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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/m²) 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 adrenocorticotrophic 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