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Peculiar Prostate Peculiarity: Exploring Ectopic Cushing's Disease in Prostate Neuroendocrine Carcinoma

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BACKGROUND

Neuroendocrine tumors (NETs) of the prostate are rare malignancies, accounting for less than 1% of all prostate cancers. These tumors can occasionally produce adrenocorticotropic hormone (ACTH), leading to ectopic Cushing's syndrome. Diagnosing a neuroendocrine tumor of the prostate that produces ACTH is challenging due to its rarity and atypical presentation. Here we discuss a case of a patient with metastatic prostate cancer who exhibited signs and symptoms of Cushing's syndrome and was subsequently found to have paraneoplastic ACTH production. The patient was subsequently treated with oral Ketoconazole and experienced improvement in symptoms.

METHODS

A 78 year old male presented with complaint of severe generalized weakness, exertional dyspnea and worsening lower body edema. Comprehensive history and physical were obtained. Past medical history included OSA, HTN, HLD, and known prostate neuroendocrine cancer. The patient additionally reported lower limb swelling, abdominal distension and weight gain of approximately 20 lbs over the course of one year. Septic workup, echocardiogram, and chest x-ray were all functionally negative. Clinical examination of cardiovascular and respiratory systems were also negative. Patient disclosed that he had been diagnosed with prostate adenocarcinoma in 2019 via MRI showing extensive abdominal metastases as well as PIRADS 5 lesions throughout the prostate itself. The patient accordingly underwent castration and ADT. Transperineal biopsy was consistent with high grade neuroendocrine carcinoma- small cell type, Ki67 > 90%.

RESULTS

When the patient was noted to have 24-hour urine cortisol of 2300, elevated random serum cortisol and ACTH, ectopic Cushing's Syndrome was confirmed and suspected to be ACTH dependent. Options for possible ACTH suppression included metyrapone and ketoconazole. The patient was started on Ketoconazole 600 mg BID via tablet and noted significant improvement in symptoms within 48 hours. Repeat urine cortisol was shown to be 1100. Given the patient's improvement, he was discharged to SAR with the goal of returning to chemotherapy afterward.

CONCLUSION

The diagnosis requires a high index of suspicion, especially in patients with Cushing's syndrome without obvious adrenal or pituitary causes. Treatment typically involves managing the hormonal effects and addressing the underlying tumor through surgical resection, radiation, or systemic

therapies such as chemotherapy or peptide receptor radionuclide therapy (PRRT). In this instance, the patient's paraneoplastic condition was suppressible. However, the standard of care mandates a multidisciplinary approach for accurate diagnosis and effective management.

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