Neuroendocrinology and Pituitary

NEUROENDOCRINOLOGY AND PITUITARY CASE REPORTS

A Follicular Stimulating Hormone Secreting Adenoma

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A gonadotroph secreting pituitary adenoma, a rare endocrinopathy, is often overlooked due to ambiguous symptoms leading to other diagnoses, such as ovarian hyperstimulation syndrome (OHSS).

A 53 year old African American woman presented for evaluation of a 4 month history of right hemianopsia. Her past medical history included a total hysterectomy and bilateral oophorosalpingectomy for recurrent ovarian cysts 11 years prior. Initial MRI:4.1x3.6x3.9 cm pituitary macroadenoma with prechiasmtic optic nerve compression. Initial laboratory studies: follicular stimulating hormone (FSH) level 186.83 mIU/mL (RR:23.0-116.3 mIU/mL) and luteinizing hormone (LH) 14.44 mIU/mL (RR:15.9-54.0 mIU/mL). Other pituitary labs were unremarkable. Debulking surgery was performed to relieve mass effect; pathology showed an FSH immunoreactive adenoma. Postoperatively, FSH was 2.55 mIU/mL and LH was 0.75 mIU/mL.

The prevalence of pituitary adenomas is 80-100 per 100,000 persons; 15-30% are non-functional. The majority of gonadotroph adenomas are difficult to identify because they may not produce biologically active hormones. Clinically functioning gonadotroph adenomas are very rare, often macroadenomas when found, most often found in reproductive age women, and preferentially secrete FSH. In reproductive age women, a FSH secreting adenomas may present clinically with OHSS, with multiple large ovarian cysts found on ultrasound for menstrual or pelvic complaints as seen in our patient. While OHSS is a known complication of assisted reproductive procedures, spontaneous OHSS is atypical and suggests need for evaluation of a FSH-secreting adenoma. Men may present with macroorchidism. Children may present with precocious puberty.

The treatment of choice is surgical removal of the adenoma, normalizing FSH to an age appropriate level. When OHSS is present, this results in a rapid decline in estradiol level and regression of ovarian cysts. Medical treatment, if surgery is contraindicated, is limited to cabergoline, which can initially decrease FSH and estradiol levels. In patients with normal pituitary function, gonadotropin-releasing hormone agonists acutely stimulate LH and FSH, thereby desensitizing GnRH receptors and decreasing gonadotropin secretion. In patients with FSH-secreting adenomas, they may have a persistent stimulatory effect, and in rare cases, result in pituitary apoplexy. Tumors that cannot be completely surgically removed may be treated by adjuvant radiotherapy or radiosurgery.

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A Rare Case of a Plurihormonal Pituitary Adenoma

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Background: Pituitary adenomas are usually benign tumors that arise from adenophypophyseal cells and produce one or two types of hormones. Plurihormonal adenomas are a rare subtype that produce two or more hormones and represent less than 1% of all pituitary adenomas.

Clinical Presentation: A 76-year-old female presented for evaluation of abnormal thyroid function test results. She was found to have an elevated free T4 of 1.92 ng/dL and total T4 of 14.4 ug/dL with an inappropriately normal TSH of 2.11 uIU/mL. Physical examination was significant for tachycardia, tremors, diaphoresis, coarse facial features, and enlarged hands. Further biochemical evaluation of her pituitary hormone levels demonstrated an elevated prolactin (PRL) of 237.2 ng/mL, elevated insulin-like growth factor 1 (IGF-1) of 787 ng/mL, normal morning ACTH of 47 pg/mL, normal morning cortisol of 17.0 ug/dL, an inappropriately suppressed FSH of <5.0 mIU/mL, an elevated alpha subunit of pituitary glycoprotein hormones (PGH) of 6.9 ng/mL, and an elevated free T4 of 3.5 ng/dL by equilibrium dialysis. She underwent an MRI of the pituitary and brain which demonstrated a pituitary adenoma measuring 1.2 x 1.3 x 1.8 cm with a portion herniating into the sella turcica with no mass effect of the optic chiasm. A formal visual field examination was normal. The patient underwent workup for Cushing's Disease with a low dose overnight dexamethasone suppression test, resulting in an appropriate response with an 8 AM cortisol of <1.0 ug/dL. Glucose suppression test confirmed the diagnosis of acromegaly with growth hormone at 120 minutes of 19.90 ng/mL. Neurosurgery performed a trans-sphenoidal pituitary adenoma resection. Thyroid function tests on post-operative day 4 demonstrated a suppressed TSH of 0.01 uIU/mL and an elevated free T4 of 2.30 ng/dL. Histological evaluation revealed dual expression of transcription factors pituitary-specific positive transcription factor 1 (PIT1) and steroidogenic factor 1 (SF1) as well as PRL, GH, TSH and FSH expression. Immunostaining for LH and ACTH were negative. Post-operative IGF-1 and GH levels were 106 ng/mL and 0.51 ng/mL at 17 weeks, respectively. Post-operative TSH normalized to 0.82 uIU/mL, free T4 normalized to 1.04 ng/dL, and PRL normalized to 8.1 ng/mL at 12 weeks. The patient remained symptom free after successful surgical resection.

Discussion: Our case demonstrates the clinical course of a unique patient with clinical and biochemical manifestations of thyroid dysfunction and acromegaly with a pituitary adenoma immunoreactive for GH, TSH, FSH, and PRL. The co-secretion of GH, TSH, PRL, and FSH as well as positivity for the alpha-subunit is extremely unusual. This case emphasizes the importance of considering pituitary abnormalities as a cause for abnormal thyroid function tests.

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