

2011 Imaging Criteria

Magnetic Resonance Imaging (MRI), Pituitary⁽¹⁾

ICD-9-CM: 88.91

CPT: 70551, 70552, 70553

I/O Setting: Outpatient

INDICATION(S)

- 100 Suspected acromegaly
- 200 Cushing's disease
- 300 Secondary (central) hyperthyroidism
- 400 Hyperprolactinemia
- 500 Secondary (central) adrenal insufficiency
- 600 Secondary (central) hypothyroidism
- 700 Hypogonadotropic hypogonadism
- 800 Suspected neurogenic diabetes insipidus
- 900 Visual field defect
- 1000 Suspected pituitary apoplexy ♦
- 1100 Abnormal sella turcica by x-ray/CT
- 1200 Known pituitary mass/cyst

- 100 Suspected acromegaly [**One**]^(2, 3, 4)
 - 110 IGF-1 (somatomedin-C) level > normal⁽⁵⁾
 - 120 GH level > 2 mcg/L after glucose load^(6, 7)
- 200 Cushing's disease [**Both**]⁽⁸⁾
 - 210 Elevated ACTH and cortisol levels⁽⁹⁾
 - 220 Suppression by high-dose dexamethasone⁽¹⁰⁾
- 300 Secondary (central) hyperthyroidism [**All**]
 - 310 Thyroid hormone > normal [**One**]
 - 311 Free T₄
 - 312 Free thyroxine index
 - 313 T₃
 - 320 Inappropriately elevated TSH⁽¹¹⁾
 - 330 Lab results confirmed ≥ 2x
 - 340 No supplemental thyroid Rx by Hx
- 400 Hyperprolactinemia [**All**]⁽¹²⁾

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- 410 Prolactin concentration > normal
- 420 TSH normal⁽¹³⁾
- 430 Medications deemed noncontributory⁽¹⁴⁾
- 440 No seizure w/in 24 hrs⁽¹⁵⁾
- 450 Patient characteristics [**One**]
 - 451 Male patient
 - 452 Female patient [**Both**]
 - 1 Serum/urine HCG [**One**]⁽¹⁶⁾
 - A) Negative
 - B) Not indicated⁽¹⁷⁾
 - 2 No childbirth/breastfeeding w/in 12 wks by Hx⁽¹⁸⁾
- 500 Secondary (central) adrenal insufficiency [**Both**]⁽¹⁹⁾
 - 510 ACTH level < normal⁽²⁰⁾
 - 520 Abnormal screening test [**One**]
 - 521 AM cortisol \leq 3 mcg/dL⁽²¹⁾
 - 522 ACTH (Cortrosyn) stimulation test abnormal⁽²²⁾
- 600 Secondary (central) hypothyroidism [**Both**]⁽²³⁾
 - 610 Thyroid hormone < normal [**One**]
 - 611 Free T₄
 - 612 Free thyroxine index
 - 620 TSH \leq normal
- 700 Hypogonadotropic hypogonadism [**One**]⁽²⁴⁾
 - 710 Male [**Both**]
 - 711 Testosterone < normal⁽²⁵⁾
 - 712 FSH/LH inappropriately low⁽²⁶⁾
 - 720 Premenopausal female [**All**]
 - 721 Clinical Sx/findings [**One**]
 - 1 Oligomenorrhea
 - 2 Amenorrhea
 - 3 Infertility
 - 722 FSH/LH inappropriately low⁽²⁷⁾
 - 723 Estrogen \leq normal (premenopausal range)
 - 724 No OCPs by Hx
 - 730 Postmenopausal female [**All**]⁽²⁸⁾
 - 731 FSH/LH inappropriately low⁽²⁷⁾
 - 732 Estrogen \leq normal (menopausal range)
 - 733 No HRT by Hx

- 800 Suspected neurogenic diabetes insipidus **[All]**⁽²⁹⁾
 - 810 Serum osmolality \geq 280 mOsm/kg
 - 820 Urine sp.gr. < 1.005
 - 830 ADH < normal
 - 840 Abnormal screening test **[One]**⁽³⁰⁾
 - 841 Water deprivation test⁽³¹⁾
 - 842 Vasopressin test⁽³²⁾

- 900 Visual field defect **[One]**⁽³³⁾
 - 910 Bitemporal hemianopsia⁽³⁴⁾
 - 920 Bilateral superior/inferior temporal visual defect
 - 930 Unilateral temporal visual defect

- 1000 Suspected pituitary apoplexy **◆**⁽³⁵⁾

- 1100 Abnormal sella turcica by x-ray/CT⁽³⁶⁾

- 1200 Known pituitary mass/cyst **[One]**^(4, 37)
 - 1210 Follow-up post neurosurgical procedure⁽³⁸⁾
 - 1220 Follow-up post medical Rx **[One]**⁽³⁸⁾
 - 1221 Radiation
 - 1222 Medication
 - 1230 New/worsening Sx/findings **◆**

Notes

(1)

The following are examples of relative and absolute contraindications to the use of magnetic resonance imaging:

- Implanted devices that are electrically or magnetically activated (e.g., cardiac pacemakers, automatic cardioverter defibrillators, drug infusion pumps, cochlear implants)
- Ferromagnetic metal objects (e.g., cerebral aneurysm clips, intraocular metallic foreign body, prostheses, screws)
- Pregnancy, first trimester
- Renal insufficiency in cases when magnetic resonance imaging is performed with gadolinium-based contrast

(2)-DEF:

Acromegaly is a disorder of GH hypersecretion most often secondary to a pituitary adenoma. Clinical signs include enlargement of the hands, feet and forehead, excessive sweating, weakness, arthralgias, new skin tags, HTN, and glucose intolerance.

(3)

Because the skeletal defects associated with acromegaly are irreversible, and larger pituitary tumors are more difficult to treat, it is best to make the diagnosis as early as possible. Diagnosis rests on the measurement of serum IGF-1 (somatomedin-C) concentrations or GH levels following oral glucose administration (Ben-Shlomo and Melmed, *Endocrinol Metab Clin North Am* 2008; 37(1): 101-122, viii).

(4)

Pituitary MRI with contrast is most sensitive for determining a pituitary source of GH oversecretion and detecting tumors as small as 2 mm. Additionally, pituitary MRI can visualize tumor dimensions, extent of tumor, and proximity to the optic chiasm (Ben-Shlomo and Melmed, *Endocrinol Metab Clin North Am* 2008; 37(1): 101-122, viii; Doerfler A and Richter G, *Clin Neuroradiology* 2008; 18(1): 5-18; Chandler and Barkan, *Endocrinol Metab Clin North Am* 2008; 37(1): 51-66, viii).

(5)

IGF-1 (somatomedin-C) is a hormone made primarily by the liver in response to GH. It is thought to be the primary factor responsible for most of the clinical manifestations of acromegaly. Serum IGF-1 concentrations (values need to be interpreted according to age and gender) are elevated in virtually all cases. Other physiologic factors that influence IGF-1 concentrations include circadian rhythm, nutrition, insulin, thyroxine, and steroid levels (Ben-Shlomo and Melmed, *Endocrinol Metab Clin North Am* 2008; 37(1): 101-122, viii).

(6)

Pituitary GH-secreting adenomas are responsible for 98% of acromegaly and almost exclusively are benign (Ben-Shlomo and Melmed, *Endocrinol Metab Clin North Am* 2008; 37(1): 101-122, viii).

(7)

Measurement of GH 60 to 120 minutes following an oral glucose load (75 to 100 g) is done in making a diagnosis of acromegaly. Normally, GH will be suppressed in this setting. In acromegaly, however, levels typically exceed 100 mcg/L at baseline and rarely will they suppress below 2 mcg/L. Because the secretion of GH is pulsatile, random GH levels are difficult to interpret and should not be used as a diagnostic assay. Instead, GH values should be measured following a glucose load. In addition, false elevation of GH levels can be found in disease states other than acromegaly (e.g., DM, renal failure, liver disease) (Ben-Shlomo and Melmed, *Endocrinol Metab Clin North Am* 2008; 37(1): 101-122, viii).

(8)-DEF:

Cushing's syndrome is a condition caused by increased levels of cortisol secreted by an adrenal mass or adenoma. Cushing's disease is one of the many causes of Cushing's syndrome and refers specifically to an ACTH-secreting pituitary adenoma. The clinical picture classically includes HTN, proximal muscle weakness, truncal obesity, hirsutism, a "buffalo hump," oligomenorrhea or amenorrhea, impotence, generalized weakness, striae, osteoporosis, hyperglycemia, and hypokalemia (Doerfler A and Richter G, *Clin Neuroradiology* 2008; 18(1): 5-18).

(9)

ACTH is the pituitary hormone that stimulates cortisol secretion from the adrenal glands. Approximately 5% to 10% of pituitary adenomas are ACTH-producing adenomas and cause elevated glucocorticoid levels (Doerfler A and Richter G, *Clin Neuroradiology* 2008; 18(1): 5-18).

(10)

Suppression of cortisol excretion with high-dose dexamethasone is useful in distinguishing Cushing's disease (an ACTH-secreting pituitary adenoma) from other forms of Cushing's syndrome. Failure to suppress plasma or urine corticosteroids generally indicates an adrenal cortical or ectopic ACTH-secreting tumor; suppression of corticosteroids supports the diagnosis of a pituitary adenoma instead. Individuals with pituitary disease should demonstrate suppression in cortisol of 50% or more (Vaughan, *Med Clin North Am* 2004; 88(2): 443-466).

(11)

Normally, TSH should be suppressed or low when thyroid levels are elevated. Elevated or inappropriately "normal" TSH levels in a hyperthyroid patient is very suggestive of a TSH-secreting pituitary adenoma. These patients warrant evaluation.

(12)

Hyperprolactinemia can be caused by a pituitary microadenoma. Some providers may elect to treat patients with hyperprolactinemia empirically with bromocriptine and follow prolactin levels before obtaining imaging studies.

(13)

Hypothyroidism is a common cause of reversibly elevated prolactin concentrations (Mancini et al., *Endocrinol Metab Clin North Am* 2008; 37(1): 67-99, viii).

(14)

Medications that can increase prolactin levels include: phenothiazine, TCAs, metoclopramide, domperidone, cimetidine, OCPs, verapamil, MAO inhibitors, protease inhibitors, SSRI's (e.g. Prozac), haloperidol, risperidone, antihypertensives (verapamil, methyl dopa, reserpine), and opiates. Illicit drugs such as cocaine can also increase prolactin levels (Mancini et al., *Endocrinol Metab Clin North Am* 2008; 37(1): 67-99, viii).

(15)

Serum prolactin levels, like all hypothalamic hormones, typically rise immediately following a seizure (Mancini et al., *Endocrinol Metab Clin North Am* 2008; 37(1): 67-99, viii). These criteria therefore require that hyperprolactinemia be documented in the absence of recent seizures before pursuing further investigation.

(16)

Pregnancy causes increased prolactin secretion.

(17)

An HCG is ordered to exclude pregnancy (intrauterine or ectopic) and is not indicated in men or women who are postmenopausal or post hysterectomy.

(18)

Pregnancy is the most common cause of hyperprolactinemia and lactation is also a common associated cause. There is no exact time frame for when prolactin levels return to normal after childbirth or breastfeeding. After delivery, the volume of the pituitary gland rapidly decreases, and normalization of the size of the gland occurs within 6 months postpartum (Mancini et al., *Endocrinol Metab Clin North Am* 2008; 37(1): 67-99, viii). It is important to note that in addition to suckling, lactation (and thus prolactin elevation) can be stimulated by just thinking about or hearing the infant cry. Several prolactin level measurements may be required to confirm a decreasing trend.

(19)

Secondary AI can be caused by the abrupt discontinuation of long-term administration of glucocorticoids, pituitary adenomas, craniopharyngiomas, pituitary surgery, lymphocytic hypophysitis, and a wide variety of other neoplastic, inflammatory, and infectious processes involving the sellar or suprasellar area. Pituitary or whole brain irradiation can cause AI up to several years after its completion (Salvatori, *JAMA* 2005; 294(19): 2481-2488).

(20)

Plasma ACTH levels will distinguish between primary and secondary adrenal insufficiency. In secondary adrenal insufficiency, plasma ACTH can be either low or inappropriately normal (when serum cortisol is reduced) (Salvatori, *JAMA* 2005; 294(19): 2481-2488).

(21)

Serum cortisol levels peak in the early morning hours. Morning plasma cortisol levels ≤ 3 mcg/dL indicate adrenal insufficiency and are felt to obviate the need for additional testing. Values ≥ 18 mcg/dL exclude the disorder. Levels between 3 and 18 mcg/dL require further evaluation. Test results should be interpreted in the context of the clinical scenario (Salvatori, *JAMA* 2005; 294(19): 2481-2488). The finding of an AM cortisol ≤ 3 mcg/dL, in conjunction with a low ACTH level, points to secondary hypoadrenalism (pituitary origin) rather than primary hypoadrenalism (adrenal origin).

(22)

The most reliable screening test for adrenal insufficiency is the measurement of plasma cortisol levels following the administration of synthetic ACTH (Cortrosyn). Normally, cortisol levels should rapidly rise (within 30 to 60 minutes) to ≥ 18 to 20 mcg/dL. Because further differentiation between primary and secondary adrenal insufficiency requires an ACTH level, it is prudent to draw a sample for ACTH determination prior to this study (Salvatori, JAMA 2005; 294(19): 2481-2488).

(23)

Secondary (central) hypothyroidism refers to hypothyroidism caused by insufficient TRH or TSH secretion. The condition stems from an abnormality in the hypothalamus or pituitary gland (e.g., infiltrative processes, congenital defect, trauma, mass lesion). Hypothalamic TRH deficiency is usually iatrogenic, resulting from a transsphenoidal neurosurgical procedure. Patients who are currently hypothyroid but had been taking suppressive doses of thyroxine within a few weeks of testing can have a laboratory profile consistent with central hypothyroidism. This occurs because of slow hypothalamic-pituitary axis recovery following discontinuation of thyroxine therapy. In this setting, repeat TFTs 6 to 8 weeks after thyroxine discontinuation is necessary before the diagnosis of central hypothyroidism can be made, as the effect may be transient. The same delayed hypothalamic-pituitary axis recovery may occur after thyroid radioablation therapy (Ross, Endocrinol Metab Clin North Am 2001; 30(2): 245-264, vii).

(24)

Hypogonadism secondary to pituitary insufficiency almost always occurs in the setting of other pituitary hormone deficiency. Onset is usually gradual with GH being the first hormone affected. GH insufficiency is then followed by insufficient secretion of the gonadotropins (FSH and LH), TSH, ACTH, and finally prolactin. Because the clinical symptoms and findings of GH deficiency are vague, many patients do not present until after one of the other pituitary hormones becomes affected. When FSH and LH become insufficient, women can develop oligomenorrhea or amenorrhea and infertility, and men may experience impotence. Both men and women may complain of decreased libido. The diagnosis is suspected when FSH and LH hormones are inappropriately low and testosterone levels (in men) or estrogen levels (in women) are depressed.

(25)

Testosterone levels can be difficult to interpret in patients with hypogonadism, reflecting the hour-to-hour fluctuation of normal levels and the variability of serum proteins which bind testosterone in certain disease states. The testosterone level may therefore be normal despite genuine hypogonadism, and it may be low in unaffected patients. An early morning total testosterone level offers the best screening method; levels below 300 ng/dL are diagnostic for hypogonadism. If the total testosterone level is normal and symptoms are present, measuring the level of free testosterone (testosterone not bound by serum proteins) should be done (Basaria and Dobs, Am J Med 2001; 110(7): 563-572).

(26)

Hypogonadotropic hypogonadism is suggested by FSH and LH levels that are inappropriately depressed in relation to an abnormally low testosterone level, since the normal response of the pituitary gland to diminished testosterone production is to produce more gonadotropins (FSH and LH).

(27)

Hypogonadotropic hypogonadism is suggested by FSH and LH levels that are inappropriately depressed in relation to an abnormally low estrogen level, since the normal response of the pituitary gland to diminished estrogen production is to produce more gonadotropins (FSH and LH).

(28)

Postmenopausal women normally have increased FSH and LH levels, reflecting the pituitary gland's response to the decrease in ovarian estrogen production. Pituitary insufficiency (i.e., hypogonadotropic hypogonadism) should therefore be suspected in postmenopausal women with low FSH and LH levels.

(29)

Diabetes insipidus refers to the passage of large volumes of dilute urine. This condition can result from insufficient secretion of ADH (central or neurogenic diabetes insipidus), renal insensitivity to ADH (nephrogenic diabetes insipidus), or compulsive consumption of copious amounts of fluid (psychogenic polydipsia). All forms of diabetes insipidus are clinically similar, and characterized by excessive thirst, polydipsia, polyuria and dilute urine.

(30)

The water deprivation and the vasopressin tests are obtained to distinguish central (neurogenic) diabetes insipidus from nephrogenic. The decision as to which provocative screening test to perform and in what order is a matter of clinical judgment.

(31)

In the water deprivation test, patients with nephrogenic diabetes insipidus show little increase in urinary osmolality with dehydration, and no further increase after vasopressin injection. Patients with central (neurogenic) diabetes insipidus will have an increased urine osmolality above that of plasma osmolality.

(32)

In the vasopressin test, urine osmolality is measured after water deprivation and the administration of vasopressin or desmopressin. Patients with central (neurogenic) diabetes insipidus will show an increase in urine osmolality >50%, while patients with nephrogenic show an increase < 50%.

(33)

The optic chiasma where the two cranial optic nerves cross is situated anterior and superior to the pituitary gland, and usually overlies the pituitary fossa. As the pituitary gland enlarges due to tumor growth, these nerves can become compressed causing visual loss. Visual field defects associated with pituitary tumors are often late findings. The field loss usually affects the temporal visual fields and may be unilateral or bilateral.

(34)-DEF:

Bitemporal hemianopsia is the loss of vision on the temporal half of the visual field in each eye.

(35)-DEF:

Pituitary apoplexy is spontaneous, hemorrhagic infarction of a pituitary tumor. It is a medical emergency which can result in partial or total pituitary insufficiency.

(36)

The sella turcica ("Turkish saddle") is the saddle-shaped transverse depression in the sphenoid bone which contains the pituitary gland. This indication refers to abnormalities of the sella turcica discovered incidentally during an imaging study performed for another purpose.

(37)

A pituitary mass may be discovered incidentally during an imaging study (e.g., CT head) performed for another reason. A recent study has shown that pituitary abnormalities are found in approximately 10% of the normal adult population. Most pituitary adenomas do not require treatment if they remain asymptomatic (Aron and Howlett, *Endocrinol Metab Clin North Am* 2000; 29(1): 205-221). Some providers may elect to follow these lesions with periodic MRI. When to order these studies is a matter of clinical judgment.

(38)-POL:

Surveillance of the pituitary mass or cyst after surgical or medical treatment is to examine the amount of residual pituitary or tumor tissue, and to look for any treatable recurrence. The interval of surveillance exams is a matter of local medical policy.