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Research Article

CHALLENGES IN THE MANAGEMENT OF ADDISON'S DISEASE-A CASE STUDY REPORT

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ABSTRACT

Addison's disease is a rare endocrine condition due to adrenal insufficiency which results in severe weight loss. The main objective of the study is to investigate the diagnosis and treatment of Addison's disease. A 35-year-old female presented to the hospital with fever, generalized weakness and blisters all over the body. She had known history of epilepsy and was in coma for 13 days in 2013. Nutritional screening was done in which there was weight loss, loss of appetite and decreased intake. Scores obtained from PGSGA and NRS 2000 depicts that the patient is nutritionally at risk. Appropriate medication and diet have to be maintained to manage Addison's disease.

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INTRODUCTION

Addison's disease is a rare endocrine disease1 in which there is destruction of the adrenal cortex with resultant inadequate secretion of the adrenal cortical hormones-cortisol, aldosterone and androgens. Thomas Addison (1855) first described the clinical features of primary adrenal insufficiency. Hence the term Addison's disease.[1]Adrenal insufficiency can result in fatigue and weakness. Adrenal insufficiency may cause persistent vomiting, anorexia, hypoglycemia, poor weight gain in children, or unexplained weight loss in an adult, malaise, fatigue, muscular weakness, hyponatremia, hyperkalemia, hypotension, hypoglycemia and especially generalized hyperpigmentation.[2]The main hormone affected in the disorder is cortisol that have an important role in the body's ability to cope with stressful situations such as infection, hypotension and surgical procedures. Addison's disease is a term restricted to primary adrenocortical insufficiency. Other secondary or tertiary causes of adrenocortical insufficiency are not included in the term - 'Addison's disease'. Primary adrenal insufficiency can be a lifethreatening disorder particularly in stressful situation, since cortisol secretion cannot be increased on demand at all.[3] The prevalence of primary adrenal insufficiency (Addison's disease) has been reported to be 39 to 60 per million population.[4]

Case presentation

A 35-year-old woman presented to the hospital with fever, generalized weakness, decreased appetite, weight loss and blisters all over the body. She had known history of epilepsy and was in coma for 13 days in 2013.Physical examination revealed generalized hyperpigmentation especially on the face, palmar creases and knuckles. Laboratory investigations and results are shown in Table 1. The patient was hyponatremic with a serum sodium of 133 mEq/L (135-150 mEq/L), magnesium 1.2 mg/dL (1.8-2.6mg/dL), calcium 2.6 mg/dL (8.8-10.6mg/dL), phosphorous 11.9 mg/dL (2.5-4.5 mg/dL), ALP 166 U/L (30-120 U/L), SGOT 72 U/L(<50U/L), total protein 5.7 g/dL(6-8g/dL), serum albumin 3.3 g/dL (3.5-5.2g/dL). Thyroid stimulating hormone was 5.86 µIU (0.34-5.60 µIU), cortisol 1.91 µg/dL, parathyroid hormone 1pg/ml (12-88pg/ml). Renal function tests were essentially normal. Contrast-enhanced computed tomography (CECT) abdomen revealed atrophied supra renal gland. CT brain has shown subtle calcifications. ECG was normal.

Table 1 Laboratory investigations

Test	Values	Reference values
Hemoglobin	10.1g/dl	12.0-15.0 g/dl
RBC	3.42mill/cumm	3.8-4.8 mill/cumm
Serum Creatinine	1.08mg/dl	0.66-1.09 mg/dl
Urea	26mg/dl	17.0-43.0mg/dl
Serum Sodium	133mEq/L	135-150 mEq/L
Serum Potassium	3.9mEq/L	3.5-5.0 mEq/L
Serum Chloride	93mEq/L	94.0-110 mEq/L
Serum Magnesium	1.2mg/dl	1.8-2.6 mg/dl
S. Calcium	2.6mg/dl	8.8-10.6 mg/dl
Serum Phosphorous	11.9mg/dl	2.5-2.6 mg/dl
T. Bilirubin	0.4mg/dl	0.3-1.2 mg/dl
D. Bilirubin	0.1mg/dl	0-0.2 mg/dl
I. Bilirubin	0.3mg/dl	-
ALP	166 U/L	30-120 U/L
SGPT	27U/L	<50 U/L

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SGOT	72U/L	<50 U/L
T. Protein	5.7g//dl	6-8 g/dl
S. Albumin	3.3g/dl	3.5-5.2 g/dl
S. Globulin	2.4g/dl	1.8-3.6 g/dl
A/G	1.4	1.2-1.5
TSH	5.86µIU/ml	0.34-5.60 µIU/ml
Cortisol	1.91 µg/dl	6.7-22.6 µg/dl (AM) Or <10 µg/dl (PM)
PTH	1.0pg/ml	12-88pg/ml
Free T4	0.94 ng/dl	0.61-1.12 ng/dl

DISCUSSION

On the basis of investigation results, the patient was diagnosed with autoimmune polyglandular syndrome, denova hypothyroid, hypoparathyroidism, primary adrenal insufficiency. She was in intensive care unit for five days and then shifted to ward for further follow up and management. Injections were given to correct all deranged parameters. Acute treatment included intravenous hydrocortisone 6hrly, with aggressive fluid resuscitation in the form of normal saline and inotropic support to treat hypotension, correction for hyperkalemia was also initiated. Patient was started with tapered dose of Intravenous Hydrocortisone. Mineralocorticoid treatment with Fludrocortisone once daily was simultaneously started. The patient was on nasogastric feeds (feeding volume 75ml/hr) yielding 1800kcal, 84 grams protein to meet nutrient requirements. Later gradually shifted to liquid diet and then to soft and normal diet.

The patient was discharged home on oral hydrocortisone and was counselled regarding the dietary management. Discharge diet sheet was given and patient was educated about the importance of compliance and follow up.

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