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.