extra-adrenal tumors do not have the enzymatic capacity to form epinephrine from norepinephrine as was exemplified by our case. For catecholamine-secreting tumors, biochemical diagnosis should be followed by radiological evaluation (typically either CT or MRI of the abdomen and pelvis) to locate the tumor. Treatment options are dependent on location of tumor, size, presence of symptoms and if there is metastatic disease present.

Neuroendocrinology and Pituitary
NEUROENDOCRINOLOGY AND PITUITARY CASE REPORTS
Perplexing Infectious Etiology of a Case of Hypopituitarism and Central Diabetes Insipidus
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Injury to the hypothalamus and both the anterior and posterior components of the pituitary gland is rare, but can result from infiltrative processes such as sarcoidosis, Langerhans cell histiocytosis, granulomatous with polyangiiitis, and lymphocytic hypophysitis. Meningitis, pituitary infection, traumatic brain injury, and surgical instrumentation are other etiologies.

A 40 year old man with mild cognitive impairment due to remote meningitis was evaluated for progressive somnolence. He was obtunded and cerebrospinal fluid (CSF) analysis revealed 11 WBC with lymphocytic pleocytosis (90% lymphocytes), highly elevated protein of 588 mg/dL (ref range 12-60 mg/dL), and low glucose of 17 mg/dL (ref range 40-70 mg/dL). MRI Brain revealed basilar meningitis/rhombencephalitis and suspected infectious vasculitis induced right middle cerebral artery territory stroke. Of note, there was substantial T2 hyperintense signaling in the hypothalamus and pituitary areas, which has been reported with tuberculosis (TB), Cocciidioidomycosis (Cocci), and the aforementioned etiologies. He received broad antimicrobials, including TB treatment and fluconazole. He developed sinus bradycardia, hypotension, hypoglycemia, and hypothermia. Labs demonstrated inappropriately normal TSH of 1.4 milU/mL (ref range 0.35-4.94 milU/mL), low free T4 (T4) of 0.50 ng/dL (ref range 0.7-1.48 ng/dL) with repeat T4 undetectably low the following day, and AM cortisol less than 0.5mcg/dL (ref range 4-22 mcg/dL). Levothyroxine and steroids were initiated. He then developed central diabetes insipidus (DI) for which DDAVP was initiated. Comprehensive infectious and autoimmune meningencephalitis workup was unrevealing. Serum and CSF tests for Listeria PCR, Cocci antibody and antigen, and TB were negative upon multiple, serial checks spanning weeks. CSF analysis one month later showed improvement in protein level (106 mg/dL), but still with elevated WBC (10 WBCs, 98% lymphocytes). MRI one month later demonstrated improvement in edema and the areas of ischemia and vasculitis were less. His adrenal insufficiency and central DI were transient and improved, no longer requiring steroids or DDAVP. He remains on levothyroxine for central hypothyroidism.

The MRI and CSF findings point to an infectious etiology for hypopituitarism and central DI. We suspect an indolent bacteria such as Listeria or a fungus, likely Cocci, or TB meningitis. Cocci seemed to provide a unifying explanation as it classically creates infarcts and causes vasculitis. However, serial CSF tests were negative for Cocci, as well as for TB and Listeria. Marked improvement on follow up MRI also makes TB meningitis less likely as imaging would not resolve so quickly. This is a mysterious case; he improved on broad antimicrobial therapy and is being monitored closely in the outpatient setting.

Neuroendocrinology and Pituitary
NEUROENDOCRINOLOGY AND PITUITARY CASE REPORTS
Perplexing Polyuria Caused by a Rare Disorder
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Background: Central diabetes insipidus is an uncommon condition characterized by polyuria and polydipsia. In adults, central diabetes insipidus is most commonly caused by a primary brain tumor, followed by idiopathic causes, head trauma, and neurosurgery. The presence of diabetes insipidus is often discovered prior to the underlying culprit and detection may reveal further pituitary dysfunction. Herein an unusual cause of central diabetes insipidus is presented. Case: A 35-year-old male was seen in consultation for polyuria. He initially presented with fevers, cloudy urine, and excess urine output. He indicated frequent water consumption, craving cold water and feeling persistently dehydrated with poor energy levels. During hospitalization, the patient had up to 9 liters of urine output daily, with low urine osmolality and intermittent hypernatremia. As patients’ labs were consistent with central diabetes insipidus a brain MRI was completed and showed a thickened enhancing infundibulum and some fullness of the right pituitary without a focal lesion noted, concerning for autoimmune or inflammatory hypophysitis. Other pituitary axes were evaluated, and patient was noted to have a low morning total testosterone and low IGF-1. Concurrently, the patient was discovered to have multiple bone lesions on an MRI abdomen and pelvis, which prompted a bone scan showing diffuse uptake in osseous structures. A PET scan was then obtained demonstrating mandibular uptake as well as hypermetabolic activity in both adrenal glands, the right iliac bone, bilateral femurs and humeri. Biopsy of the mandibular lesion was performed, and the specimen revealed chronic xanthogranulomatous and lymphocytic inflammation consistent with a diagnosis of Erdheim-Chester disease. The patient was discharged on desmopressin and a biologic agent for treatment of Erdheim-Chester disease.

Clinical Lesson: Erdheim-Chester disease is a rare non-Langerhans histiocytic multisystem disorder that often presents with skeletal, neurologic, endocrine, cutaneous, cardiac and renal abnormalities. There is a slight male predominance of the disorder and diagnosis occurs between the 5th and 7th decade of life. Erdheim-Chester disease is a form of histiocytosis with a histologic hallmark of xanthomatous infiltration of tissues by CD68-positive foamy histiocytes. This case reflects the diagnostic delay often associated with the condition. Early identification is essential to organize a multidisciplinary team to ensure accurate diagnosis and to initiate appropriate therapy. Presently interferon-alpha