but was misdiagnosed as gastroparesis or anxiety and was prescribed medications for the same (pantoprazole, sucralfate, and Lexapro).
On presentation this time, she was hypotensive and tachycardic but partially responsive to IV fluids. Her BMI was 15.31 and the abdominal exam was benign. Her basic labs showed Na 125, K 4.9, Calcium 10.5, Cr 1, WBCs 9K, lipase 8, normal LFTs, TSH 6.96, Free T4 1.18. Infectious workup was done and the patient was started on empirical antibiotics. Negative infectious workup, hypotension partially responsive to IV fluid, along with hyponatremia and borderline high potassium level prompted us to check Cortisol. We found Cortisol of 1.3(very low for the degree of her illness). ACTH stimulation test with 250mcg IV cosyntropin showed cortisol of 1.3 at 30 minutes and 1.1 at 60 minutes confirming the diagnosis of adrenal insufficiency. Further workup revealed a positive 21 hydroxylase antibody and ACTH level of 322 (high due to lack of negative feedback to the pituitary). She was comprehensively tested for other autoimmune diseases which showed positive Endomysial IgA and Tissue Transglutaminase antibodies indicating asymptomatic celiac disease. She improved drastically after starting hydrocortisone. TSH was repeated in 3 months which was normal.
Conclusion: The onset of chronic adrenal insufficiency is very gradual with vague presentation and it may go undetected unless illness or other stress precipitates acute crisis as in our patient. Since autoimmune adrenalitis is the most common cause of primary adrenal insufficiency (Addison's disease), patients with a confirmed diagnosis should also undergo workup for other autoimmune diseases.

Adrenal

ADRENAL CASE REPORTS

Adrenal Mass Hemorrhage Clinically Mimicking a Pheochromocytoma

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Background: A pheochromocytoma is diagnosed clinically using correlation of clinical, imaging, and laboratory studies. We report the case of an adrenal mass hemorrhage that presented with significantly elevated urine metanephrines mimicking a pheochromocytoma.
Clinical Case: A 59-year-old healthy woman presented to the ED with chest pain, left flank and upper quadrant pain associated with diaphoresis and palpitations. Vital signs were significant for a pulse of 92 bpm, and a BP of 213/88 mm Hg. Physical exam revealed tenderness to palpation on the left upper quadrant and left costovertebral angle. Laboratory tests were significant for an elevated D-dimer (2,449 ng/mL, reference range 215–499 ng/mL). CT abdomen with IV contrast showed a 3.5 x 2.9 cm round mass abutting the lateral limb of the left adrenal gland with surrounding fat stranding with suspicion of inflammation or hemorrhage. 24-hour urine metanephrines showed elevated metanephrine (7,227 mcg/24hr; reference range <400 mcg/24 hr) and normetanephrine (1,209 mcg/24hr; reference range 900 mcg/24 hr). In the setting of up trending cardiac enzymes and inferior-lateral ST segment depression, a cardiac catheterization was performed which was unrevealing. She was discharged from the hospital and referred to endocrinology. On that visit, ~ 1 month after the patient was admitted to the hospital, plasma metanephrines were ordered which showed mildly elevated plasma metanephrine (0.83 nmol/L; reference range <0.5 nmol/L) and normetanephrine (1.2 nmol/L; reference ranges <0.9 nmol/L). An MRI abdomen with and without contrast revealed a 2.7 x 2.2 x 1.8 cm nodule arising from the lateral limb, with loss of signal on in-phase images suggestive of blood products. Plasma metanephrines and MRI findings ruled out the diagnosis of a pheochromocytoma.
Conclusion: To our knowledge, few cases of an adrenal mass hemorrhage clinically mimicking a pheochromocytoma have been reported. Although the inpatient clinical presentation of our patient was consistent with this, the outpatient plasma metanephrines and MRI were not. An adrenal adenoma hemorrhage should be considered as a potential differential diagnosis for elevated metanephrines, which can clinically mimick a pheochromocytoma.
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Adrenal

ADRENAL CASE REPORTS

Adrenoleukodystrophy and Secondary Hyperaldosteronism; A Clinical Demonstration of the Zona Glomerulosa Persistence

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Adrenoleukodystrophy (ALD) is a peroxisomal disorder which leads to the accumulation of very long-chain fatty acids in all tissues. Age at onset of symptoms vary depending on the phenotype severity. It can present with progressive symptoms of neurological defects and/or primary adrenal insufficiency.
We report a case of 31 yo man diagnosed in childhood with ALD and treated with hydrocortisone and fludrocortisone who eventually developed resistant hypertension due to secondary hyperaldosteronism.
He was diagnosed with ALD at 9-year-old and have received an allogenic hematopoietic cell graft 4 years later. After transplantation, he developed a bronchiolitis obliterans which was treated with high dose of glucocorticoids for 3 years.
In 2014, at first evaluation with our team, he was on hydrocortisone 10mg/m^2 and fludrocortisone 0.05 mg daily. Fludrocortisone was started at 14 yo for a clinical suspicion of mineralocorticoids deficiency. At that time, the patient was normokaliemic (3.8 mmol\L), his aldosterone was,