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# Addison's Disease With Acute Adrenal Crisis – A Case Report

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**ABSTRACT:**
Addison's disease being a rare endocrine disorder however, if it lands a patient into critical care unit as a primary pathology is a rare of the rarest . Addison's disease is characterized by a primary adrenal cortical insufficiency which is distinct from other forms of secondary adrenal insufficiency resulting from defects anywhere in hypothalamo – pitutary adrenal axis.
We are hear reporting a case of addison's disease 33 years old male who presented to our hospital with features consistent with acute adrenal insufficiency as hypotension, hyponatremia, hypoglycemia with gastrointestinal symptoms and fever.
This case adds to literature emphasizing the need for keeping a high index of suspicion for adrenal insufficiency when features are consistent with it amongst the other possible spectrum of other possible differentials presenting to ICU.

## I. INTRODUCTION :

Addison's disease is a rare endocrine disorder.[1] Primary adrenal insufficiency occurs due to total or near total destruction or dysfunction of one or both adrenal gland. Adrenal gland don't make enough of the hormones cortisol and aldosteron. Secondary adrenal insufficiency occurs when pitutary gland doesn't make enough of the hormone ACTH and results as adrenal gland don't make enough cortisol, Addisons disease term is restricted to only primary adrenal insufficiency. It is life threatening condition in stressful situation as cortisol secretion can not be increased or demand at all. Addisons disease has incidence of 0.8 million and a prevalence of 40-110 per million in the USA and European countries.[2]

## II. CASE REPORT:

A 33 years old man was referred to hospital by local physician with history of altered sensorium since 2 days patient also had history of abdominal pain loose motion since 10 days for which he had taken medication for 2 days, after that having constipation since 5 days. He lost 5 kg weight in previous 6 months, he had taken antitubercular medicine empirically for 2 months started by local hospital, sleep habit is disturbed since 1 month.

Patient also had complaint of bodyache, decrease appetite. No history of visual di disturbance. No past history of tuberculosis, diabetes mellitus, thyroid disorder. No history of alcohol intake, smoking, drug addiction, on general physical examination, the patient was drowsy, thin built, appeared weak and dehydrated, drooling of saliva from mouth, pulse of 58 bpm which was low volume, blood pressure of 90/60mmHg in supine position and respiratory rate 22 breath/min. Patient was febrile (101oF) . No other finding as pallor, lymphadenopathy, skin pigmentation. Remaining physical examination were normal. On systemic examination patient was drowsy and arousable, no focal neurological deficit, perabdomen there were diffuse mild tenderness allover abdomen, however, no guarding rigidity. Cardiovascular and respiratory system examination normal. Investigation where done for further evaluation.

Laboratory investigation showed a haemoglobin 11.2 gm/dl, total leucocyte count 3450µl, platelet 1,38 lac/cu.mm. The patient was hyponatremic as serum sodium was 122 mmol/l (135-149) previous documented sodium level was 117mmol/l, serum potassium was 4.74 (3.8 - 5.2mmol/l), serum chloride was 95.8 (100-105)mmol/l, liver functions test and renal function test were normal. Blood sugar level was 72 mg/dl. Routine examination of urine had not significant finding. Chest X-ray, USG abdomen was normal.

During the course of ICU, patient had neck rigidity and decreased sensorium. So we did MRI which was normal. The test result of montoux test was also negative. On 3rd day of admission patient had repeated episode of hypoglycemia despite supplement of dextrose infusion and hyponatremia despite of treated with 3% saline. Mean while for evaluation of hyponatremia we did serum osmolarity which was 270 mosm/L and urine osmolarity, which was 273 mosm/L, urinary sodium was 80mmol/L. So differential diagnosis kept in mind as addision's disease. Early morning 8a.m. serum cotrisol level measure and it came as 0.585µg/dl (6.42–21.00), cosyntropin test was performed found to be positive. Post-cosyntropin

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cortisol level was found to be 8µgm/dl. normally (16.0 – 18.0), after 60 minutes of stimulation. Thyroid function test are in normal range. Adrenocorticotropin (ACTH) was 13.8 pg/ml.

Contrast enhanced computerized tomography scan of the abdomen to visualised the adrenal gland showed significant thinning of bilateral adrenal glands without evidence of abnormal enhancing soft tissue lesion or calcification.

In this case, the absence of other pathology and suggestive MRI finding, autoimmune adrenalitis might have been the most likely cause of primary adrenal insufficiency. The patient was diagnosed as addison's disease. The patient was appropriately managed on IV saline infusion and dextrose infusion. Initially we gave injectable hydrocortisone 100mgthrice a day until he was stabilized. The patient showed improvement on third day of steroid treatment as serum electrolytes blood pressure blood sugar where within normal range. Patient was discharge on tablet prednisolone 40mg once a day and Tab. Fludrecortisone 100mg twice a day. Patient was also puton multivitamin calcium supplements.

## III. DISCUSSION:

In addison's disease, adrenal glands are unable to produce sufficient quantity of glucocorticoid, mineralcorticoid and androgen hormones. Most common cause of this rare endocrinopathy is bilateral destruction of adrenal gland due to autoimmune adrenalitis and tuberculosis. Tuberculosis is the most common cause of addison's disease in developing country like India and in Western World. Most common is autoimmune adrenalitis out of which 60-70% are associated with autoimmune polyglandular syndrome (APS)[3].

The incidence is 0.6 per 1,00,000 of the population per year. In great britain 39 cases per 1 million population and 60 cases per million population in Denmark.

Morbidity and mortality associated with addison's usually are due to failure or delay in making the diagnosis or failure to institute adequate glucocorticoid and mineralocorticoid replacement. Even after diagnosis and treatment, the risk of death is more than 2 fold higher in patients with addions's disease, cardiovascular, malignant, infectious disease, sepsis are responsible for the higher mortality rate.[4]

Common features of adrenal insufficiency include weight loss, anorexia, nausea, vomiting, lethargy, fatigue features of mineralocorticoid insufficiency – including postural hypotension, muscle cramps, abdominal discomfort and salt craving are more pronounced.

Skin pigmentation mostly over skin creases and scars, extensor surface of elbow, knuckles, lips and gingival mucosa is present in 90% of people with pri-adrenal failure hypoglycemia or reduction in insulin repairment in individual with diabetes mellitus. Adrenal insufficiency may associated with acute hemodynamic decompensation like hypovolaemia, hypotension, tachycardia, frequently with disorientation or impaired consciousness. It is known as adrenal crisis. Adrenal crisis occur in response to major stress, infection or trauma. It is medical emergency because individual with primary adreanal insufficency resulting in electrolyte and fluid volume disturbance. 90% of patient having hyponatremia other laboratory abnormalities like hypercalcemia, hypoglycaemia, normochromic normocytic anaemia, eosinophilia and lymphocytosis in secondary adrenal insufficency. Electrolyte imbalance is in less amount, features of pituatary disease like central hypothyroidism, hypogonadotropic hypogonadism, hyper prolactinaemia, visual field defect.[5]

Diagnosis of addison's disease done by cosyntropin test, cortisol level measured before starting of steroid therapy. Morning serum cortisol less than 100mmol/L strongly suggest adrenal failure. In autoimmune addison's disease plasma renin and ACTH is useful, as elevation of these hormone is present.

In case of low or normal ACTH, a pitutary magnetic resonance imaging scan should be obtained along with measurement of anterior pitutary hormone. Calcium level and thyroid function test should be done. Once primary adrenal insufficiency is the diagnosis contrast enhanced CT scan to visualise adrenal gland should be done. If adrenal glands are normal, screening for adrenal autoantibodies and measurement of very long chain fatty to rule out adrenoleukodystrophy.

Treatment started as soon as suspecting adrenal crisis without any delay. Treatment consist of volume replenishment give dextrose to correct hypoglycemia and glucocorticoid replacement therapy with IV hydrocortisone 100mg bolus and 50-100 mg hydrocortisone every 6 hourly over the 24 hours till patient stabilize.

Dexamethasone 4mg IV bolus considred in emergency when emergent steroid administration required. It is long acting and does not interfere with biochemical assay of endogenious glucocorticoid production.

In maintenance phase, these patient requires hydrocortisone5-25mg/day and

predinisolone 3-5 mg/day. Dose should be adjusted according to clinical response normalization of electrolytes abnormalities.

Fludrocortisone 0.05 – 0.02 mg daily should be administered to keep plasma renin level in the reference range patient should be educated regarding need of doubling steroid dose in time of stress and trauma and need urgent hospitalization. Calcium supplement should be added in patient receiving 30mg or more oral glucocorticoid as bone metabolism is affected.

## IV. CONCLUSION:

This case presented with feature of gastrointestinal disease and altered sensorium with fever. The presence of hypoglycemia, hypotension, hyponatremia leads to suspicion of addison's disease. Addison's disease can develop into an adrenal crisis if not recognized and promptly treated. Delayed recognition and treatment of hypoglycemia can cause neurological consequences. Supraphysiologic glucocorticoid replacement can leads to cushing syndrome. Delayed diagnosis leading to increased morbidity and mortality thus physician should keep a high index of suspicion for adrenal insufficiency.

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