sympathetic chain (1). The tumours commonly produce one or more catecholamines: epinephrine, norepinephrine, and dopamine leading to a classic triad of presentation with pounding headache, profuse sweating, and palpitations occurs in spells; However, one in 10 patients may be completely asymptomatic and the diagnosis of PPGL is frequently missed (2).

**Care Report:** A 29 years old African female presented with one-month history of throbbing headache, palpitations, profuse sweating, and unintentional weight loss. Her previous medical history and family history was unremarkable. She was found to have accelerated hypertension and a small lacunar infarct and some suspected subarachnoid hemorrhage on MRI head but was clinically silent. Investigation for secondary causes of hypertension revealed high metanephrine in urine and her imaging showed giant adrenal mass suggestive of pheochromocytoma.

CT Abdomen of large left adrenal necrotic solid mass lesion with heterogeneous enhancement of its solid components measuring 6.5 x 6.1 x 7 cm in maximum AP, TR and CC dimensions respectively.

She was scheduled for open resection and left adrenalectomy after approximately 10 day and was treated with high-sodium diet, alpha blockers, and beta blockers perioperatively. Histopathology examination revealed Pheochromocytoma measuring 8 x 7 x 5 cm with diffuse growth >10% of tumor volume with central necrosis; PASS = 4, which has malignant potential. She recovered well post operatively and molecular genetic testing was considered, but it was not available.

**Conclusion:** Pheochromocytoma is a rare cause of secondary hypertension with a variable clinical presentation. Episodes of tumor catecholamine release, are thought to be responsible for the high prevalence of cardiovascular emergencies, such as myocardial infarction, heart failure, and stroke as a complication of hypertensive crisis. Timely diagnosis and treatment are crucial to prevent life-threatening complications.

**Adrenal**

**ADRENAL CASE REPORTS**

**Glucocorticoids and Pheochromocytoma - a Recipe for Disaster?**

Nikhitha Chandrashekar, MD, Arnold Allan Asp, MD.

Gundersen Health System, La Crosse, WI, USA.

**Background:** Pheochromocytomas are catecholamine-secreting tumors that arise from the chromaffin cells of the adrenal medulla. The catecholamine release manifests as episodic headaches, diaphoresis, and palpitations which can lead to hypertensive emergency known as a pheochromocytoma crisis (PC). This crisis can be precipitated by commonly used medications including corticosteroids, metoclopramide, and anesthetic agents like ketamine and halothane. **Case report:** We present a 60 year old Caucasian male with a past medical history significant for SVT on metoprolol and diltiazem and long-standing hypertension. He was admitted directly from the clinic to the ED after complaining of severe abdominal pain, multiple episodes of vomiting, and diaphoresis following prednisone intake for a presumed gout flare. He had a blood pressure of 180/100 and tachycardic to 120s on arrival. He was also found to be hypoxemic with a chest x-ray concerning for pulmonary edema along with evidence of a demand related type II NSTEMI. He underwent a CT abdomen pelvis with contrast which showed evidence of high grade distal ileum obstruction with no obstructing mass or inflammatory process. There was also 5-cm solid right adrenal mass which was initially thought to be an incidentaloma. Following stabilization, he underwent an exploratory laparotomy which did not show an obvious mechanical etiology of bowel obstruction. This raised concerns for a pheochromocytoma. Oral verapamil with doxazosin was started to provide appropriate alpha blockade. Subsequently, metoprolol was reintroduced for beta blockade. He was officially diagnosed with glucocorticoid-induced pheochromocytoma after his labs returned with elevated levels of serum metanephrines 43.7 (0.00 - 0.49 nmol/L), serum normetanephrine 22.2 (0.00 - 0.89 nmol/L), 24-hr urine epinephrine 1021 ug/g (0 - 20 ug/g), and 24-hr urine norepinephrine 838 ug/g (normal: 0 - 45 ug/g). Ultimately 3 weeks later he underwent successful R sided adrenalectomy and is doing well.

**Conclusion:** This case demonstrates a rare but life-threatening adverse reaction associated with commonly used glucocorticoids in a patient with an undiagnosed pheochromocytoma. Pheochromocytomas are exceedingly rare and can go undiagnosed in up to 50 percent of patients (1). Failure or delays in diagnosis of PC can lead to increased morbidity and mortality. When managing a pheochromocytoma crisis, it is imperative to avoid selective beta-blockers, as it can worsen hypertension due to unopposed alpha agonism by the secreted catecholamines. This also demonstrates the importance of keeping a broad differential in order to avoid anchoring bias, particularly when there is conflicting clinical information. **References:** 1. Sutton MG, Sheps SG, Lie JT. Prevalence of clinically unsuspected pheochromocytoma. Review of a 50-year autopsy series. Mayo Clin Proc. 1981 Jun;56(6):354–60. PMID: 6453259.
been rarely described in the literature. **Clinical Case:** A 15-year-old male with classic CAH diagnosed since birth when he had vomiting and darkly pigmented skin. He was on regular follow-up with his endocrinologist in his home country. For an unclear reason, he was on hydrocortisone 40 mg daily in divided doses. The patient presented to the emergency department with nausea, vomiting, loose stool, dizziness, and fatigue for 5 days. On examination, he was conscious and oriented but looked tired. He had a temperature of 36.8°C, blood pressure (supine) 132/78 mmHg (standing blood pressure could not be taken because the patient was dizzyy), pulse rate 130/minute, and respiratory rate 17/minute. Otherwise, the physical exam was unremarkable. Initial laboratory investigations showed sodium 114 (136–145 mmol/L), potassium 6.0 (3.5–5.1 mmol/l), chloride 83 (102–104 mmol/l) and Bicarbonate 14 (22–29 mmol/L). Serum creatinine, complete blood count, C-reactive protein (CRP) and procalcitonin levels were within normal. He was treated for AC with stress doses of hydrocortisone intravenously, and normal saline intravenous infusion. He showed a gradual improvement of his symptoms with normalization of the electrolytes. Thus, he was switched to oral hydrocortisone replacement 15 mg am and 10 mg in the afternoon. Nevertheless, his pulse rate was 105 - 110 / min. Therefore, thyroid function test (TFT) was done and revealed TSH 0.3 (0.5–4.3 mIU/L) and FT4 30.6 (12.9–20.6 pmol/L). Thyroid uptake scan showed a mildly enlarged thyroid gland with homogeneous and slightly increased radiotracer uptake suggestive of GD. Thus, propranolol and carbimazole were prescribed in addition to hydrocortisone. Then, the patient was discharged after proper education about precipitating factors of AC. Later on, the patient appeared for his clinic appointment, he was clinically well with normal vital signs, serum electrolytes, and TFT. Propranolol was stopped, carbimazole dose was adjusted, and he was maintained on hydrocortisone.

**Conclusion:** Unrecognized Graves’ disease was the precipitating factor of the adrenal crisis in this patient with CAH. Despite the rarity of the association, a high index of clinical suspicion for unusual acute stressors is very important for proper management of AC and to prevent future recurrence.

**Adrenal**

**ADRENAL CASE REPORTS**

**Hyperaldosteronism in a Patient With Gastrointestinal Potassium Wasting**

Samuel Amankeah, MD, Henry G. Fein, MD.

Sinai Hospital of Baltimore, Baltimore, MD, USA.

**Background:** Medical conditions causing hypokalemia can be masked by a diet very rich in potassium. The following case presents a patient who developed new onset symptoms of hypokalemia after immigrating to the United States. **Clinical Case:** A 35 year old man, native of Mali, had a history of intermittent muscle spasms and serum potassium of 3.0 mmol/L first recognized elsewhere in 2019. He was not on any home medications. He was admitted with postprandial generalized abdominal pain, diarrhea, and bright blood in his stools. Serum potassium was 2.7 (nl 3.5–5.1mmol/L). He required at least 120 mEq of intravenous and oral potassium chloride per day while hospitalized to achieve and maintain serum potassium in the normal range. Colonoscopy found segmental colitis and it was thought that his hypokalemia was due to gastrointestinal losses. However, studies demonstrated urinary potassium wasting. A 1.9 cm nodular mass of the left adrenal gland was found on CT of the abdomen, and the differential diagnosis was expanded to include hypokalemia secondary to primary hyperaldosteronism or renal tubulopathies such as Bartter and Gitelman syndromes. The patient was normotensive and had biochemical findings that were consistent with Bartter syndrome including metabolic alkalosis, hypercalciuria, elevated urine sodium and chloride, and normal serum magnesium levels. However, he had low plasma renin activity with an elevated serum aldosterone on three tests over two months (the last test was more than one month after normalization of bowel function). On all of these, the aldosterone to renin ratio was greater than 20 ng/mL/hour. The persistent suppression of plasma renin with elevated aldosterone in the setting of left adrenal mass narrowed the differential diagnosis to primary hyperaldosteronism, as elevated rennin would be expected in Bartter syndrome. Since discharge, he has received 80 meq of daily oral potassium in divided doses, which has kept serum potassium at or below the lower limit of normal. Further management will consist of either pharmacologic or surgical treatment with or without adrenal venous sampling. **Conclusion:** The patient had hypokalemia for which an endocrine etiology could have been easily overlooked and attributed to gastrointestinal losses. This case demonstrates the very close clinical similarities between primary hyperaldosteronism and renal tubulopathies. However, there are biochemical patterns that can be relied on to help differentiate amongst these disorders. This patient with primary hyperaldosteronism may not have been hypokalemic in his native country due to consuming a diet rich in potassium.

**Adrenal**

**ADRENAL CASE REPORTS**

**Hypercalcemia in an Infant With Pseudohypoaldosteronism Type 1**

Henry-Jeng, DO, Julia Rodica Broussard, MD.

Children’s Mercy Hospital, Kansas City, MO, USA.

**Background:** Pseudohypoaldosteronism type 1 (PHA1) is an aldosterone resistance syndrome due to insensitivity of target tissues to aldosterone action, with supraphysiologic aldosterone and renin levels. PHA1 presents usually in infancy and is divided into autosomal dominant (AD) and autosomal recessive (AR) form. A secondary form of PHA1 associated with UTI and/or renal malformations was described. In AD PHA1, salt loss is due to renal mineralocorticoid resistance while hyponatremia in AR PHA1 is caused by multi-organ salt loss. PHA1 has variable signs/symptoms associated with hyponatremia and hyperkalemia; thus, this clinical picture can be attributed to more common conditions such as dehydration, poor feeding, congenital adrenal hyperplasia. **Clinical Case:** A 5-month old male was admitted for airway evaluation. He was a 23-week gestation preemie, with chronic