Adult E-Poster

EP_A075

DEFYING THE PROGNOSIS: LONG-TERM SURVIVAL IN ADVANCED ADRENOCORTICAL CARCINOMA WITH MULTIMODAL THERAPY

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INTRODUCTION/BACKGROUND

Adrenocortical carcinoma (ACC) is a rare and aggressive malignancy with poor prognosis. Long-term survival is challenging due to high recurrence rates and limited treatment options. A multimodal treatment strategy includes incorporating surgical resection, systemic therapy and radiotherapy. Stereotactic body radiotherapy (SBRT), a highly precise form of radiation therapy, targets tumours effectively with minimal surrounding damage.

CASE

A 33-year-old Chinese female presented with a one-month history of low back pain and constitutional symptoms. The abdominal CT scan revealed a 15 x 15 cm left adrenal mass with inferior vena cava (IVC) thrombosis and lung metastases. She underwent extensive surgery with complete surgical resection of the adrenal tumour, including left adrenalectomy, IVC thrombectomy, splenectomy and lung metastasectomy. Histopathologic examination confirmed metastatic ACC with Ki-67 proliferation of 40%. One month post-operatively, mitotane was initiated and titrated to a maximum dose tolerable at 3 g daily. Sorafenib was trialed but discontinued after four months due to adverse effects. Seven months post-operation, the PET scan revealed FDG-avid in the right upper lobe lung nodule with active IVC thrombus. A multidisciplinary team deemed the thrombus inoperable. Hence, she underwent 10 cycles of SBRT. A repeated FDG PET scan 8 months later showed a right upper lobe nodule and IVC thrombus resolution. At 4- and 6-years post-surgery, the PET dotatate scan revealed a dotatate-avid lesion at the right upper and left upper lobes. Hence, we proceeded with a biopsy, and the HPE examination showed only benign findings. Subsequent FDG-PET/CT scans revealed FDG-avid hypermetabolic activity in the lungs, consistent with bronchiectasis and plate atelectasis, but no signs of local recurrence.

CONCLUSION

Despite the typically poor prognosis of advanced ACC, this patient achieved long-term survival beyond eight years through a comprehensive, individualised treatment strategy, including complete surgical resection, systemic therapy, targeted SBRT and close multidisciplinary follow-up. This case highlights the potential role of SBRT in managing ACC and underscores the importance of coordinated, patient-specific oncologic care.

EP_A076

HYPOKALEMIA AS A HIDDEN CAUSE OF CUSHING DISEASE

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INTRODUCTION/BACKGROUND

Cushing disease is caused by an adrenocorticotropic hormone (ACTH)-secreting pituitary adenoma, and it comprises 70% of endogenous Cushing syndrome. Cushing disease is rare and is associated with high morbidity and mortality. The diagnosis is often delayed due to its manifestations of variable clinical features. Cortisol also has mineralocorticoid activity, and hypokalemia occurs when severe hypercortisolism occurs.

CASE

A 34-year-old female with a past medical history of type 2 diabetes mellitus, hypertension and polycystic ovarian syndrome. She was admitted for the first time 5 years ago for uncontrolled diabetes mellitus and complained of subacute onset of recurrent bilateral lower limb weakness and excessive weight gain. There was no history of neck trauma, gastrointestinal losses, thyrotoxic symptoms and treatment with corticosteroids. Physical examination revealed classical cushingoid features with truncal obesity, thin limbs, moon face, facial acne, dorsocervical fat pad and purplish abdominal striae. She was hypokalemic with serum potassium of 2.6-2.8 mmol/L and alkalotic with a bicarbonate level of 32 mmol/L. The thyroid function test and serum magnesium were normal. She received both enteral and parenteral potassium supplementation. Cushing syndrome was considered and further evaluation confirmed ACTH-dependent Cushing syndrome with a non-suppressed overnight dexamethasone test with raised serum cortisol, 24-hour urinary cortisol and ACTH. The pituitary MRI showed a microadenoma (8.2 x 9.4 x 8.3 mm). She was started on steroidogenesis inhibitors (ketoconazole) preoperatively. She underwent trans-

sphenoidal surgery (TSS), and histopathology confirmed a pituitary adenoma. However, she still had persistent Cushing disease post-operatively with non-suppressed serum cortisol, poor glycemic control with HbA1c of 11-13% and mild hypokalemia. A repeat pituitary MRI was scheduled, and a repeat TSS is likely warranted.

CONCLUSION
Although hypokalemia is not a determining feature of CD, it can be a significant presentation. Hence, a high index of clinical suspicion of the possible etiologies in evaluating hypokalemia is essential.

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A RARE CASE OF THIOAMIDE-INDUCED PANCYTOPENIA

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INTRODUCTION
Thioamides play a central role in the management of hyperthyroid disorder due to their efficacy and relatively lower risk of adverse events. While serious adverse effects are relatively uncommon, the more frequently reported are agranulocytosis, hepatotoxicity and vasculitis. Notably, propylthiouracil has been associated with a higher incidence and severity of agranulocytosis and hepatic dysfunction compared to carbimazole. We report a case of a patient with toxic multinodular goitre who developed pancytopenia shortly after initiation of various thioamide agents.

CASE
A 72-year-old female with toxic multinodular goitre developed recurrent neutropenic sepsis following exposure to multiple thioamides. She was initially treated with carbimazole but was complicated with neutropenic sepsis after 2 weeks of treatment; hence, she was switched to cholestyramine and prednisolone. Due to a lack of clinical response, propylthiouracil was introduced, resulting in initial improvement but with subsequent pancytopenia. Iodine therapy was then attempted but failed to produce clinical benefit. A low dose of methimazole was initiated as a final medical option, which eventually precipitated a third episode of neutropenic sepsis. In all three episodes, she was treated with appropriate antibiotics and received granulocyte-colony stimulating factor (G-CSF) support, leading to hematologic recovery. Extensive work-up excluded other potential causes of pancytopenia. Eventually, despite persistently elevated thyroid hormone levels and being at a high risk of intra-operative thyroid crisis, she underwent a successful semi-emergency total thyroidectomy following a multi-disciplinary team discussion.

CONCLUSION
This case highlights a rare and potentially life-threatening complication associated with thioamides, distinct from more commonly observed isolated agranulocytosis, emphasising the need for heightened vigilance when prescribing these medications.

EP_A078

PROLONGED HYPOTHYROIDISM AS A RARE COMPLICATION AFTER ANTITHYROID TREATMENT FOR A PATIENT PRESENTING WITH THYROID STORM

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INTRODUCTION/BACKGROUND
Hypothyroidism rarely occurs following anti-thyroid therapy (ATT). We present a case of prolonged hypothyroidism following ATT for thyroid storm.

CASE
A 50-year-old female presented to the emergency department with a 3-week history of failure symptoms, 10 kg weight loss and diarrhoea. She was in respiratory distress, hypotensive with a high fever and had atrial fibrillation in rapid ventricular response (170 beats/min) with congestive heart failure. She had no goitre or ophthalmopathy. She was diagnosed with thyroid storm (Burch-Wartofsky Score 90) with free T4 79.9 pmol/L and TSH <0.005IU/L. Despite prompt initiation of carbimazole, IV hydrocortisone, Lugol's iodine, non-invasive ventilation, IV amiodarone and electrical cardioversion, she suffered cardiorespiratory arrest. She was revived after cardiorespiratory resuscitation, intubation and triple inotropic support. Her 21-day ICU stay was eventful with multiorgan failure (ischaemic hepatitis, cardiogenic shock, oliguric kidney injury) complicated by nosocomial infection, critical illness myopathy and bedsore. She spent three months in the hospital, including one month of inpatient rehabilitation. Thyroid-wise, she responded to ATT with fT4 dropping to 36 pmol/L on day 3 of admission. All ATT was discontinued on day 11 when fT4 was reduced to 3.64 pmol/L and TSH <0.005 IU/L. On Day 28, her fT4 remained suppressed, reaching a nadir of 1.36 pmol/L (TSH 0.084IU/L, fT31.60 pmol/L [normal