Diseases of the Sella Turcica and Parasellar Region

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Learning Objectives

- Acquire knowledge of the micro-anatomy of the parasellar region.
- Learn the basic physiology of the hypothalamic–pituitary axis.
- Gain an understanding of the diagnostic features of the most common lesions arising from each structure in this region.
- Become proficient in establishing a concise differential diagnosis.

Key Point

- Knowledge of the anatomy of the pituitary gland, pituitary stalk, cavernous sinuses, suprasellar cistern, optic chiasm, and arteries of the central skull base, and their relationship to one another, is essential for accurate diagnosis.

1.1 Introduction

A large variety of disorders affect the pituitary gland and surrounding region. By far the commonest disorders are pituitary adenomas and Rathke's cleft cysts. Magnetic resonance imaging (MRI) is the imaging modality of choice in the investigation of pituitary pathology. Patients are referred for MR imaging of the pituitary most commonly on the basis of clinical and laboratory findings, although given the frequency of MR imaging of the brain, it is also not uncommon to discover lesions in this region as incidental findings. Computed tomography (CT) is used in occasional cases where intra-lesional calcifications or bone destruction is suspected.

1.2 Pituitary Adenomas

Despite the many advances in MR imaging over the past decade, the imaging work-up of pituitary disease remains largely unchanged. T1-weighted images are obtained in the sagittal and coronal plane as well as a T2-weighted sequence in the coronal (occasionally sagittal). Contrast-enhanced sequences are also done in many, but not all, centers on a routine basis. Many investigators also perform dynamic contrast-enhanced T1-weighted imaging, and occasionally delayed enhanced images, especially if the patient is a surgical candidate and conventional imaging is negative or equivocal.

Pituitary microadenomas commonly exhibit hypointensity to the normal adenohypophysis on T1-weighted images. The lesions are round or oval in shape; more commonly than not, the pituitary infundibulum is deviated away from the side of the lesion. Occasionally, pituitary adenomas may exhibit hyperintensity on T1-weighted images, generally reflecting internal hemorrhage. Such hemorrhage is more common in prolactinomas, oftentimes spontaneous or following pharmacotherapy.

The signal intensity on T2-weighted images of microadenomas is variable, although if focal hyperintensity to the normal gland is seen, a microadenoma is likely. Hypointensity on T2-weighted images is also a helpful feature. In the clinical and laboratory scenario of excess growth hormone, a mass of focal T2 hypointensity is highly correlated with a diagnosis of a densely granulated GH-secreting adenoma.

In many cases of prolactinoma, unenhanced T1- and T2-weighted imaging is normal. If the lesion is not obvious, contrast enhancement is indicated. As the pituitary is a circumventricular organ and not subject to the blood–brain barrier, a half dose of paramagnetic contrast is generally sufficient. Following the administration of contrast, a hypoin-

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tense lesion either surrounded by, or superiorly capped by normal avidly enhancing pituitary tissue will reveal a microadenoma or other lesions in the differential diagnosis. As well, delayed contrast-enhanced imaging may inadvertently obscure microadenomas by virtue of their slower "wash-in" curve, relative to normal pituitary. Uncommonly, a small tuft of intra-lesional enhancement may be seen within an otherwise hypointense (lesser enhancing) microadenoma. Dynamic contrast-enhancing imaging may be useful for some microadenomas, especially ACTH-secreting tumors.

In contradistinction to the microadenoma, pituitary macroadenomas (defined as tumors greater than 10 mm in size) often extend beyond the confines of the sella. Most commonly, such tumors extend cephalad toward the optic chiasm and hypothalamus or laterally into the cavernous sinus. While the delineation of such extension has always been important to the surgeon, with the increasing adoption of transnasal endoscopic surgical approaches to the sella and parasellar regions, the description of such extension has become a routine component of the preoperative neurosurgical checklist.

Macroadenomas that extend cephalad are commonly bilobed in shape, constrained at the waist by the diaphragma sellae. Unlike their microadenoma counterparts which are more commonly of one signal intensity, macroadenomas usually are of multiple signal intensities due to the mixed solid and cystic nature of these lesions. Solid components tend to be slightly hypointense on T2 and T2-weighted images and exhibit some contrast enhancement. Cystic and/or necrotic components exhibit variable signal, but often contain proteinaceous debris which may show some hyperintensity on T1-weighted images. On T2-weighted images, hyperintensity within the cystic or necrotic components of an adenoma is common. Occasionally, an intra-tumoral, intracystic component may reveal a fluid-fluid level, perhaps reflecting intra-lesional hemorrhage.

Usually the posterior lobe hyperintensity on T1-weighted images is positioned slightly behind the adenohypophysis, often within a cup-shaped depression of the dorsum sellae. With a macroadenoma the normal neurohypophysis may be compressed, laterally displaced, or completely absent from the normal location. In many cases compression of the infundibulum is disruptive enough that the neurohypophyseal function is displaced upstream, with a "new" bright spot more cephalad along the infundibulum.

Cavernous sinus involvement may be due to compressive growth and consequent medial and occasionally lateral dural reflection or due to true invasion of the sinus. On imaging, invasion is suggested by tumor appearing to encircle the cavernous internal carotid artery. Invasion is excluded if normal pituitary tissue is seen between the mass and the sinus.

There is a correlation between prolactin levels and adenoma size. However, given two prolactinomas of equal size, the hypointense tumor on T2-weighted images usually secretes more prolactin than its counterpart. Medical treatment based on bromocriptine decreases adenoma volume drastically; occasionally, bromocriptine-induced hemorrhage can be seen.

While prolactinomas and growth hormone (GH)-secreting adenomas are usually located laterally in the sella turcica, ACTH-secreting adenomas in Cushing's disease, usually smaller in size and more difficult to detect, are more often located in the midline. Because of the severe prognosis of this disease and the surgical possibilities, ACTH-secreting lesions require the most detailed and exhaustive imaging.

GH-secreting adenomas have the unique characteristic of exhibiting hypointensity on T2-weighted images in two-thirds of cases, usually the densely granulated subtype. Spontaneous infarction or necrosis of GH-secreting adenomas is far from exceptional. Some cases of acromegaly that were detected late in the course of the disease exhibited an enlarged, partially empty sella turcica, lined with adenomatous tissue that proved difficult to analyze. Medical treatment based on octreotide analogs (somatostatin) decreases the size of the adenoma by an average of 35% and brings the level of somatomedin C back to normal in 50% of cases.

Macroadenomas can be nonfunctioning, but they can also be prolactin-secreting adenomas, gonadotroph adenomas, and growth hormone-secreting adenomas. The greater their size, the more heterogeneous they are, as areas of cystic necrosis are caused by poor tumoral blood supply. Gonadotroph adenomas are often massive and have a strong tendency to recur.

Hemorrhage occurs in all or parts of 20% of all pituitary adenomas, but it is usually occult. Pituitary apoplexy, with severe headache, cranial nerve paralysis, and severe hypopituitarism, is generally caused by massive hemorrhage within a pituitary macroadenoma. Smaller scale hemorrhage occurs much more often and can be seen within pituitary adenomas.

Surgery of the sella and parasellar region is currently usually undertaken via the transsphenoidal and transnasal endoscopic. The transnasal endoscopic approach to the sella has been more widely adopted for a number of reasons. Multiple instruments can be utilized, including angled endoscopes. A transnasal mucosal flap can be created at the time of surgery. This flap has been very successful at limiting complication of CSF leak. And the exposure can be lateralized to extend to parasellar lesions, as well as extended posteriorly through the clivus to reach the prepontine cistern, the basilar artery, and the anterior brainstem.

Preoperative CT is often routinely performed in some centers. CT will reveal bony anatomy including the number and location of intra-sphenoidal septae and their insertions, the presence of Onodi cells, and the degree of aeration of the sphenoid sinus, all of which may influence the surgical approach, patient safety and ultimately, the patient outcome.

1.3 Postoperative Sella Turcica and Pituitary Gland

The surgical cavity immediately after surgery is often filled with packing material after transsphenoidal resection of a pituitary adenoma. Surgicel is frequently used and is impregnated with blood and secretions. The presence of packing material, secretions, and periadenomatous adhesions usually keeps the cavity from collapsing in the days and weeks that follow surgery. Blood, secretions, and packing material slowly involute over the following 2–3 months. Even after a few months, fragments of blood-impregnated Surgicel can still be found in the surgical cavity. If the diaphragm of the sella turcica is torn in the course of surgery, fat or muscle implants are inserted by the surgeon to prevent the occurrence of a cerebrospinal fluid fistula. Their resorption takes much longer. Implanted fat involutes slowly and may exhibit hyperintensity on the T1-weighted image up to 2–3 years after surgery. Postoperative MRI 2–3 months after surgery is useful to monitor further development of a resected adenoma. An earlier MRI examination performed 48 hours after surgery checks for potential complications and may visualize what appears to be residual tumor, i.e., a mass of intensity identical to that of the adenoma before surgery that commonly occupies a peripheral portion of the adenoma. This early investigation is extremely helpful to interpret the follow-up MR images. At this stage, the remaining normal pituitary tissue can be characterized: it is usually asymmetrical, and a hyperintense area is frequently observed at the base of the deviated hypophyseal stalk, due to an ectopic collection of neurohypophyseal secretory vesicles. The 2-month follow-up MRI examination is essential to check for residual tumor. Late follow-up MRI, after 1–2 years or more, usually demonstrates adenoma recurrence as a rounded or convex progressively enlarging mass.

1.4 3T MRI and DWI for Pituitary Imaging

The improved SNR of 3T scanners relative to 1.5T can be traded off for thinner image slices and smaller voxels, thereby offering improved spatial resolution at comparable SNR. Hence, some microadenomas may be detected at 3T that are invisible at 1.5T. Also, the cavernous sinus wall can be depicted more consistently. These facts favor the performance of pituitary exams on 3T MRI. DWI and ADC images have been applied to pituitary imaging as aid to determining tumor consistency and thereby aiding surgical planning. Early evidence suggests that soft adenomas with high cellularity and scant fibrous stroma have low ADCs, whereas firm adenomas, with low cellularity and abundant fibrous stroma, have high ADCs. DWI obtained without echo planar imaging offers improved imaging at the skull base. Such TSE-based DWI is increasingly supplanting echo planar DWI in pituitary imaging.

Key Point

- The most common lesions in the sella turcica are pituitary adenomas and Rathke's cleft cysts. Most of these are visible on non-contrast MRI.

1.5 Craniopharyngioma

Craniopharyngiomas are epithelial-derived neoplasms that occur exclusively in the region of the sella turcica and suprasellar cistern or in the third ventricle. Craniopharyngiomas account for approximately 3% of all intracranial tumors and show no gender predominance. Craniopharyngiomas are hormonally inactive lesions, although compression of the stalk may result in diabetes insipidus. They have a bimodal age distribution; more than half occur in childhood or adolescence, with a peak incidence between 5 and 10 years of age; there is a second smaller peak in adults in the sixth decade. The tumors vary greatly in size, from a few millimeters to several centimeters in diameter. The center of most is in the suprasellar cistern. Infrequently, the lesions are entirely within the sella or in the third ventricle.

Most discussions of craniopharyngiomas in the literature are confined to the most frequent form, the classic *adamantinomatous* type, but a distinct *papillary* type is also recognized. Typically, adamantinomatous craniopharyngiomas are identified during the first two decades of life. These children most often present with symptoms and signs of increased intracranial pressure: headache, nausea, vomiting, and papilledema. Visual disturbances due to compression of the optic apparatus are also frequent but difficult to detect in young children. Others present with pituitary hypofunction because of compression of the pituitary gland, pituitary stalk, or hypothalamus. Rarely, adamantinomatous craniopharyngiomas are found outside the suprasellar cistern, including the posterior fossa, pineal region, third ventricle, and nasal cavity (sphenoid sinus).

Adamantinomatous tumors are almost always grossly cystic and usually have both solid and cystic components. Calcification is seen in the vast majority (~90%) of these tumors. Commonly, these calcifications can be identified on MR scans as low signal nodular excrescences of the wall of the primary lesion. Occasionally, the calcifications are difficult to discern; in these cases, CT will prove helpful. Extensive fibrosis and signs of inflammation are often found with these lesions, particularly when they are recurrent, so that they adhere to adjacent structures, including the vasculature at the base of the brain. Optic tract edema on T2-weighted images is a common associated finding that is not commonly seen with other suprasellar masses. Due to the inflammatory

and fibrotic nature of this lesion, recurrence is common, typically occurring within the first 5 years after surgery.

The most characteristic MRI finding is a suprasellar mass that is heterogeneous and contains a cystic component that is well defined, internally uniform, and hyperintense on both T1- and T2-weighted images. Almost always, an adamantinomatous craniopharyngioma that presents with large cystic components in the middle cranial fossa and elsewhere can be traced back to the suprasellar region, where a more solid, enhancing component of the lesion can be seen. On rare occasions, the cyst is absent and the solid component is completely calcified. These calcified types of tumors can be entirely overlooked on MRI unless close scrutiny is paid to subtle distortion of the normal suprasellar anatomy. Contrast medium administration causes a moderate degree of enhancement of the solid portion of the tumor, which otherwise may be difficult to see.

Papillary craniopharyngiomas are typically found in adult patients. These lesions are solid, without calcification, and may be found within the third ventricle. Although surgery remains the definitive mode of therapy for all craniopharyngiomas, as papillary variants are encapsulated and are readily separable from nearby structures and adjacent brain, they are generally thought to recur much less frequently than the adamantinomatous type.

In distinction from their adamantinomatous counterpart, MRI typically shows papillary craniopharyngiomas as solid lesions. Occasionally, cysts may be seen, although they are unlikely to be dominant cysts as in the adamantinomatous variety. These lesions demonstrate a non-specific signal intensity pattern, without the characteristic hyperintensity on T1-weighted images of the cystic component of adamantinomatous tumors. Like all craniopharyngiomas, papillary lesions typically enhance.

1.6 Rathke's Cleft Cyst

Symptomatic cysts of Rathke's cleft are less frequent than craniopharyngiomas, although asymptomatic Rathke's cysts are a common incidental finding at autopsy. In a recent evaluation of 1000 non-selected autopsy specimens, 113 pituitary glands (11.3%) harbored incidental Rathke's cleft cysts. These cysts are predominantly intrasellar in location. Of incidental Rathke's cysts larger than 2 mm in a large autopsy series, 89% were localized to the center of the gland, whereas the remaining 11% extended to show predominant lateral lesions. In that series, of all incidental pituitary lesions localized to the central part of the gland, 87% were Rathke's cysts. Others may be centered in the suprasellar cistern, usually midline and anterior to the stalk. Rathke's cysts are found in all age groups. They share a common origin with some craniopharyngiomas in that they are thought to originate from remnants of squamous epithelium from Rathke's cleft. The cyst wall is composed of a single cell layer of columnar, cuboidal, or squamous epithelium on a basement membrane. The epithelium is often ciliated and may contain goblet cells. The cyst contents are typically mucoid, less commonly filled with serous fluid or desquamated cellular debris. Calcification in the cyst wall is rare.

Most Rathke's cleft cysts are small and asymptomatic, incidentally discovered at autopsy. Symptoms occur if the cyst enlarges sufficiently to compress the pituitary gland or optic chiasm and rarely, secondary to hemorrhage. The cysts with mucoid fluid are hyperintense on T1- and T2-weighted images. Those with serous cysts match the signal intensity of cerebrospinal fluid (CSF). Those containing cellular debris pose the greatest difficulty in differential diagnosis for they resemble solid nodules. Rathke's cleft cysts do not typically enhance. However, occasionally there may be thin marginal enhancement of the cyst wall. This feature can be used to advantage in difficult cases to separate these cysts from craniopharyngiomas.

1.7 Meningioma

Approximately 10% of meningiomas occur in the parasellar region. These tumors arise from a variety of locations around the sella including the tuberculum sellae, clinoid processes, medial sphenoid wing, and cavernous sinus. Meningiomas are usually slow-growing lesions that present because of compression of vital structures. Patients may suffer visual loss because of ophthalmoplegia due to cranial nerve involvement, proptosis due to venous congestion at the orbital apex, or compression of the optic nerves, chiasm, or optic tracts.

Meningiomas are most frequently isointense—and less commonly hypointense—to gray matter on unenhanced T1-weighted sequences. Approximately 50% remain isointense on the T2-weighted sequence, whereas 40% are hyperintense. Since there is little image contrast to distinguish meningiomas from brain parenchyma, indirect signs such as a mass effect, thickening of the dura, buckling of adjacent white matter, white matter edema, and hyperostosis are important diagnostic features. Other diagnostic signs include visualization of a cleft of CSF separating the tumor from the brain (thus denoting that the tumor has an extra-axial location) and a clear separation of the tumor from the pituitary gland (thus indicating that the tumor is not of pituitary gland origin). The latter sign is particularly well assessed on sagittal views of planum sphenoidale meningiomas. A peripheral black rim occasionally noted at the edges of these meningiomas is thought to be related to surrounding veins. Hyperostosis and calcification are features that may be apparent on MRI but are better assessed with CT. Vascular encasement is not uncommon, particularly with meningio-

mas in the cavernous sinus. The pattern of encasement is of diagnostic value. Meningiomas commonly constrict the lumen of the encased vessel. This is rare with other tumors. As on CT, the intravenous administration of contrast medium markedly improves the visualization of basal meningiomas. They enhance intensely and homogeneously, often with a trailing edge of thick surrounding dura (the "dural tail sign").

1.8 Chiasmatic and Hypothalamic Gliomas

The distinction between chiasmatic and hypothalamic gliomas often depends on the predominant position of the lesion. In many cases, the origin of large gliomas cannot be definitively determined, as the hypothalamus and chiasm are inseparable; therefore, hypothalamic and chiasmatic gliomas are discussed as a single entity. The vast majority (75%) of these tumors occur in the first decade of life, with equal prevalence in males and females. There is a definite association of optic nerve and chiasmatic gliomas with neurofibromatosis, more so for tumors that arise from the beginning of optic nerve rather than from the chiasm or hypothalamus.

Tumors of chiasmal origin are also more aggressive than those originating from the optic nerves and tend to invade the hypothalamus and floor of the third ventricle and cause hydrocephalus. Patients suffer from monocular or binocular visual disturbances, hydrocephalus, or hypothalamic dysfunction. The appearance of the tumor depends on its position and direction of growth. It can be confined to the chiasm or the hypothalamus; however, because of its slow growth, the tumor usually attains a considerable size by the time of presentation, and the site of origin is frequently conjectural. Smaller nerve and chiasmal tumors are visually distinct from the hypothalamus, and their site of origin is more clear-cut. From the point of view of differential diagnosis, these smaller tumors can be difficult to distinguish from optic neuritis, which can also cause optic nerve enlargement. The clinical history is important in these cases (neuritis is painful, tumor is not) and, if necessary, interval follow-up of neuritis will demonstrate resolution of optic nerve swelling.

On T1-weighted images, the tumors are most often isointense, while on T2-weighted images they are moderately hyperintense. Calcification and hemorrhage are not features of these gliomas but cysts are seen, particularly in the larger hypothalamic tumors. Contrast enhancement occurs in about half of all cases. Because of the tumor's known propensity to invade the brain along the optic radiations, T2-weighted images of the entire brain are necessary. This pattern of tumor extension is readily evident as hyperintensity on the T2-weighted image; however, patients with neurofibromatosis (NF) present a problem in differential diagnosis. This relates to a high incidence of benign cerebral hamartomas and atypical glial cell rests in NF that can exactly mimic glioma. These both appear as areas of high signal intensity on T2-weighted images within the optic radiations. Lack of interval growth and possibly the absence of contrast enhancement are more supportive of a diagnosis of hamartoma, while enhancement suggests glioma.

1.9 Metastases

Symptomatic metastases to the pituitary gland are found in 1–5% of cancer patients. These are primarily patients with advanced, disseminated malignancy, particularly breast and bronchogenic carcinoma. The vast majority will succumb to their underlying disease before becoming symptomatic of pituitary disease. Autopsy series have demonstrated a much higher incidence, but these by and large are small and asymptomatic lesions. Intrasellar and juxtasellar metastases arise via hematogenous seeding to the pituitary gland and stalk, by CSF seeding, and by direct extension from head and neck neoplasms. There are no distinctive MRI characteristics of metastases, although infundibular involvement is common, and bone destruction is a prominent feature of lesions that involve skull base. Occasionally, leptomeningeal enhancement of posterior fossa sulci may be visualized on the postcontrast images which lends credence to the diagnosis in those cases of CSF tumor seeding, although this finding also invokes the differential diagnosis of sarcoidosis and tuberculosis.

1.10 Infections

Infection in the suprasellar cistern and cavernous sinuses is usually part of a disseminated process, or occurs by means of intracranial extension of an extracranial infection. The basal meninges in and around the suprasellar cistern are susceptible to tuberculous and other forms of granulomatous meningitis. The cistern may also be the site of parasitic cysts, in particular (racemose and subarachnoid) neurocysticercosis. In infections of the cavernous sinus, many of which are accompanied by thrombophlebitis, the imaging findings on CT and MRI consist of a convex lateral contour to the affected cavernous sinus with evidence of a filling defect after contrast administration. The intracavernous portion of the internal carotid artery may also be narrowed secondary to surrounding inflammatory change.

Infections of the pituitary gland itself are uncommon. Direct viral infection of the hypophysis has never been established and bacterial infections are unusual. There has been speculation that cases of acquired diabetes insipidus may be the result of a select viral infection of the hypothalamic supra-optic and paraventricular nuclei. Tuberculosis and

syphilis, previously encountered in this region because of the higher general prevalence of these diseases in the population, are now uncommon. Gram-positive cocci are the most frequently identified organisms in pituitary abscesses. Pituitary abscesses usually occur in the presence of other sellar masses such as pituitary adenomas, Rathke's cleft cysts, and craniopharyngiomas, indicating that these mass lesions function as predisposing factors to infection.

There are a few reports on CT of pituitary abscesses. These indicate that the lesion is similar in appearance to an adenoma. As a result of the frequent coincidental occurrence of abscesses with adenomas, and because of their common clinical presentations, the correct preoperative diagnosis of abscess is difficult and rarely made. Non-contrast MRI demonstrates a sellar mass indistinguishable from an adenoma. With intravenous administration of contrast medium, there is rim enhancement of the mass with persistence of low intensity in the center. Occasionally, pituitary abscesses are unrelated to primary pituitary lesions. In these cases, erosion of the bony sella from an aggressive sphenoid sinusitis may be the route of infection.

1.11 Noninfectious Inflammatory Lesions

Lymphocytic hypophysitis is a rare, noninfectious inflammatory disorder of the pituitary gland. It occurs almost exclusively in women and particularly during late pregnancy or in the post-partum period. The diagnosis should be considered in a peripartum patient with a pituitary mass, particularly when the degree of hypopituitarism is greater than that expected from the size of the mass. It is believed that, if untreated, the disease results in panhypopituitarism. Clinically, the patient complains of headache, visual loss, failure to resume menses, inability to lactate, or some combination thereof. Pituitary hormone levels are depressed. CT and MRI demonstrate diffuse enlargement of the anterior lobe without evidence of any focal abnormality or change in internal characteristics of the gland. The distinction between simple pituitary hyperplasia and lymphocytic hypophysitis may be difficult on MRI alone.

Sarcoid afflicting the hypothalamic–pituitary axis usually manifests itself clinically as diabetes insipidus, or occasionally as a deficiency of one or more anterior lobe hormones. Low signal intensity on T2-weighted images is one finding that occurs in sarcoid with some frequency, but rarely in other diseases, with few exceptions (other granulomatous inflammatory diseases, lymphoma, some meningiomas). This low signal finding may aid in differential diagnosis. Also, the presence of multiple, scattered intra-parenchymal brain lesions should raise the possibility of the diagnosis, as should diffuse or multifocal lesions of the basal meninges. The latter are best defined on coronal contrast-enhanced T1-weighted images.

Tolosa–Hunt syndrome (THS) refers to a painful ophthalmoplegia caused by an inflammatory lesion of the cavernous sinus that is responsive to steroid therapy. Pathologically, the process is similar to orbital pseudotumor. Imaging in this disorder is often normal, or may show subtle findings such as asymmetric enlargement of the cavernous sinus, enhancement of the prepontine cistern, or abnormal soft tissue density in the orbital apex. The lesion resolves promptly with steroid therapy. Hypointensity on T2-weighted images may be observed; since this observation is uncommon in all but a few other diseases (e.g., meningioma, lymphoma, and sarcoid), it may be helpful in diagnosis. Clinical history allows further precision in differential diagnosis: meningioma does not respond to steroids, while lymphoma and sarcoid have evidence of disease elsewhere in almost all cases.

1.12 Vascular Lesions

Saccular aneurysms in the sella turcica and parasellar area arise from either the cavernous sinus portion of the carotid artery or its supra-clinoid segment. These are extremely important lesions to identify correctly. Confusion with a solid tumor can lead to surgical catastrophes. Fortunately, their MRI appearance is distinctive and easily appreciated. Aneurysms are well defined and lack any internal signal on spin echo (SE) images, the so-called signal void created by rapidly flowing blood. This blood flow may also cause substantial artifacts on the image, usually manifest as multiple ghosts in the phase-encoding direction, and in itself is a useful diagnostic sign.

Thrombus in the aneurysm lumen fundamentally alters these characteristics, the clot usually appearing as multilamellated high signal on T1-weighted SE images, partially or completely filling the lumen. Hemosiderin from superficial siderosis may be visible in the adjacent brain, evident as a rim of low signal intensity on T2-weighted SE images, or on gradient echo (GE) or susceptibility-weighted images. If confusion exists as to the vascular nature of these lesions, MR or CT angiography is used to confirm the diagnosis, define the neck of the aneurysm, and establish the relationship of the aneurysm to the major vessels.

Carotid cavernous fistulas are abnormal communications between the carotid artery and cavernous sinus. Most cases are due to trauma; less frequently they are "spontaneous." These spontaneous cases are due to a variety of abnormalities, including atherosclerotic degeneration of the arterial wall, congenital defects in the media, or rupture of an internal carotid aneurysm within the cavernous sinus. Dural arteriovenous malformations (AVMs) of the cavernous sinus are another form of abnormal arteriovenous (AV) communication in this region.

On MRI, the dilatation of the venous structures, in particular the ophthalmic vein and cavernous sinus, is usually clearly vis-

ible. The intercavernous venous channels dilate in both direct and indirect carotid cavernous fistulae and may also be seen on MR images. Furthermore, the internal character of the cavernous sinus is altered; definite flow channels become evident secondary to the arterial rates of flow within the sinus. The fistulous communication itself is most often occult on MRI. The pituitary gland has been noted to be prominent in cases of dural arteriovenous fistula without evidence of endocrine dysfunction. The exact mechanism of pituitary enlargement is not known; however, venous congestion is a postulated cause.

Cavernous hemangiomas are acquired lesions and not true malformations. However, there have been reports of extra-axial cavernous hemangiomas occurring in the suprasellar cistern. Of importance is that one of these hemangiomas did not have the features usually associated with, and so highly characteristic of, cavernous hemangiomas in the brain. The atypical appearance of extra-axial cavernous hemangiomas indicates that some caution must be exercised in the differential diagnosis of parasellar masses, because even though cavernous hemangiomas in this location are rare, failure of the surgeon to appreciate their vascular nature can lead to unanticipated hemorrhage. Cavernous hemangiomas should at least be considered in the differential diagnosis of solid, suprasellar masses that do not have the classic features of more common lesions, in particular craniopharyngiomas or meningiomas. Furthermore, T2-weighted images should be a routine part of the MRI protocol for suprasellar masses because visualization of a peripheral dark rim may be the only sign of the nature of the lesion.

Other vascular abnormalities of the sella include unilateral tortuous or bilateral "kissing" internal carotid arteries, and medial trigeminal artery. While the former are relatively straightforward on imaging, the medial trigeminal artery is worth remembering. Much like with the intrasellar aneurysm, with the medial trigeminal artery, neurosurgical catastrophes can occur if the presence of an intrasellar artery is not identified. This artery will arise from the medial aspect of the cavernous carotid artery and will course directly posteriorly through the gland and through the dorsum sellae to reach the basilar artery. Approximately 40% of trigeminal arteries arise medially. In addition, patients with trigeminal arteries are at increased risk of associated intracranial aneurysm. Finally, congenital absence of the internal carotid artery and asymmetric pneumatization of the sphenoid and sella can pose confusing images.

Key Point

- Carotid artery aneurysms and anomalies can mimic intrasellar and parasellar mass lesions. Arterial lesions must be considered in the differential diagnosis of lesions in this area. MRA and/or CTA can be used to confidently confirm or exclude the presence of arterial lesions.

1.13 Other Conditions

Many other lesions may involve the sella turcica and parasellar region. These include mass lesions such as germinoma, epidermoid, dermoid, teratoma, schwannoma, chordoma, ecchordosis, choristoma, arachnoid cyst, hamartoma, IgG4-related disease, and Langerhans cell histiocytosis. Also, there are several important metabolic conditions that may cause pituitary dysfunction or MRI-observable abnormalities in and around the sella. These include diabetes insipidus, growth hormone deficiency, hemochromatosis, hypermagnesemia, and hypothyroidism. Space limitations preclude their further discussion in this synopsis.

1.14 Conclusion

There are nine or ten important structures in and around the sella turcica. There are only 3–4 types of pathology that arise from each of these structures, except in rare circumstances. Knowledge of these structures and their relationship to one another, and familiarity with these pathologies results in high probability of correct diagnosis.

Take Home Messages

- The vast majority of lesions arising in the sella turcica are pituitary adenomas or Rathke's cleft cysts.
- The most important lesions to not misdiagnose are aneurysms and arterial anomalies.
- Anatomic relationships are key to diagnosis.
- Knowledge of basic physiology helps with diagnosis.