding sellar bone.
and α-fetoprotein in cerebrospinal fluid or serum, these markers are present in approximately 30% of dysgerminomas that contain other malignant components. Histologic examination shows a granulomatous infiltrate around germ cells. Lymphocytic infiltrate can make the diagnosis more difficult. Because of a germinoma’s localization and multicentricity, surgery rarely is curative and shunting often indicated.

Germinomas usually respond well to chemotherapy and radiotherapy. Remission is achieved in 75% to 80% of cases. As germinomas are sensitive to radiotherapy, a therapeutic trial with a fractionated dose of 1500 cGy can be performed, a positive response indicative of germinoma (Fig. 2) [2,6].

Teratoma. Teratomas are benign tumors derived from the pluripotential cells from all three embryologic layers: ectoderm, mesoderm, and endoderm. Intracranial teratomas are rare, comprising approximately 0.5% of all intracranial tumors. Mature teratoma is characterized by one or two fully differentiated embryologic layers; immature teratoma is formed by embryonic elements from one or more layers. These tumors are found most commonly in the pineal region, followed by the suprasellar and hypothalamic regions, and rarely in the sellar region. They occur more frequently in children and young adults. Teratomas can involve the pituitary gland primarily or secondarily, by invasion. Pituitary dysfunction is seen commonly and depends on tumor growth.

Teratoma appears in imaging assessments as a well-delineated mixed cyst with calcification. This tumor can undergo ossification, teeth formation, or malignant transformation.

Fig. 2. Coronal MRI sellar T₁-weighted images depicting a germinoma: pretreatment (A) and after radiotherapy (B).
Mature teratomas are benign and usually radioresistant; therefore, surgery is recommended. Immature teratomas, in contrast, are aggressive and have metastatic potential [7].

*Other tumors*

**Meningioma.** Meningiomas are benign tumors originating from arachnoid cells and frequently adhere to the dura mater. They account for 20% of all intracranial tumors. Meningiomas that arise from tuberculum sella, the planum sphenoidale, or the diaphragma sella can appear as suprasellar mass. Intrasellar meningioma is rare and originates from arachnoid tissue in a herniated pouch. They are more common in 40 to 50 year olds and they predominate in women. Visual loss is the most common symptom.

CT shows a hyperdense lesion originating from the tuberculum sella, planum sphenoidale, or sphenoidal ridge with hyperostosis. In MRI, meningiomas typically appear as isointense on precontrast T1-weighted images and with a bright enhancement after gadolinium contrast. A dural tail sign, which is a thickening of the dura in continuity with convexity on contrast T1 images, is suggestive of meningioma [8].

For patients who have benign meningiomas that are resectable with minimal morbidity or that have significant mass effect, excision is the treatment of choice, occasionally followed by fractionated radiotherapy [9].

**Glioma.** Optic nerve gliomas are rare and comprise 3.5% of intracranial tumors in children and 1% of intracranial tumors in adults, in whom they have malignant behavior. Approximately 30% are associated with von Recklinghausen’s disease (neurofibromatosis type 1).

In children, the most common symptoms are visual loss, headache, and proptosis, whereas in adults, the initial symptom is impaired vision and retroorbital pain. Diencephalic syndrome, characterized by failure to gain weight, diabetes insipidus, and visual loss, occurs if there is hypothalamic tumoral invasion [2]. Imaging examinations show a tumor with origin in chiasm or optic nerve, classically a hypointense lesion on T1 images with contrast enhancement.

Most optic gliomas in children are low-grade astrocytomas; high-grade lesions are seen more commonly in adults.

Radiotherapy plays a primary role in management. Surgery can be performed on exophytic chiasmal tumors causing mass effect or hydrocephalus. Chemotherapy can delay other aggressive therapies, such as radiation or surgery, in young children until they progress neuropsychologically [10].

**Ependymoma.** Ependymomas are glial neoplasms arising from the ependyma of cerebral ventricules, the spinal cord central canal, or cells of the terminal ventricle in the terminal filum. Pituitary fossa is a rarely documented site of these tumors and only four cases are described in the literature.
Ependymomas appear as hyperdense lesions in CT with low-density areas suggestive of cystic or necrotic regions. These areas are seen as low-attenuation regions in MRI. There are no specific features in imaging examinations. Surgical removal is the treatment of choice. Some investigators suggest radiotherapy [11].

**Astrocytoma.** Hypophyseal stalk and posterior pituitary can be a host to all tumors that originate from glial cells. Pituicytoma is a rare primary tumor of the neurohypophysis that also can be located at the pituitary stalk. Pituicytoma usually occurs in young to middle-aged women and the most common symptoms are headache and hypopituitarism. Visual loss is described infrequently. On MRI, lesions with low intensity on T1-weighted images with homogeneous enhancement and low to intermediate intensity on T2-weighted images usually are found. Special attention to the posterior pituitary is the key in diagnosis. Although histologically benign, the location and vascular nature of these tumors can make surgical resection difficult. Cure can be achieved by total excision [12,13].

**Lymphoma.** Lymphomas can involve the pituitary gland primarily or secondarily, by metastatic spread. Primary pituitary lymphomas are rare [14]. Giustina and colleagues [15], in 2001, described 14 cases of primary pituitary lymphomas. The investigators observed that primary pituitary lymphomas are twice as common in men and occur most often around the sixth decade of life. Pituitary adenomas, lymphocytic hypophysitis, and AIDS are considered pituitary lymphoma risks. Patients can present with hypopituitarism, diabetes insipidus, and other neurologic signs due to mass effect.

Surgery, radiotherapy, and chemotherapy can be used to treat these tumors.

**Metastatic tumors.** Metastasis to the pituitary is an infrequent clinical problem with surgical prevalence less than 1% of sellar and parasellar masses. Usually, metastatic tumors affect patients in the sixth or seventh decade of life with no gender predominance. Breast and lung cancer are the most common primary neoplasms, accounting for two thirds of the cases, followed by gastrointestinal and prostate cancer, but virtually any neoplasm can metastasize to pituitary. Confirming these data, in 380 cases of metastases to pituitary, 39.7% had primary tumor origin in the breast, 23.7% in the lungs, 6.3% in the gastrointestinal system, and 5% in the prostate [16]. In general, there are other metastatic sites at the time of diagnosis, and pituitary metastasis is not the first manifestation. Pituitary metastasis was a presenting sign of extrapituitary malignancy in 43.7% of cases, however, in a series of 190 cases compiled by Komminos and colleagues [16].
In patients who have metastasis to the pituitary, the most common symptoms are diabetes insipidus in 45.2%, visual deficiency in 27.9%, anterior pituitary deficiency in 23.6%, other cranial nerve paresis in 21.6%, and headache in 15.8% [16]. Syndrome of inappropriate antidiuretic hormone secretion or diabetes insipidus also is described. Hyperprolactinemia resulting from stalk compression is encountered in 6% of cases. There are no specific radiologic findings in radiologic assessment.

A rapidly growing sellar tumor or a sudden onset of diabetes insipidus, ophthalmoplegia, or headaches in patients over age 50 suggests metastases to the pituitary. Diagnosis is important to avoid unnecessary surgery in patients who are severely compromised and prognosis is determined by the primary tumor.

Extremely rare nonadenomatous pituitary tumors. Hypothalamic hamartomas can occur in sellar regions. They are associated with precocious puberty. Gangliocytomas are benign tumors composed of neuronal tissue that can originate in sellar or parasellar areas. They sometimes are associated with functioning pituitary adenomas [3]. Other tumors, such as schwanommas [17], melanocytic tumor [18], chondrosarcoma [19], and plasmacytoma [20], are rare and appear as case reports in literature.

Inflammatory lesions

Sarcoidosis

Sarcoidosis is a chronic granulomatous disease of unknown origin affecting mainly young and middle-aged adults. The organs involved most commonly are the lungs, skin, and lymph nodes [21]. This disorder can affect individuals of both genders and almost all ages [22]. Endocrine disease is rare in sarcoidosis; hypothalamus and pituitary are the glands affected most commonly [23].

Diabetes insipidus is reported in approximately 25% to 33% of all neurosarcoidosis cases [24]. Hyperprolactinemia also occurs commonly as the result of loss of dopaminergic inhibition [23]. Anterior pituitary deficiency, mainly hypogonadism, can be present. Other hypothalamic disturbances may occur less frequently. These disturbances are attributed to granulomatous invasion of the hypothalamus-pituitary axis. Visual field defects can occur with mass expansion [21]. Psychiatric disturbances and seizures can occur. Rarely, multiple lesions mimicking multiple sclerosis, spinal cord abnormalities, and peripheral neuropathy may be present. Symptoms of neurosarcoidosis often occur after more common manifestations, such as lungs and lymph nodes involvement [22]. In patients suspected of neurosarcoidosis, cerebrospinal fluid examination, including angiotensin-converting enzyme, cytology, and tumor markers, is indicated [25].

MRI findings can show the absence of normal hyperintense signal of the neurohypophysis on T1-weighted views. The pathologic lesions are
isointense on T₁-weighted images and enhance with gadolinium [3,21,23]. In some patients, the diagnosis is made only by biopsy of the granulomatous lesion [25].

Corticosteroids are the therapy of choice, but various adjuvant immunosuppressants are used [21,23,25].

Wegener’s granulomatosis

Wegener’s granulomatosis (WG) is a multisystemic disorder characterized by systemic vasculitis and necrotizing granulomas in the respiratory tract and the kidney. They also can be present in the orbits, heart, skin, and joints [26,27]. Pituitary involvement is rare and affects predominantly the posterior pituitary as central diabetes insipidus; that usually occurs after pulmonary or kidney lesions [28,29]. Rarely, partial or total anterior pituitary abnormality can be present. The suggested causes of this complication include vasculitis of pituitary blood vessels, granulomatous lesions in situ, or encroachment on the nervous system by adjacent nasal or paranasal granuloma [27]. A combination of respiratory tract involvement, positive antineutrophil cytoplasmic antibodies (ANCA), and increased proteinase 3-ANCA suggests a diagnosis of WG [27].

MRI usually shows an enlarged pituitary gland with homogeneous enhancement and thickening of infundibulum, especially in the superior portion [26].

The majority of patients respond to medical management, such as corticosteroids and immunosuppressors associated with hormonal replacement. Several cases show complete resolution of diabetes insipidus and control of WG. Others patients have demonstrated persistent diabetes insipidus in spite of improvement of peripheral manifestations of the disease and even diminution in hypothalamic granulomatous lesions [27–29].

Sphenoidal sinus mucocele

Mucocele of the sphenoid sinus is a lesion resulting from a chronic obstruction of the sinus that leads to accumulation and dehydration of secretions [30]. Congenital anomaly, trauma, inflammatory conditions, and previous surgery of sphenoid sinus may predispose to mucocele development [31].

Rarely, mucocele can extend to the pituitary fossa, parasellar and suprasellar regions, nasopharynx, orbits, clivus, or ethmoid air cells [31]. Headache frequently is present and can be severe. Visual impairment resulting from direct nerve compression and ocular palsies can occur. Exophthalmos is common, present in approximately half of patients [3]. Hypopituitarism is less common, but hyperprolactinemia is described [32].

Because of its high protein content, the most characteristic mucocele appearance on MRI is a mass with a homogeneously hyperintense T₁ signal [30]. Contrast enhancement usually is absent or there may be a thin peripheral
rim (Fig. 3). Erosion of the walls of the sphenoid bone occasionally may be present [3,30]. Transssphenoidal surgery offers excellent prognosis.

*Langerhans’ cell histiocytosis*

Langerhans’ cell histiocytosis or class I histiocytosis is a rare disease that affects the reticuloendothelial tissue [33] and is characterized by aberrant proliferation of specific dendritic cells, called Langerhans’ cells, belonging to the monocyte-macrophage system; these cells can infiltrate and destroy many sites, such as bone, lung, skin, hypothalamic-pituitary axis, and, less frequently, liver, spleen, and lymph nodes [34]. The pathogenesis still is unclear, but some investigators have demonstrated clonal proliferation, suggesting a neoplastic disorder. Immunohistochemical features, such as S-100 protein, a CD1a antigen, are characteristic of Langerhans’ cells [35]. The incidence is 3 to 4 cases per million per year in children younger than 15 years old and with prevalence 2 times more frequent in men than in women [33]. Only 30% of the cases reported are in adults [34]. Diabetes insipidus is a common symptom and the most prevalent endocrinopathy in these patients, occurring in 10% to 50% of cases; it nearly always occurs concomitant to or after other manifestations of this disease, but it can be the first or an isolated symptom [33,34,36]. In childhood, Langerhans’ cells histiocytosis is the second most common cause of diabetes; this diagnosis needs to be sought actively in childhood-onset diabetes insipidus [37]. Anterior pituitary dysfunction is less frequent [38]. On MRI, pituitary stalk thickening (> 3.5 mm) and the absence of the hyperintense signal of the normal posterior pituitary gland (bright spot) are the most common findings. An isolated hypothalamic lesion can be present [37,38]. Pituitary stalk biopsy is not recommended routinely in lesions smaller than 7 mm because of the risks [37]. The diagnosis may be based on the symptoms, imaging techniques, and surgical biopsy of other involved sites. There are reports

![Fig. 3. Mucocele: sagittal T1-weighted MRI (A) and contrast-enhanced coronal T1-weighted MRI (B) depicting a thin peripheral and regular rim.](image)
of spontaneous resolution of Langerhans’ cell histiocytosis; therefore, simple observation can be reasonable. Radiotherapy may be useful in some cases of diabetes insipidus and to control mass growth. High doses of corticosteroids and chemotherapeutic agents, such as vincristine, vinblastina, etoposide, and cyclosporine, can yield partial response or decrease the progression of disease. Surgery is limited to lesions with rapid progression and compression of neural structures.

Hypophysitis

Primary hypophysitis is an unusual disorder characterized by focal or diffuse inflammatory infiltration and varying degrees of pituitary gland destruction [39]. The natural history still is not understood completely, and the incidence of this disease is unknown. The number of published case reports recently has increased, probably because of the widespread use of MRI and transsphenoidal surgical biopsies [40].

Clinically, primary inflammatory hypophysitis is classified into three types: lymphocytic hypophysitis, granulomatous hypophysitis, and xanthomatous hypophysitis [40].

Lymphocytic hypophysitis, often referred to as autoimmune hypophysitis, is an inflammatory disorder and the most common among the primary cases of hypophysitis (Fig. 4) [40,41]. This type may be subclassified into three types: lymphocytic adenohypophysitis, lymphocytic infundibuloneurohypophysitis, and lymphocytic panhypophysitis. Histologic assessment shows adenohypophyseal and neurohypophyseal infiltration by lymphocytes and fibrosis [40]. In granulomatous hypophysitis, the pituitary presents diffuse collections of multinucleated giant cells, histiocytes, lymphocytes, and plasma cells [42]. As granulomatous hypophysitis and autoimmune hypophysitis can occur together, some investigators suggest that the two disorders represent a spectrum of autoimmune manifestation [40].

Fig. 4. Contrast-enhanced coronal T₁-weighted MRI from a male patient who had lymphocytic hypophysitis (A) shows almost complete shrinkage of mass after high glucocorticoid dose (B).
Xanthomatous hypophysitis is rare. Cystic-like areas of liquefaction, infiltrated by lipid-rich foamy histiocytes and lymphocytes, are present in the pituitary gland. Xanthomatous hypophysitis can be considered an inflammatory response to ruptured cysts components [43]. Necrotizing hypophysitis is a rare entity, having been demonstrated histologically only in two patients. The pituitary is destroyed by diffuse necrosis with lymphocytes, plasma cells, and eosinophile infiltration [40].

Clinical presentation is variable and can be similar to symptoms observed in clinically nonfunctioning pituitary adenoma. Therefore, visual impairment and severe headache due to expanding lesion are the most common and usually the initial complaints [39,40].

Ophthalmologic disturbances include visual field impairment and decreased acuity secondary to compression of the optic chiasm by suprasellar mass expansion. Parasellar expansion is less frequent, leading to paresis or palsy of the III, IV, and VI cranial nerves [42]. Partial or complete hypopituitarism may present initially as corticotropin deficiency, followed by thyrotropin, gonadotropins, growth hormone, and prolactin [40,41]. The primary inflammatory hypophysitis with involvement of posterior pituitary and infundibular stem is present with diabetes insipidus [44]. In contrast to other cases of hypophysitis, lymphocytic adenohypophysitis is strikingly associated with pregnancy, for unknown reasons [41]. Pituitary apoplexy in patients who have lymphocytic hypophysitis is described in the literature [45].

MRI often shows symmetric enlargement of the pituitary gland with homogeneous contrast enhancement. Thickened infundibular stalk and loss of the hyperintense bright spot signal of the posterior pituitary often are seen [39,40,46]. The definitive diagnosis is obtained only by microscopic examination of the pituitary tissue.

High doses of corticosteroids are described to elicit response (see Fig. 4), but when the dose is tapered, the risk for recurrence is high. Transsphenoidal surgery may be indicated in cases of mass effect or corticosteroids therapy failure [39,41]. Successful stereotactic radiotherapy in two resistant cases was published [47].

Infectious lesions

Pituitary abscess

Pituitary abscesses are a rare but potentially life-threatening disease comprising less than 1% of all pituitary diseases and 0.27% of pituitary surgeries. Primary abscesses comprise two thirds of the cases and occur in a previously normal pituitary, whereas secondary abscesses arise in an already compromised pituitary gland (adenoma or Rathke’s cleft cyst). Clinical manifestations are nonspecific and diabetes insipidus appears in half of the patients [48,49].

Fever and leukocytosis are seen in only one third of cases, and predisposing factors, such as sinusitis, cavernous sinus thrombophlebitis, or pituitary
surgery, usually are absent [50,51]. The most common clinical feature is headache, followed by visual complaints and hypopituitarism.

On MRI, a round sellar cystic lesion, hypo- or isointense on T₁ precontrast imaging and hyper- or isointense on T₂ imaging with peripheral gadolinium enhancement, is compatible with pituitary abscess.

Diagnosis usually is made during surgical exploration when pus is found in a cystic lesion. Culture of the abscess material identifies pathogens in only one half of the cases. The most common pathogens are gram-positive bacteria, but gram-negative bacteria, anaerobic bacteria, and fungus also can be found. The source of infection can be hematogenous spread or contiguous infection, although the infection source cannot be identified in all cases. Mortality can reach 30% to 50% of cases when it is complicated by meningitis.

Treatment consists of surgical drainage and antibiotic administration for 2 to 6 weeks. Transsphenoidal approach avoids cerebral contamination and for this reason is the preferred approach. Surgical drainage and antibiotics diminish mortality to less than 10% [52].

Tuberculosis

Intracranial tuberculosis accounted for 30% to 50% of intracranial lesions before the arrival of antibiotic therapy; however, nowadays, it accounts for 0.15% to 4% of the cases. Pituitary tuberculomas are rare. Most patients have other signs of active tuberculosis, but this not always is true. Patients are affected at any age and there is female predominance. Visual loss and headache are the most common symptoms.

MRI shows thickening of pituitary stalk in some cases. Biopsy shows caseating necrosis, and specific antibiotic therapy is mandatory [53].

Other infections

Fungal infections (aspergillosis and coccidiomycosis) [54], cysticercosis [55], and neurosyphilis [56] are rare.

Vascular lesions

Intrasellar aneurysms

Intrasellar aneurysm can mimic pituitary adenoma and imaging techniques are essential for distinguishing between the two disorders before surgery. Intense homogeneous blush on CT images with contrast suggests an aneurysm [3]. On conventional spin-echo MRI, the aneurysm appears black because of flow void, has well-defined margins, and is contiguous to vessel [57].

There are different surgical approaches to the intrasellar aneurysm, such as endovascular coiling.

Pituitary apoplexy

Pituitary apoplexy is a life-threatening condition caused by infarction or hemorrhage into a growing pituitary adenoma [58,59].
Pituitary apoplexy usually occurs in macroadenomas of any histologic subtype [60] and rarely is described in the normal pituitary gland, craniopharyngiomas, or lymphocytic hypophysitis [45,59]. Precipitating factors [60] may include reduced pituitary blood flow (hypotension or hemodialysis, angiography, or spinal anesthesia resulting from decreased intracranial pressure) or radiotherapy resulting from vascular damage. Myocardial infarction, anticoagulants, infection, stimulation of pituitary gland as happens in increased estrogen milieu, dynamic testing using gonadotropin-releasing hormone or thyrotropin-releasing hormone, and the use of bromocriptine all are implicated [58,60]. There is no evidence that hypertension and diabetes are more prevalent in patients who have pituitary apoplexy.

Clinical presentation is variable and sometimes apoplexy is asymptomatic [58]. Headache is the most prevalent complaint (76% to 87%); visual deficits are present in 56% to 62% of cases, ocular palsies in 40% to 45% [45,59], and diabetes insipidus in 8% [60]. Pituitary hypersecretion can be “cured” [61,62].

CT is the most important tool in the acute setting (24–48 hours), after contrast administration showing a high-intensity or heterogeneous gland with or without evidence of subarachnoid hemorrhage [63]. On a recent MRI, pituitary hemorrhage appears hypointense in T1- and T2-weighted images, but after a week, high signal intensity is evident on T1-weighted images [64].

Medical treatment with stress doses of glucocorticoids is mandatory [58]. Patients presenting with visual loss or consciousness impairment must be operated on immediately, with better results achieved when the surgery is done fewer than 7 days after the complaints started. In some cases, patients can be managed conservatively with corticosteroids and close clinical and imaging observation [65].

**Miscellaneous**

**Arachnoid cyst**

A true intrasellar arachnoid cyst is rare. It is believed to arise from an arachnoid herniation into the pituitary fossa as a result of incompetence of the diaphragma sellae after trauma or as a result of an adhesive arachnoiditis secondary to infection. On MRI, signal intensity is similar to cerebrospinal fluid and contrast enhancement of the cyst wall usually is not found [2–4].

**Pathologic pituitary hyperplasia**

Physiologic enlargement of the pituitary may be observed during puberty and pregnancy. Primary hypothyroidism can cause pituitary hyperplasia, reverted with levothyroxine replacement. In patients who have symmetric pituitary enlargement and hypothyroidism, finding of high thyrotropin establishes the diagnosis. Growth hormone–releasing hormone and
corticotropin-releasing hormone neoplasias also are causes of pituitary hyperplasia [3].

**Double pituitary**

Duplication of the pituitary is rare and few cases are reported in the literature. Its pathogenesis is considered to result from notochordal anomaly and can be associated with other malformations, such as absence of corpus callosum and of midline commissures in both cerebral hemispheres, frequently being incompatible with life [66]. Double pituitary may occur, however, as an isolated anomaly and without symptoms, as just an incidental finding (Fig. 5) [67].

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**References**