

One day PTA, due to poor oral intake, he did not take his replacement therapy tablets: hydrocortisone, L- thyroxine and desmopressin. Upon arrival, he was noted to be hypotensive with a blood pressure of 70/50 mmHg and a heart rate of 110 bpm. His O₂ saturation at room air was 80%. He appeared to be dehydrated with dry tongue. His GCS on arrival was E3V5M6. The abnormalities of his blood investigations were urea of 6.4 mmol/L, creatinine of 292 umol/L and sodium of 150 mmol/L. ECG showed sinus tachycardia with features of acute right ventricular strain pattern with S1Q3T3. Bedside echocardiogram showed features of acute PE with a dilated right ventricle and the presence of McConnell's sign. A CTPA showed evidence of bilateral main pulmonary artery saddle embolism with RV thrombus. He was then referred to the National Heart Institute (IJN) for EKOS and catheter-guided thrombolysis where he was successfully treated.

CONCLUSION

The case illustrates the importance of severe hyponatremia and dehydration as predisposing factors for venous thromboembolism.

EP_A115

CENTRAL SEROUS CHORIORETINOPATHY (CSCR): AN UNCOMMON MANIFESTATION OF CUSHING'S SYNDROME

<https://doi.org/10.15605/jafes.039.S1.126>

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INTRODUCTION/BACKGROUND

Central serous chorioretinopathy (CSCR) has been identified as a rare clinical presentation linked to elevated cortisol levels, both in overt Cushing's syndrome and in subclinical cases of hypercortisolism.

We report a case of Cushing's syndrome with uncommon presentation.

CASE

A 42-year-old female with pre-existing diabetes mellitus, hypertension and class III obesity came to the ophthalmology clinic for blurring of vision. Upon presentation, her blood pressure was 198/100 mm Hg and her blood glucose was 20 mmol/L. She was therefore admitted due to hypertensive emergency and uncontrolled

diabetes mellitus. As the patient exhibited stigmata of Cushing's syndrome, further investigations revealed unsuppressed serum cortisol level after an overnight low dose (1mg) dexamethasone suppression test (ODST), elevated 24-hour urinary cortisol 1912 nmol/24hours, elevated plasma adrenocorticotrophic hormone (ACTH): 14.8 pmol/L, elevated serum dehydroepiandrosterone sulphate (DHEAS): >27 umol/L and elevated serum testosterone: 5.59 nmol/L. Eye assessment with fundoscopy and optical coherence tomography was suggestive of CSCR. Magnetic resonance imaging (MRI) revealed a left lateral pituitary microadenoma. She was treated with steroid-lowering therapy and scheduled for eye laser treatment by a retina surgeon.

CONCLUSION

When CSCR is diagnosed, it is important to consider a work-up for Cushing's syndrome due to the association between high cortisol levels and CSCR. Laser therapy is one of the treatment options for CSCR while addressing the underlying cause.

EP_A116

POSTERIOR STALK INTERRUPTION SYNDROME: A PECULIAR PRESENTATION OF AN UNCOMMON DISEASE

<https://doi.org/10.15605/jafes.039.S1.127>

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INTRODUCTION/BACKGROUND

Posterior stalk interruption syndrome (PSIS) is a rare anatomical congenital anomaly that is characterised by a radiological triad of a thin or interrupted pituitary stalk, an absent or ectopic posterior lobe and anterior lobe hypoplasia or aplasia. Patients typically manifest with anterior pituitary hormone deficiencies at varying ages of presentation ranging from infancy to early adulthood.

CASE

We present a 15-year-old female who was initially referred for thrombocytopenia and hepatosplenomegaly. Further evaluation revealed that she also had short stature and primary amenorrhea. Antenatal history was unremarkable with no reported obstetrics complications. Clinical examination is consistent with Tanner Stage 1 with a height measuring below the third centile for her age.

Initial work-up with peripheral blood, iron studies and haemoglobin analysis confirmed true thrombocytopenia and ruled out haemoglobinopathies. Screening of the anterior pituitary hormone profile revealed that she had hypothyroidism, hypocortisolism, growth hormone deficiency, low gonadotropins levels with normal prolactin. MRI demonstrated a small anterior pituitary measuring 0.4 cm, with an ectopic posterior lobe and the infundibulum was not visualised. Her presentation, complemented with the biochemical and radiological findings confirmed a diagnosis of PSIS. She was started on hormonal replacement therapy.

CONCLUSION

PSIS occurs in about 0.5/100,000 births. The pathogenesis is largely unknown, though genetic factors and obstetric trauma are considered potential contributors. Common presentation includes short stature and delayed puberty. A combination of clinical assessment and biochemical tests is required to form a suspicion with MRI as the confirmatory test, differentiating PSIS from other pituitary pathologies. Treatment involves replacing the deficient hormones.

EP_A117

A RARE CASE OF KALLMAN SYNDROME IN A FEMALE

<https://doi.org/10.15605/jafes.039.S1.128>

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INTRODUCTION/BACKGROUND

Kallman Syndrome, a rare genetic disorder referring to hypogonadotropic hypogonadism, is associated with anosmia or hyposmia. It is due to abnormal migration of gonadotrophin-releasing hormone-producing neurons. The reported incidence is 1 in 8000 in men and is 5 times rarer in women. We reviewed case notes, investigation results, and imaging studies and discussed treatment options based on literature review and treatment availability.

CASE

A 19-year-old female was referred to endocrinology for primary amenorrhoea with pituitary microadenomas. The patient was born to non-consanguineous parents. She is the second child with 3 healthy siblings. The patient cannot smell since childhood. On physical examination, Tanner's staging of the breasts and pubic hair were 3 and 1, respectively, with no axillary hair. Perineal examination revealed a not well-formed labia majora with well-formed labia minora. Urethra and vaginal orifice were seen. She had bilaterally small fingers. Ultrasound of the abdomen showed a small

uterus at 2.1 x 1.1 cm with no ovaries seen. MRI of the brain showed bilateral pituitary microadenomas measuring 5 x 4 x 3 mm and 5 x 4 x 2 mm on the right and left side of the anterior pituitary lobe, respectively. Unfortunately, the olfactory bulb was not assessed. Hormonal assays identified a hypogonadotropic hypogonadism profile with total serum testosterone <0.24 nmol/L (NR: 0.29-1.21 nmol/L), serum oestrogen <43.3 pmol/L (NR: 59.1-874.6 pmol/L), serum luteinizing hormone 0.11 IU/L (NR: 1.0-52.5 IU/L), serum follicular stimulating hormone 1.02 IU/L (NR: 2.2-10.1 IU/L). Serum prolactin was normal at 79.23 uIU/mL. The patient was started on oestrogen pills and started to have fullness and tenderness in her breasts. She was referred to the Genetic Clinic for genetic studies.

CONCLUSION

Patients presenting with primary amenorrhoea and anosmia should prompt suspicion of Kallman Syndrome. Laboratory and radiological evaluation may be helpful as genetics confirmation will take time. Early detection and initiation of hormonal treatment will enable the progression of the secondary sexual characteristics. However, achieving fertility will still be a challenge depending on the availability of gonadotrophins or pulsatile GnRH therapy.

EP_A118

AN AGGRESSIVE CATECHOLAMINE-SECRETING GLOMUS PARAGANGLIOMA: A CASE REPORT

<https://doi.org/10.15605/jafes.039.S1.129>

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INTRODUCTION/BACKGROUND

Head and neck paragangliomas (HNPGs) are commonly detected at the carotid artery bifurcation and carotid body but may arise in the middle ear. HNPG presents as slow-growing, painless neck mass. Majority are non-functional with approximately 5% being biochemically active. HNPGs are generally locally invasive, and destructive and up to 19% may be malignant. Management for this type of paraganglioma is difficult and requires a multidisciplinary approach. We present a patient with a huge and locally aggressive functioning left glomus-jugulotympanic-paraganglioma with significant management challenges.

CASE

A 33-year-old male presented with left ear pulsatile tinnitus which was treated as left otitis media. Despite the persistent symptoms and progressive hearing