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Addison's disease Masquerading As Tuberculosis

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ABSTRACT: We herein report a case of Addison's disease characterized by atypical hyperpigmentation, noted as exacerbation of the pigmentation of freckles and the occurrence of new freckles. When exacerbation of the pigmentation of the freckles and/or the occurrence of new freckles is noted with persistent hypotension and hyponatremia, Addison's disease should be considered as part of the differential diagnosis. In addition, the presence of active tuberculosis needs to be assumed whenever we face patients with Addison's disease, despite its rarity.

KEY WORDS: Addison's disease (AD), adrenal insufficiency, pulmonary tuberculosis

I. INTRODUCTION

Addison's disease is a rare endocrinal disorder that was first described by Thomas Addison in 1855 [1]. The two most common causes of Addison's disease are autoimmune adrenalitis and tuberculosis [2]. As a result of the recent substantial reduction in the incidence of tuberculosis in developed world, the number of cases of Addison's disease caused by tuberculosis has also significantly decreased [3].Though in developing countries like India, tuberculous etiology is found in 47% cases.[4]

Generalized cutaneous hyperpigmentation is a characteristic physical finding of Addison's disease [5]. Generalized hyperpigmentation is observed on sun-exposed skin and over pressure areas, such as the elbows and knees, in most cases [6]. In addition, tuberculosis is inactive in the majority of patients with Addison's disease.

The incidence of adrenal tuberculosis and Addison's disease has declined over the past decade due to introduction of Antituberculous Therapy. Since symptoms are non-specific, diagnosis is often delayed and patients may first present with a life-threatening crisis.

II. CASE REPORT

A 32-year-old man presented to Respiratory Medicine OPD of our hospital, with history of taking ATT from past one month. On presentation, he had malaise, and anorexia from past 1 month, acute onset left sided chest pain, shortness of breath, vomiting and fever from past 1 day. He also complained of loss of appetite and weight loss from past 2 months. He gave history of suffering from Varicella 3 months ago. He showed freckles on his face and trunk that had been darkening every day, since resolution of Varicella. The patient had not given any remarkable medical history or known allergies. He was not taking any medications, did not smoke or drink alcohol, but was a tobacco chewer.

On a physical examination, the patient's weight was 46 kg, and his height was 160 cm. His blood pressure was 90/60 mmHg; pulse, 102 beats per minute; body temperature, 36.4℃; respiratory rate, 21 breaths per minute; and oxygen saturation level, 99% while breathing ambient air. A skin examination revealed brown freckles mainly on his face, chest and palms. His physical examination findings were otherwise normal.

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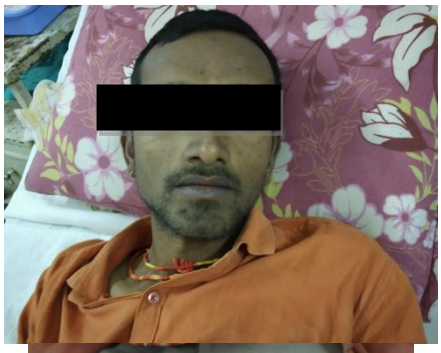
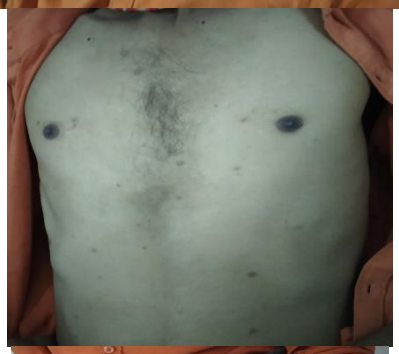
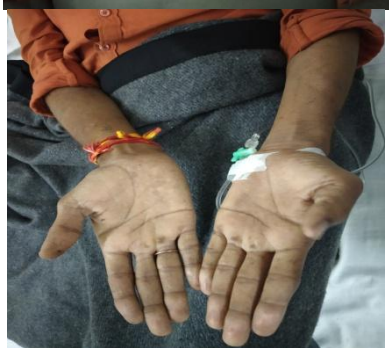
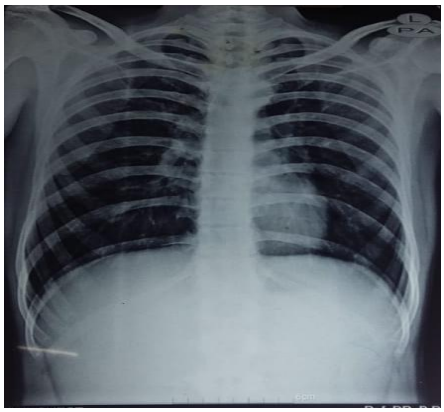






Laboratory data showed a white blood cell count of 6,900/µL, hemoglobin level of 09 g/dL, platelet count of 1.66 lac/cmm, Erythrocyte sedimentation rate of 15, albumin level of 2.88 g/dL, creatinine level of 0.79 mg/dL, sodium level of 118mEq/L, potassium level of 4.9 mEq/L, chloride level of 83 mEq/L fasting blood glucose level of 51 mg/dL.

4 samples of Sputum AFB came out to be negative. Chest X Ray was within normal limit.



USG Whole Abdomen showed moderate splenomegaly. ECHO was done which was within normal limit.

Since patient's chest X-ray and Sputum AFB were within normal limit, and taking vomiting, hyperpigmentation, hypotension and hyponatremia into account, we decided to investigate for Addison's disease.

S. cortisol level came out to be 1.14 µg/mL (normal range, 4.0-19.3 µg/mL), and adrenocorticotropic hormone (ACTH) level came out to be 944 pg/mL (normal range, 7.2-63.3 pg/mL).

Based on negative sputum sample, normal chest x ray, Pulmonary TB was ruled out completely.

And based on Cortisol and ACTH level, we decided to carry out CT Abdomen with contrast to look at Adrenal Glands. CT Abdomen showed borderline hepatomegaly and moderate splenomegaly, bilateral adrenal glands were normal.

Intravenous saline and intravenous hydrocortisone were administered to treat the hyponatremia and adrenal insufficiency, resulting in the marked amelioration of his symptoms, such as general malaise and anorexia, vomiting and hyponatremia.

Thus, on the basis of clinical, laboratory, radiological and microbiological findings, diagnosis of Addison's disease, probably due to autoimmunity, were made.

III. DISCUSSION

Addison's disease is a rare disease that affects 1 in 100,000 people [7]. Autoimmune diseases account for 70-90% of the underlying conditions of Addison's disease, while tuberculosis constitutes only 7-20% of cases [8], and the incidence of Addison's disease has been decreasing along with the decline of tuberculosis cases though in developing world, Tuberculosis is still very

prominent. The interval between tuberculosis and the onset of Addison's disease averages 32±15 years [3].Most cases of tuberculosis are inactive when Addison's disease develops. Furthermore, a previous report found that out of 94 Addison's disease patients had active tuberculosis [7].. Although the presence of active tuberculosis is rare in terms of the statistics for Addison's disease caused by tuberculosis, it needs to be considered whenever we treat patients with Addison's disease caused by tuberculosis.

No recent data are available about the incidence of Addison's disease caused by TB. However, as the incidence of TB has declined, the incidence of Addison's disease secondary to it is diminishing. In the developing world, where there is high incidence of tuberculosis, tuberculous adrenalitis is still the major cause of Addison's disease. According to a retrospective study, tuberculosis was found in 47% patients, suffering from Addison's disease.[4]

Common clinical features of Addison's disease are chronic malaise, fatigue, weakness, anorexia, weight loss, and hyperpigmentation[9]. Hyperpigmentation is observed in areas exposed to the sun. such as face, elbows and neck, due to ACTH melanogenesis[10]. The present patient demonstrated freckles on his face, and new freckles had appeared.

Thorn et al. reported eosinophilia to be a marker of adrenal insufficiency [11], which was not found in our patient. Hills et al. suggested that eosinophilia associated with Addison's disease was probably due to the low levels of circulating steroids [12].Though, a previous study reported the eosinophil count to be greater than 500/mm^3 in less than 20% of patients with adrenal insufficiency [13]. Evidence are still lacking to determine the differences between Addison patients with and without eosinophilia.

The serum cortisol concentration, serum ACTH concentration, and rapid ACTH stimulation test are important for the diagnosis of adrenal insufficiency. The present patient demonstrated a significant decrease in the cortisol level and an increase in the ACTH level, meeting the diagnostic criteria for Addison's disease without the rapid ACTH stimulation test.

Severe adrenal cortical insufficiency is characterized by vomiting, a fever, disturbance of consciousness, and shock and can be life-threatening if immediate steroid replacement therapy is not performed. In the present case, the patient complained of general malaise with low blood pressure, vomiting, fever and marked weight loss, and was considered to have severe adrenal cortical insufficiency. Therefore, we replaced steroid replacement therapy to restore the adrenal function.

IV. CONCLUSION

We herein reported a case of Addison's disease masquerading as tuberculosis, accompanied by hyperpigmentation. To our knowledge, due to prevalence of Tuberculosis, it still is major reason of Addison's disease. When progressive darkening of freckles and/or the emergence of freckles are noticed, Addison's disease should be considered as part of the differential diagnosis. In addition, the presence of active tuberculosis needs to be considered whenever we treat patients with Addison's.

CONFLICT OF INTEREST

None

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