Pituitary Apoplexy

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The posterior lobe is an extension of the hypothalamus and secretes two hormones: antidiuretic hormone (or arginine vasopressin) and oxytocin. The pituitary stalk and the portal vessel pass through a small diaphragm that separates the sella turcica from the middle fossa. This anatomic arrangement places the pituitary at risk for infarction or hemorrhage when a mass increases pressure in the sella or compresses the stalk and vessels. Higher intrasellar pressures are associated with poor outcomes.

Pituitary tumors are common and many are asymptomatic. They are classified by size (microadenoma, <10 mm; macroadenoma, >10 mm) and by the hormone produced. Of tumors that cause clinical symptoms, the most commonly secreted hormones are PRL, which leads to hypogonadism; GH, which promotes acromegaly; and ACTH, a cause of Cushing disease.

Tumors involved in apoplexy are typically nonfunctional and unsuspected macroadenomas. In patients undergoing an endocrine stimulation test for hypogonadism, hypothyroidism, or adrenal insufficiency, apoplexy may occasionally develop secondary to stimulation of a macroadenoma. Treatment of a pituitary tumor can also precipitate apoplexy, particularly in cases of surgery, irradiation, or bromocriptine administration. Other reported risk factors include pregnancy (Sheehan syndrome), head trauma, recent cardiac surgery, anticoagulation, hypertension, diabetic ketoacidosis, and ovarian stimulation medications.

Most patients with pituitary apoplexy have no identifiable risk factor. Apoplexy may occur in normal glands.

Presenting Signs and Symptoms

CLASSIC

The findings in patients with pituitary apoplexy vary from mild headache to sudden collapse and coma. Most patients exhibit severe frontal or retroorbital headache, vomiting, impaired visual acuity, visual field defects, hypopituitarism, and subsequent adrenal crisis. A minority have oculomotor palsy, obtundation, meningismus, blindness, or long-tract signs. The visual field deficit is classically bitemporal upper quadrantanopia or hemianopia. Associated cerebral infarction occasionally occurs secondary to vasospasm from subarachnoid hemorrhage or direct compression of the internal carotid artery by tumor. Table 170.1 lists the frequency of signs and symptoms reported in four case series.

Epidemiology

Pituitary adenomas are common, with a prevalence of 3% to 27% in various autopsy series. They are rarely diagnosed in life, with a reported incidence of 4 per 100,000 in a Finnish population and a prevalence of 77 per 100,000 in a British one. Apoplexy occurs in a minority of such lesions and can occasionally be seen with normal glands. Because of the relative rarity of this condition, pituitary apoplexy may be confused with more common entities such as subarachnoid hemorrhage. Delay in diagnosis and treatment may lead to blindness, permanent cranial nerve palsies, or death.

Pathophysiology

The two lobes of the pituitary gland sit within an enclosed space known as the sella turcica. Blood supply to this gland is one of the richest of all mammalian tissues.

The anterior lobe receives the portal hypophyseal vessel from the hypothalamus. Differentiated cells in the anterior lobe secrete specific hormones, including growth hormone (GH), adrenocorticotropic hormone (ACTH), prolactin (PRL), thyroid-stimulating hormone (TSH), and gonadotropins: luteinizing hormone (LH) and follicle-stimulating hormone (FSH).
SECTION XVI  METABOLIC AND ENDOCRINE DISORDERS

DIAGNOSTIC TESTING

Visual acuity and confrontational visual field testing should be performed when the diagnosis of pituitary apoplexy is considered. Samples should be obtained for electrolytes, renal function, clotting screen, blood counts, random cortisol, thyroxine, TSH, PRL, insulin-like growth factor, GH, LH, FSH, and testosterone in men and estradiol in women. A non–contrast-enhanced computed tomography scan of the head will exclude the diagnosis of acute subarachnoid hemorrhage. Endocrine simulation testing could worsen the condition and should be deferred. Computed tomography is not sufficiently sensitive to exclude a pituitary process. Contrast-enhanced, diffusion-weighted magnetic resonance imaging allows the best visualization of pituitary tumors and details of the hemorrhage and infarction within them (Fig. 170.1, A and B).

MANAGEMENT

Parenteral hydrocortisone (100 mg every 6 hours) or dexamethasone (4 mg every 12 hours) is given to decrease tumor edema and treat impending adrenal crisis. Obtunded or comatose patients should be intubated.

There is controversy regarding conservative treatment with steroids versus surgical intervention. Patients with minimal or improving visual symptoms are sometimes managed medically. Definitive treatment is pituitary decompression, most often through a transsphenoidal approach. The outcome depends on the severity of symptoms at initial presentation.
Patients with subacute or chronic panhypopituitarism should be evaluated by an endocrinologist for initiation of therapy and further management. Asymptomatic pituitary tumors can be safely followed in the outpatient setting because such cases rarely progress to apoplexy.

**REFERENCES**


**SUGGESTED READINGS**


REFERENCES