been rarely described in the literature. **Clinical Case:** A 15-year-old male with classic CAH diagnosed since birth when he was 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 dizzy), 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.

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**Adrenal**

**ADRENAL CASE REPORTS**

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

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**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 male, 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 3.5–5.1 mmol/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 rennin 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 rennin 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**

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**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
A 60-year-old woman with type 1 diabetes mellitus, past history of breast cancer, degenerative disc disease, hypertension and hirsutism accompanied by elevated pregnenolone, 17-hydroxyprogrenolone, 11-deoxycorticisol and dehydroepiandrosterone-sulfate, however, normal baseline as well as stimulated 17-hydroxyprogesterone, raise concern for co-occurrence of partial 3-betahydroxysteroid dehydrogenase deficiency in addition to non-classic 11-betahydroxylase deficiency. Management with mineralocorticoid receptor antagonist helped control hypertension.

Conclusions: Hypertension and hirsutism accompanied by elevated pregnenolone, 17-hydroxyprogrenolone, 11-deoxycorticisol and dehydroepiandrosterone-sulfate, however, normal baseline as well as stimulated 17-hydroxyprogesterone, raise concern for co-occurrence of partial 3-betahydroxysteroid dehydrogenase deficiency in addition to non-classic 11-betahydroxylase deficiency. Management with mineralocorticoid receptor antagonist helped control hypertension.

Adrenal
ADRENAL CASE REPORTS

Hypertension and Hirsutism in a Young Female: A Rare Form of Congenital Adrenal Hyperplasia?
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Background: Rare forms of congenital adrenal hyperplasia (CAH) especially can be a diagnostic and management challenge. We present a case of hypertension and hirsutism with elevated mineralocorticoid and sex steroid precursors concerning for non-classic rare form of CAH.

Clinical Case: A 22 y/o female was evaluated in our endocrinology clinic for primary hypertension and hirsutism (modified Ferriman-Gallwey score of 12) but without menstrual irregularity, clitoromegaly or deepening of voice. Family history was significant for hypertension and hirsutism in her mother. Renal and pelvic ultrasonographies were normal. CT abdomen showed thickened bilateral adrenal glands. Case detection testing was positive with PAC/PRA of 19.4/0.4 however intravenous saline suppression test suppressed aldosterone to 5 ng/dL, hence ruled out primary aldosteronism. Cortisol suppressed to 0.6 mcg/dL after 1 mg overnight Dexamethasone, hence ruled out Cushing’s syndrome; prolactin and TSH were normal, serum HCG was undetectable. This prompted work up for other endocrine causes of hypertension and hirsutism which revealed follicular phase elevated dehydroepiandrosterone-sulfate of 772 mcg/dL (35–430), pregnenolone of 603 ng/dL (15–132), 17-hydroxyprogrenolone of 1516 ng/dL (<226), 11-deoxycorticisol of 39.4 ng/dL (<32); but normal 17-hydroxyprogesterone of 36.8 ng/dL (15–70), androstenedione of 1.12 mg/mL (0.26–2.14) and free testosterone of 26 ng/dL (9–44). 250-mcg ACTH administration stimulated cortisol to 24.6 mcg/dL, pregnenolone to 1478 ng/dL, 17-hydroxyprogrenolone to 1716, 11-deoxycorticisol to 64.2 ng/dL but 17-hydroxyprogesterone only stimulated to 59.9 ng/dL. We initiated spironolactone at 12.5 mg increased to 25 mg daily, which normalized her blood pressure. She was counseled regarding its teratogenicity but is she declined birth control, as she was not engaging in heterosexual intercourse. Urinary steroid analysis and genetic testing were pending.

Conclusions: Hypertension and hirsutism accompanied by elevated pregnenolone, 17-hydroxyprogrenolone, 11-deoxycorticisol and dehydroepiandrosterone-sulfate, however, normal baseline as well as stimulated 17-hydroxyprogesterone, raise concern for co-occurrence of partial 3-betahydroxysteroid dehydrogenase deficiency in addition to non-classic 11-betahydroxylase deficiency. Management with mineralocorticoid receptor antagonist helped control hypertension.