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Liza Das, Kirti Gupta, Pinaki Dutta

ABSTRACT

The pituitary-hypothalamus unit functions as the central regulator of multiple endocrine glands and determines several physiological functions. Disorders of the hypothalamus-pituitary unit can be secondary to congenital or developmental causes, genetic syndromes, or acquired causes. Functionally, any of these can lead to under- or overfunctioning of a single cell type or multiple cell subtypes of the pituitary. Underfunctioning of the pituitary is termed hypopituitarism, which may be a result of transcription factor defects, trauma including birth trauma, tumors and nontumoral causes (infective, inflammatory, traumatic, and degenerative).

Pituitary overfunctioning is usually due to tumoral causes (monoclonal or polyclonal proliferation of cells) or pituitary hyperplasia (in case of certain syndromic causes of acrogigantism). Rarely, the hormonal production is from an ectopic source causing acromegaly or Cushing's syndrome due to ectopic secretion of growth hormone-releasing hormone (GHRH) or corticotropin-releasing hormone (CRH), respectively. Over 90–95% pituitary adenomas are sporadic while the rest 5–10% have an underlying genetic background. Identification of a genetic cause warrants surveillance for other organ involvement, which may appear over time or perform cascade screening to identify relatives at a presymptomatic or early symptomatic stage.

"Be like Pituitary gland, be master of all (glands), size does not matter but most importantly, do follow your supreme master (Hypothalamus)"

—Anonymous

INTRODUCTION

The pituitary gland is a midline structure located within the sella turcica recess of the sphenoid bone at the base of the brain. It is regarded as the master endocrine gland, which controls a multitude of physiological processes, such as growth, puberty, metabolism, energy regulation, fluid balance, and fertility. It integrates signals from the central nervous system which are relayed to it from the hypothalamus and secretes various hormones from different cell populations. These hormones then act on target organs and regulate physiological processes. In some instances, the hormone is also trophic for the target gland.

The unifying feature of all hormone axes (hypothalamus-pituitary-target organ) is a negative feedback system in all cases, except estrogen which works in a feed-forward cycle for luteinizing hormone (LH) in the preovulatory phase of the menstrual cycle. This principle of feedback not only governs physiology but has clinical relevance in terms of diagnostic tests in various pituitary tumor associated endocrinopathies. Therefore, stimulation tests with appropriate pharmacological agents are used to diagnose hormone deficiencies while suppression tests are used to identify autonomous hormone excess states.

ANATOMY AND ONTOGENY

The pituitary is composed of adenohypophysis which develops from the oral ectoderm (or Rathke's pouch) and grows cranially to meet the neurohypophysis which develops from the neural ectoderm and grows caudally. The intermediate lobe develops from the Rathke's pouch and the infundibulum (stalk), which connects the hypothalamus to the pituitary as well as the posterior pituitary develops from the diencephalon. The anterior lobe comprises about two-thirds of the pituitary and the rest is constituted by the posterior lobe.

A sequence of tightly regulated cellular and molecular events subsequently begins to guide the formation of differentiated cell types within the anterior pituitary. They secrete specific hormones; prolactin (PRL) from lactotrophs, growth hormone (GH) from somatotrophs, LH and follicle-stimulating hormone from gonadotrophs, adrenocorticotropic hormone (ACTH) from corticotrophs, and thyroid-stimulating hormone (TSH) from thyrotrophs. Other cells in the anterior pituitary include the folliculostellate cells, which are regarded as the stem cells, enabling pituitary plasticity in case of physiological conditions like enlargement of the pituitary during

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pregnancy and lactation. The intermediate lobe contains melanotrophs, which secrete pro-opiomelanocortin (POMC). The stalk consists of a neurovascular bundle (axons from the hypothalamus and hypophyseal portal system) and transmits releasing hormones to act on the pituitary. This stalk traverses the dura mater to join the pituitary, which is an extradural structure. The neurons (magnocellular) from the hypothalamus include the supraoptic and paraventricular nuclei that secrete vasopressin and oxytocin, respectively. These neurons project to the posterior lobe, which acts as a reservoir for these hormones. The posterior pituitary also contains pituicytes, specialized glial cells, which account for a variety of posterior pituitary tumors. The median eminence, at the base of the hypothalamus, contains a capillary bed into which the parvocellular neurons secrete hypophysiotropic (releasing hormones), which pass through the portal system to reach and stimulate the anterior and intermediate lobes. Thus, the pituitary gland synchronizes multiple physiological processes including growth, reproduction, and homeostasis through central hypothalamic and peripheral hormonal signals.

PHYSIOLOGY

The major physiological functions of the anterior, intermediate, and posterior lobes and distribution of the different cell populations are summarized in **Tables 1** & **2** and **Figure 1**.

TABLE 1 Hormones of the pituitary gland with their role in normal physiology.

Hormone	Target organ/cell	Effects on target organ	Downstream endocrine effects
Anterior lobe			
GH	Liver, skeleton, and soft tissues	• IGF-1 generation (from the liver) • Nutrient metabolism	• Linear growth directly and via IGF-1 • Protein synthesis • Lipolysis • Anti-insulin, causing glucose intolerance • Water, sodium, and phosphate retention
ACTH	Adrenal cortex	• Cortisol production • Adrenocortical development	• Resistance to physiologic stress • Fuel metabolism • Maintenance of vascular tone • Immunity
LH/FSH	Testis/ovaries	• Secretion of testosterone and estrogen/progesterone • LH—ovulation, maintenance of corpus luteum in females, and testosterone production in males • FSH—follicular development in females and spermatogenesis in males	• Onset and progression of sexual development in puberty • Adult sex steroid production • Fertility
TSH	Thyroid gland	• Induction of T4 secretion • Trophic for thyroid follicles	• Homeostasis including BMR • Cellular metabolism • Growth
PRL	Breast	• Galactopoiesis • Lactation	
Intermediate lobe (rudimentary in humans)			
POMC	• CLIP • γ-LPH • β- endorphin • α-MSH • β-MSH	Only some amount of β-endorphin is secreted	
Posterior lobe			
ADH/AVP	Renal collecting ducts	• V1a (vascular smooth muscle) • V1b (anterior pituitary) • V2 (renal collecting ducts)	• Principal action is on water homeostasis and maintenance of osmolality • Pressor action • ACTH secretion
Oxytocin	Suckling breast	Lactation	Myoepithelial cell contraction in the breast for milk "let-down", bonding, and love

(ACTH: adrenocorticotropic hormone; ADH: antidiuretic hormone; AVP: arginine vasopressin; BMR: basal metabolic rate; CLIP: corticotropin-like intermediate lobe peptide; FSH: follicle-stimulating hormone; GH: growth hormone; LH: luteinizing hormone; LPH: lipotropin hormone; MSH: melanocyte-stimulating hormone; POMC: pro-opiomelanocortin; PRL: prolactin; TSH: thyroid-stimulating hormone)

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TABLE 2 Distribution of cells in the pituitary.

Cell type	Proportion of cells in the pituitary	Location and distribution
Somatotrophs	~40–50%	Lateral aspect
Lactotrophs	~20–25%	Posterolateral aspect
Corticotrophs	~10–20%	Posteromedial aspect
Thyrotrophs	~5%	Anteromedial aspect
Gonadotrophs	~10%	Distributed all throughout the pituitary

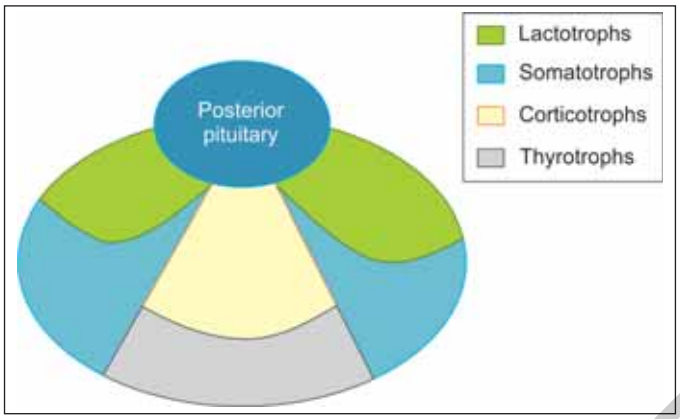


FIGURE 1: Schematic representation of distribution of cells in the pituitary.

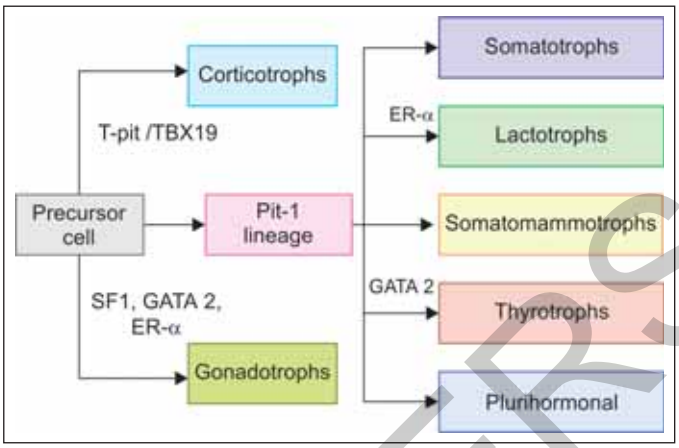
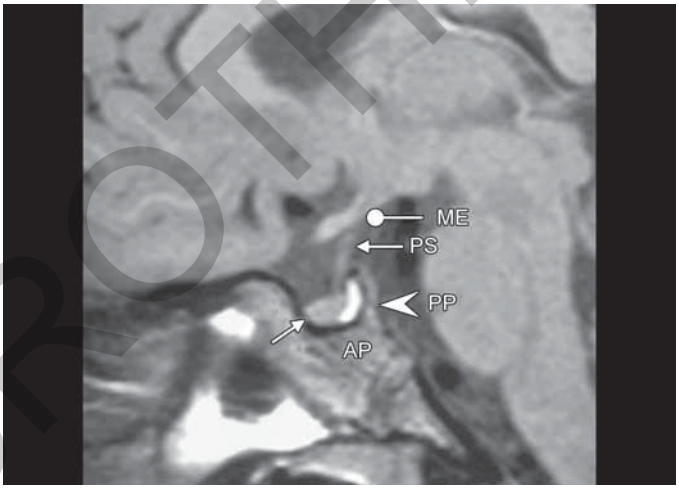


FIGURE 2: Lineage of pituitary cell development and role of transcription factors.



(AP: anterior pituitary; ME: median eminence; MRI: magnetic resonance imaging; PP: posterior pituitary; PS: pituitary stalk)

FIGURE 3: Normal landmarks of the sellar-suprasellar region on sagittal section MRI.

PATHOLOGY

Disorders of the hypothalamus and pituitary gland include congenital/developmental and acquired causes. Congenital/developmental disorders of the hypothalamus-pituitary unit are predominantly caused by defects in the various transcription factors involved in the development of the pituitary and its constituent cells (**Fig. 2**). Acquired causes including infective, inflammatory, infiltrative, degenerative, traumatic, or neoplasms can cause hypopituitarism. On the other hand, endocrinopathies associated with pituitary tumors result in clinical syndromes associated with the excess of one or multiple hormones.

CONGENITAL DISORDERS

Defects in the orchestrated development of the pituitary gland, primarily of the transcription factors, result in hypopituitarism. Depending upon the involved factor, it may result in isolated or multiple pituitary hormone deficiency (MPHD). Risk factors for hypopituitarism include breech presentation and birth asphyxia. Clinical presentation may include hypoglycemia, pathological jaundice, and micropenis. The radiological tetrad of empty sella, hypoplastic anterior pituitary, redundant or absent stalk, and ectopic or absent posterior pituitary bright spot is the characteristic feature. GH deficiency is the most common and other hormone deficiencies in the absence of GH deficiency (except TBX19/T-Pit) are undescribed. Moreover, there is a correlation between radiological abnormalities and severity of GH deficiency.

Normal anatomic landmarks of the pituitary are shown in **Figure 3**. Magnetic resonance imaging (MRI) of a neonate depicting physiological T1 hyperintensity of the entire gland is shown in **Figure 4**. Individual cases of MPHD with clinical and radiological aspects of diagnosis are shown in **Figures 5** and **6**.

Apart from transcription factor defects, other congenital anomalies of the hypothalamus-pituitary unit include hypothalamic malformations such as hamartoma which can manifest as precocious puberty with or without gelastic seizures (**Fig. 7**). It may be seen in association with GLI3 mutations as part of the Pallister–Hall syndrome which also includes polydactyly and cleft lip/palate. Congenital defects also include other hamartomas and persistent craniopharyngeal canal (**Fig. 8**).

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FIGURE 4: This sagittal magnetic resonance image of a neonate highlights that unlike adult pituitary where T1 hyperintensity is restricted to the posterior pituitary, the entire gland (including anterior pituitary) shows T1 hyperintensity.

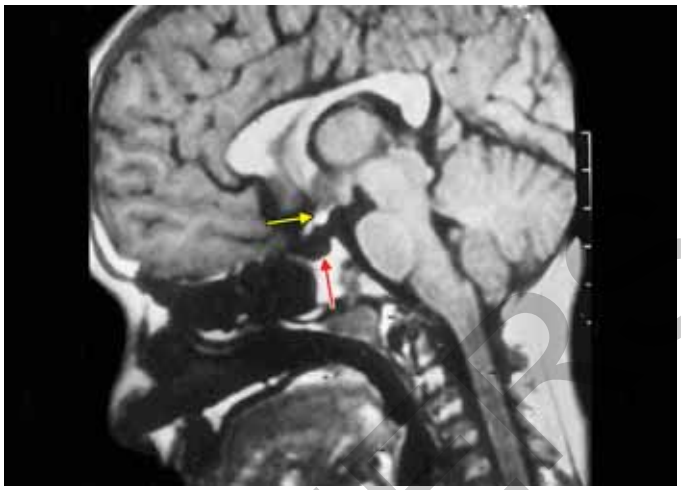

FIGURE 5: Magnetic resonance imaging (MRI) tetrad in transcription factor defects depicting small sella, hypoplastic anterior pituitary (red arrow), absent stalk, and ectopic posterior pituitary bright spot (yellow arrow).



FIGURES 6 A to C: Clinical defects noted in children with multiple pituitary hormone deficiency (MPHD). Depicted here are the **(A)** single central incisor, **(B)** hexadactyly, and **(C)** clinodactyly in different patients with MPHD.

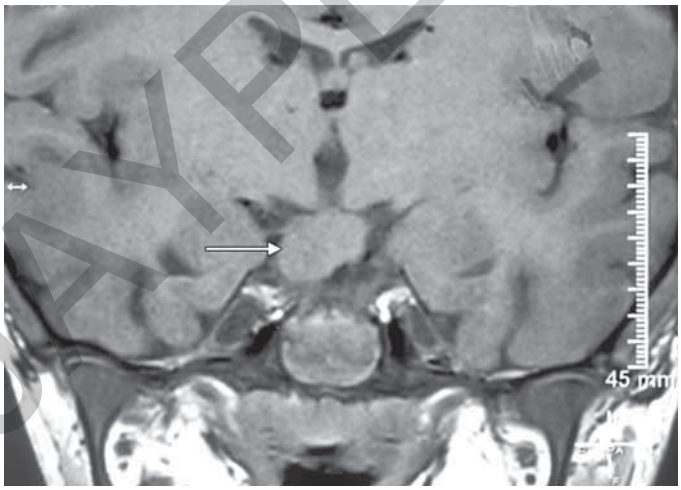

FIGURE 7: Magnetic resonance imaging (MRI) showing a hypothalamic mass consistent with hypothalamic hamartoma. He was treated with gonadotropin-releasing hormone (GnRH) agonist (leuprolide) monthly injections and letrozole (aromatase inhibitor).

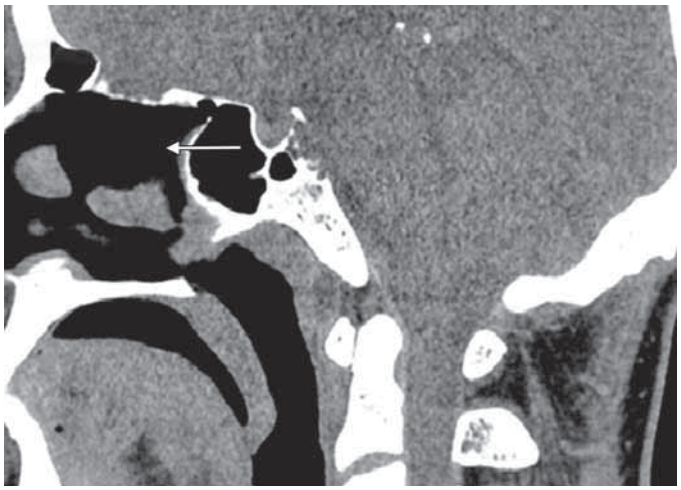

FIGURE 8: Sagittal computed tomography (CT) scan: Erosive lesion is appreciated on the dorsum sella in the retroclival location (arrow) reaching up till the posterior pituitary typical of ecchordosis physaliphora (a congenital benign hamartomatous lesion derived from notochord remnants).

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ACQUIRED DISORDERS

Among acquired causes of hypopituitarism, infective, inflammatory (primary or secondary to mass lesion or systemic inflammation), traumatic, and degenerative causes may lead to hypopituitarism in varying degrees. The degree and type of hormone deficiencies are variable and there may be discordance between clinical and radiological findings.

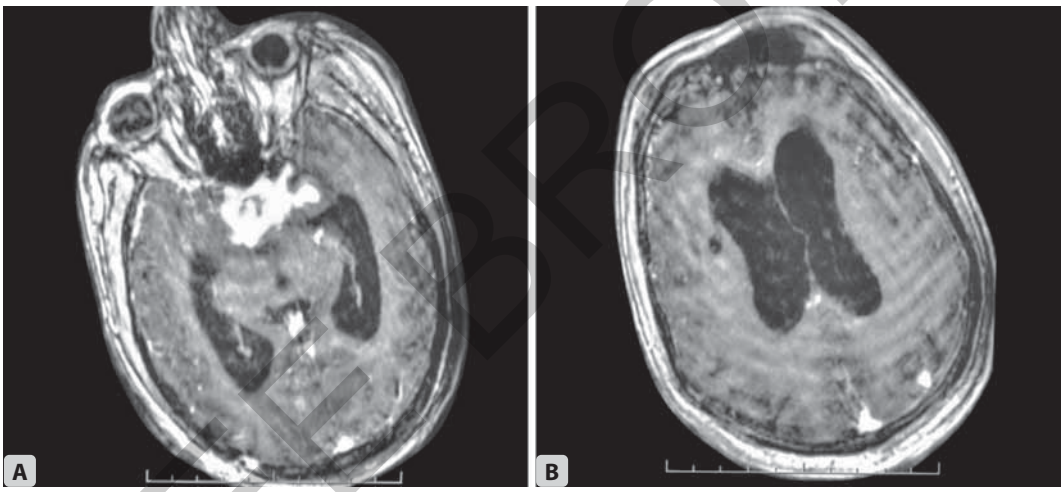
INFECTIVE

The risk factors for infections are meningitis, paranasal sinusitis (direct spread), immunocompromised host (hematogenous spread), and surgery. Tuberculosis may involve the pituitary gland either directly by tuberculomas or indirectly by hypophysitis (**Figs. 9** and **10**). Nearly 80% patients of tubercular meningitis are reported to have hypopituitarism at presentation with hyperprolactinemia and secondary hypoadrenalism being the most common patterns of presentation. Persistence of these abnormalities in nearly half of those following completion of antituberculous therapy suggests evaluation of hypothalamic–pituitary–adrenal (HPA) axis in all patients. Among other infections, syphilis (gumma), parasitic involvement by neurocysticercosis, *Pneumocystis carinii* and toxoplasma, viruses [herpes simplex virus (HSV), Varicella-zoster virus (VZV), and cytomegalovirus (CMV)] and fungi (*Candida* and *Aspergillus*) are also described.

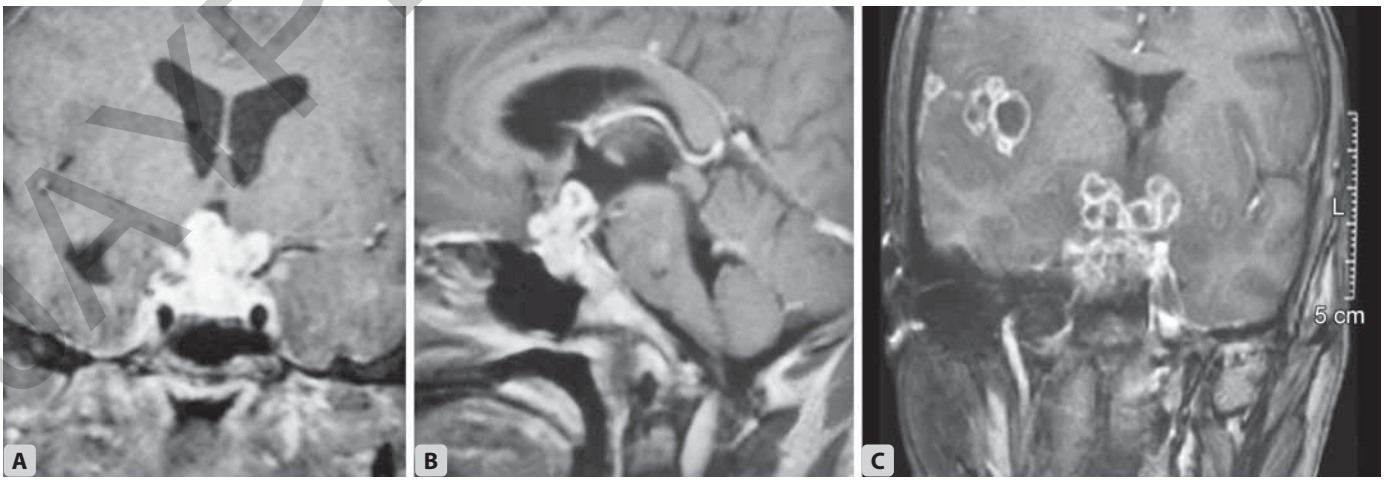
INFLAMMATORY

Primary hypophysitis: Primary inflammatory involvement of the pituitary is termed hypophysitis, which is of four types, as summarized in **Table 3**. **Figure 11** depict individual case of primary hypophysitis.

Secondary hypophysitis: Hypophysitis may be secondary to a number of conditions, including tumors, drugs, and hematopoietic malignancies, including lymphoma. Emerging causes include drug-induced inflammation secondary to drugs viz. immune checkpoint inhibitors (ICP) (anti-PD-1/anti-PD-L1). They cause an idiosyn-



FIGURES 9 A & B: **Basal exudates, hydrocephalus, and hypophysitis in tuberculous meningitis (TBM).**



FIGURES 10 A to C: **(A and B)** Gadolinium-enhanced magnetic resonance imaging (MRI) in the coronal and sagittal planes show suprasellar tuberculomas causing compression and encasement of the infundibulum and chiasma leading to vision loss; **(C)** MRI in the coronal plane in another patient shows conglomerate tuberculomas in the sellar-suprasellar region.

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TABLE 3 Patterns and spectrum of primary hypophysitis with diagnostic and management aspects.

Type of hypophysitis	Characteristic finding(s)	Imaging findings	Age and gender	Extrapituitary involvement	Management
LYH/ autoimmune hypophysitis	• ACTH, LH/FSH deficiency • Associated with pregnancy • Diffuse involvement of pituitary by lymphocytes and other inflammatory cells, later causing pituitary fibrosis and atrophy	• Diffuse pituitary enlargement (isointense on T1) • Stalk thickening • Contrast enhancement	• F:M 3:1 3rd to 4th decade • 75–85% of cases	• Hashimoto's • APS II • GD	• High-dose glucocorticoids • Surgery in nonresponders
IgG4/ plasmacytic	• Variable • Mononuclear infiltrate of plasma cells and lymphocytes and at least 10 IgG4 positive plasma cells/ high-power field (HPF) along with storiform fibrosis	Simultaneous involvement of sinuses and/or skull base can point towards IgG4 subtype	• M:F 2:1 • 6th to 7th decade • 30% of cases	• Retroperitoneal fibrosis • Exophthalmos	• High-dose glucocorticoids • AntiCD20 therapy (rituximab), mycophenolate or surgery in nonresponders
Granulomatous	Infiltration of gland by granulomas composed of multinucleated giant cells and histiocytes	• Diffuse pituitary enlargement (isointense on T1) • Stalk thickening	• F:M 3:1 • 5th decade • 20% of cases	-	• Glucocorticoids less effective • Surgery is preferred
Xanthomatous	Variable infiltrate of lipid laden foamy macrophages, histiocytes without granulomas	Hypointense on T1	• F:M 3:1 • 4th decade • 3–5% of cases		• Glucocorticoids less effective • Replacement of hormone deficiencies • Data is scarce
Necrotizing	Diffuse nonhemorrhagic necrosis with inflammatory infiltrate	Enlarged gland without contrast enhancement	<1% of cases		Replacement of hormone deficiencies
ICP induced (anti CTLA4 and anti PD-1/ PD-L1)	• ACTH, LH/FSH uncommon deficiency • PRL excess • Idiosyncratic reaction • Complement fixation • Hypersensitivity reactions (type II, IV)	Diffuse enlargement of the pituitary, contrast enhancement		• Thyroiditis or hypo/ hyperthyroidism • Hypoadrenalism (primary) • Fulminant diabetes • Nonendocrine associations like hepatitis	• Hormone replacement in mild cases • High-dose glucocorticoids in moderate-to-severe cases • In most cases, the drug can be continued

(ACTH: adrenocorticotropic hormone; APS: autoimmune polyglandular syndrome; FSH: follicle-stimulating hormone; GD: Graves' disease; ICP: immune checkpoint inhibitor; LH: luteinizing hormone; LYH: lymphocytic hypophysitis; PD-1/PD-L1: programmed death 1/programmed death ligand 1)

cratic pattern of hypophysitis, predominantly with hypocortisolism, hypogonadism, and hypothyroidism. Hyperprolactinemia and diffuse enlargement as well as contrast enhancement of the pituitary are classical of lymphocytic hypophysitis (LYH). Diabetes insipidus (DI) is also more common with LYH than with ICP-induced hypophysitis. Some cases of hypophysitis secondary to primary conditions like tumors or systemic inflammation are shown in **Figures 12** and **13**.

Isolated hypothalamic involvement, though rare, is also reported, leading to deficiencies of trophic hormones and consequently hypopituitarism (**Fig. 14**).

Among other causes of hypophysitis due to secondary or systemic causes, sarcoidosis, Langerhans cell histiocytosis, Wegener's granulomatosis, and lymphomatous involvement are rare but reported.

NEOPLASTIC

Pituitary adenomas are the third most common intracranial tumors, accounting for nearly 15–25% of all cases. A change in nomenclature to pituitary neuroendocrine tumors (PitNETs) has been recently suggested. Nonfunctioning pituitary adenomas (NFPAs) are the most common subtype and include gonadotropinomas, silent pituitary adenomas, or null cell adenomas. After the proposition of the transcription factor-based classification, it has been suggested that PitNETs form a continuum, varying from null cell adenomas to silent and whispering

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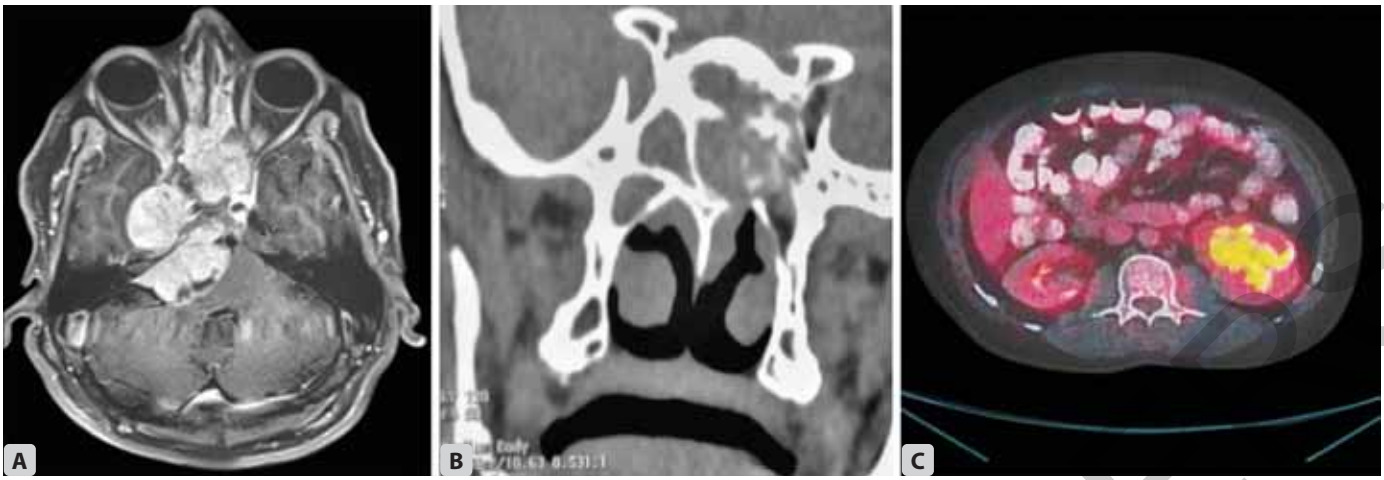
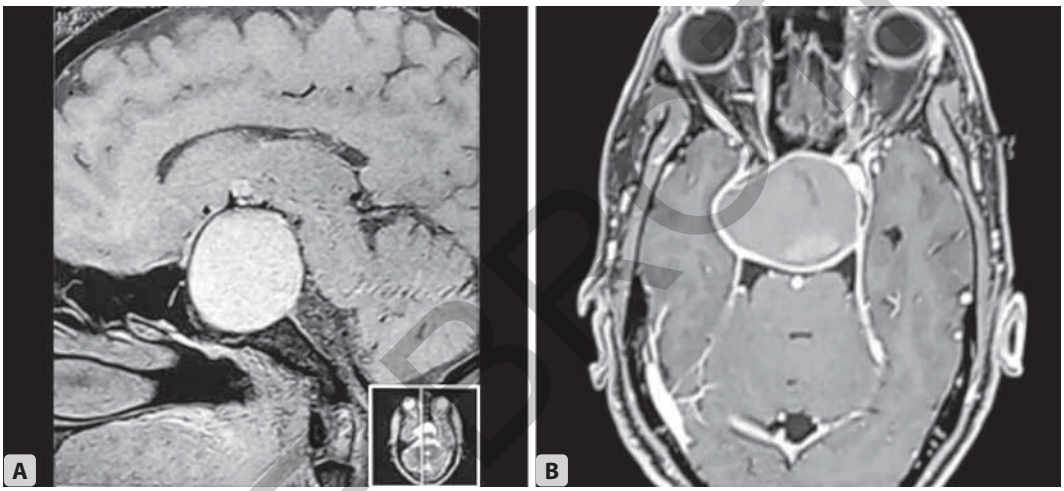


FIGURES 11 A to C: A 50-year-old man who presented with hemiparesis and involvement of multiple lower cranial nerves (VII, VIII, IX, and XI) was found to have an infiltrating mass in the sellar region on magnetic resonance imaging (MRI) with concomitant sinus involvement. Biopsy revealed immunoglobulin G4 (IgG4)-related disease and fluorodeoxyglucose positron emission tomography (FDG PET) showed focal lesion in the right kidney.



FIGURES 12 A & B: A 45-year-old male presented with headache, blurring of vision, and was found to have panhypopituitarism on evaluation. Magnetic resonance imaging (MRI) sagittal image depicts a large, cystic mass compressing the optic chiasma and extending up to the floor of the third ventricle with peripheral rim enhancement noted in the axial section. Following replacement with hydrocortisone, thyroid hormone, and testosterone, he was subjected to excision of the mass by transsphenoidal route and histopathology revealed xanthgranulomatous hypophysitis.

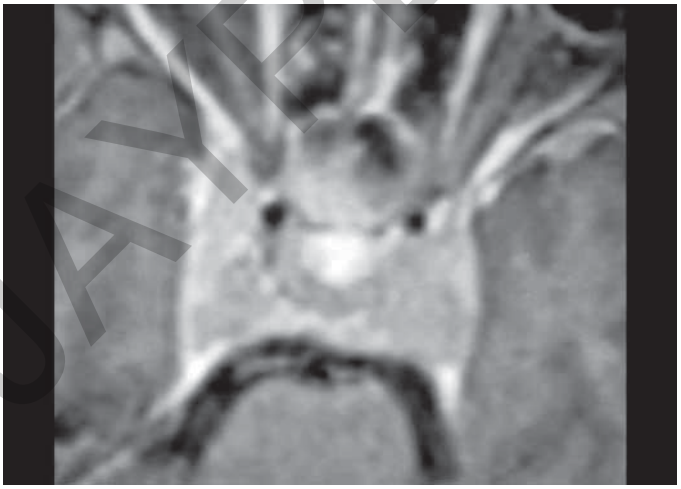


FIGURE 13: Diffuse sheet-like sellar-parasellar soft tissue extending on either side and partly encasing the internal carotid artery (ICA) without luminal compromise on this postcontrast T1 magnetic resonance (MR) image suggests a pliable tumor typically seen in lymphoma.

tumors to functional tumors (**Fig. 15**). Among the functional tumors, prolactinomas are the most common followed by somatotropinomas, corticotropinomas, and thyrotropinomas.

Various endocrinopathies are associated with these PitNETs due to hormonal hypersecretion associated with proliferation of specific subtypes of cells (**Table 4**). Mixed or multiple hormone production may also be present, for example, PRL hypersecretion in acromegaly (due to acidophil stem cell adenoma, mammosomatotroph adenoma, or mixed GH and PRL secreting adenoma). In case of other tumors, immunohistochemistry helps in diagnosis of multiple hormone positivity in a given tumor.

All tumors with macroadenomas should be subjected to assessment of visual acuity, visual fields at baseline, and at follow-up to determine remission for planning adjuvant therapy and/or surveillance for recurrence.

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FIGURES 14 A & B: A 50-year-old female presented with cognitive dysfunction and diencephalic syndrome. She had past history of bronchial asthma for which she was on inhaled steroids intermittently. She was found to have multiple pituitary hormone deficiency (MPHD) and central diabetes insipidus. Magnetic resonance imaging (MRI) T1-weighted sagittal section showing an intensely enhancing lesion in the hypothalamus with sparing of the pituitary. She was managed with high-dose glucocorticoids, which were tapered, besides hormone replacement for MPHD and oral desmopressin.

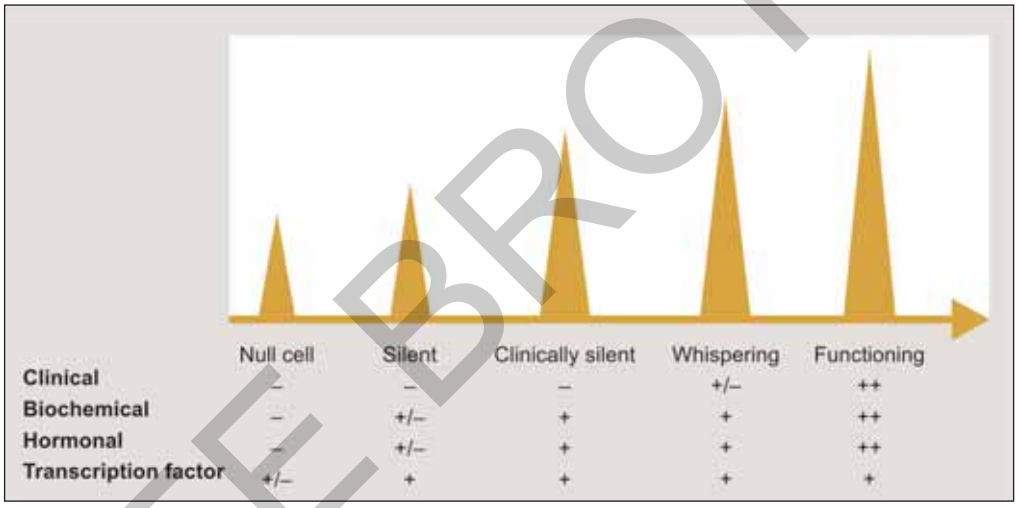

FIGURE 15: Proposed spectrum of pituitary neuroendocrine tumors (pituitary adenomas) based on clinical and biochemical features, hormonal immunohistochemistry, and transcription factors.

Source: Adapted from Drummond J, Roncaroli F, Grossman AB, Korbonits M. Clinical and pathological aspects of silent pituitary adenomas. The Journal of Clinical Endocrinology and Metabolism. 2019;104(7):2473-89.

TABLE 4 Investigations for hormone hypersecretion syndromes in pituitary adenomas.

PitNET-associated endocrinopathy	Screening/Confirmatory test and further testing
Prolactinoma	• Prolactin (in dilution), pooled sample preferable • CEMRI sella • Rule out secondary causes in case of equivocal prolactin elevation (<200 ng/mL)
Acromegaly	• IGF-1 (above the age matched range) • GH not suppressible to 1 ng/mL on 75 g OGTT • CEMRI sella
Cushing's disease	• Screening tests (two of four including UFC, ONDST, LNPC/LNSC, LDDST) • ACTH dependency • CEMRI sella • IPSS (+/-) • CECT chest and abdomen, Ga68 DOTANOC (to rule out ectopic source of ACTH excess)

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