value, 85.4 nmol/L, after optimization of diabetes. She underwent endoscopic transsphenoidal surgery with normalization of blood sugar post-surgery. Blood pressure was normal throughout.

CONCLUSION

False negative or normal IGF-1 levels may result in patients with hepatic or renal failure, hypothyroidism, malnutrition, use of oral estrogen, severe infection, and poorly controlled diabetes mellitus. Hence, a low or normal IGF-1 does not exclude acromegaly in patients with a high index of suspicion and warrants further investigation.

EP_A129

BIG AND BLURRY: GIANT PROLACTINOMA CASE SERIES

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INTRODUCTION

Giant prolactinomas represent 2–3% of prolactin-secreting pituitary adenomas and show a male predominance. They present with mass effect symptoms and hypogonadism, but may be overlooked, leading to delayed diagnosis. Although dopamine agonists are first-line therapy, management remains challenging due to the large size and invasive behavior. This study aims to describe the clinical and radiological features, treatment modalities, and outcomes of three cases of giant prolactinomas. We retrospectively reviewed three men with giant prolactinomas, including their clinical, biochemical, and radiological features, along with treatment and outcomes.

CASES

Three male patients aged 35–59 years with giant prolactinomas were included. Two patients presented with visual disturbances, headache, and features of hypogonadism, while one patient had acute confusion and visual loss secondary to obstructive hydrocephalus requiring ventriculoperitoneal shunt insertion. Imaging in all cases demonstrated large invasive pituitary macroadenomas with extensive local extension. Baseline serum prolactin levels were markedly elevated, ranging from 86,568 to 441,116 uIU/mL (86–324 uIU/mL). All patients had secondary hypogonadism, while secondary hypothyroidism and hypocortisolism were each identified in two patients. One patient also had poorly controlled diabetes mellitus at presentation. Dopamine agonist therapy was initiated as the primary therapy for all the patients. Two patients developed cerebrospinal fluid leak following initiation of low-dose dopamine agonist therapy (one patient received cabergoline 0.25 mg weekly, while another received 0.5 mg weekly), which resolved spontaneously with conservative management. Serum prolactin levels decreased markedly, with significant improvement in symptoms related to mass effect following treatment.

CONCLUSION

Giant prolactinomas may present with significant mass effect and multiple pituitary hormone deficiencies. Dopamine agonists remain the cornerstone of management and can result in substantial biochemical and clinical improvement even in large invasive tumors. However, rapid tumor shrinkage may lead to complications such as cerebrospinal fluid leak, highlighting the importance of close monitoring during treatment initiation.

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BEYOND THE INFARCT: THE SILENT SELLAR SURPRISE

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INTRODUCTION

Pituitary apoplexy is a rare, life-threatening endocrine emergency caused by acute hemorrhage or infarction of the pituitary, often in the setting of a pre-existing adenoma (2–12%). While classically presenting with sudden headache, visual loss, and altered consciousness, it can occasionally mimic an acute stroke. Occurrence after coronary procedures is exceptionally rare, particularly when accompanied by cerebral infarction. We report a middle-aged male who developed an acute stroke following percutaneous coronary intervention (PCI) and subsequently had panhypopituitarism secondary to pituitary apoplexy.

CASE

A 50-year-old male, with underlying diabetes mellitus, hypertension, dyslipidemia, and chronic kidney disease stage G3A, initially admitted to a private centre for PCI, however, complicated with acute left-sided weakness, headache, and reduced consciousness 2 days post-procedure. He had a history of progressive left visual loss for 3 months. Examination revealed Glasgow Coma Scale E2V4M4, blood pressure 118/82 mmHg, left-sided power 4/5, and temporal pallor of the left optic disc. His blood sugar was 15.5 mmol/L.

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)