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Case Report

Primary adrenal insufficiency presenting with neuropsychiatric illness

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ABSTRACT

Adrenal insufficiency is characterized by decreased production of glucocorticoids from adrenal cortex, either due to a primary adrenal or a secondary pituitary etiology. Neuropsychiatric symptoms and signs could be the first presenting symptoms or the only presentation of adrenal insufficiency and are relatively less explored in patients with adrenal insufficiency and less understood. Patients with chronic psychiatric symptoms, namely depression and anxiety should be evaluated for association with adrenal insufficiency.

Keywords: Adrenal insufficiency, Glucocorticoids, Neuropsychiatric, Pituitary

INTRODUCTION

Chronic adrenal insufficiency has a constellation of typical symptoms and signs including fatigue, weakness, weight loss, nausea, vomiting and abdominal pain. The neuropsychiatric aspect of the condition remains to be thoroughly investigated. Illness started with cardinal symptoms like dizziness, generalized weakness, later progressing to neuropsychiatric symptoms.[1] We present case report of a 26-year-old female presenting with headache, dizziness, low mood, crying spells and seizure like activity, worked up with repeated brain imaging, EEGs, on antiepileptic treatment, finally diagnosed with chronic adrenal insufficiency.

CASE REPORT

A 26-year-old female with no known comorbidities, presented to our casualty with complaints of intermittent dizziness for 10 months. Dizziness and headache. Headache was diffuse, intermittent, moderate to severe in intensity and increased in intensity and duration gradually. Later in the course, headaches were associated with clenching of teeth and crying spells and outstretching of upper and lower limbs. There was no associated history of tongue bite, up rolling of eyes, urinary incontinence or any post ictal confusion or deficit. History of generalized weakness with low mood was present for 10 months. History of weight loss of approximately 5 kg was present in 8 months, with no associated history of loss of appetite, however she complained of nausea on and off. History of hair loss was present for 4-5 months. There was no history of fever, joint pain, rashes, oral ulcers, neck rigidity or photophobia. In view of above-mentioned complaints, she was thoroughly evaluated by a neuro-physician on multiple occasions MRI brain was done three times in last nine months, which showed normal study on all three occasions. VEP, H reflex and nerve conduction study were also normal. She was put on anti-epileptic treatment by an outside physician in view of seizure like activity, since her symptoms did not subside, she was later started on antipsychotic treatment by her physician. However, despite antipsychotic and antiepileptic treatment her symptoms persisted, and she presented to our hospital with a witnessed episode of crying spell and outstretching of hands and feet. On general physical examination, she was conscious, oriented to time, place and person and had hyperpigmentation around elbows and in the nape of neck. Her physical parameters were BP-90/50 mmhg, PR-80 beats/min, oxygen saturation-98% on room air. Systemic examination was insignificant. Initial

investigations showed Hb 12 g/dl, TLC-11.78 thous/ul, platelets-306 thous/ul, ESR-18 mm in first hour, CRP and procalcitonin were negative. RBS-105 mg/dl serum creatinine-0.64 mg/dl, serum calcium-9.4 mg/dl, sodium/potassium levels-139/4.85 meq/l. Electroencephalography was done, which showed normal study. Autoimmune work up was done and c-ANCA, p-ANCA and Antinuclear antibody profile were negative. Mantoux test and TB QuantiFERON gold test were both negative. Ultrasound abdomen showed chronic cholelithiasis. In view of persistent low blood pressure and low mood 8 am and 4 pm cortisol levels were done which were less than 3 meq/l. Subsequently cosyntropin stimulation test with 250 ug cosyntropin were done and 8 and 9 am cortisol levels showed subnormal elevation after cosyntropin stimulation. Corticosteroid therapy was started and the patient's condition improved significantly.

Table 1: Routine investigations.

Tests		Results
CBC	Hb	12 gm/dl
	TLC	11.78×10^3/mm^3
	Platelets	306×10^3 thousand/mm^3
KFT	Creatinine	0.64 mg/dl
	Na/K	139/4.85 meq/l
LFT	Total/direct bilirubin	1.00/0.9 mg/dl
	total protein/ albumin	4.2/2.4 gm/dl
	SGOT/SGPT	35/22 IU/l
	ALP/GGT	257/64 IU/l
PT/INR/APTT		12.4 s/1.11/36.3 s

DISCUSSION

Adrenal insufficiency is an uncommon condition, resulting from inadequate glucocorticoid secretion from adrenal glands. Either hypothalamic-pituitary axis disturbance or primary adrenal failure itself is the cause for this condition. The hormones released from adrenals are crucial for energy, salt and fluid homeostasis. Clinical picture may vary from asymptomatic hormonal dysfunction to adrenal crisis.[2] It usually presents with a constellation of symptoms including fatigue, weakness, weight loss, nausea, vomiting, abdominal pain. Our patient had few of these symptoms with neuropsychiatric symptoms like low mood, headaches, crying spells and clenching of teeth, with outstretching of hands and feet. Presentation with neuropsychiatric symptoms and signs in adrenal insufficiency is rare and has not been thoroughly investigated.[3] Prompt diagnosis and management of adrenal insufficiency are important since adrenal crisis may prove fatal for the patient if unrecognized. Adrenal crisis is more common in primary AI than in central AI because of concomitant loss of mineralocorticoids. It presents with acute deterioration of health, involving hypotension, acute abdominal symptoms, which resolve after corticosteroid therapy.[4] Skin pigmentation is a significant feature to differentiate primary from central AI, which is due to activation of melanocortin 1 receptors by elevated levels of ACTH. Prominent skin hyperpigmentation prompts us for working up the patient for primary adrenal insufficiency.[5] Random cortisol levels within normal ranges may not be able to exclude adrenal insufficiency, as results towards lower limit of normal range may indicate early adrenal insufficiency and warrants concomitant ACTH stimulation testing to rule out primary adrenal insufficiency.[6] Autoimmune workup of the patients with primary adrenal insufficiency should be done, since autoimmunity is one of the most common causes of Addison's disease and is associated with other autoimmune disease processes, namely vitiligo, type 1 diabetes mellitus, polyendocrinopathies, autoimmune thyroid disease.[7] This may be one of the many cases in which diagnosis of adrenal insufficiency is missed in the pretext of psychiatric symptoms and treatment for psychiatric illness may not be enough to make life easy for these patients. Treatment with glucocorticoids generally does wonders for these patients.[8] Furthermore, loss of glucocorticoids also results in decreased responsiveness to angiotensin through RAAS system and adrenergic hormones. Also, mineralocorticoid deficiency should also be evaluated in patients with primary adrenal insufficiency, as zona glomerulosa is also involved in the disease process in primary adrenal insufficiency.[9] Treatment with glucocorticoids and regular follow up is important in these patients as non-compliance to treatment can cause adrenal crisis and can be life threatening.

CONCLUSION

To conclude, neuropsychiatric symptoms, may be the first presentation or may present in addition to the typical signs and symptoms of adrenal insufficiency and warrant evaluation for the same. Our patient was evaluated for neurological and psychiatric signs, and was put antiepileptics and antipsychotics respectively, but finally improved on corticosteroid therapy after the diagnosis of adrenal insufficiency.

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