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.

In March 2026, he presented with new onset right-sided weakness and facial asymmetry. Initial neurological examination revealed right-sided motor power of 4/5. Computed tomography of the brain showed no acute intracranial hemorrhage but confirmed multiple old infarcts and small vessel ischemic changes. Computed tomography angiography revealed intracranial atherosclerotic disease with 25–50% stenosis of the cavernous segments of both internal carotid arteries and mild irregularities of posterior circulation vessels without evidence of large vessel occlusion or aneurysm.

Radiation-induced vasculopathy is a recognized delayed complication of cranial irradiation, often presenting 5–25 years after treatment. Pathophysiological mechanisms include endothelial injury, accelerated atherosclerosis, and progressive arterial stenosis. In rare cases, cranial irradiation may also lead to secondary Moyamoya syndrome, characterized by progressive stenosis of the intracranial internal carotid arteries with development of collateral vessels. Although this patient's vascular imaging did not demonstrate the characteristic collateral network seen in Moyamoya syndrome, it remains an important differential diagnosis in young patients presenting with recurrent stroke after cranial irradiation. In this patient, young age, prior cranial irradiation, multifocal infarcts, and coexisting radiation-induced cavernomas strongly suggest radiation-related cerebrovascular disease as a major contributor to recurrent stroke.

CONCLUSION
Long-term survivors of intracranial germinoma treated with cranial radiotherapy remain at risk of delayed cerebrovascular complications. Early recognition and aggressive secondary stroke prevention, along with continued neurological surveillance, are essential to reduce morbidity in this population.

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ASSOCIATION BETWEEN MEN1 GENE AND AIHA

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INTRODUCTION
Multiple endocrine neoplasia type 1 (MEN1) is a rare autosomal dominant syndrome caused by mutations in the tumor suppressor gene MENIN, classically characterized by endocrine tumors of the parathyroid glands, pancreas, and pituitary. Beyond tumorigenesis, emerging evidence suggests a role for MENIN in immune regulation, with deficiency linked to $CD4^+$ and $CD8^+$ lymphocyte dysfunction and predisposition to autoimmunity. While autoimmune conditions such as thyroiditis and pernicious anemia have been described in MEN1, an association with autoimmune hemolytic anemia (AIHA) has not been previously reported. We describe a rare case of MEN1 associated with warm AIHA, highlighting a potential link between endocrine tumorigenesis and immune dysregulation.

CASE
A 56-year-old female presented with a 4-month history of lethargy, anorexia, weight loss, and painless jaundice. She is para 6 + 1, with no history of anemia in pregnancy, prior blood transfusions, or family history of hematological disorders. Examination revealed mild pallor, jaundice, and hepatomegaly without splenomegaly.

She had a prior diagnosis of MEN1, with two hallmark features: primary hyperparathyroidism and a pancreatic neuroendocrine tumor. She underwent pancreatic enucleation, hemithyroidectomy, and hemiparathyroidectomy; histopathology demonstrated a benign thyroid nodule and parathyroid hyperplasia.

Whole exome sequencing identified no pathogenic MEN1 mutation but revealed a c.1621A>G variant, classified as a non-deleterious polymorphism. Variants in TP53 and BRCA1 were also detected without phenotypic expression. Surveillance colonoscopy and mammography were unremarkable.

Laboratory findings were consistent with warm AIHA, including elevated lactate dehydrogenase, indirect hyperbilirubinemia, low haptoglobin, reticulocytosis, and a positive direct Coombs test (immunoglobulin G). Peripheral blood film was nonspecific. She responded well to a tapering course of prednisolone.

CONCLUSION
This case highlights a possible association between MEN1 and autoimmune hemolysis. The presence of the MEN1 c.1621A>G variant, alongside TP53 and BRCA1 variants, raises the possibility of modifier effects influencing immune dysregulation. Further studies are needed to clarify this relationship.