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Pituitary Adenoma

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Continuing Education Activity

Pituitary adenomas are common, usually benign tumors of the anterior pituitary that present with symptoms of hormone excess, hormone deficiency, or mass effect. Microadenomas are often incidental findings, while macroadenomas may cause visual impairment, headaches, or hypopituitarism from compression of surrounding structures. Functioning adenomas, including prolactinomas, corticotrope adenomas, and somatotrope adenomas, produce characteristic clinical syndromes such as infertility, Cushing disease, and acromegaly, which are associated with significant long-term complications if untreated. Diagnosis relies on pituitary protocol, magnetic resonance imaging, and comprehensive endocrine testing. Management involves individualized therapy, including medical treatment, transsphenoidal surgery, or radiotherapy, depending on tumor type and functional status.

This activity enhances clinician competence in recognizing pituitary adenomas' pathophysiology, clinical presentation, and management. Participants improve their ability to interpret diagnostic studies, implement evidence-based treatment strategies, and counsel patients regarding long-term monitoring, recurrence, and therapy effects. Interprofessional collaboration among endocrinologists, neurosurgeons, neuro-ophthalmologists, radiation oncologists, nurses, and pharmacists strengthens outcomes through coordinated evaluation, treatment optimization, and patient education. Enhanced communication and shared expertise promote early detection of complications, personalized care planning, and improved quality of life for patients with pituitary tumors.

Objectives:

- Identify clinical presentations and imaging findings suggestive of pituitary adenomas to enable timely diagnosis.
- Interpret biochemical test results, including hormone levels, to differentiate functioning from nonfunctioning pituitary adenomas.
- Evaluate the risks, benefits, and expected outcomes of surgical, medical, and radiotherapeutic options for patients with pituitary adenomas.
- Collaborate within an interprofessional team, including endocrinologists, neurosurgeons, neuro-ophthalmologists, nurses, and pharmacists, to optimize coordinated care and improve patient outcomes.

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Introduction

Pituitary adenomas are tumors of the anterior pituitary, most of which are indolent and benign. They are classified based on size or cell of origin. Based on size, pituitary adenomas are categorized as microadenomas (<10 mm), macroadenomas (≥10 mm), and giant adenomas (>40 mm). Functionally, they may be divided into hormone-secreting (functional) adenomas, which

produce excess amounts of one or more anterior pituitary hormones, and nonfunctional adenomas, which do not secrete biologically active hormones.

Nonfunctioning tumors can cause symptoms through mass effect, including compression of normal pituitary tissue or surrounding structures, leading to hormone deficiencies or visual impairment. Evaluation and management of pituitary adenomas typically require an interdisciplinary team involving endocrinologists, neurosurgeons, ophthalmologists, and radiation specialists when indicated.[1][2]

Etiology

The pathogenesis of pituitary adenoma remains incompletely understood; epigenetic changes that disrupt cell cycle regulation appear central to tumor development.[3] Familial cases represent 5% of all pituitary tumors.[4] Several germline mutations are implicated in the syndromic and nonsyndromic forms of familial pituitary adenomas:

- **Multiple endocrine neoplasia type 1 (*MEN1*)**: *MEN1* is a tumor suppressor gene. Loss-of-function mutation in this gene leads to tumor formation in the parathyroid, pancreatic, and pituitary glands.[5]
- **Multiple endocrine neoplasia type 4 (*MEN4*)**: *MEN4* involves mutations in the cyclin-dependent kinase inhibitor 1B (*CDKN1B*) gene, leading to pituitary tumors, hyperparathyroidism, and neuroendocrine tumors in the testes and cervix.[6]
- **Carney complex**: The Carney complex is caused by germline mutations in the tumor suppressor gene *PRKAR1A*, which result in primary pigmented nodular adrenocortical disease, testicular tumors, thyroid nodules, spotty skin hyperpigmentation, and acromegaly.[7]
- **Familial isolated pituitary adenomas**: This hereditary condition refers to pituitary adenomas that occur in 2 or more family members without other syndromic features. These tumors, which are often aggressive and appear at a young age, are typically growth hormone- or prolactin-secreting adenomas. Familial isolated pituitary adenomas are linked to mutations in the aryl hydrocarbon receptor–interacting protein (*AIP*) gene, X-linked acrogigantism due to *GPR101* duplication, or cases without identifiable gene mutations.[8]
- **Succinate Dehydrogenase Mutation (*SDHx*)**: *SDHx* mutations are associated with paragangliomas, pheochromocytomas, and pituitary adenomas.
- **Neurofibromatosis type 1**
- **DICER1 syndrome**: Germline mutations in the *DICER1* gene predispose to conditions such as infant pulmonary blastoma, differentiated thyroid cancer, nasal hamartomas, sex cord tumors, and pituitary blastoma.

Somatic mutations are reported in 40% of pituitary adenomas:

- **Guanine nucleotide–binding protein, α-stimulating complex locus (*GNAS*) mutation**: This somatic gain-of-function mutation, commonly found in growth hormone-producing tumors, affects the *GNAS* gene, which encodes Gsα subunits.
- **Ubiquitin-specific protease 8 (*USP8*) mutation**: In Cushing disease, somatic gain-of-function mutations in *USP8* lead to increased recycling of epidermal growth factor.
- **Phosphoinositide 3-kinase catalytic subunit (*PIK3CA)* mutation**: Point mutations in the *PIK3CA* gene are reported across all types of pituitary adenomas.

- **Splicing factor 3 subunit B1 *(SF3B1R625H)*:** Somatic mutation in *SF3B1R625H is* identified in 20% of prolactinomas and is potentially associated with aggressive behavior.

Epidemiology

Pituitary adenomas are often found incidentally during imaging.[9] Most incidental findings are microadenomas, which are usually clinically silent. Due to their frequently insidious presentation, small size, and incidental detection, estimating the true prevalence of pituitary adenomas in the general population remains challenging. Reported prevalence varies depending on the source. Results from a meta-analysis showed an average frequency of pituitary adenomas of 16.7%, with a prevalence of 14.4% in autopsy studies and 22.5% in radiological series.[10] Data from the Central Brain Tumor Registry of the United States indicate that pituitary tumors account for 17.2% of all primary brain tumors.[11]

In contrast, clinically relevant pituitary adenomas secrete hormones, causing recognizable syndromes or grow large enough to cause compressive symptoms. Prolactinomas are the most common subtype. Clinically relevant pituitary adenomas occur at a significantly lower rate, with a prevalence of 89.1 per 100,000 persons, of which 47.8% are classified as macroadenomas.[9][12][13][14]

- Prolactinomas (approximately 50% of cases)
- Nonfunctioning adenomas (30%)
- Somatotroph adenomas (11%)
- Corticotroph adenomas (5%)
- Thyrotroph and gonadotroph adenomas (rare) [9][12][13][14]

Sex distribution varies by the subtype of pituitary adenoma. Prolactinomas are most commonly diagnosed in women of reproductive age, whereas acromegaly is more often diagnosed in men. Pituitary incidentalomas are more commonly detected in adults aged 30 to 60 and are relatively rare in children. In pediatric populations, Rathke cleft cysts are the most frequent incidental sellar mass, followed by nonfunctioning pituitary adenomas.[15]

History and Physical

The clinical presentation of pituitary adenoma depends on tumor size and functional status. A comprehensive history and physical examination are needed to determine the tumor's systemic impact.[1][2][11][16][17][18] Pituitary microadenoma is usually found incidentally on an MRI of the brain. Patients are usually asymptomatic unless the tumor is hormonally active. In contrast, pituitary macroadenoma presents with a mass effect, potentially resulting in hormonal deficiency or excess. Pituitary apoplexy, a rare but acute hemorrhage into a pituitary adenoma, presents with symptoms of a mass effect, including headaches, vision changes, and hormonal deficiency.

Symptoms From Mass Effect

- **Visual impairment:** Visual impairment occurs in approximately 40% to 60% of patients from suprasellar extension of the pituitary adenoma, which compresses the optic chiasm, leading to visual field defects (bitemporal hemianopia). Bitemporal defect is the most prevalent pattern, followed by homonymous defects. Involvement of the oculomotor nerve causes diplopia, and invasive tumors may compromise cranial nerves IV, V, and VI.[16][19][20]

- **Headache:** Headache is a nonspecific and commonly reported symptom in pituitary adenoma.[20]
- **Hormonal deficiency:** One or more anterior pituitary hormonal deficiencies can be observed in patients with pituitary macroadenoma:
    - Gonadotropin deficiency presents as amenorrhea in women and erectile dysfunction in men.
    - Adults ' growth hormone (GH) deficiency leads to fatigue and weight gain.
    - Thyroid-stimulating hormone (TSH) deficiency symptoms are weight gain, fatigue, cold intolerance, and constipation.
    - Adrenal corticotropic hormone (ACTH) deficiency presents with fatigue, arthralgia, weight loss, hypotension, dizziness, nausea, vomiting, and abdominal pain.

Functioning Adenomas

The clinical presentation depends on the hormone secreted:

- **Prolactin-secreting adenoma:** Elevated prolactin levels suppress the gonadotropin levels, leading to infertility, decreased libido, and osteoporosis in men and women. Women present with amenorrhea and galactorrhea, while men present with erectile dysfunction and gynecomastia.
- **Growth hormone-secreting adenoma (acromegaly):** The clinical presentation is characterized by headaches, vision changes, an increase in ring or shoe size, arthritis, carpal tunnel syndrome, and hyperhidrosis. Characteristic physical findings include coarse facial features, frontal bossing, prognathism, macroglosia, and skin tags. Comorbidities include hypertension, cardiomyopathy, obstructive sleep apnea, and multiple colonic polyps.
- **Adrenal corticotropic hormone-secreting adenoma (Cushing disease):** Presents with weight gain, muscle weakness, mood disorders, easy bruising, and multiple fractures. Physical examination findings include moon facies, facial plethora, supraclavicular fat, ecchymoses, and purple striae on the abdominal area and armpits.
- **Thyroid-stimulating hormone-secreting adenoma:** Patients typically present with symptoms including palpitations, arrhythmias, and weight loss. Upon physical examination, they may have tremors and a goiter.

Evaluation

The initial evaluation of a pituitary adenoma involves a pituitary protocol MRI and comprehensive hormonal assessment.

Imaging

While many pituitary adenomas are detected incidentally on routine head CT scans, contrast-enhanced MRI provides superior resolution and is the gold standard. Gadolinium-enhanced coronal and sagittal T1-weighted images are recommended to differentiate an adenoma from other sellar lesions, such as an aneurysm, Rathke cleft cyst, or hemorrhage. A pituitary-dedicated MRI provides information on the tumor size, shape, consistency, optic chiasm compression, and cavernous sinus invasion.

Hormonal Evaluation

The Endocrine Society clinical practice guidelines recommend a complete biochemical assessment in all patients with incidentally discovered pituitary adenomas. This testing includes serum prolactin, thyrotropin (TSH), free thyroxine (T4), insulin-like growth factor-1 (IGF-1), GH, follicle-stimulating hormone (FSH), estradiol, testosterone, ACTH, morning cortisol, and a basic metabolic panel.[1][2][11][16][17][19][21]

Evaluation for hormonal excess:

- **Prolactin:** The prolactin level ideally should be obtained in the morning. A serum prolactin level of less than 200 ng/mL is seen in microadenoma, stalk effect from macroadenoma, hypothyroidism, renal failure, pregnancy, lactation, chest wall trauma, or use of medications including antipsychotics, antidepressants, opiates, and antiemetics. A prolactin level greater than 200 ng/mL is usually associated with macroadenoma. If a patient is asymptomatic, consider macroprolactin (also known as "big prolactin") an inactive form of prolactin. Detection of macroprolactin is essential to avoid unnecessary treatment. The diagnosis of macroprolactin involves precipitation of the serum with polyethylene glycol. The presence of free prolactin at levels greater than 60% confirms the diagnosis of a prolactinoma. Consider the hook effect in patients with a giant adenoma and mildly elevated prolactin, an assay artifact that can lead to falsely low levels.
- **IGF-1/GH:** Serum IGF-1 is the preferred screening test for the diagnosis of acromegaly, and should be interpreted using age- and sex-adjusted reference ranges. Isolated GH measurement, fasting or random, is not helpful due to the pulsatile secretion and short half-life (14 minutes). If the IGF-1 level is mildly elevated or equivocal, a 75-g oral glucose tolerance test may be performed. A nonsuppressed GH level greater than 0.4 to 1 ng/mL (assay-dependent) with hyperglycemia confirms acromegaly. However, IGF-1 and GH levels can vary in several conditions and due to medications. Both IGF-1 and GH levels can be elevated in late puberty, pregnancy, and with exogenous testosterone administration. IGF-1 and GH decrease with increasing age and obesity. IGF-1 levels are reduced in anorexia, liver and kidney disease, and poorly controlled diabetes. Oral estrogen contraceptives decrease IGF-1 levels, but transdermal estrogen usually does not.
- **Cortisol:** Morning or random cortisol is not diagnostic due to the diurnal rhythm. The screening tests for Cushing disease include late-night salivary cortisol, 24-hour urine-free cortisol, or a 1-mg overnight dexamethasone suppression test. If performed accurately, late-night salivary cortisol has a sensitivity and specificity greater than 90%, but it is invalid in patients with disrupted sleep cycles. Urine-free cortisol requires accurate 24-hour urine collection with creatinine. The results can be affected by renal impairment, urine volume, and body mass index. The overnight dexamethasone suppression test involves administering 1 mg of dexamethasone at 11:00 PM, followed by an early morning cortisol measurement. A cortisol level of 1.8 µg/dL or greater suggests hypercortisolism. False-positive results may occur with oral estrogen and drugs that induce dexamethasone metabolism (eg, phenytoin, phenobarbital). Interpretation of the screening test should account for confounding factors such as exogenous steroids, depression, excess alcohol intake, and oral contraceptives. Once hypercortisolism is confirmed biochemically, ACTH levels are obtained to establish the etiology. Hypercortisolism with elevated ACTH suggests a corticotrope adenoma versus ectopic Cushing syndrome. Because ACTH-producing adenomas are small, an MRI of the brain may yield normal findings in approximately 50% of patients with these tumors. For suspected microadenomas measuring less than 6 mm or for cases with inconclusive imaging, inferior petrosal sinus sampling is recommended to differentiate between ectopic and pituitary Cushing syndrome. This procedure involves catheterizing the bilateral petrosal sinuses with measurement of ACTH before and after vasopressin stimulation. In patients with Cushing

disease, corticotropin-releasing hormone and vasopressin are overexpressed; therefore, vasopressin increases ACTH levels. A central-to-peripheral ACTH gradient greater than 2 at baseline and 3 after vasopressin indicates a pituitary source.

- **TSH/free T4:** A thyrotropin-secreting adenoma will produce elevated free T4 and T3 with inappropriately normal or elevated TSH levels.

Evaluation for hormonal deficiency:

- **Gonadotropin (luteinizing hormone [LH]/follicle-stimulating hormone [FSH])**: Low estradiol or testosterone levels with normal or low LH and FSH levels suggest hypogonadotropic hypogonadism. Interpretation of sex hormone levels is inaccurate in women if they are on oral contraceptives. Postmenopausal women have elevated FSH levels; thus, inappropriate low gonadotropin levels indicate pituitary dysfunction.
- **IGF-1/GH:** In adults, isolated GH deficiency should be further evaluated with provocative testing, such as insulin-induced hypoglycemia or glucagon stimulation tests. Conditions such as poorly controlled diabetes, malnutrition, sepsis, hypothyroidism, and hepatic or renal failure can impact the IGF-1 and GH levels.
- **Cortisol:** Early morning cortisol levels can help assess the function of the hypothalamic-pituitary-adrenal axis. A morning cortisol level greater than 10 µg/dL suggests an intact hypothalamic-pituitary-adrenal axis function. A cosyntropin stimulation test should be performed if morning cortisol levels are equivocal or low.
- **TSH/free T4:** Low free T4 with normal or low TSH suggests secondary hypothyroidism.

Treatment / Management

The management of pituitary adenomas requires collaboration between an endocrinologist and a neurosurgeon to develop an individualized, patient-centric approach.[1][2][11][17][19][21][19][22][23][24]

Treatment of Nonfunctioning Adenomas

Transsphenoidal resection is recommended in patients with macroadenomas and the following:

- Visual field deficit due to a tumor
- Other visual abnormalities, such as ophthalmoplegia
- Compression of the optic nerves or chiasm on imaging
- Pituitary apoplexy with visual disturbance
- Loss of endocrine function
- Progressive tumor growth

Improvement in visual symptoms and hormonal dysfunction occurs in most patients after surgery. Radiotherapy is considered for patients with residual or recurrent tumors. For nonfunctioning adenomas that do not require surgical management, regular evaluation by endocrinology is essential to monitor tumor growth and potential hypopituitarism. The timing of repeat MRI depends on the adenoma size, patient age, and tumor consistency. A follow-up MRI can be performed 2 to 3 years after the initial scan for microadenomas. For macroadenomas located more than 5 mm from the optic chiasm, a follow-up MRI is recommended after 1 year. The interval can be extended to every 2 to 3 years if the tumor remains stable. However, for macroadenomas located within 5 mm of the optic chiasm, the first follow-up MRI should be

conducted within 6 to 12 months after surgery. If the tumor remains stable, subsequent MRIs are recommended every 1 to 2 years.

Treatment of Functioning Adenomas

Prolactin-secreting adenoma:

The primary goals of treating prolactin-secreting adenomas are to restore gonadal function and reduce tumor size. Observation with periodic monitoring of prolactin level may be appropriate for asymptomatic individuals with microadenoma.[25]

- **Medical therapy:** Dopamine agonists (DAs) are the primary treatment for prolactin-secreting tumors. The currently available DAs are cabergoline and bromocriptine. Cabergoline is more than 90% effective in normalizing prolactin levels and decreasing tumor size. The adverse effects of DAs are postural hypotension, valvular cardiac abnormalities, and compulsive behavior or mood changes. DAs can be discontinued after 2 years of treatment if the MRI brain findings do not demonstrate a visible tumor. However, annual monitoring of serum prolactin levels is recommended due to the risk of recurrence or tumor growth following cessation of treatment.
- **Surgery:** Transsphenoidal surgery is reserved for prolactin-secreting tumors that are resistant to medical treatment, patients who develop adverse effects from dopamine agonists, and tumors larger than 1 cm in patients desiring pregnancy. In recently updated guidelines, surgery can also be considered in patients with microprolactinomas or a well-encapsulated macroprolactinoma.
- **Radiation therapy:** Radiotherapy is occasionally used for aggressive prolactinomas after surgeries and medical treatment have failed to control the size.

GH-secreting adenoma:

The goal of treatment is to decrease growth hormone levels to less than 1 μg/L and normalize IGF-1 levels.[26][27]

- **Medical therapy:** Medical treatment is indicated in patients with persistently elevated IGF-1 levels 3 months postoperatively or for nonsurgical candidates with invasive tumors. Somatostatin analogs (SSAs) are the mainstay of treatment for acromegaly, as they act on somatostatin receptors to decrease GH secretion. SSAs are available as long-acting depot injections (octreotide, lanreotide, and pasireotide) or oral octreotide capsules. The adverse effects of SSAs include cholecystitis, abdominal cramps, flatulence, diarrhea, and alopecia. Pasireotide long-acting release can lead to hyperglycemia in 50% to 70% of patients. DAs like cabergoline are used for mildly elevated IGF-1 levels postoperatively or as an adjunct therapy with SSAs. If the GH level remains elevated, GH receptor antagonists (pegvisomant) can be used in combination with SSAs or alone.
- **Surgery:** Transsphenoidal surgery is the first-line treatment for GH-secreting tumors. Normalization of IGF-1 levels is achieved in 80% to 90% of patients with microadenomas and 40% to 60% of patients with macroadenomas.
- **Radiation therapy**: Radiation therapy may be considered as an adjunct for patients with persistently elevated IGF-1 levels after surgery; however, clinical effects may take several years to manifest.

ACTH-secreting adenoma:

Treatment aims to rapidly decrease cortisol levels and reduce the associated complications and mortality.[28]

- **Surgery:** Transsphenoidal surgery is the first-line treatment for Cushing disease. The cure rates are 70% to 90%. Remission is typically defined by a cortisol level of less than 2 µg/dL (55 nmol/L). Patients often require temporary glucocorticoid replacement therapy until the hypothalamic-pituitary-adrenal axis recovers, which may take up to 12 months. Monitoring morning cortisol levels every few months after discontinuation of glucocorticoids helps assess recovery.
- **Medical therapy:** In Cushing disease, medical treatment is used for persistent disease, recurrence, or as a bridge to definitive treatment. Medical therapy includes pituitary-directed therapy, adrenal steroidogenesis inhibitors, and glucocorticoid receptor antagonists. Pituitary-directed therapy includes dopamine agonists (such as cabergoline) and somatostatin analogues (such as pasireotide). Adrenal steroidogenesis inhibitors include metyrapone, mitotane, levoketoconazole, osilodrostat, and etomidate. The glucocorticoid receptor blocker mifepristone is approved for use in Cushing syndrome with hyperglycemia.
- **Bilateral adrenalectomy:** Bilateral adrenalectomy is considered in patients with persistent or recurrent hypercortisolism refractory to surgery and medical therapy. The procedure provides immediate control of cortisol excess but results in adrenal insufficiency requiring lifelong treatment. Nelson syndrome, which is radiological pituitary tumor enlargement, occurs in 25% to 40% of patients after adrenalectomy.
- **Radiation therapy:** Radiation is an adjunctive treatment for residual or recurrent disease. The effects are delayed, often requiring months to years for biochemical remission.

TSH-secreting adenoma

Transsphenoidal surgery is the initial preferred treatment, leading to a cure in 50% to 90% of patients. Control of hyperthyroidism preoperatively avoids thyroid storm. Presurgical euthyroidism is achieved by using antithyroidal medical therapy such as methimazole or SSAs. Patients who are not cured by surgery can be treated with SSA therapy alone or combined with radiotherapy to decrease TSH levels and tumor size.

Differential Diagnosis

The differential diagnosis includes other sellar or suprasellar masses:

- Arachnoid cyst
- Basilar artery thrombosis
- Brainstem glioma
- Cavernous sinus syndrome
- Cerebral venous thrombosis
- Craniopharyngioma
- Dermoid cyst
- Ependymoma
- Glioblastoma multiforme
- Leptomeningeal carcinomatosis
- Low-grade astrocytoma

- Meningioma
- Primary central nervous system lymphoma
- Rathke cleft cyst
- Tuberculous meningitis

Prognosis

The prognosis of pituitary adenoma largely depends on whether the tumor is functioning or nonfunctioning. Nonfunctioning adenomas and prolactinomas have an excellent prognosis if treated promptly with surgery or medical therapy. In contrast, functioning adenomas like Cushing disease and acromegaly are associated with several comorbidities and long-term complications. Increased mortality is associated with delayed medical or surgical treatment, especially in patients with Cushing disease.

Deterrence and Patient Education

Pituitary adenomas are common, usually benign tumors that may cause symptoms through a mass effect or by affecting hormone levels. Proper evaluation requires an interdisciplinary team to manage pituitary disease, including an endocrinologist, neurosurgeon, neuro-ophthalmologist, and occasionally a radiation oncologist. Patient education is a key component of management. Patients should be instructed on when to seek medical care after a diagnosis of pituitary adenoma. Education about the adverse effects of medical therapy is also important in helping to prevent complications and mortality.

Enhancing Healthcare Team Outcomes

Criteria defining Pituitary Centers of Excellence were released in a statement by the Pituitary Society in 2017. These guidelines recommend a structured approach with a leading team comprising an endocrinologist and a neurosurgeon working in close collaboration during the initial evaluation. Centers of excellence should also have supporting units of neuroradiology, neuropathology, radiation oncology, and neuro-ophthalmology.[29]

Specialty care nurses monitor patients, provide patient and family education, and help coordinate care. Pharmacists review medication dosages and check for drug interactions. If the tumor is not benign, a board-certified oncology pharmacist should consult with the oncology clinician team to assist with selecting the appropriate agent. Through these interprofessional efforts, patient outcomes will continue to improve.

Review Questions

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