Nonspecific Symptoms? Expect the Unexpected. Central Adrenal Insufficiency in a Type 1 Diabetic with Presyncope and Labile Glucose Levels

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Case Presentation

A 62-year-old man with a past medical history significant for type 1 diabetes mellitus diagnosed in 2009 and eczema presented to the emergency department for lightheadedness, leg weakness, and hypo- and hyperglycemia. He had begun experiencing episodes of progressively worsening lightheadedness and bilateral leg weakness 2 months prior to admission but denied any loss of consciousness. He was previously hospitalized for lightheadedness one month prior, which was resolved with fluid resuscitation and no further revealing workup. Interestingly, at home, his blood glucose readings ranged from 60-500 mg/dL, and he also reported a 40-pound weight loss over the past year. The patient had been using insulin over the counter and did not consistently check daily blood sugars, self-adjusting his insulin doses based on subjective lightheadedness or high blood sugar readings. On the day of admission, he presented to clinic for complaints of falls and generalized weakness and was found to be hyperglycemic to 556 mg/dL with an HgA1c of 11.5% . He was sent to the emergency department, where he was hypotensive and fluid responsive. Physical exam was significant for weak appearance and orthostatic hypotension. All other pertinent ROS were normal. The patient was admitted to the medicine team for workup of lightheadedness and optimization of insulin regimen/blood glucose control. Of note, the patient was hyponatremic, had a lactate of 2.7, and an AKI, all of which resolved with appropriate fluid resuscitation. CT scan of the head did not show abnormal pathology. The patient continued to have labile glucose levels, and on hospital day 3, the endocrinology service was consulted. The patient was placed on a custom sliding scale and had further workup revealing a cortisol level diagnostic of adrenal insufficiency. Cosyntropin stimulation test, 21-hydroxylase antibodies, aldosterone level, renin activity, T4, and TSH were ordered thereafter. Cosyntropin stimulation test confirmed the diagnosis, and ACTH level was 1.7 pg/mL (low). The patient improved on a regimen of 50 µg fludrocortisone and 15 mg hydrocortisone in the morning and 10 mg in the evening. The patient denied previously using any corticosteroid medications, and pituitary MRI the following day showed no abnormalities. The patient had a final diagnosis of central AI/idiopathic ACTH deficiency and discharged with appropriate steroids and endocrinology follow-up.

Discussion

- Adrenal insufficiency (AI), both primary and central, can present with nonspecific signs/symptoms of fatigue, weight loss, GI symptoms, myalgias, arthralgias, psychiatric symptoms, postural hypotension (presyncope/ syncope), hyperpigmentation, hypoglycemia, etc.
- Delayed diagnosis of AI is not uncommon, owing to the broad, nonspecific symptoms with which it presents.
- Once AI is suspected, workup is performed to determine the etiology (primary vs central). This involves obtaining a morning cortisol, an ACTH level, and, if the cortisol level is low, performing a cosyntropin stimulation test. ACTH levels help determine the level of AI as primary or central.
- The treatment for AI is glucocorticoid therapy for all patients as well as mineralocorticoid therapy for those with primary AI. Hydrocortisone is the preferred glucocorticoid due to its partial mineralocorticoid activity.
- In patients with any autoimmune disease, the differential diagnosis for a new presenting problem should be expanded to include other autoimmune processes.
- Our case emphasizes the importance of having a low index of suspicion for AI as an etiology for presyncope, orthostatic hypotension, and falls.