up to T4. During admission, she developed polyuria with hypernatremia. Laboratory examinations showed sodium of 150 mmol/L (NR: 136-145), serum osmolality of 318 mOsm/kg (NR: 275-295), urine osmolality of 142 mOsm/kg (NR: 275-295). Renal function, serum calcium, potassium and glucose were all normal. Anterior pituitary hormone panel showed panhypopituitarism with TSH: 0.24 m IU/L (NR: 0.27-4.2), FT4: 11.7 pmol/L (NR: 12-22), FSH: 7.35 IU/L (NR: 25.8-134) and LH: 5.92 IU/L (NR: 7.7-58.3). FSH and LH were low despite the patient being post-menopausal. The cortisol axis was not assessed as the patient was on dexamethasone. Cranial and pituitary MRI revealed parietal and occipital skull lesions, small right temporal and frontal brain lesions, and thickened pituitary stalk measuring 0.5 cm with a non-enhancing posterior pituitary lesion suggestive of metastasis.

Central diabetes insipidus occurs in less than 1% of patients with primary pituitary adenoma, while it is the presenting symptom in 70–80% of patients with pituitary metastasis. The radiological diagnosis is based on MRI which can highlight an iso-intense or hypo-intense mass on T1-weighted images with a high-intensity signal on T2-weighted images, a homogeneous enhancement with gadolinium and loss of the high signal pituitary signal intensity on T1-weighted images.

CONCLUSION

The prognosis for metastases to the pituitary gland is poor due to the presence of advanced neoplastic disease. Posterior pituitary lesions are rare, therefore being a red flag for metastasis.

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NEUROSARCOIDOSIS PRESENTING WITH CENTRAL DIABETES INSIPIDUS AND SECONDARY AMENORRHEA

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INTRODUCTION/BACKGROUND

Sarcoidosis is a multisystem granulomatous inflammatory disorder with common involvement of the lungs, lymph nodes and heart. Neurosarcoidosis, especially hypothalamic-pituitary involvement, is a rare phenomenon (<10% of sarcoidosis patients). Recognizing this holds significance in guiding investigations and early commencement of treatment.

CASE

A 32-year-old nulliparous female with no prior medical illness, presented to the ophthalmology clinic with 6 months history of painless red eye, without cough or constitutional symptoms. She was managed as a case of left eye anterior uveitis. Further assessment to exclude tuberculosis revealed perihilar lobulated opacities on CXR, negative serial sputum AFB and positive tuberculin skin test (Mantoux) of 16mm. CT of the thorax showed multiple lung nodules with lymphadenopathies involving cervical, mediastinal, hilar and axillary regions. Bronchoscopy was negative for TB PCR and MTB culture had no growth. She was treated for latent PTB for 3 months. Further history revealed secondary amenorrhea and polyuria with polydipsia. Pituitary workup confirmed hypogonadotrophic hypogonadism [serum FSH: 2.9 IU/L, LH: 1.13 IU/L, estradiol: 52.8 pmol/L]. After an overnight water fasting, serum osmolality increased to 301 mOsm/kg, with hypernatremia (serum Na: 151 mmol/L) and markedly diluted urine (urine osmolality: 45 mOsm/kg). These were all suggestive of central diabetes insipidus. Other pituitary hormones were normal [serum cortisol: 573.5 nmol/L, TSH: 1.916 m IU/L and prolactin: 331.1 mU/L]. Pituitary MRI revealed an absence of a posterior pituitary bright spot, without a pituitary lesion. Since TB has been ruled out, multi-systemic sarcoidosis was considered. Serum angiotensin-converting enzyme was elevated at 72.80 U/L (NR 8-52). A respiratory consult concluded a stage 2 pulmonary sarcoidosis. She was started on oral desmopressin 60 mcg BID and hormone replacement therapy. Oral prednisolone at a dose of 0.5 mg/kg/day was initiated for sarcoidosis treatment.

CONCLUSION

Neurosarcoidosis is often diagnosed late due to a low index of suspicion. It should be included in the differential diagnosis when patient presented with hypothalamic-pituitary disorder as it is associated with higher morbidity. Corticosteroids and simultaneous hormonal therapy remain the mainstay of treatment.