Urgent computer tomography scan of the brain demonstrated a right basal ganglia infarct with sellar mass measuring 15 × 15 × 20 mm extending into the suprasellar cistern with mass effect. Magnetic resonance imaging confirmed multifocal acute and chronic infarcts with an acute pituitary hemorrhage compressing the optic chiasm. Biochemistry showed mild acute kidney injury (urea 11.7 mmol/L, creatinine 119 μmol/L) without electrolyte imbalance, thrombocytopenia, or anemia. Hormonal studies revealed partial hypopituitarism with normal thyroid function, secondary adrenal insufficiency (serum morning cortisol 40 nmol/L), and central hypogonadism (serum testosterone 2.2 nmol/L). Hydrocortisone was initiated inpatient, followed later by testosterone replacement and antiplatelet therapy during follow-up.

CONCLUSION

This case illustrates pituitary apoplexy with an atypical presentation: acute neurological deficits following invasive cardiovascular procedures. It underscores the importance of multidisciplinary management involving endocrinologists, cardiologists, neurologists, and neurosurgeons to optimize outcomes. Early recognition, prompt neuroimaging, and timely treatment are vital to prevent irreversible neurological and endocrine complications.

EP_A131

AVP DEFICIENCY AS THE INITIAL MANIFESTATION OF MULTISYSTEM LANGERHANS CELL HISTIOCYTOSIS: A DIAGNOSTIC ODYSSEY

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INTRODUCTION

Arginine vasopressin (AVP) deficiency is an uncommon but important presentation of infiltrative hypothalamic–pituitary disorders. In adults, isolated AVP deficiency with pituitary stalk thickening is diagnostically challenging, especially in the absence of systemic disease. Langerhans cell histiocytosis (LCH) is a rare cause and may precede systemic involvement by several years.

CASE

We report a 42-year-old male who presented in 2017 with polyuria and polydipsia, with urine output of up to 10 L/day. AVP deficiency was confirmed by the water deprivation test, and desmopressin was initiated. Initial pituitary magnetic resonance imaging (MRI) was normal, baseline anterior pituitary hormonal evaluation was unremarkable, and contrast-enhanced computed tomography (CECT) of the thorax and abdomen showed no abnormalities.

Repeat pituitary MRI 1 year later demonstrated loss of the posterior pituitary bright spot with pituitary stalk thickening. In the absence of systemic involvement, a presumptive diagnosis of lymphocytic hypophysitis was made, and pituitary biopsy was deferred due to the high procedural risk.

Serial pituitary MRIs over the following years showed persistent infundibular thickening and continued absence of the posterior pituitary bright spot, without interval progression or development of additional hormonal deficiencies. The patient remained clinically stable until June 2024, when new-onset left hip pain prompted MRI, revealing a heterogeneous mass involving the femoral neck and intertrochanteric region with a pathological fracture. Histopathological examination following wide resection confirmed LCH, with negative BRAF V600 mutation. Postoperative PET scan revealed a multisystem disease involving the skeleton, lymph nodes, spine, and gastrointestinal tract, with no bone marrow involvement. He subsequently completed six cycles of intravenous methotrexate and cytarabine.

CONCLUSION

Adult-onset AVP deficiency may be the earliest manifestation of occult multisystem Langerhans cell histiocytosis. This case highlights the importance of long-term follow-up and reconsideration of the initial diagnosis when new systemic features emerge.

EP_A132

A RARE CASE OF PITUITARY APOPLEXY ASSOCIATED WITH MIDDLE CEREBRAL ARTERY INFARCT: A CORRELATION OR COINCIDENCE?

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INTRODUCTION

Pituitary apoplexy is a rare, life-threatening condition resulting from hemorrhage or infarction of the pituitary gland, most commonly in patients with pre-existing pituitary tumors. It typically presents with a sudden headache, visual disturbance, ophthalmoplegia, and altered mental status. An uncommon but serious complication is ischemic stroke in the middle cerebral artery (MCA)

territory, particularly in the absence of direct internal carotid artery (ICA) compression.

CASE

We report a case of a 36-year-old male who presented with headache, visual impairment, and fever. Initial computed tomography (CT) brain imaging demonstrated a heterogeneous sellar lesion with peripheral calcification measuring 2.4 × 3.2 × 2.7 cm, suggestive of a pituitary mass with possible apoplexy, without evidence of acute cerebral infarction. The patient subsequently developed dysarthria, hemianopia, and reduced consciousness, prompting repeat neuroimaging. Follow-up CT revealed a large hypodense area in the right fronto-parieto-temporal region consistent with ischemic infarction. Magnetic resonance imaging confirmed an acute infarct in the right MCA territory without hemorrhagic transformation. A sellar-suprasellar mass measuring 2.3 × 2.8 × 3.8 cm was identified, consistent with a pituitary macroadenoma with intratumoral hemorrhage compressing the optic chiasm, but without direct right ICA compression. Time-of-flight mineralocorticoid receptor antagonists demonstrated attenuated flow in the right ICA (C2–C7), suggesting intracranial ICA thrombosis and reduced perfusion in the right MCA and its branches. Laboratory evaluation revealed hyperthyroidism and hypocortisolism, with no evidence of coagulopathy. The patient was treated with corticosteroid replacement and carbimazole and referred for neurosurgical management.

CONCLUSION

This case highlights a rare association between pituitary apoplexy and MCA stroke, possibly mediated by vasospasm, inflammation, or hypercoagulability. More research is needed to understand this connection and improve treatment strategies.

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DEFYING THE SCALPEL: MANAGEMENT OF PITUITARY APOPLEXY WITH HYDROCORTISONE

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INTRODUCTION

Pituitary apoplexy is an acute endocrine emergency, as early recognition may influence the outcomes, often presenting with sudden headache, visual disturbances, ocular palsies, vomiting, or altered consciousness. Given that urgent surgical decompression is a common management, emerging evidence supports corticosteroid therapy in our patient with significant neuro-ophthalmic deficits.

CASE

A 65-year-old male with hypertension, dyslipidemia, type 2 diabetes mellitus, chronic kidney disease stage III, and ischemic heart disease presented with a 4-day history of fever, vomiting, bifrontal headache, and generalized abdominal pain. Initially, he was treated for presumed intra-abdominal sepsis. On day 3 of admission, he developed acute right-sided complete ptosis with ophthalmoplegia involving cranial nerves III, IV, and VI. Prior to that, he had also reduced morning erections for 6 months before presentation. Biochemical evaluation revealed markedly reduced total testosterone (0.33 nmol/L) with inappropriately low–normal gonadotropins (luteinizing hormone 1.5 IU/L, follicle-stimulating hormone 2.3 IU/L), in keeping with secondary hypogonadism. Adrenocorticotropic hormone was suppressed, indicating secondary adrenal insufficiency. Thyroid function was preserved. Overall findings suggested partial hypopituitarism. Magnetic resonance imaging confirmed a cystic pituitary lesion measuring 1.4 × 2.6 × 1.8 cm with cavernous sinus involvement and features of pituitary apoplexy. His Pituitary Apoplexy Score was 4. He was offered surgical intervention but was not keen. He was treated with intravenous hydrocortisone, with rapid clinical improvement, including near-complete resolution of right eye ptosis and restoration of extraocular movements within 3 days, and was able to recover without surgery. The hydrocortisone was gradually tapered, and the patient was discharged well with Endocrine follow-up.

CONCLUSION

Timely corticosteroid therapy alone can result in rapid, near-complete neurological recovery in pituitary apoplexy, even with multiple cranial nerves involvement. In carefully selected patients without visual field compromise, conservative management may safely obviate the need for urgent surgical intervention, emphasizing the importance of early recognition and individualized treatment strategies.