on clinical criteria (thickened skin, periosteal reaction, and finger clubbing). PDP is a genetic disorder of prostaglandin metabolism with variable expression and incomplete penetrance. It manifests after puberty and affects males more than females with a 7:1 ratio. PDP has been recently shown to go through an inflammatory phase, in which patients may benefit from immunosuppressants or NSAIDs. PDP should be distinguished from hypertrophic pulmonary osteoarthropathy, thyroid acropachy, and acromegaly. Our patient had a normal chest radiograph and normal hormone levels (TSH, T4, and IGF-1). The patient received topical steroids and antibiotics with artificial tears for his eyes, which resulted in a good response, and was referred to a rheumatology clinic for further management. **Clinical Lesson:** PDP can be mistaken for acromegaly or thyroid acropachy and should be considered in any patient with acromegaloid features with normal IGF-1 and no evident pituitary pathology. Awareness of this condition can help in reaching the diagnosis promptly.

Neuroendocrinology and Pituitary

NEUROENDOCRINOLOGY AND PITUITARY CASE REPORTS

Panhypopituitarism Presenting as Progressive Visual Acuity Deterioration Secondary to Suprasellar Germinoma

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Background: Overall the incidence of germ cell tumors worldwide is 0.1% with 60% comprising of germinomas. These may present in the suprasellar region a third of the time and thus impart variability in its presenting characteristics. **Clinical Case:** A 27-year male presented with progressive blurring of vision with incongruent visual acuity and refraction grade with optic disc pallor assessed with normal angiography. Patient also presented with increasing docility, sluggishness and more withdrawn behavior paired with polyuria, polydipsia and cold intolerance. Progressive worsening of vision led to cranial CT revealing an enhancing hyper density in the right caudate head later elucidated via MRI with several interspersed non-enhancing foci in the right caudate nucleus, both internal capsules with associated mild mass effect. The largest component located in the right capsular region with measurement of 21 x 21 x 16 mm with enhancing lesions in the hypothalamus and pituitary stalk. Endocrine evaluation revealed low serum cortisol at 91.03nmol/L (NV 138-685 nmol/L), responsive to ACTH stimulation test (65.94nmol/L to 387nmol/L), high prolactin at 856.9 mIU/L (42.4-296.8mIU/L) and low FT4 8.62pmol/l (9.01-19.05 pmol/L) despite normal TSH 1.528uIU/ml (0.35-4.94 uIU/mL). Optic nerve atrophy was now attributed to compressive intracranial mass. Secondary adrenal insufficiency was managed with hydrocortisone 50mgIV every 8 hours, and central hypothyroidism was managed with levothyroxine 75mcgtab 1tab once a day. Increased urinary output assessed as diabetes insipidus was given desmopressin 60mcg/tab ½ tab 2x a day at this time. Stereotactic biopsy of the brain revealed a germinoma of the right caudate lobe. Given the nature of the intracranial mass with high sensitivity to chemotherapeutic and radiotherapy, a multidisciplinary approach to treatment was taken with radiotherapy, hormonal and steroid replacement. Patient underwent external beam radiation therapy of the brain and spine for a total of 51 treatments allowing for improvement of visual acuity to counting, with polyuria less than 2-3 diaper changes per day. **Conclusion:** Optic disc pallor is a clinical sign that indicates optic nerve atrophy reflective of the optic tract. This may be an important clinical sign to increase clinical suspicion for intracranial mass lesion especially with correlation to manifestation of pituitary hormone deficiencies.

Neuroendocrinology and Pituitary

NEUROENDOCRINOLOGY AND PITUITARY CASE REPORTS

Paraganglioma

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Introduction: Paragangliomas are rare neuroendocrine tumors arising from extra-adrenal medullary neural crest derivatives. The terms pheochromocytoma and paraganglioma are often used interchangeably because morphologically and functionally these entities are almost the same. However, paragangliomas that arise in the adrenal medulla are called pheochromocytomas and those outside the adrenal gland are called paragangliomas. Paragangliomas are often discovered incidentally during imaging studies performed for other reasons. We report a case of a patient who had incidental finding of retroperitoneal paraganglioma on imaging done for evaluation of thigh and gluteal cellulitis.
Clinical Case: A 24-year-old female presented with chief complaints of episodic headaches, multiple skin abscesses, fever and malaise. She has a history of Type 1DM and had been non-compliant on insulin. Vitals on presentation was significant for tachycardia with heart rate of 124, blood pressure of 119/81, respiratory rate of 16. Initial labs were consistent with DKA. CT abdomen and pelvis done for further evaluation of gluteal and thigh abscess showed incidental finding of a 3.4 cm retroperitoneal/para-aortic well-circumscribed rim-enhancing mass with central hypoenhancing component, suspicious for neoplasm such as paraganglioma. Biochemical testing was performed. 24-hour urine catecholamine levels obtained showed elevated urine norepinephrine level of 1008µg/day (reference range 15-100µg/day), urine dopamine 410µg/day (reference range 65- 400µg/day), urine epinephrine less than 2µg/day (reference range 0-20µg/day). Total urine catecholamines was elevated 1008µg/day (reference range 15-100µg/day). These results confirmed diagnosis of paraganglioma. Treatment options were discussed with the patient including surgery for removal of paraganglioma which she has currently declined.
Conclusion: Diagnosis of a paraganglioma can usually be made using biochemical and radiographic testing. All patients with paraganglioma should be tested for hypersecretion of catecholamines in a 24-hour urine or serum collection, even if they do not present with a clinical picture of catecholamine hypersecretion. Importantly these

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 polyangiitis, 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), Coccidioidomycosis (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 mclU/mL (ref range 0.35-4.94 mclU/mL), low free T4 (fT4) of 0.50 ng/dL (ref range 0.7-1.48 ng/dL) with repeat fT4 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 meningoencephalitis 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 5^{th} and 7^{th} 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