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Original Research Paper

Medicine

ADDISON'S DISEASE SECONDARY TO TUBERCULOSIS OF ADRENAL GLAND: A RARE CASE REPORT

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ABSTRACT

Background: Addison disease is a relatively uncommon disorder that, if not diagnosed and treated properly, can have fatal implications. Because of the disease's insidious nature, many individuals do not receive a diagnosis until an acute adrenal crisis occurs.

Introduction: We describe a rare case of Addison's Disease Secondary to Tuberculosis of Adrenal Glands.

Diagnosis: Addison's Disease was diagnosed from Clinical Features, Biochemical Laboratory Parameter and CECT abdomen.

Interventions: Several drugs are used to treat the disease, including Glucocorticoids Like Prednisolone and Mineralocorticoid Like Fludrocortisone.

Outcomes: Under the medicine treatment of Prednisolone and Fludrocortisone Addison disease has very good prognosis and good compliance.

KEYWORDS : Addison's disease, Tuberculosis, Mineralocorticoids, Glucocorticoids

INTRODUCTION:

Addison disease is a relatively uncommon disorder that can have serious effects if it is not diagnosed and treated early. Because of the disease's insidious nature, many patients do not receive a diagnosis until a severe adrenal crisis has developed.

Addison disease is a rare condition.
The annual incidence rate is 0.6 per 100,000 people. At any one time, the overall number of people affected by this illness ranges between 4 and 11 per 100,000 of the population. Adults often present between the ages of 30 and 50. Incidence is more common in women than men.

Addison disease is a life-threatening illness that necessitates timely diagnosis and treatment. There is a considerable risk of morbidity and mortality if the diagnosis is delayed.

Case report:

A 37 years old patient presented with
Skin pigmentation since 3-4 months
Generalised weakness since 15 days.

Patient was apparently alright 3-4 months back than started noticing darkening of skin. Skin pigmentation was generalised and was present all over body. It was insidious in onset and progressive in nature. Patient also complains of generalised weakness with easy fatiguability during this period which was insidious in onset and progressive in nature now since 15 days patient is unable to walk and do his routine activity by himself and needs support.

- Patient also gives history of weight loss.
- Patient Occasional alcohol drinker,
- No h/o fever, chills, rigors, night sweats, evening rise of temperature.
- No h/o pain in abdomen, loss of appetite, loose motion, vomiting.
- No h/o bleeding manifestation from natural orifice
- Non-Diabetic, Non-Hypertensive
- No history suggestive of Pulmonary Kochs in past.
- Patient on admission vitals
- Bp-70/50mmhg
- Sugar -68mg/dl.
- Pulse: 104bpm, regular
- Spo2- 97% Room Air
- Temperature- 98.4 F

On examination Patient had generalised skin pigmentation with systemic examination within normal limits.

Routine Biochemical investigation revealed
Serum Electrolytes: K-5.8mEq/L, Na-122mEq/L, Calcium-10.9
HIV: Non-Reactive

8AM Serum cortisol:
3microg/dL (normal levels: 5-23microg/dL)
8AM Serum ACTH levels: 442pg/dL (normal levels:10-60pg/dL)

Random blood Sugar - 68mg/dl.
ECG: 104 bpm, Normal Axis /no ST T wave changes.
Chest X Ray PA: suggestive of Mild Cardiomegaly with
USG Abdomen and pelvis: Normal

Computed Tomography with Contrast of Abdomen:
Bilateral adrenal gland appears heterogeneously enhancing with few areas of non enhancement seen likely necrotic area. Tiny foci of coarse calcification likely granulomas. Mostly likely these findings represent sequele of Tuberculosis.

Fig.1 On admission

Fig 2. After 6 month of treatment.

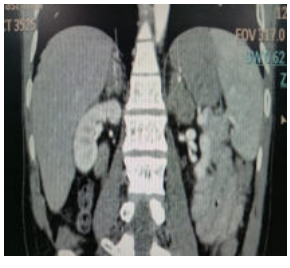

Fig.3

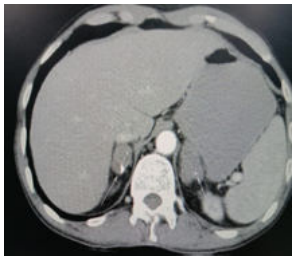

Fig.4

Fig 3 and Fig 4: Contrast Enhanced abdominal computed tomography image showing enlargement of the bilateral adrenal glands with calcification.

Management:

Patient was resuscitated with IVF, IV D25, inotropic support on admission and intravenous hydrocortisone. Later on, patient was tapered from inotropes. Patient was also given tablet fludrocortisone 0.1mg od, tablet prednisolone 40mg od. Patient did not receive any antitubercular Medication as calcified granuloma was present in adrenal gland with no active tubercular lesion. Patient was discharged on tablet fludrocortisone 0.1mg od and tablet prednisolone 40mg od which he was advised to take lifelong.

DISCUSSION:

Addison disease is characterised by a slow and gradual emergence of non-specific symptoms, which often leads to a misdiagnosis. The signs and symptoms may develop with time, making early detection difficult. To avoid misdiagnosis, maintain a high level of clinical suspicion. The diagnosis is often made only after the patient has experienced an acute adrenal crisis (hypotension, hyponatremia, hyperkalemia, and hypoglycemia). A stressful disease or triggering circumstances such as infection, trauma, surgery, vomiting, or diarrhoea can cause this. Cortisol and mineralocorticoid insufficiency might be revealed by significant stress or illness. Addison disease can strike anyone at any age, but it is most common in the second or third decades of life. Fatigue, generalised weakness, weight loss, nausea, vomiting, stomach discomfort, dizziness, tachycardia, and/or postural hypotension are some of the first symptoms. Hyperpigme ntation is a common symptom that affects almost all individuals. It's normally widespread, but it's more noticeable in sun-exposed and high-pressure places. The causes are elevated ACTH and melanocyte-stimulating hormone. ACTH is thought to bind to the melanocyte receptors, which are involved in pigmentation. Palmar creases, gingival mucosa, lips (especially vermilion border), elbows, knuckles, posterior neck, breast areola, nipples, and nail beds show increased hyperpigmentation. Because ACTH and MSH levels are low in secondary insufficiency, hyperpigmentation does not occur. It's possible that multiple new nevi will appear. Decreased or sparse axillary and pubic hair may occur in female patients.

When analysing a collection of non-specific symptoms, a strong index of suspicion for Addison disease is required due to its varied presentation. Unusual exhaustion, a loss of appetite, chronic stomach pain, or weight loss are some of these symptoms. In Addison disease, hyponatremia with or without hyperkalemia and/or hypotension is common. Severe dehydration, disorientation, refractory hypotension, and shock are common symptoms of Addisonian crisis. Primary adrenal insufficiency is more likely to cause it than secondary adrenal insufficiency.

The causes of Addison's disease are autoimmune adrenalitis, primary tubercular adrenalitis, secondary metastasis, meningococcus infection and CMV adrenalitis in HIV positive patients.

In a patient with Addison's disease, the differential diagnosis includes.

Sepsis

Many symptoms of sepsis are similar to those of adrenal insufficiency. Weakness, weariness, vomiting, hypotension, and shock are among symptoms. Low cortisol response to ACTH stimulation test and low ACTH level validate the diagnosis of primary adrenal insufficiency.

Shock (due to any cause)

Plasma cortisol level with shock suggests adrenal insufficiency.

Hypothyroidism

Fatigue can be evident in hypothyroidism, just as it can be in adrenal insufficiency. It is, however, linked to weight gain. Both conditions should be distinguished by cortisol levels.

To differentiate these disorders, a thorough history and physical, as well as laboratory and imaging workup, are required.

Addison's disease is treated by replacing glucocorticoids and mineralocorticoids in the body for the rest of the life.

CONCLUSION:

The treatment for Addison disease is glucocorticoid and mineralocorticoid replacement for the rest of one's life. To avoid over- or under-treatment with glucocorticoids, a careful balance is essential. As a result, cautious observation is essential. Overuse of glucocorticoids has been linked to obesity, diabetes, and osteoporosis. Overuse of mineralocorticoids can lead to hypertension. As a result, both medications must be monitored on a frequent basis.

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