Current Pain and Headache Reports (2019) 23: 7
https://doi.org/10.1007/s11916-019-0740-x

NEUROIMAGING (L. MECHTLER, SECTION EDITOR)

Sellar and Parasellar Pain Syndromes

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Published online: 2 February 2019

Abstract

Purpose of Review Sellar and parasellar lesions are numerous and varying in terms of their patholphysiology and physical and radiographic characteristics but often incite pain syndromes that are similar in semiology. The goal of this review was to familiarize the reader with a variety of sellar and parasellar lesions grouped together based on common clinical symptomatology, with a focus on important imaging characteristics that are often distinguishing features diagnostically.

Recent Findings In most cases, tissue acquisition via surgical resection or stereotactic biopsy are the mainstay for definitive diagnosis of sellar and parasellar lesions. With advances in MRI technology in particular in terms of resolution and the inclusion of new techniques including dynamic imaging with delayed contrast, imaging studies of lesions in the sellar and parasellar regions have become increasingly important for diagnostic purposes, with pituitary adenomas and schwannomas as prime examples. In the case of chordoid gliomas, molecular features of the tumor also help distinguish it from other disease processes similar in presentation, which have dramatic impacts on management. Advances in surgical approaches and radiation techniques offer more precise and targeted therapy to lesions in an area with increased risk of clinical morbidity given the high concentration of critically important structures that must be spared during treatment.

Summary Sellar and parasellar lesions have the potential to cause significant morbidity and mortality, highlighting the importance of clinical recognition of warning signs/symptoms, obtaining high-quality imaging studies in various modalities for diagnostic purposes, and prompt management which often involves a multimodal approach that includes surgical resection, radiation, and/or medical therapy. Future advanced imaging techniques will only improve presurgical diagnostic accuracy and lead to more prompt and efficient management.

Keywords Sellar and parasellar lesions · Sellar and parasellar imaging · Intracranial pressure headaches · Chemical meningitis

Introduction

The pituitary gland is nestled within sella turcica and flanked by parasellar soft tissue structures, intricate bony architecture, and complex dural attachments and invaginations. This area is prone to various pathologies that can cause a constellation of signs and symptoms, the most common of which are headaches of various etiologies. In this article, after a brief review of pituitary anatomy and basic imaging tenets of the sellar and parasellar regions, various pathological lesions will be discussed with respect to clinical symptoms, with an emphasis on headache and related pain symptoms. Representative images will be included highlighting characteristic radiographic features of diagnostic value.

Pituitary Anatomy

The pituitary gland resides within the sella turcica, suspended in place by the infundibulum. It consists of the anterior pituitary or adenohypophysis derived from Rathke's pouch, and the posterior pituitary or neurohypophysis, which originates from neural ectoderm. The adenohypophysis consists of five subtypes of secretory cells that release growth hormone, follicle-stimulating/luteinizing hormones, adrenocortical-stimulating hormone, thyroid-stimulating hormone, and prolactin hormone, respectively. These cells are stimulated by the

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hypothalamus, which drives pituitary signaling of target organs. The neurohypophysis includes the infundibulum and the pars nervosa, a collection of hypothalamic axon terminals that store oxytocin and vasopressin, hormones which are released directly into the hypophyseal portal system. The median eminence, a bulge along the inferior hypothalamus, is an area devoid of blood–brain barrier allowing for entry of hypothalamic hormones into the circulation. A potential space known as Rathke's cleft lies between the adenohypophysis and neurohypophysis, along with a collection of cells along the border of these two structures known as the pars intermedia, involved in the production of melatonin. The anatomic composition of the sellar and parasellar regions is shown in Fig. 1.

Pituitary Imaging

High-resolution MRI is best suited to visualize soft tissues and CSF within the sellar and parasellar regions. T1-weighted imaging is often best suited for anatomical visualization; on this sequence, the adenohypophysis is isointense relative to gray matter, while the neurohypophysis is normally hyperintense and is often referred to as the pituitary "bright spot." Coronal views are informative when evaluating normal structures within sellar and parasellar regions, along with regional pathology. As per above, given the absence of the blood–brain barrier at the median eminence, this structure and the adjacent neurohypophysis normally take up contrast. While MRI provides more detail, plain film radiography and CT are used to visualize normal bony architecture and areas of calcification and other structural aberrations in the context of pathology including bone remodeling, erosion, invasion, and/or destruction.

Increased Intracranial Pressure Headaches

Large midline mass lesions in the suprasellar space can obstruct the foramen of Monro on one or both sides and cause a noncommunicating or obstructive hydrocephalus from the resultant elevated intracranial pressure. Headaches are a hallmark symptom of an obstructive hydrocephalus; they are often holocephalic but frontally predominant and may be exacerbated by leaning forward or lying supine. They are often most severe in the morning, and patients will often be woken from sleep with increasing pressure. In the cases of sellar lesions, concomitant symptoms often include neck pain, nausea/vomiting, and double vision in addition to bitemporal visual field deficits with associated compression of the optic chiasm. Sellar lesions with lateral extension or parasellar-based lesions can also precipitate headaches particularly with extension into the cavernous sinus laterally, causing supraorbital and/or retro-orbital headaches most commonly. Generally speaking, in cases of severe hydrocephalus causing headaches and altered mental status, management initially often involves placement of an external ventricular drain to drain CSF and relieve pressure in a controlled fashion so as to prevent brain tissue from coming into contact with the drain and obstruct flow. Definitive management involves surgical resection of lesions in the sellar/parasellar regions.

Pituitary Adenomas

Pituitary adenomas are sellar masses classified by size and function. Macroadenomas are greater than 1 cm in size and tend to be nonfunctional, meaning they do not secrete excess hormones from the anterior pituitary. They are typically hyperdense on CT imaging and isointense with gray matter on T1-weighted sequences and do not contrast enhance. They

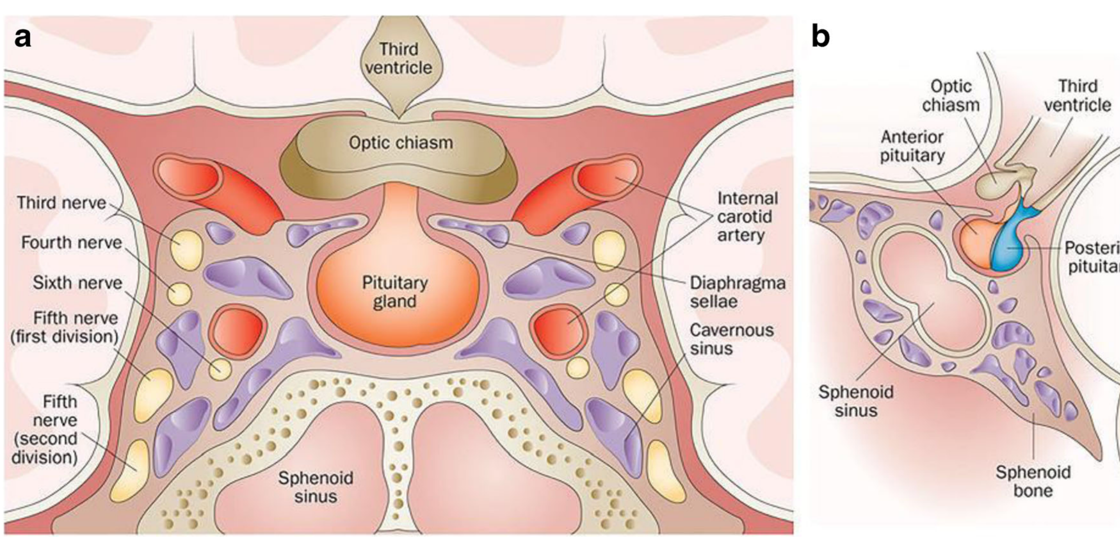

Fig. 1 The anatomic composition of the sellar and parasellar regions

often will extend in to the suprasellar space taking on an hourglass shape when visualized in the coronal and sagittal planes, and cause unilateral or bilateral obstructive hydrocephalus with resultant headaches and nausea. They also can cause oculomotor nerve compression and encasement of the internal carotid artery with lateral extension into the cavernous sinus, as well as compression of the optic chiasm anteriorly. Functional adenomas actively secrete specific hormones produced in the anterior pituitary; these tend to be microadenomas predominantly, which by definition are less than 1 cm in size, are often hyperintense on T2-weighted sequences and sometimes enhance on T1-weighted sequences with IV contrast as demonstrated on dynamic imaging with delayed contrast uptake [1•] as seen in Fig. 2. Management typically involves trans-sphenoidal resection when able. For unresectable or partially resected macroadenomas, either high-dose stereotactic or conventional fractionated radiation therapy is also a consideration [2]. In the case of functionally active large and/or unresectable prolactinomas pretreatment with cabergoline, a long-acting dopamine receptor agonist that exerts a measure of inhibitory control through the release of dopamine, can result in prolonged and sustained tumor shrinkage [3]. There has been some evidence that cabergoline can also precipitate tumor shrinkage in adenomas deemed nonfunctional by hormonal testing [4].

Craniopharyngiomas

Craniopharyngiomas are benign suprasellar tumors that originate from a remnant of the anterior wall of Rathke's pouch known as the pars tuberalis, which encircles the infundibulum of the pituitary. They can also cause obstructive hydrocephalus and headaches related to elevated intracranial pressure, along with a bitemporal hemianopsia due to anterior–superior compression of the optic chiasm. Craniopharyngiomas have unique imaging features, comprised of both solid and cystic components with fluid high in protein and lipids, and calcifications along the cystic rim visualized on a noncontrast head CT. Cystic fluid is often hyperintense on T1- and T2-weighted MRI sequences, while the solid component and cystic rim typically enhance with contrast. A representative MR image is shown in Fig. 3. Management includes surgical resection, often via endoscopic endonasal approach [5], followed by radiation therapy for residual disease. These lesions can recur/reaccumulate, in which case aspiration of the fluid contents to relieve pressure and radiation if initially deferred due to presumed gross total resection are considerations. Fluid contents have been known to leak and cause chemical meningitis [6], which is discussed in more detail later in this article.

Meningiomas and Schwannomas

Meningiomas develop in the subdural space from arachnoid cap cells and can arise from the sellar or parasellar dura in addition to the dura within the cavernous sinus itself, while schwannomas originate from Schwann cells of cranial nerves III, IV, V (1), V (2), or VI, all of which traverse through the cavernous sinus. Resultant headaches from these lesions are most commonly associated with either growth of cavernous sinus-based schwannomas or meningiomas or lateral extension of sellar/parasellar meningiomas into the cavernous sinus, which can result in increased intracranial pressure. The headaches tend to localize to the affected side and are often supraorbital or retro orbital in nature initially. Both meningiomas and schwannomas can contain calcifications in up to 20% of cases [7] which can be visualized on a noncontrast head CT scan, in addition to bony erosions and/or hyperostosis along the adjacent skull. Meningiomas and schwannomas are often isointense or hypointense on MRI imaging, sometimes with the classic dural tail best visualized in sphenoid wing meningiomas along the skull base as seen in Fig. 4. Both enhance with contrast homogeneously,

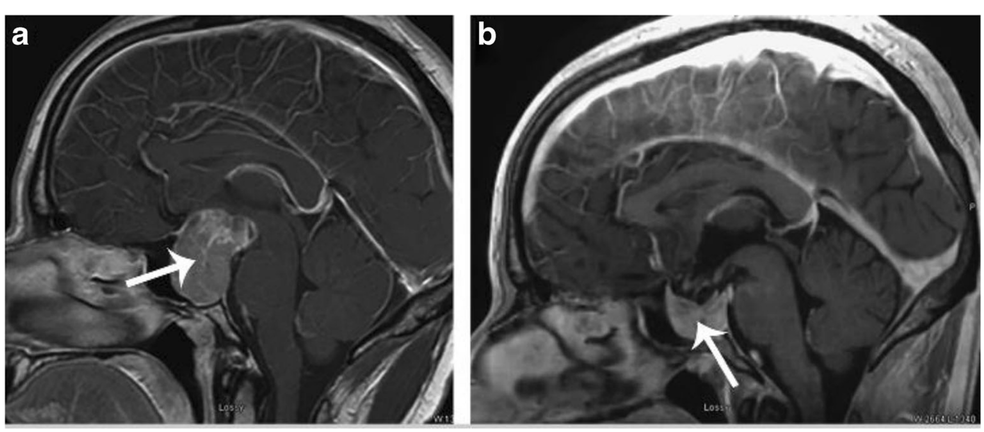

Fig. 2 **a** Sagittal T1-weighted postcontrast brain MRI demonstrating a homogenously enhancing pituitary mass (arrow). **b** The same mass, a pituitary adenoma, is seen postoperatively (arrow) in the T1-weighted MRI with contrast

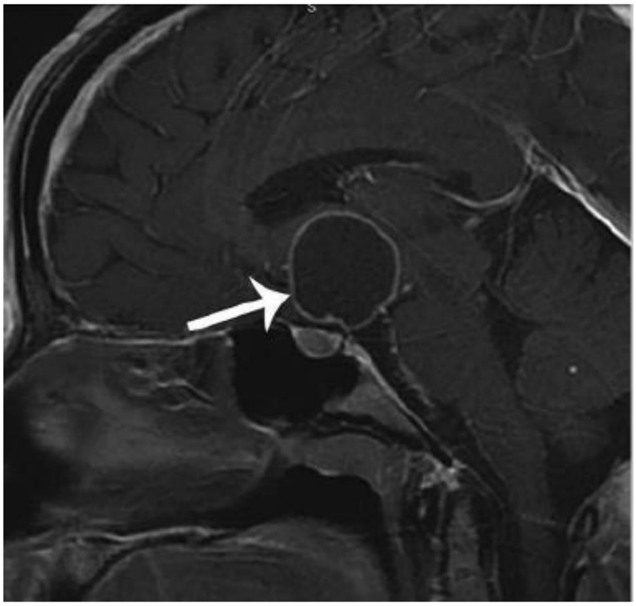

Fig. 3 Sagittal T1-weighted image with contrast shows a cystic lesion with a faint rim of enhancement (arrow) along with a solid component inferiorly, consistent with a craniopharyngioma

although schwannomas tend to show more delayed contrast enhancement often visible on dynamic contrast MRI [8]. More aggressive atypical and anaplastic variants often demonstrating rim enhancement and vasogenic edema. Management is typically surgical resection when able, which is definitive in the case of benign lesions if a gross-total resection can be achieved; for surgically inaccessible lesions, radiation therapy is the mainstay. For atypical and malignant meningiomas, adjuvant radiation therapy is usually indicated even in settings of a complete resection given increased risk of recurrence.

Primary Germ Cell Tumors

Primary germ cell tumors include both germinomas and nongerminomatous germ cell tumors (NGGCTs). NGGCTs are midline lesions that tend to grow more rapidly, while germ cell tumors tend to grow more slowly. Both can occupy the suprasellar cistern, causing an obstructive hydrocephalus and associated headache. These tumors are typically hyperdense on CT and mildly hyperintense on T2-weighted imaging on MRI, while on T1-weighted imaging, they are isointense but do enhance homogeneously with IV contrast. Given both chemo and radiosensitivity of both germinomas and NGGCTs, whole ventricular radiation with boost to the primary tumor bed is the mainstay of treatment, with preceding neoadjuvant chemotherapy in the case of all NGGCTs and select pure germinomas [9].

Mesenchymal Tumors

Mesenchymal tumors evolve from either connective or vascular/lymphatic tissues. The most common tumors include chondrosarcomas, chordomas, and plasmacytomas. Chondrosarcomas are cartilage-based malignant tumors that usually paramedian in origin, arising most commonly petro-occipital fissure. They can cause headaches in the context of elevated intracranial pressure given their expansile nature, and regional pain with dural/bony invasion. There have been reported cases of more midline chondrosarcomas occupying the sellar/parasellar regions mimicking a pituitary macroadenoma and causing ventricular obstruction/hydrocephalus in addition [10]. Unlike benign cystic lesions with calcifications in the periphery, over 50% of chondrosarcomas have central

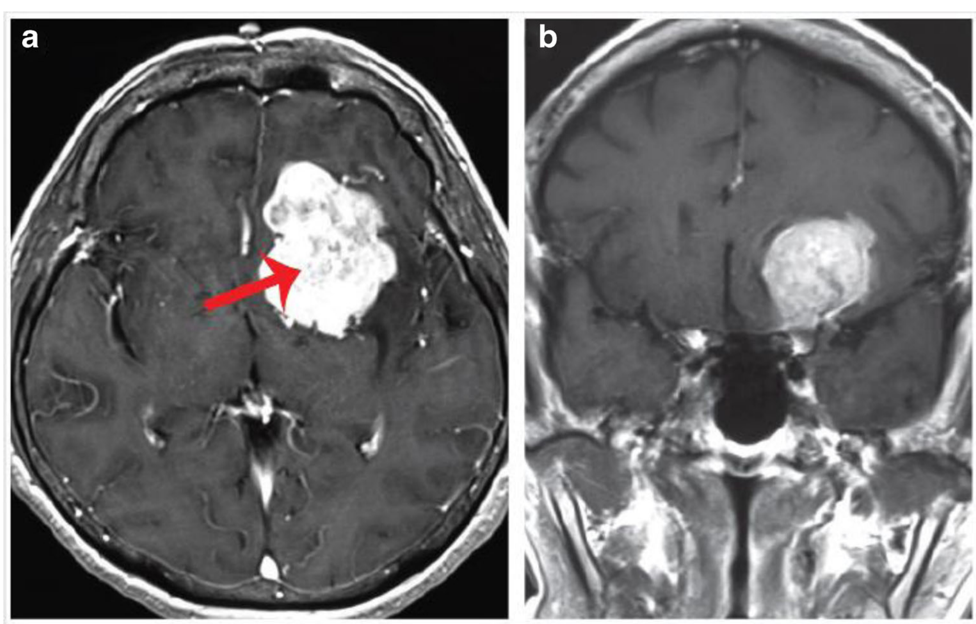

Fig. 4 Axial T1-weighted contrast MRI demonstrates a meningioma arising from the medial portion of the greater sphenoid wing, seen axially (**a**) with extension to parasellar region and middle cranial fossa (arrow) best visualized coronally (**b**)

calcifications that are best visualized on CT [11]. T2-weighted MRI sequences often demonstrate a chondroid matrix, with heterogeneous contrast enhancement noted on T1-weighted sequences with contrast.

Chordomas arise from remnants of the primitive notochord and occur along the midline, unlike paramedian chondrosarcomas. They too are aggressive and expansile, often causing bony destruction of the skullbase [12]. They usually arise from the clivus, and as such can cause a deeply rooted headache and double vision in the context of a regional abducens palsy, and ultimately can precipitate an obstructive hydrocephalus with compression of the aqueduct of Sylvius. They often enhance intensely with contrast, and on T2-weighted MR sequences chordomas are hyperintense, and when visualized in the sagittal plane, they sometimes take on the shape of a thumb print, commonly referred to as the "thumb sign" (Fig. 5).

Plasmacytomas are uncommon intracranial tumors that often originate from the clivus with sellar extension [13] and occupation of the neighboring cavernous and/or sphenoid sinus with ongoing growth, sometimes resembling nonfunctional macroadenomas in this regard. They are comprised of abnormal plasma cells with a high proportion of light chain immunoglobulins. Headaches are commonly related to obstruction of CSF flow and resultant hydrocephalus/elevated intracranial pressure. They typically homogenously enhance on T1-weighted images with contrast.

Mesencymal tumors are typically resected either open (transcranial/transfacial) or using endoscopic endonasal approach, which is often the modality of choice for chordoma resection in particular as per above. Adjuvant radiation therapy is advised after resection of chordomas given their aggressive nature, and most craniopharygiomas with the exception of low-grade tumors that have been completely resected. The detection of an intracranial plasmacytoma necessitates assessment of serum and urine electrophoresis and bone marrow biopsy for workup of multiple myeloma, which is commonly associated with CNS plasmacytomas although isolated brain lesions in the absence of systemic disease have been described [14].

Astrocytomas

Hypothalamic gliomas are rare tumors histologically consistent with juvenile pilocytic astrocytomas, often occurring in patients with NF1, more common in children than adults. They can obstruct CSF flow within the third ventricle with significant upward extension, causing hydrocephalus and elevated intracranial pressure headaches. They appear hypointense on T1-weighted images and hyperintense on T2-weighted images, and contain both cystic and contrast-enhancing solid components.

Pituicytomas are infiltrative and often expansile glial tumors arising from neurohypophysis, appearing heterogeneously hyperintense on T2-weighted images. A hallmark finding of a bright spot representing the displaced neurohypophysis is sometimes seen on T1-weighted imaging [15] as seen in Fig. 6. While they are often asymptomatic, or more commonly associated with diabetes insipidus with compression of the neurohypophysis, given their location they can

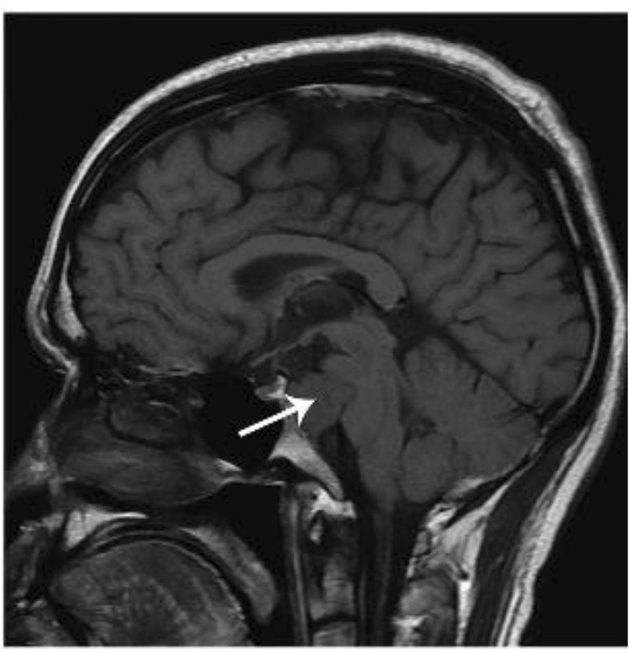

Fig. 5 Clival chordoma with characteristic thumb sign, whereby the mass compresses the brainstem posteriorly on sagittal T1-weighted imaging

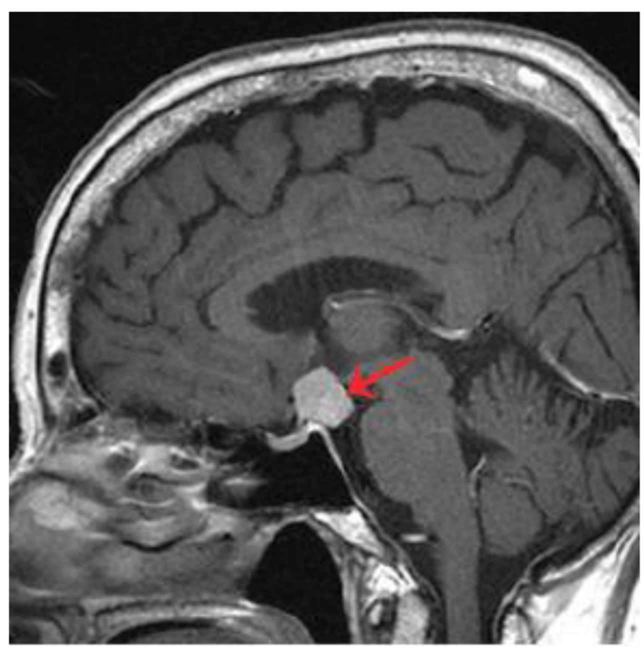

Fig. 6 Pituicytoma occupying the sella as seen on a contrast T1-weighted MRI (arrow), with a characteristic bright spot in the superior portion of the mass, the compressed and deviated neurohypophysis

grow superiorly and precipitate an obstructive hydrocephalus, leading to headache as a presenting feature.

Chordoid gliomas originating in the third ventricle and/or sellar region can also precipitate obstructive hydrocephalus. They appear isointense on T1-weighted MRI with strong uniform contrast enhancement. Treatment for these lesions is usually a combination of surgical resection and adjuvant stereotactic radiation therapy particularly in the context of residual disease [16]; newer research looking at genomic sequencing of these tumors suggests the potential for targeted therapy via MEK inhibition in addition [17•].

Parasellar arteriovenous fistulas cause retrograde high-flow venous shunting of blood between the internal carotid artery and ipsilateral cavernous sinus, causing orbital venous congestion and resultant proptosis, orbital edema, cranial nerve palsies, and elevated intracranial pressure. CT angiography (CTA) often demonstrates subarachnoid vein enlargement and increased tortuosity (Fig. 7). Retrograde venous drainage/edema are seen on MRI in patients with carotid-cavernous fistulas in particular [18]. Cerebral angiography remains the gold standard for evaluation of arteriovenous malformations. Definitive management usually involves embolization or microsurgical fistula resection/repair.

Lymphocytic hypophysitis often occurs in the peripartum setting of pregnancy, thought to be a paraneoplastic phenomenon with abnormal immune targeting of pituitary gland, and has been associated more recently with checkpoint blockade therapy, often at least partially resolving with cessation of therapy [19]. It results in infiltration of the pituitary gland by lymphocytes, plasma cells, and macrophages. Patients can often present with severe, diffuse intractable headaches due to elevated intracranial pressure, and sometimes periorbital pain with extension into the cavernous sinus. Imaging findings are often subtle with thickening of the pituitary stalk best visualized on MR imaging [20] as seen in Fig. 8. Treatment is limited to high-dose glucocorticoid therapy to limit mass effect in addition to removal of the offending agent in iatrogenic cases.

Meningitis

Meningitis is inflammation of the meninges, which can cause skull-base headaches, nuchal rigidity, nausea/vomiting, fevers, and altered mental status. Aseptic meningitis refers to nonbacterial causes, including iatrogenic causes, the most common of which is drug induced. In the case of cystic and/or vascular lesions in the sellar and parasellar regions, there is the risk of a so-called chemical meningitis with cyst rupture and dissemination of sometimes caustic fluid contents into the ventricular system, or with tumor hemorrhage in this same vein. Empiric antibiotic/antiviral therapy is usually indicated at presentation; these drugs can be discontinued with negative cultures/PCR, at which point treatment is largely supportive.

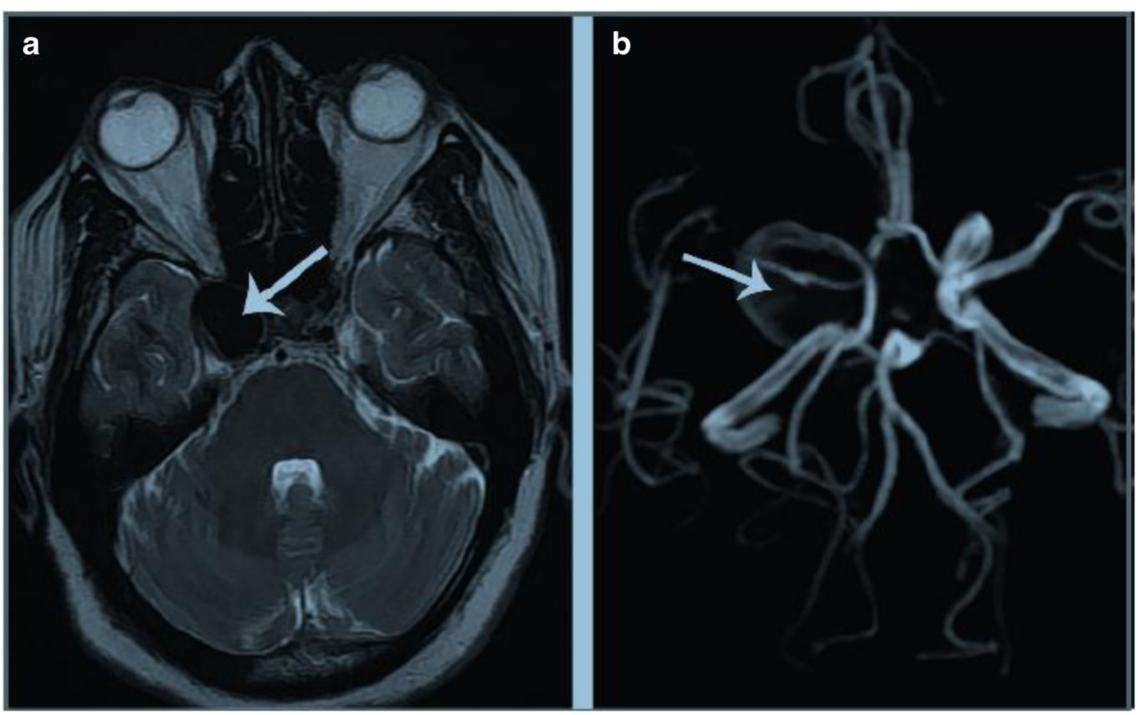

Fig. 7 Intracavernous internal carotid artery aneurysm. **a** Axial T2-weighted MRI demonstrates ovoid T2 hypointensity within the right cavernous sinus. Flow void is seen within the cavity of the lesion shows turbulent blood flow (arrow). **b** Magnetic resonance angiography (MRA) delineates the site of origin of the aneurysm from its parent vessel (arrow)

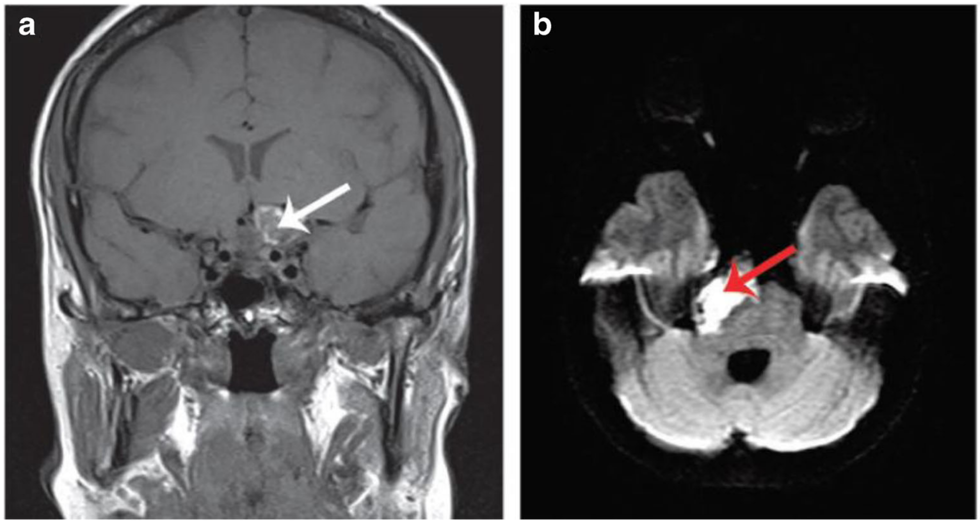

Fig. 8 Dermoid cysts (**a**, arrow) often contain peripheral calcifications, while epidermoid cysts (**b**, arrow) often appear bright on diffusion-weighted imaging

Corticosteroids can be of benefit in counteracting meningeal inflammation and associated symptoms.

Dermoid Cysts

Dermoid cysts are midline lesions that contain squamous epithelial cells, hair follicles, and sebaceous structures akin to teratomas, arising when embryonic ectoderm is ensnared with closure of the neural tube during development. Cyst walls often contain calcifications that can be seen on CT similar to craniopharyngiomas, while the contents are typically hyperintense on T1-weighted imaging given high fat content [21]. Neither the wall nor the cystic contents enhance. Surgical resection is the mainstay of treatment and must be approached carefully as dermoid cysts are prone to rupture, which can cause a chemical meningitis characterized by severe headaches, nuchal rigidity, nausea and vomiting, and sometimes provoke seizures. Leptomeningeal enhancement after cyst rupture is best seen on T1-weighted imaging with contrast (Fig. 8**a**).

Epidermoid Cysts

Epidermoid cysts can arise from the suprasellar cistern, though they are more commonly parasellar in location. Like dermoid cysts, they can form at the time of neural tube closure, or due to traumatic injury often in a postsurgical setting. Epidermoid cysts are more homogenous in composition, containing only squamous epithelial cells along with fluid filled with cholesterol and keratin crystals. Capsules of epidermoid cysts are also calcified lending to visualization on CT imaging. On MRI, they are hyperintense on T2-weighted imaging and bright on diffusion-weighted imaging (DWI), which is a hallmark finding [22] (Fig. 8b). Epidermoid cysts are usually surgically resected. Cystic fluid is usually caustic, fluid leakage can cause local inflammation and resultant damage to surrounding tissues, and a more diffuse meningitis in the case the sellar lesions with ventricular invasion [23]. Symptoms are usually responsive to steroid therapy.

Rathke's Cleft Cysts

Rathke's cleft cysts are cystic lesions whose walls are comprised of ciliated columnar epithelial cells. They often contain an intraluminal nodule visible on CT or MR imaging, appearing hyperdense on CT and are variably hyperintense on both T1- and T2-weighted imaging depending on protein content [24] as seen in Fig. 9. These lesions arise when the Rathke cleft, an outgrowth derived from the lumen of the embryonic structure known as the Rathke pouch does not appropriately regress. Larger/expanding cysts are removed by transsphenoidal surgery often endoscopically [25]. In addition to symptoms related to mass effect, Rathke's cleft cysts are prone to both hemorrhagic and nonhemorrhagic expansion and rupture akin to an apoplectic event; leakage of the cystic contents can cause chemical meningitis.

Arachnoid Cysts

Arachnoid cysts are slow-growing lesions arising in both the sellar and parasellar regions. They are isointense relative to CSF on T1- and T2-weighted images without DWI changes. They can cause adaptive bony erosion similar to benign meningiomas best visualized on CT imaging. The need for surgery is related to exertional mass effect of arachnoid cysts; surgical options include craniotomy/cystectomy, fenestration of the cyst into the subarachnoid space or less commonly cyst-peritoneal shunting. In addition to general meningeal

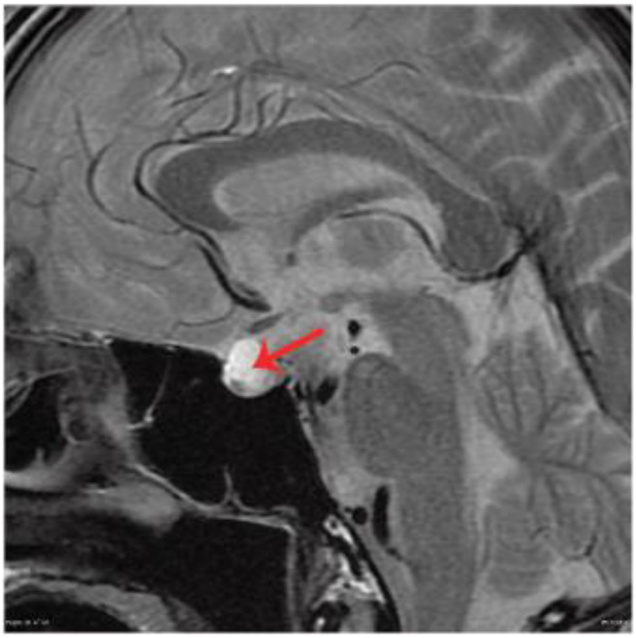

Fig. 9 Sagittal T2-weighted MRI showing a Rathke cleft cyst with characteristic intracystic nodule (arrow)

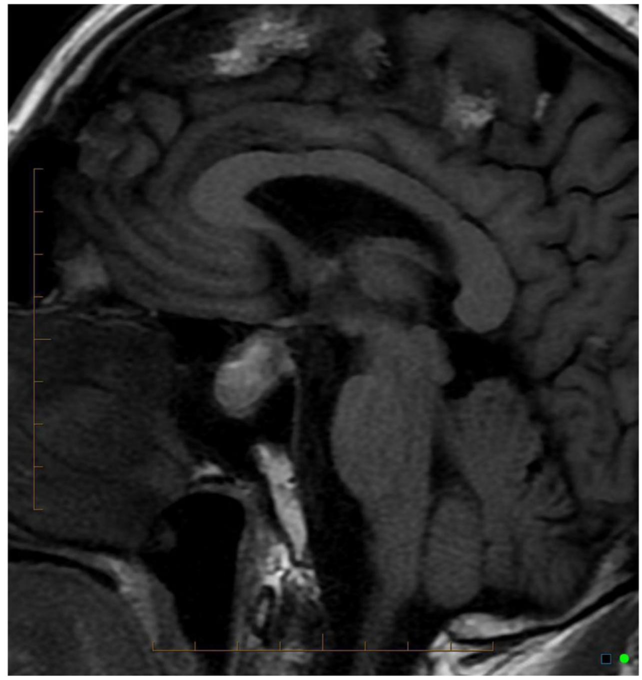

Fig. 10 Sagittal T1-weighted noncontrast MRI showing hyperintense pituitary apoplexy with residual blood visible in the sella

irritation, rupture of arachnoid cysts has been known to cause acute subdural hygroma formation and resultant headaches, nausea, and altered mental status, which often required emergent evacuation [26].

Pituitary Apoplexy

Pituitary apoplexy refers to spontaneous hemorrhage of the pituitary gland. Common causes of apoplexy include rupture of a pituitary adenoma in the setting of longstanding bromocriptine use for treatment of a prolactinoma, hemorrhagic infarction during the postpartum period (Sheehaan syndrome), and sudden changes in intracranial pressure. This is often associated with a severe "thunderclap" unrelenting headache, ophthalmoplegia/decreased visual acuity, and sometimes coma/death. Iatrogenic causes include prior exposure to radiation therapy and recent conventional cerebral angiography. Blood in the sellar region appears hyperdense on CT and correspondingly hyperintense on T1-weighted MR as seen in Fig. 10, with more prominent peripheral enhancement. Infarcted portions of the pituitary with Sheehan syndrome are often DWI positive [27].

Neurosarcoidosis

Neurosarcoidosis is a condition where noncaseating granulomas within arise within the central nervous system, resulting in both parenchymal infiltration and leptomeningeal spread. Pituitary involvement can cause ventricular obstruction and resultant ICP headaches, while leptomeningeal involvement often causes meningeal symptoms including skull base headaches, nuchal rigidity, and nausea/vomiting. MRI allows for the best visualization; affected areas may appear hypointense on T1-weighted imaging and hyperintense on T2-weighted imaging. With administration of contrast, pachymeningeal enhancement is often seeing along with leptomeningeal thickening. Tagged white blood cell scanning can also be informative given the leukocytic predominance. Treatment includes high-dose corticosteroids as first-line therapy, and a steroid sparing immunosuppressant in less frequent cases. Placement of an EVD is sometimes indicated in cases of hydrocephalus necessitating urgent reduction in ICP; surgical resection/debulking is usually not recommended given the steroid sensitivity (Fig. 11).

Pituitary Abscess

Primary pituitary abscesses are rare, when isolated are often unclear in origin though sometimes they can be seen in cases of a more widespread bacterial meningitis. Headaches can be related to mass effect, and patients with concomitant meningitis often have more classic skull base headaches, nuchal rigidity, and nausea. On MR imaging, the abscess rim is often DWI positive and enhances with

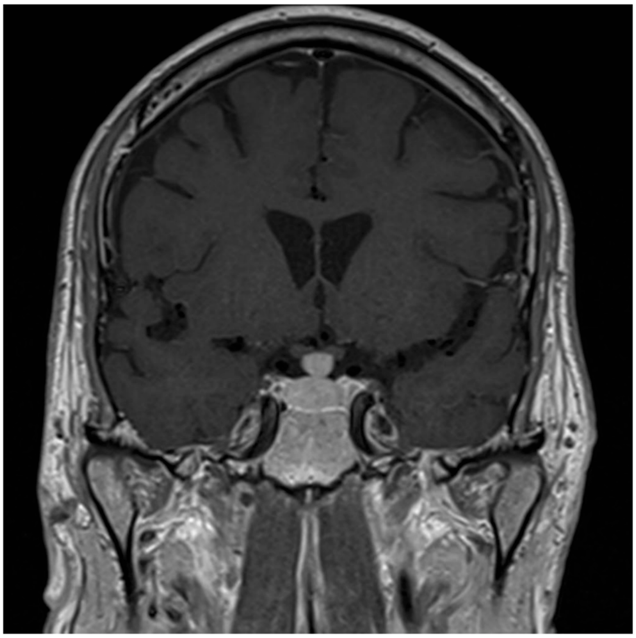

Fig. 11 Neurosarcoid lesion in the sella and suprasellar region, homogeneously enhancing with contrast on T1-weighted MR imaging with contrast

contrast, with a more central area of necrosis [28]. Management often includes careful surgical resection to avoid rupture/dissemination, in addition to empiric broad spectrum antibiotic therapy orally before surgery, intravenous therapy perioperatively (30 min before to 2 weeks postsurgery) and more directed maintenance oral antibiotic therapy based on antimicrobial susceptibilities of the abscess culture.

Conclusion

Pathology in the sellar and parasellar regions are numerous, including benign and malignant tumors, cystic lesions of various etiologies, vascular lesions and associated hemorrhagic complications, inflammatory conditions with neurospecificity, and of course infectious entities as outlined in this review. One commonality amongst the various pathologies is their presentation, specifically with respect to headache-related pain. The high risk of ventricular obstruction makes increased headaches and associated symptoms a usual presentation of sellar/parasellar disease, and many of the disease processes with fluid (cysts, vascular lesions) have the proclivity to cause a chemical meningitis. Management is usually multimodal in nature, including acute measures such as decompression/resection and EVD placement to relieve intracranial pressure, focal radiation therapy to prevent recurrence/regrowth, and medical therapy including hormone agonists and antibiotics where appropriate.

Compliance with Ethical Standards

Conflict of Interest Ajay P. Abad declares no conflict of interest.

Human and Animal Rights and Informed Consent This article does not contain any studies with human or animal subjects performed by any of the authors.

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