compression and obstructive hydrocephalus. Further workup revealed markedly elevated prolactin levels at 36,872 m IU/L suggestive of a giant prolactinoma.

She was started with cabergoline 0.25 mg twice per week which was gradually titrated to 0.5 mg five times per week. However, the prolactin levels only reduced slightly to 31,365 mIUs/L after 2 months of therapy. A craniotomy resection of the tumour was done given medical treatment failure and the mass effect of the lesion. Post-operation histopathological examination confirmed a prolactinoma. Unfortunately, she developed a large cerebral infarct post-operatively and eventually succumbed due to aspiration pneumonia.

CONCLUSION

Giant prolactinoma is a rare condition which is potentially treatable with dopamine agonists; however, cystic macroprolactinomas tend to be resistant to medical therapy. In such patients, surgical resection is often needed. However, a transcranial tumour resection posts higher risks and potential complications. Young patients with giant prolactinoma often require genetic testing for aryl hydrocarbon receptor-interacting protein (AIP) mutation and multiple endocrine neoplasia 1 (MEN1) mutation.

EP_A135

CYCLICAL CUSHING'S DISEASE WITH DISSEMINATED TUBERCULOSIS

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INTRODUCTION/BACKGROUND

Corticotroph adenomas account for the majority of cases of cyclical Cushing's Syndrome (CS). We described a middle-aged female with cyclical Cushing's Disease (CD) complicated with disseminated tuberculosis (TB) infections involving lymph nodes, the brain and the colon.

CASE

A 30-year-old Malay female presented with classical CS symptoms: weight gain of 10 kg, acne eruption, abdominal striae, proximal myopathy, and skin bruising. Her random 4 pm serum cortisol was 1003 nmol/l and 24-hour urine cortisol levels were elevated 3 times above the upper limit. However, a month later, her overnight dexamethasone test (ODST) was suppressed at 38.4 nmol/l with a serum ACTH of 1.8 pmol/L, and her symptoms had resolved. Three months later, she had a recurrence with an unsuppressed low-dose dexamethasone test and elevated 24-hour urine cortisol. She was diagnosed with cyclical CD following high serum ACTH level 20.5 pmol/L (1.6–13.9 pmol/L). Further testing was planned, but she was found to be pregnant. Her disease remained quiescent throughout her pregnancy. Postpartum, her CS symptoms and hypercortisolaemia recurred along with hypokalaemia and prediabetes, though her blood pressure was normal.

Post-partum, she underwent total thyroidectomy and left central lymph node dissection for a suspicious thyroid nodule with thyrotoxicosis. Histopathology revealed left micropapillary thyroid carcinoma with chronic granulomatous changes in the lymph nodes, consistent with TB. Pituitary MRI revealed a pituitary microadenoma measuring 0.7 cm and a tuberculoma in the cerebellum. TB meningitis was confirmed after an MTB GeneXpert test was performed on her cerebrospinal (CSF) fluid and yielded a positive result. CT scan of the abdomen and pelvis showed features suggestive of TB in the gut, and a colonoscopy revealed multiple transmural ulcers with positive MTB PCR results. Anti-TB therapy was initiated, and a multidisciplinary meeting recommended pituitary surgery after the intensive phase of anti-TB therapy.

CONCLUSION

This case illustrates the complexities in managing CD which may be cyclical and further complicated with severe opportunistic infections.

EP_A136

UNVEILING SIADH: A CASE REPORT OF HYPONATREMIA SECONDARY TO ABIRATERONE THERAPY

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INTRODUCTION/BACKGROUND

Hyponatremia is an uncommon complication of abiraterone treatment with an incidence rate of 0.4%-5%. Syndrome of inappropriate antidiuretic hormone secretion (SIADH) as a complication of abiraterone therapy is even rarer, and to our knowledge, this is the first-ever report on abiraterone-induced SIADH in a patient with metastatic hormone-sensitive prostate cancer (mHSPC).

CASE

An 83-year-old Chinese male with newly diagnosed mHSPC presented acutely confused and lethargic with symptomatic hyponatremia (serum sodium of 126 mmol/L) following two weeks of oral abiraterone 1 g daily. The trial of intravenous fluids appeared to worsen hyponatremia, whereby serum sodium dropped further to 117 mmol/L. His vital signs were otherwise stable, with serum potassium, urea, creatinine, glucose, thyroid function and other electrolytes within normal range. His urine sodium was 138 mmol/L, urine osmolality was 259 mOsm/kg, serum osmolality was 259 mOsm/kg, and early morning serum cortisol was 396 nmol/L. Contrast-enhanced CT scan of the brain ruled out intracranial lesions or brain metastasis. Diagnosis of SIADH was made and the patient was put on fluid restriction. Within 4 days, his serum sodium improved to 125 mmol/L. Abiraterone was withheld and was subsequently discontinued.

CONCLUSION

Abiraterone is a selective, irreversible androgen biosynthesis inhibitor used for the treatment of metastatic prostate cancer and it has shown to improve the rate of overall survival. The prevalence of SIADH among patients receiving abiraterone therapy is not well established and the exact mechanism by which abiraterone induces SIADH remains elusive. It is hypothesized that abiraterone's inhibition of CYP17A1, an enzyme crucial for androgen synthesis, may lead to dysregulation of ADH release, subsequently giving rise to SIADH.

Further research is needed to fully understand this relationship. Prompt recognition and management of abiraterone-induced SIADH are crucial to prevent associated complications.

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THE MALADIES OF CUSHING SYNDROME

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INTRODUCTION/BACKGROUND

Ectopic ACTH secretion (EAS) occurs in 1.6-6% of cases of small-cell lung carcinoma (SCLC). Diagnosis can be challenging due to a wide variety of clinical manifestations.

CASE

A 58-year-old male smoker presented with acute respiratory distress. He had a 2-month history of worsening dyspnoea, generalized oedema and reduced effort tolerance. At presentation, he was tachypnoeic with an oxygen saturation of 90%, blood pressure of 187/94 mm Hg and heart rate of 103 beats/minute. Examination revealed bilateral lung crepitations with generalized pitting oedema. A CXR showed multiple ill-defined lung masses with pulmonary infiltrates. Laboratory results revealed hypokalaemia (potassium 2.4 mmol/L) with metabolic alkalosis (pH 7.58, HCO 46.9). A CT TAP was consistent with lung malignancy with lymphangitis, carcinomatosis and bone and liver metastases. He was oxygen-dependent and had persistent hyperglycaemia (HbA1c 7.7%) and hypertension with hypokalaemia requiring >3 antihypertensive agents. Despite not being cushingoid-looking and with no hyperpigmentation, ectopic CS was suspected and confirmed with grossly elevated cortisol (>1740 nmol/L) and ACTH level 57.1 pmol/L (1.6- 13.9), non-suppressible by high dose dexamethasone suppression test (cortisol >1750 nmol/L, ACTH 57.9 pmol/L). Lung biopsy confirmed SCLC. Spironolactone and ketoconazole were started, with improvement in BP and metabolic parameters. Cortisol level reduced to 352 nmol/L within 3 weeks of treatment. He was planned for palliative chemotherapy but desaturated further. Therapeutic anticoagulant, IV piperacillin-tazobactam and Pneumocystis pneumonia (PCP) treatment with clindamycin and primaquine were added. PCR for PCP returned as positive later. Due to his poor ECOG score, family opted for conservative treatment and he succumbed later on palliative care.

CONCLUSION

Presence of profound hypokalaemia, hypertension with oedema and new-onset diabetes with lung malignancy should alert the clinicians to possible EAS. The intense hypercortisolism in EAS requires prompt treatment to reduce hypercortisolism and targeted therapy for associated co-morbidities. Early diagnosis is important as EAS confers poorer prognosis to SCLC and is associated with more extensive disease and reduced response to first-line treatment.