Adult E-Poster

she was referred to an endocrinologist. Further evaluation included a morning serum cortisol level at 9 am, which was normal, ruling out adrenal insufficiency. However, serum aldosterone was low with inappropriately low renin levels despite elevated potassium. Mild metabolic acidosis was present. These findings strongly suggested a diagnosis of HH. The patient was managed through dietary potassium restriction, discontinuation of NSAIDs, initiation of fludrocortisone for potassium excretion, and sodium bicarbonate to correct the metabolic acidosis with a close watch on renal function and potassium levels. Within two weeks, her potassium levels normalized and remained stable thereafter. Hyperkalaemia was ultimately attributed to HH, likely caused by a combination of chronic diabetes with CKD and the use of NSAIDs, all leading to juxtaglomerular apparatus dysfunction.

CONCLUSION

This case highlights the importance of considering HH in persons with diabetes or CKD with unexplained hyperkalaemia despite a normal or mildly impaired renal function and initiating the appropriate management to prevent potentially life-threatening arrhythmias.

EP_A020

THYROTOXICOSIS WITH DISCORDANT THYROID FUNCTION TESTS: A RARE PITUITARY TUMOR PRESENTING WITH THYROTOXIC CARDIOMYOPATHY

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INTRODUCTION

Thyrotoxicosis can lead to life-threatening complications, including thyroid storm and thyrotoxic cardiomyopathy. Discordant thyroid function tests (TFTs) in severe thyrotoxicosis raise suspicion for atypical causes such as assay interference, pituitary pathology, or ectopic thyrotropin (TSH) secretion.

CASE

A 29-year-old male presented with a two-week history of cough, dyspnea, and palpitations. On admission, he was hemodynamically stable but had bibasal fine crepitations, bilateral pedal edema and signs of thyrotoxicosis (agitation, fine tremors, and hyperreflexia). Cardiac monitoring revealed atrial fibrillation with a heart rate >150 bpm. Thyroid function tests showed discordant TSH 14.92 Miu/L, T4 65.9 pmol/L and T3 13.47 pmol/L levels. He was treated as a case of thyroid storm with thyrotoxic cardiomyopathy.

Echocardiography confirmed heart failure with reduced ejection fraction (20%) and pulmonary artery systolic pressure of 48 mm Hg. Further workup ruled out Group 2, 3, 4 Pulmonary Hypertension (PH) and no invasive right heart catheterization was done. Assay interference was excluded, thyroid autoantibodies were negative and other pituitary hormones were normal. Pituitary MRI showed a large pituitary mass, raising suspicion for the presence of a TSH-secreting pituitary adenoma.

He is currently managed with the anti-thyroid drug methimazole, anti-heart failure medications and anticoagulation by a multidisciplinary team.

TSH-secreting pituitary adenomas (TSHomas) are rare, causing autonomous TSH secretion unresponsive to negative feedback. Unlike resistance to thyroid hormone (RTH), TSHomas typically present with overt hyperthyroidism and tumor-related symptoms (visual defects, headaches, anterior pituitary dysfunction). Atrial fibrillation and heart failure are rare in TSHomas but were prominent in this case. Transsphenoidal surgery is the preferred treatment, resulting to thyroid function normalization in 80% of cases. However, TSHomas often exhibit aggressive invasion, affecting surgical success. If surgery fails, somatostatin analogs (SSAs) can normalize TSH and reduce tumor size.

CONCLUSION

This case highlights the need to consider causes of atypical thyrotoxicosis when TFTs are discordant. Early recognition and a multidisciplinary approach are crucial for managing thyrotoxic cardiomyopathy and its underlying etiology.

EP_A021

ADULT LANGERHANS CELL HISTIOCYTOSIS WITH MULTISYSTEM INVOLVEMENT

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INTRODUCTION/BACKGROUND

Langerhans cell histiocytosis (LCH) is a rare, heterogenous disease with a wide range of manifestations, from unifocal lesions to multisystem involvement. Commonly affected

sites include the bone, lungs, pituitary gland, liver, bone marrow, and skin. We report a case of adult-onset LCH presenting with unifocal bony lesions and hypophysitis.

CASE

A 34-year-old female presented in June 2022 with polyuria and polydipsia for four months. Serum sodium was 148 mmol/L, serum osmolality 309 mOsmol/kg and urine osmolality 62 mOsmol/kg. Water deprivation test confirmed central diabetes insipidus. In September 2022, she complained of amenorrhea for three months but denied headaches, visual disturbances, galactorrhea or significant weight changes. Menstrual cycles were previously regular. She had two children, with her last childbirth two years prior. Anterior pituitary function was consistent with central hypogonadism (LH: 2.1 IU/L, FSH: 5.3 IU/L). Autoimmune screening and tumour markers were negative. Magnetic resonance imaging of the pituitary revealed a thickened pituitary stalk and the absence of bright spot in the posterior pituitary. No biopsy was performed. Hence, she was treated for hypophysitis and given desmopressin as well as estradiol.

Six months later, she developed left shoulder pain. Radiographs revealed a 3.0 x 4.6 x 5.4 cm lytic lesion in the left scapula. She underwent curettage and excision, and histopathological examination confirmed LCH with neoplastic cells expressing CD1a and S100. The patient did not consent for chemotherapy. One year after diagnosis, she developed central hypothyroidism (free T4: 8.2 nmol/L, TSH: 0.12 mU/L), and was started on levothyroxine. During her latest follow-up, she was asymptomatic, with no new lesions on imaging. Cortisol axis remains intact.

CONCLUSION

Adult-onset LCH is rare and biopsy remains the gold standard for diagnosis. This case highlights the diagnostic challenge of distinguishing LCH from other causes of hypophysitis. A comprehensive systemic evaluation is crucial for accurate diagnosis and assessment of disease extent.

EP_A022

GLUCOCORTICOID-INDUCED UNMASKING OF CRANIAL DIABETES INSIPIDUS IN HYPOPHYSITIS: A CASE SERIES

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INTRODUCTION/BACKGROUND

Hypophysitis is a rare inflammatory condition of the pituitary gland that can mimic other sellar masses. Glucocorticoid (GC) therapy remains the mainstay of treatment, but in some cases may unmask underlying posterior pituitary dysfunction such as cranial diabetes insipidus (DI). We report two distinct cases of hypophysitis in young women in which GC therapy revealed subclinical DI.

CASE

Case 1: A 25-year-old female presented with headache, visual blurring, and polyuria. Magnetic resonance imaging showed pituitary stalk thickening and a sellar mass suggestive of lymphocytic hypophysitis. Initial endocrine evaluation showed isolated hyperprolactinemia. Following high-dose GC therapy, she developed cranial DI confirmed biochemically and treated successfully with desmopressin. Follow-up MRI showed resolution of pituitary swelling.

Case 2: A 22-year-old female with bullous skin lesions and chronic otorrhea was diagnosed with multisystem Langerhans Cell Histiocytosis (LCH). Chemotherapy with vinblastine and high-dose dexamethasone led to diabetic emergencies and the onset of cranial DI. Magnetic resonance imaging revealed infundibular involvement and empty sella. Desmopressin was initiated and doses of steroids and chemotherapy were tapered, resulting to clinical improvement.

These cases highlight the phenomenon of GC-induced unmasking of DI in patients with hypophysitis. Inflammatory edema may initially obscure AVP dysfunction, which becomes apparent only after anti-inflammatory treatment. A high index of suspicion and close monitoring for polyuria following GC initiation are essential. Magnetic resonance imaging remains a valuable diagnostic tool, although radiological differentiation from other pituitary pathologies may be challenging.

CONCLUSION

Glucocorticoid therapy in hypophysitis can unmask subclinical cranial DI. Clinicians should be vigilant for evolving symptoms post-therapy. Early recognition and treatment of DI can significantly improve patient outcomes.