ADDISON’S DISEASE- A CASE REPORT
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Abstract:
Addison’s disease is a rare endocrine disease which is commonly due to autoimmune adrenalitis and tuberculosis. Usual manifestations involve chronic fatigue, muscle weakness, loss of appetite, nausea, vomiting, diarrhea, hypotension and hyper pigmentation of skin. A 64 year old female, presented with complaints of cough with expectoration, fever, breathlessness, darkening of skin and diarrhea. The treatment given for the patient was Inj. Dexamethasone for 4 days. Her symptoms resolved gradually after starting the therapy.

Keywords: Addison’s disease, Cortisol, Autoimmune Adrenalitis, Treatment, Dexamethasone.

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INTRODUCTION:
Addison’s disease which is also referred to as primary hypoaldrenism, is caused by a total (or) near total destruction of both adrenal cortices. [1] This results in decreased secretion of the adrenal cortical hormones - cortisol, aldosterone and androgen. The symptoms of adrenal insufficiency is characterized by weight loss, muscle weakness, fatigue, low blood pressure, nausea, vomiting, diarrhoea and darkening of skin in both exposed and non-exposed parts of body.[2] The deficiency in Adrenocroticotrophic hormone (ACTH), can produce hypocortisolism, which is known as secondary adrenal insufficiency. Addison’s Disease is a term restricted to primary adrenocortical insufficiency.[3] The prevalence of Addison disease has been reported to be 39-60 per million population.[4] Addison disease is usually found in association with other disease conditions, therefore, proper knowledge of the disease can lead to prompt diagnosis and earlier management once the diagnosis is made.[5] Treatment for Addison’s disease includes hormone replacement therapy to normalize the steroid level, so the treatment involves both oral and injection corticosteroids.

CASE REPORT:
A 64 year old female was hospitalized due to cough with expectoration, fever for past 10 days followed by breathlessness, muscle weakness, diarrhoea 2 episodes per day and darkening of skin. Past medication and medical history were not recorded. On general examination patient was conscious and comfortable. But in systemic examination patient was found to have crepts with decreased breath sounds in right infra axillary and infra scapular region. Parahilar opacities was seen in Chest X-ray, by considering the symptoms patient was diagnosed with bronchial Asthma initially.

Laboratory investigation showed a low level of WBC - 2.7x10^9/L and slightly increased PLT level of 314x10^9/L. She also showed anemic parameters with a hemoglobin level of 5.9g/dl, with decreased value of MCV – 65.6 fl, MCH – 16.5 pg and normal RBC count. Peripheral smear examination revealed patient had microcytic hypochromic anaemia.

As the patient showed symptoms like darkening of skin, muscle weakness and diarrhoea, physician advised to check the cortisol level to rule out other endocrine diseases. Cortisol level was found to be 1.33µg/dl, lower than the normal. In accordance with the symptoms and the cortisol level, patient was diagnosed with Addison’s disease.

Treatment was started with Intravenous (IV) saline infusion, Ringer lactate, Cefazolin IV 1.5gm BD, Pantoprazole IV 40mg OD, Metronidazole IV 500mg TDS, Cap. Racecadotril TDS, Neb. Duolin and Budecort Q12H, Tab. Fours B OD, Tab. Dolo 650mg TDS. On the next day, Injection Cefazolin was changed to Injection Ceftriaxone 1gm BD. Cap. Acebrophylline and Tab. Mucolite were added to the therapy for expectoration as it aggravated day by day. Inj. Dexamethasone 1 ml OD was added to the therapy, but after two days it was stopped. Injection Dexamethasone was restarted again as the patient was diagnosed with Addison’s Disease. Blood transfusion was done as the patient was Anaemic. Discharge medications were shown in Table 1:-

<table>
<thead>
<tr>
<th>Sl. No.</th>
<th>Discharge Medication</th>
<th>Dose</th>
<th>Frequency</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Tab. Pantoprazole</td>
<td>40mg</td>
<td>OD</td>
</tr>
<tr>
<td>2</td>
<td>Tab. Fours B</td>
<td></td>
<td>OD</td>
</tr>
<tr>
<td>3</td>
<td>Tab. Wysolone</td>
<td>5mg</td>
<td>OD</td>
</tr>
<tr>
<td>4</td>
<td>Cap. AB-Flo</td>
<td>100mg</td>
<td>BD</td>
</tr>
</tbody>
</table>

DISCUSSION:
Thomas Addison first described the clinical features of primary adrenal insufficiency in 1855. Characteristics of the disease result from a variety of pathological processes.

Addison’s disease usually manifest with diverse and non-specific clinical features.[3,4] The disease may present with signs of acute abdominal or neurological disease, along with depression and decreased responsiveness.[6] The most precise sign is hyperpigmentation of the skin and mucosal surfaces associated with fatigue and weight loss. Addison’s disease also account for the delay in the diagnosis in some cases.[1,4]

The diagnosis of Addison’s disease is done by short consyntropin test, where cortisol values are measured after IV or IM administration of ACTH. Second step to measure plasma ACTH levels to determine primary or secondary cause of adrenal insufficiency.[2] The mainstay of therapy for Addison’s disease is steroids such as dexamethasone and prednisolone.

In this case the treatment was started with volume replenishment and glucocorticoid replacement.
therapy, like Inj. Dexamethasone as it is the core therapy for this disease. After starting the treatment patient condition got improved as the days progressed. Inj. Metronidazole and Cap. Racecadotril was given to treat diarrhoea. Acute kidney injury was reversed after volume replacement. The patient was also diagnosed with Microcytic anemia and Bronchial Asthma. Asthma was treated with Inj. Ceftriaxone, Neb. Duolin and Budecort, Cap. Acebrophylline and blood transfusion was done for Anemia.

CONCLUSION:
Addison’s disease is an endocrine disorder affecting the adrenal glands. As per the studies, the nature of the disease, signs and symptoms vary from patient to patient. Due to its varying nature there is a delay in the diagnosis of the disease. In most of the cases, diagnosis is made after a patient experience an acute adrenal crisis. This disease form can be often precipitated by any infection or stress conditions in an undiagnosed patient. In this study patient was diagnosed in accordance with the progressed symptoms and lower cortisol level. This case highlights that the healthcare professional should be aware of the atypical presentation and basic investigations, as these may hint towards the proper diagnosis of the Addison’s disease.

REFERENCES: