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CUSHING DISEASE MASQUERADING AS POLYCYSTIC OVARY SYNDROME: A DIAGNOSTIC PITFALL IN SEVERE HYPERANDROGENISM

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INTRODUCTION
Polycystic ovary syndrome (PCOS) is the most common cause of hyperandrogenism in women of reproductive age. However, several endocrine disorders, particularly Cushing disease (CD), can closely mimic the clinical, biochemical, and radiological features of PCOS. This overlap may lead to misdiagnosis and delayed recognition of hypercortisolism, with significant metabolic and reproductive consequences.

CASE
We report a 24-year-old female with young-onset diabetes mellitus who was referred for endocrine co-management during admission for recurrent mons pubis and labial abscesses with poorly controlled glycemia. She had a 5-year history of progressive hirsutism, oligomenorrhoea, scalp hair loss, significant weight gain, and insulin resistance, and had previously been labelled as having PCOS during adolescence, with subsequent default of follow-up. On examination, she was obese (body mass index 33 kg/m^2) with plethoric facies, acanthosis nigricans, proximal myopathy, and hirsutism (Ferriman–Gallwey score 10), without overt virilization or acromegalic features.

Biochemical evaluation demonstrated severe hyperandrogenism with markedly elevated total testosterone (7.05 nmol/L), suppressed gonadotropins, and adrenocorticotropic hormone (ACTH)-dependent hypercortisolism. Cortisol failed to suppress on low-dose dexamethasone testing, and 24-hour urinary free cortisol was markedly elevated (>4,900 nmol/24 h). Pelvic ultrasonography and computed tomography imaging showed polycystic ovarian morphology without evidence of an ovarian mass. Pituitary magnetic resonance imaging revealed a small right-sided pituitary microadenoma measuring 2.6 × 3.7 mm. Inferior petrosal sinus sampling demonstrated a central-to-peripheral ACTH gradient with adequate prolactin ratios, confirming pituitary CD.

CONCLUSION
This case highlights how Cushing disease can closely mimic PCOS, including polycystic ovarian morphology and hyperandrogenism. Progressive symptoms, severe biochemical androgen excess, and marked insulin resistance should prompt evaluation for secondary causes of hyperandrogenism, particularly hypercortisolism, to avoid delayed diagnosis and prolonged morbidity.

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CRISIS IN THE MASTER GLAND: A CASE SERIES OF PITUITARY APOPLEXY

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INTRODUCTION
Pituitary apoplexy is a rare but potentially life-threatening endocrine emergency caused by hemorrhage or infarction of the pituitary gland. Its presentation often mimics other acute neurological conditions, posing diagnostic and management challenges. We report a case series of three patients presenting with similar neuro-ophthalmic complaints but differing in symptom onset and radiological features.

CASES
The first case was a 25-year-old obese female who presented with acute headache, fever, and right eye ptosis with complete ophthalmoplegia for 2 days. Imaging demonstrated a heterogeneous pituitary macroadenoma with superimposed hemorrhage. Cortisol, prolactin, and insulin-like growth factor-1 levels were low. She received hydrocortisone replacement and underwent left pterional craniotomy with tumor debulking, resulting in marked visual improvement.

The second case involved a 59-year-old male who presented with headache and bilateral blurred vision for 1 week, followed by acute right-sided ptosis. Imaging showed a heterogeneous sellar-suprasellar mass compressing the optic chiasm. He had central hypocortisolism, hypothyroidism, and hyponatremia. Surgical intervention was declined, and outpatient follow-up showed stable neuro-ophthalmic findings.

The third case was a 30-year-old female who presented with a 2-week history of headache and right-sided blurred vision with temporal hemianopia. Imaging revealed a sellar-suprasellar mass with fluid-fluid levels compressing the optic chiasm. She had central hypocortisolism, hypothyroidism, and hypogonadism. Hydrocortisone

replacement was initiated, followed by transsphenoidal surgery with tumor debulking. Her vision improved after the surgery.

CONCLUSION

Pituitary apoplexy may present with similar clinical features despite differing onset and radiological characteristics. Early corticosteroid therapy is essential, while surgical intervention should be reserved for patients with severe or progressive neuro-ophthalmic deficits. This case series highlights the importance of individualized, multidisciplinary management to achieve favorable outcomes.

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COEXISTENCE OF NONFUNCTIONING PITUITARY ADENOMA AND GRAVES' DISEASE: A DIAGNOSTIC CHALLENGE

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INTRODUCTION

Nonfunctioning pituitary adenomas (NFPA) may cause central hypothyroidism due to pituitary compression, often presenting with low thyroid-stimulating hormone (TSH). However, suppressed TSH in this setting should not automatically be attributed to pituitary dysfunction, as primary hyperthyroidism—such as Graves' disease—may rarely coexist. Distinguishing between these disorders is essential to avoid misdiagnosis and inappropriate management.

CASE

A 42-year-old female presented with intermittent headache, visual field impairment, palpitations, fine tremors, and weight loss. Physical examination revealed visual field deficits and no goiter.

Laboratory evaluation showed cortisol level of 1 µg/dL (normal: 3.7–19.4 µg/dL), luteinizing hormone 1.62 mU/L (normal: 2.4–12.6 mU/L), follicle-stimulating hormone 5.01 mU/L (normal: 3.5–12.5 mU/L), free thyroxine 4 28.32 pmol/L (normal: 12–22 pmol/L), TSH 0.02 µIU/mL (normal: 0.27–4.2 µIU/mL), and prolactin 70.84 ng/mL. Thyrotropin receptor antibody (TRAb) was 3.53 IU/L, confirming Graves' disease.

Contrast-enhanced brain magnetic resonance imaging demonstrated a pituitary macroadenoma (2.13 × 2.28 × 3.05 cm) with optic chiasm compression.

The patient was diagnosed with NFPA, Graves' disease, secondary adrenal insufficiency, possible hypogonadotropic hypogonadism, and hyperprolactinemia likely due to the stalk effect.

Preoperative management included hydrocortisone replacement and antithyroid therapy. The patient subsequently underwent transsphenoidal surgery with appropriate perioperative care. Postoperatively, no new pituitary hormone deficiencies were observed. She was maintained on thiamazole 10 mg daily with clinical improvement and remains under regular follow-up.

CONCLUSION

This case highlights a rare but clinically important coexistence of NFPA and Graves' disease. Suppressed TSH in patients with pituitary adenoma should not be assumed to reflect pituitary dysfunction without thorough evaluation. Comprehensive thyroid assessment is crucial to ensure accurate diagnosis and appropriate management.

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RECURRENT ISCHEMIC STROKE TWO DECADES AFTER CRANIOSPINAL RADIOTHERAPY FOR SUPRASELLAR GERMINOMA: A CASE OF SUSPECTED RADIATION-INDUCED CEREBROVASCULAR DISEASE

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INTRODUCTION

Cranial radiotherapy is an established treatment for intracranial germinoma but may lead to late cerebrovascular complications including radiation-induced vasculopathy, cavernoma formation, and premature intracranial atherosclerosis. These complications may manifest many years after treatment and represent an important cause of stroke in young adult survivors of brain tumors.

CASE

We report a 39-year-old male with a history of suprasellar germinoma treated with transsphenoidal surgery followed by craniospinal radiotherapy in 2006. He subsequently developed panhypopituitarism requiring lifelong hormone replacement therapy. In October 2024, he experienced a cerebrovascular accident resulting in mild residual right-sided weakness. Brain magnetic resonance imaging demonstrated multifocal old infarcts, small vessel disease (Fazekas grade 2), and multiple susceptibility artifacts suggestive of radiation-induced cavernomas.