Imaging of the Pituitary: Microsurgical Anatomy, Mass Lesions and Differential Diagnosis

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Learning objectives

Understanding high resolution imaging anatomy of the pituitary gland, sella, suprasellar and parasellar region and imaging findings in common mass lesions.

Background

Introduction

The pituitary gland situated within the sella turcica (meaning Turkish Saddle), is also known as the "Master Control Gland" as it controls most of the body's endocrine functions through the hypothalamic-pituitary axis. The pituitary comprises of two main lobes coming from different embryological origins (anterior and posterior) which secrete a variety of hormones. Anatomy of the sellar, parasellar and suprasellar region is complex and needs thorough understanding in evaluating pituitary mass lesions and offering relevant differential diagnosis of sellar or suprasellar origin of mass lesions.

Multimodality imaging of the pituitary includes Computed Tomography (CT) and Magnetic resonance imaging (MRI). CT scan, though less frequently used for evaluating sellar and parasellar lesions, is a useful examination in depicting soft tissue calcification, bony destruction, acute bleed and surgically relevant bony anatomy. MR is the "gold standard" and modality of choice for evaluating pituitary related abnormalities, as the soft tissue contrast enables differentiation of the anterior from posterior lobe and clear visualisation of the infundibulum.

Dynamic MRI of pituitary is helpful in identifying microadenomas like prolactinomas which usually enhance to lesser extent as compared to normal pituitary which is outside the blood brain barrier. MR is critical in assessing macroadenomas and its extension to the cavernous sinus, sphenoid sinus and optic chiasm. Pituitary gland is best visualised in coronal and sagittal plane with typical MR imaging parameters of thin slice 2 mm, small FOV of 180 and dynamic imaging with contrast with 3-4 temporal sample points. 3-Tesla MRI with high field strength offers an improved image quality and spatial resolution. Diffusion-weighted imaging (DWI) is useful in early detection of acute pituitary infarction and can also help in characterising tumour components and consistency of macroadenomas.

The most common abnormalities that arise in the pituitary gland are pituitary adenoma, Rathke's cleft cyst and craniopharyngioma and they can be easily differentiated with MRI.
Anatomy of the pituitary gland, sellar region and suprasellar region

(Fig. 1 on page 4, Fig. 2 on page 4, Fig. 3 on page 5 and Fig. 4 on page 5)

The sellar region is an anatomically complex area bounded by the sphenoid sinus anterolaterally, the cavernous sinuses laterally, the suprasellar cistern, diaphragma sellae and hypothalamus superiorly and the dorsum sellae and brainstem posteriorly.3

The pituitary gland is divided into two main lobes coming from separate embryological origins. The anterior lobe (adenohypophysis) arising from Rathke’s pouch which originates rostral to the oropharyngeal membrane11 consists of the pars tuberalis, pars intermedia and pars distalis. The pars distalis is the largest part of the pituitary gland containing specialised epithelial cells that secrete:

- Growth hormone (GH, somatotropin) secreted by somatotrophs
- Thyroid stimulating hormone (TSH) secreted by thyrotrophs
- Adrenocorticotropic hormone (ACTH) secreted by corticotrophs
- Follicular stimulating homrone (FSH) secreted by gonadotrophs
- Leutinizing homrone (LH) secreted by gonadotrophs
- Prolactin (PRL) secreted by lactotrophs

Mammosomatotrophs are also present, these cells can secrete both prolactin and growth hormone.

The pars tuberalis is part of the adenohypophysis that surrounds the anterior aspect of the infundibular stalk. While the pars intermedia is a thin layer of epithelial cells (a vestigial structure) between the pars distalis and neurohypophysis arising from the posterior wall of Rathke’s pouch and may give rise to Rathke’s Cleft Cysts (also known as pars intermedia cysts).9,11

The posterior pituitary (neurohypophysis or pars nervosa) is a direct extension of the hypothalamus that does not synthesise any hormones but rather releases oxytocin and antidiuretic hormone (vasopressin) synthesised in the hypothalamus and travel down the hypothalamo-hypophyseal tract.3,9,11

The suprasellar region is a cerebrospinal fluid filled space containing the optic chiasma, pituitary infundibulum and the circle of Willis. This space is superior to the sellar region, hence any pathology within this region may extend into the sellar region.
Normal variation of the pituitary gland can occur. This is usually dependent upon the age and gender of the patient.

- A normal adult pituitary usually measures 8mm or less
- A paediatric pituitary usually measures 6mm or less in greatest height
- During adolescence (puberty), postpartum or pregnancy, the pituitary can measure up to 10mm and 12mm respectively

Images for this section:

![Fig. 1: Sagittal CT slice demonstrating the sella turcica.](image)
**Fig. 2:** Sagittal section of the normal anatomy of a pituitary gland and its surrounding structures.

**Fig. 3:** Coronal section of a normal pituitary gland and its surrounding structures.
**Fig. 4:** (A) The normal anatomy of the pituitary gland. (B) The six cell types of adenohypophysis.
Imaging findings OR Procedure details

Imaging Modalities

Computed Tomography (CT)

CT is a useful technique that is readily available allowing characterisation and differential diagnoses for most processes quickly. Specifically, CT allows visualisation of the sellar floor and bony involvement or pathology which is useful in surgical planning for treatment of the pituitary pathologies within this article, despite being inferior to MRI in soft tissue definition. CT also remains the modality of choice for patients who are unable to undergo MRI (cardiac pacemaker or claustrophobia).

Magnetic Resonance Imaging (MRI)

Assessment of the pituitary under MRI is best done with 1.5T or 3T scanner using protocols appropriate for the region and suspected pathology. These should take into account the field of view, slice thickness, sequences for complete assessment of the condition as well as the use of contrast (gadolinium enhanced). Sagittal and coronal planes are most useful with thin slices (3mm or less) without any gaps between slices will allow small structures to be appropriately visualised.

The composition of lesions usually differ to that of a healthy pituitary gland, brain tissue and cerebrospinal fluid, allowing a variation in the T1 and T2 weighted relaxation times and hence characterisation of a lesion on MRI. The administration of gadolinium chelate may also further enhance the contrast between normal and abnormal tissue and allowing a more confident diagnosis of the condition (e.g. microadenoma).

Pre-contrast T1 and T2 weighted spin echo images are usually acquired with both dynamic and routine post-contrast images and delayed scans after 30-60 minutes of contrast administration done in one study to confirm a condition confidently. In addition to this, T2 weight gradient echo images and diffusion weighted images are also used and play an important role in the diagnosis of certain conditions.

Nuclear Isotope (Radionuclide) Imaging

Radionuclide techniques are limited and not commonly used in the diagnosis of pituitary pathologies. Using pharmaceuticals (\(^{111}\)Indium labelled Octreotide) that specifically bind to pituitary receptors, sellar masses (such as non-functioning adenomas) can be detected
with single photon emission tomography (SPECT). However, other parasellar tumours such as meningiomas may express somatostatin receptors and also have octreotide uptake, rendering the technique limited in usefulness.

Positron emission tomography (PET) using $^{18}$F-fluro-deoxy-D-glucose (FDG) in assessing biological activity of pituitary tumours are also limited since majority of pituitary masses are slow growing and hence not metabolically active. Tracers such as $^{11}$C-methionine also have limited use, however the short half-life and high cost of production does not make this technique practical or economical in clinical practice. $^{19}$

**Classification of Pituitary Pathology**

**Fig. 5 on page 16**

**Congenital/Developmental Conditions**

**Rathke’s Cleft Cyst (Fig. 6 on page 16, Fig. 7 on page 17 and Fig. 8 on page 18)**

Also known as Pars Intermedia Cysts (due to the location of the cyst, usually within the pars intermedia region of the pituitary gland), these are non-neoplastic, sellar or suprasellar (with purely suprasellar cysts being rare) epithelium lined cysts arising from Rathke’s pouch. RCC are more common in females than males and rarely occur in paediatrics, since the cysts appear to enlarge during life (instead of regressing normally). Clinical presentations for RCC are usually asymptomatic, however if enlarged, they can cause visual disturbances, pituitary dysfunction and headaches. Symptomatic Rathke’s cleft cysts account for up to 9% of symptomatic sellar lesions. $^{6,23,24}$

On CT, RCCs appear typically as non-calcified lesions with homogenous hypointensity, but may also be of mixed attenuation (isointense and hypointense) or have small calcifications within the lesion wall. These cysts do not enhance with contrast.

The signal characteristics of these cysts on MRI vary depending on the composition of the cyst (mucoid or serous). RCCs are usually sharply circumscribed, unilocular and located within the midline (unlike cystic adenomas). T1WI can be hyperintense or hypointense depending on the protein content of the cyst (high protein content gives a hyperintense signal). T2WI have variable intensities, most RCCs are hyperintense, however some can be isointense or hypointense. There is no contrast enhancement of the cyst, however a thin enhancing rim may be present due to the compression of surrounding pituitary tissue. Fluid levels may also be seen, especially in cases where haemorrhage is present. $^{22,23,25}$
Pituitary Ectopia (Fig. 9 on page 19)

Pituitary ectopia typically occur as an ectopic posterior pituitary gland, thought to be caused by the incomplete downward extension of the hypothalamus (pituitary infundibulum) resulting in the inability of growth hormone produced by the hypothalamus to reach its target organs sufficiently and is a common cause of pituitary dwarfism. This condition is a congenital abnormality commonly presenting with features of decreased growth hormone (pituitary dwarfism) and neonatal hypoglycaemia, however it can also have associations with other central nervous system malformations.

MRI is the "gold standard" and only modality that is appropriate in identifying posterior pituitary ectopia, with sagittal midline T1WI images being the most diagnostic. Features that indicate pituitary ectopia include hyperintense T1 signal of a 3-8mm nodule at the floor of the third ventricle (median eminence) and an absent posterior pituitary bright spot. 28,29,30,31

Pituitary Neoplasm

Adenoma

A microadenoma is a lesion less than 10mm and a macroadenoma is a lesion that is more than 10mm in its greatest dimension. Adenomas can be functional or non-functional, as determined by whether or not it produces a hormone or not. The prevalence of these tumours range from 14 to 22.5% based on autopsy and imaging findings. 4

A functional adenoma can then be further classified by the hormone that it secretes, the most common being a prolactinoma secreting prolactin and causing amenorrhea or galactorrhea in females and gynaecomastia, hypogonadism or impotence in males. Non-functioning adenomas can be asymptomatic until they cause mass effect on surrounding structures and result in cranial neuropathy.

MRI allows localisation of the lesion, assessment of regional extension/invasion and surveillance/monitoring of patients after treatment. 4 Microadenomas tend to present as functional tumours from excess hormone production, while patients with macroadenomas tend to present with non-functional tumours due to symptoms relating to compression of adjacent structures, mass effect or increased intracranial pressure. 6

Microadenoma (Fig. 10 on page 20, Fig. 11 on page 21, Fig. 12 on page 22, Fig. 13 on page 23 and Fig. 14 on page 24)
Although most adenomas are detectable on non-contrast enhanced images, microadenomas may only become visible after contrast administration. These tumours may be isointense as surrounding tissue on T1 and variable on T2. Most tumours are hyperintense on T2 and tend to be soft and readily resected during surgery, however hypointense T2 signals microadenomas can also occur and these tend to be firmer and more adherent to the surrounding structures during surgical removal.

It is also important to note that prolactinomas tend to be hyperintense with high T2 signal while most growth hormone secreting adenomas have low T2 signals. Administration of gadolinium chelate is can assist in diagnosis since microadenomas are relatively hypoenhancing or isoenhancing relative to the normal pituitary during the wash-in phase of contrast. Dynamic studies are also useful for demonstrating the differential uptake of contrast between a microadenoma and normal pituitary tissue, with some tumours also retaining contrast on delayed images compared to the normal tissue and hence show a hyperenhancing tumour compared to the normal gland.

**Macroadenoma (Fig. 15 on page 25, Fig. 16 on page 25, Fig. 17 on page 26 and Fig. 18 on page 27)**

These tumours can be visualised without the use of gadolinium chelate due to the size of the tumour, with MRI being the primary diagnostic modality of the lesion. MRI is important to evaluate the potential suprasellar extension, mass effect on the optic chiasm, lateral infiltration into the cavernous sinus and inferior invasion of the clivus or sphenoid sinus in macroadenomas.

On sagittal T1WI the mass cannot be separated from the pituitary gland and as it grows, it may extend into the suprasellar region or erode the sellar floor (enlarging the sellar turcica). Often a "Figure Eight" or "Snowman" appearance in the coronal plane is seen due to the constriction of the waist of the lesion by the diaphragma sellae. In rare cases it can also mimic an aggressive skull base process caused by caudal growth into the basisphenoid bone (this is usually called an invasive macroadenoma) which can be functional or non-functional.

Macroadenomas are usually isointense to grey matter on T1WI and T2WI, but can also be heterogenous in signal because of internal haemorrhage, cystic changes or necrosis. There is usually mild to moderate enhancement and occasionally smooth dural enhancement which can mimic a meningioma.

On dynamic imaging, non-adenomatous pituitary tissue will enhance before the adenoma and is usually superiorly or posteriorly displaced to the mass. Mass effect on the
optic chiasma is best visualised on coronal T2WI and cavernous sinus invasion should also be assessed. Since cavernous sinus invasion will determine whether the tumour should be completely resected versus debulked with surveillance imaging or coexistent radiotherapy.  

On CT, a macroadenoma will appear:

- isodense to grey matter
- noncalcified
- as a solid mass that moderately enhances with contrast

As a macroadenoma outgrows its blood supply, it becomes heterogeneous in attenuation as a result of necrosis or haemorrhage.

Cavernous sinus invasion is an important consideration in complete surgical tumour resection, as up to 21% of macroadenomas can have invasion. 

Pituitary Apoplexy (Fig. 17 on page 26, Fig. 18 on page 27 and Fig. 19 on page 27)

Pituitary apoplexy is a rare and acute syndrome that can potentially be fatal, caused by sudden haemorrhaging and/or infarct of the pituitary gland, generally within an undiagnosed pre-existing adenoma. However, patients can have apoplexy without previous pituitary pathology, such as Sheehan syndrome characterised by pituitary infarction occurring in post or peripartum women with hypovolemia. In rare cases, it can also occur after the initiation of bromocriptine or cabergoline for treatment of a prolactin secreting adenoma. 

Clinical presentations of this condition is an onset of acute headache (occurring in more than 80% of patients), nausea, visual impairment (present in more than half of patients with pituitary apoplexy), ophthalmoplegia, altered mental state, panhypopituitarism or endocrine deficiencies caused by acute haemorrhagic or ischemic/necrotic pituitary infarction. 

CT is the initial emergency examination for the clinical presentation of pituitary apoplexy since it is similar to other acute conditions (i.e. subarachnoid haemorrhage). CT is not sensitive for pituitary apoplexy detection and features depend on the subtype of apoplexy (haemorrhagic or ischemic/necrotic) and timing of imaging relative to onset. The sella may appear normal or show a hypodense or hyperdense sellar mass. CT is most useful in the acute setting (24-48 hours), after this time the blood intensity decreases and the lesion
may be difficult to detect. Post-contrast images may show a rim (ring) of enhancement which may be a sign of pituitary apoplexy. Hence, CT is usually used to exclude other diagnoses with MRI to give more details about the suspected apoplexy.

MRI is the preferred and gold standard for evaluating apoplexy as it can identify haemorrhagic areas as well as the relationship of the lesion to surrounding anatomy. Evaluating mass effect on the optic chiasm or involvement of the cavernous sinus is also critical. T1 and T2 weighted image features are dependent upon the subtype of apoplexy and timing of imaging which varies due to the haemoglobin state of oxygenation of blood degradation.

A fluid level may also be present within the mass, indicating haemorrhage but may not indicate a pituitary apoplexy. Thickening of the sphenoid sinus mucosa is highly indicative of pituitary apoplexy and can be related to venous engorgement. T2 weighted gradient echo images are the most sensitive neuroimaging technique in identifying brain haemorrhages.¹

**Craniopharyngioma (Fig. 20 on page 28 and Fig. 21 on page 29)**

A craniopharyngioma is a tumour that arises from the squamous epithelial remnants of the Rathke pouch. These tumours are the most common non-glial tumours within paediatrics, but can also be present in adults (peak incidences of presentation occurring between the ages of 5 and 14 years, and less frequent between the ages of 40 and 74). Clinical presentations of a craniopharyngioma is dependent upon its size and location, which can include headaches, nausea, visual disturbances (occurring in 20% of paediatric patients and 80% of adults with craniopharyngiomas), hydrocephalus or hypothalamic/pituitary abnormalities.⁴,³⁶,³⁷,³⁸

These tumours have 2 major histological subtypes - Adamantinomatous and Papillary. The adamantinomatous type is the most common and usually presents in paediatric patients as a multiloculated, cystic, solid, calcified mass. While the papillary subtype presents mostly in adults as a solid, less commonly calcified mass.⁴,⁶,³⁶,³⁷,³⁸

Craniopharyngiomas usually arise from the pituitary infundibulum within the suprasellar region and occasionally the sellar region, however they can arise anywhere along the craniopharyngeal duct either as a pure adenomatous, pure papillary or transitional (mixed) lesion.

On CT, the adamantinomatous type appears as an isodense to hypodense, multiloculated, sellar/suprasellar mass having cystic (occasionally also solid)
components. A nodular or thin border of calcification of the cystic components with heterogeneous enhancement is often noted. While the papillary type is usually solid with cystic components, isodense, non-calcified and homogeneous in enhancement. 21,37,38

However, MRI can be variable due to the cholesterol, protein and haemorrhage content of the cysts. Cystic signal is hyperintense in comparison to CSF on T1WI and mixed or hyperintense on T2WI. There may also be fluid levels present. Solid components are isointense to hypointense on T1WI and homogeneous and hyperintense on T2WI. There can be heterogenous enhancement of both the solid components and cystic walls. Calcification may appear as hypointense foci or gradient echo blooming on T1 or T2WI. These lesions do not follow fat signals on fat suppression sequences and usually enhances, which make it easily identifiable from other lesions (such as Dermoid cysts). 4, 6, 21, 22,36,37,38

Pituicytoma ( Fig. 22 on page 30 and Fig. 23 on page 30 )

Pituicytomas are rare tumours that arise from pituicytes, which are specialised glial cells within the neurohypophysis and infundibulum of the pituitary gland. These tumours arise in adults (typically 50 years old) and are more predominant in females than males. Patients with a pituicytoma can present with endocrine dysfunction or from mass effects of surrounding structures, however many are asymptomatic with the lesion being an incidental finding.

These tumours are usually well circumscribed, spindle or stellate shaped and rarely have necrosis or cystic degeneration. On CT, the masses are homogenous lesions within the pituitary fossa or suprasellar region ranging in size. MRI of pituicytomas typically show an isointense solid mass with an absent posterior pituitary bright spot on T1WI and bright enhancement with contrast. On T2WI, the mass is usually heterogeneous and can be isointense to hypointense. 25,26,27

Pituitary Secondary Neoplasm

Pituitary Metastases

Pituitary metastases are rare and can be misdiagnosed as pituitary adenomas instead. Common metastases are breast cancer in females and lung cancer in males as primary tumours. Diagnosis of a pituitary metastasis include additional enhancing foci within the calvarium, skull base or intracranial structures with patients knowing that they have a metastatic disease. Due to the blood supply of the posterior pituitary gland, it is more
frequently involved in metastatic processes than the anterior lobe. Imaging will usually show a locally invasive and growing lesion within or separable from the pituitary.\textsuperscript{4,6}

**Meningioma ( Fig. 24 on page 30 )**

Meningiomas are the most common meninges tumour. These tumours are non-glial neoplasms arising from the arachnoid cap cells of the meninges and are typically benign but can be malignant (although very rare). Meningiomas can be located anywhere that meninges are found, including the intrasellar region and mimic an adenoma, since a meningioma can arise from the tuberculum sellae, diaphragm sellae, sphenoid wing, cavernous sinus dura or planum sphenoidale.\textsuperscript{4,6,32,33}

The first modality often used to assess meningiomas is CT, which can show a solid, extra axial, hyperdense mass with or without areas of cystic degeneration or necrosis. Other associated findings can include calcification, hyperostosis and pneumosinus dilatans, with homogenous or heterogenous enhancement.

On MRI, most meningiomas are isointense to grey matter on both T1 and T2WI. Heterogenous T2WI signals may be present due to the calcium (hypointense), cystic changes (hyperintense) or haemorrhage (variable). The tumour will also have intense post-contrast enhancement. Other features that can support a diagnosis of meningioma include:

- hyperostosis
- a thick, enhanced dural tail
- encasement
- narrowing
- potential occlusion of the cavernous or supraclinoid segments of the internal carotid artery\textsuperscript{4,6,32,33}

**Miscellaneous Conditions**

**Empty Sella ( Fig. 25 on page 31 )**

Also known as "Empty Pituitary Fossa", is when the pituitary fossa is largely empty of pituitary tissue and replaced by cerebrospinal fluid (CSF) instead. This condition can be classified into two categories - primary and secondary.

Primary empty sella is a condition that is not caused by a pre-existing condition while secondary is caused by a pre-existing identifiable condition (such as prior tumours, radiotherapy, surgery, haemorrhage).
CT and MRI have similar findings, in that the images show a pituitary fossa filled with CSF of variable size and the pituitary infundibulum is demonstrated as coursing through the space. This feature is known as the "infundibulum sign" and excludes a cystic mass.\textsuperscript{4,6,34}

**Lymphocytic Hypophysitis**

Clinically, lymphocytic hypophysitis cannot be differentiated from a non-functioning pituitary tumours. Any female presenting with a sellar mass during pregnancy or within the first year after postpartum should be suspected to have lymphocytic hypophysitis. Diagnosis is only certain through histological assessment, however MRI features that are indicative of lymphocytic hypophysitis can determine whether a patient would benefit more from medical or surgical treatment.

Such features include:

- symmetric enlargement of the gland
- homogenous enhancement
- intense contrast enhancement
- thickening and enhancement of pituitary infundibulum
- loss of posterior pituitary bright spot
- enhancement of adjacent dura to the pituitary mass
- intact sellar floor

which is in contrast with a pituitary macroadenoma which are usually asymmetric, heterogenous, less contrast enhancement, rarely involves the stalk, preservation of the posterior pituitary bright spot and an eroded sellar floor. However, a thickened pituitary infundibulum can also be indicative of germinoma, lymphoma, tuberculosis, sarcoidosis or Langerhans cell histiocytosis.\textsuperscript{3}

**Differential Diagnoses**

( Fig. 24 on page 30, Fig. 26 on page 32, Fig. 27 on page 33 and Fig. 28 on page 32 )

The imaging analysis of pituitary conditions can fall into one of four categories that are defined by the location/region of the lesion. This method of evaluation along with the knowledge of common and uncommon conditions in that region and the patient's presentation will allow a concise list of differential diagnoses to be made. The differential diagnoses for each of these regions are defined below.

**Sellar Region**

- Pituitary Adenoma
• Craniopharyngioma

Parasellar Region
• Arachnoid Cyst
• Meningioma

Suprasellar Region
• Aneurysm
• Hypothalamic-chiasmatic glioma
• Meningioma
• Dermoid/Epidermoid Cyst

Infrasellar Region
• Macroadenoma
• Clivus Chordoma
• Nasopharyngeal carcinoma

Images for this section:

**Fig. 5: Classification of Pituitary Pathology**
Fig. 6: Rathke's Cleft Cyst Small
Fig. 7: Rathke's Cleft Cyst Medium
Fig. 8: Rathke's Cleft Cyst Large
Fig. 9: Ectopic Posterior Pituitary
Fig. 10: Microprolactinoma
Fig. 11: Microadenoma causing Acromegaly
Fig. 12: Exophytic Microadenoma
Fig. 13: Microadenoma causing Acromegaly Preoperative
Fig. 14: Microadenoma causing Acromegaly Postoperative

Fig. 15: Macroadenoma
Fig. 16: Macroadenoma Large
**Fig. 17:** Macroadenoma with Apoplexy

**Fig. 18:** Macroadenoma with Apoplexy causing fluid-fluid level
Fig. 19: Sheehan's Syndrome
Fig. 20: Admantinomatous Craniopharyngioma
**Fig. 21:** Papillary Craniopharyngioma

**Fig. 22:** Pituicytoma

**Fig. 23:** Pituicytoma
Fig. 24: Meningioma with secondary extension in to the sella
**Fig. 25:** Primary Empty Sella Syndrome

**Fig. 26:** Hypothalamic-chiasmatic Glioma
Fig. 28: Clivus Chordoma extending into the Sella
Fig. 27: Suprasellar Dermoid
Conclusion

Understanding anatomy of the sella and pituitary gland is of crucial importance in accurate localisation of the mass in this location. It helps to discern if the mass is of sellar origin arising from the pituitary gland itself or a secondary extension in to the sella. This relationship determines the differential diagnosis.

Hi-resolution imaging with MRI is of paramount importance in diagnosis of pituitary tumors. Nature of the mass and its size also determines medical vs. surgical management. Preoperative planning and vital information like cavernous sinus extension, optic chiasm compression and vascular invasion can be precisely demonstrated on MRI which in turn influences the neurosurgical outcome.

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