Hypopituitarism

Series Editors:
Bryan McIver, MB, PhD
Consultant in Endocrinology
Mayo Clinic and Foundation
Rochester, MN

Paul R. Conlin, MD
Assistant Professor of Medicine
Harvard Medical School
Director, Endocrinology,
Diabetes and Metabolism Training Program
Brigham and Women’s Hospital
Boston, MA

Contributor:
Matthew H. Corcoran, MD
Assistant Professor of Clinical Medicine
Section of Endocrinology
University of Chicago Hospital
Chicago, IL

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Hypopituitarism refers to the decreased secretion of one or more anterior pituitary hormones. The clinical presentation depends on how rapidly the anterior lobe is affected, the specific cells involved, and the severity of the functional impairment.

In the classic case of anterior pituitary hormone loss, gonadotropins are affected first, followed by growth hormone (GH), thyroid-stimulating hormone (TSH; thyrotropin), and finally adrenocorticotrophic hormone (ACTH; corticotropin). Hypopituitarism may develop acutely, such as pituitary apoplexy following hemorrhage into a preexisting pituitary adenoma. In contrast, radiation therapy exerts its effects slowly, and the hormone deficiency may not manifest clinically for months to years.

In assessing the clinical presentation of a patient with hypopituitarism, one should consider the loss of each anterior pituitary hormone individually. With some exceptions, the loss of production of an anterior pituitary hormone results in clinical manifestations similar to those arising from failure of the target gland the pituitary hormone controls. The most common presenting symptom of hypopituitarism in men and premenopausal women is hypogonadism, secondary to gonadotropin deficiency or hyperprolactinemia. The failure to lactate following parturition may indicate a lack of prolactin secretion, the only known clinical manifestation of prolactin deficiency. ACTH deficiency causes clinical manifestations of cortisol deficiency and adrenal insufficiency. TSH deficiency results in symptoms of thyroxine deficiency and hypothyroidism. GH deficiency presents as short stature in children; adverse consequences in adults may include an increase in fat mass and a diminution of lean muscle mass, a decrease in bone mineral density, and a diminished sense of well-being.

If hypopituitarism is the result of a pituitary or sellar mass, there may be central symptoms related to the mass as well as its direction of extension. Symptoms include headache, visual loss secondary to superior extension and involvement of the optic chiasm, cranial nerve involvement and ophthalmoplegia secondary to lateral extension into the cavernous sinus, and epistaxis or rhinorrhea secondary to inferior extension.

A variety of conditions may affect the pituitary gland or hypothalamus to cause hypopituitarism. More than 50% of cases are caused by benign pituitary macroadenomas or their treatment. Hypothalamic lesions also may produce hypopituitarism. The hyposecretion of pituitary hormones typically has no diagnostic value in differentiating between hypothalamic and pituitary causes of hypopituitarism. The exception is the development of spontaneous diabetes insipidus, suggesting a hypothalamic disease. Because vasopressin-producing neurons terminate in the median eminence, pituitary lesions alone will not cause diabetes insipidus. In contrast, the hypersecretion of a specific pituitary hormone identifies the lesion causing hypopituitarism as a pituitary adenoma, as well as the type of adenoma. It should be noted, however, that a prolactin level between 20 and 200 ng/dL may result from a lactotroph adenoma or another mass causing a stalk effect, interrupting dopamine’s inhibitory role in prolactin secretion.

This manual provides an overview of the clinical and endocrine approach to partial pituitary failure and panhypopituitarism. The clinical work-up of potential hormone deficiencies using both static and dynamic endocrine testing are discussed, and key points regarding appropriate hormone replacement therapies are addressed.