A 60-year-old man with progressive malaise, fatigue and decreased libido

A 60-year-old man sought endocrine evaluation because of a 5-year history of progressive malaise, fatigue, declining potency and libido. He had a history of stable ischemic heart disease (underwent triple-vessel bypass in 1999), hypertension, hyperlipidemia and remote depression (took antidepressants until about 10 years ago). He was taking atenolol, ASA, ramipril and atorvastatin. The man denied headaches or visual symptoms, and his physical examination revealed no obvious visual field defects, diplopia, nystagmus, acromegaloid or cushingoid features. He had central obesity, mild bilateral gynecomastia without galactorrhea, normal male facial features and axillary chest and pubic hair, and a normal right testicle but small left testicle (about 2 cm long). His levels of total testosterone (6.3 [normally 8.3–28.7] nmol/L), free testosterone (13.7 [normally 23–54] nmol/L), follicle stimulating hormone (FSH; 4.3 [normally 1–11.6] IU/L) and luteinizing hormone (LH; 2.6 [normally 0.9–12.3] IU/L) were consistent with secondary hypogonadism. His thyroid function was normal, as were his glucose, prolactin, calcium, electrolyte and hemoglobin levels. Although the result of a cosyntropin test (plasma cortisol measurement before and after injection of synthetic adrenocorticotropic hormone [ACTH]) was normal, the patient had a low-normal morning cortisol measurement of 275 (normally 265–800) nmol/L. An MRI scan of the head revealed a giant (4 × 2.5 × 2.2 cm) pituitary macroadenoma compressing the optic chiasm and extending above the sella turcica (Fig. 1). Although the patient had no visual symptoms, detailed ophthalmologic assessment revealed a right superior temporal quadrantic defect attributed to compression of the left optic tract.

The patient underwent transnasal transsphenoidal surgery to remove most of the tumour, which was confirmed pathologically to be a null-cell adenoma (a nonfunctional pituitary tumour). Postoperatively, desmopressin therapy was initiated to treat diabetes insipidus. Intramuscular testosterone therapy was started, with shots given every 3 weeks, and 6 weeks after surgery the patient felt more energetic, more potent and had increased libido. His vision was unaffected. Because of some suprasellar extension of the tumour not being amenable to surgical removal, the patient returns for follow-up serial MRI scanning, with the option of stereotactic gamma-knife radiosurgery in the future.

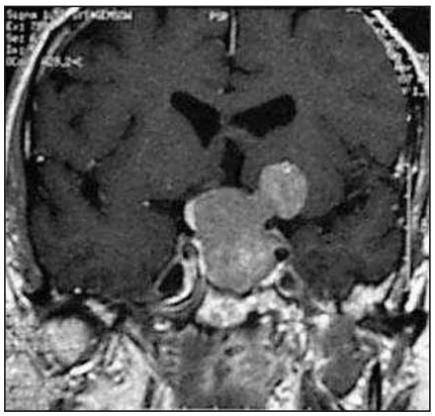

Image courtesy of Dr. Usman Chaudhry

The pituitary gland is a tiny (12 × 8 mm) structure central to all endocrine functions. The differential diagnosis of sellar masses includes pituitary adenomas, craniopharyngiomas, meningiomas, germinomas, metastases, sarcoidosis, tuberculosis, histiocytosis X and vascular lesions.[1] The estimated mean annual incidence of pituitary tumours is 0.5–7.4 per 100 000 people.[2] Pituitary tumours are usually slow growing, benign, monoclonal neoplasms, yet they are often associated with significant morbidity because of local effects of the mass, or excessive or deficient hormone secretion.[3,4]

Two-thirds of pituitary tumours are considered nonfunctional adenomas. The other third are functional (hormone secreting) and hypersecrete one or more of prolactin, growth hormone and ACTH and, rarely, FSH, LH and thyroid stimulating hormone. Although functional adenomas can present with mass effects, they are usually small (< 1 cm).

Nonfunctional pituitary adenomas, also called null-cell adenomas, are the most common macroadenomas (> 1 cm). Although gonadotroph cells give rise to these tumours, most secrete FSH and LH inefficiently while often hypersecreting the subunits (mostly the alpha subunit) of these hormones. Nonfunctional adenomas usually present with local mass effects (e.g., optic chiasm compression), neurologic symptoms (cranial nerve III, IV and VI palsies) and pituitary hormone deficiencies (e.g., hypogonadism). Pituitary apoplexy, a life-threatening sudden hemorrhage or infarction of a pituitary adenoma characterized by severe headache, nausea, vomiting, ophthalmoplegia and reduced level of consciousness, can occur in patients with large pituitary adenomas who suddenly deteriorate clinically.[5,6]

Except for prolactinomas, which are primarily managed medically, pituitary adenomas are treated by surgical resection, with adjuvant radiation therapy sometimes offered to patients with residual adenoma tissue.[7] Postoperatively, vision improves in 70% of cases with preoperative visual field deficits, and 50% of patients with preoperative pituitary hormone deficiencies will recover spontaneously, thus avoiding life-long substitution therapy.[8] About 15% of nonfunctional pituitary adenomas excised surgically will recur within 5 years.

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