

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, multi-disciplinary 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 428.32 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 co-existence 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.