PICTORIAL CME

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Primary Adrenal Tuberculosis Presenting as Addison's Disease

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A 30-year-old gentleman presented to our department with complaints of diffuse hyperpigmentation of skin for one year along with nausea, vomiting and loss of appetite for 8 months. He also had lost almost 25 kgs of weight during this period. There was no history of fever, any drug intake or tuberculosis in the past. On examination; his blood pressure was 84/50 mmHg, BMI – 17.1 kg/m^2 and he had diffuse hyperpigmentation of skin, including oral mucosa and palmar creases (Figs. 1 - 3). Rest of the systemic examination was normal.

Blood investigations showed normal haemogram with raised ESR, renal function tests were mildly deranged (urea-42 mg/dl, creatinine – 1.4 mg/dl, uric acid – 11 mg/dl), he also had hyponatraemia and hyperkalaemia (serum sodium – 131 mEq/l and potassium – 5.5 mEq/l). We had a strong suspicion of Addison's disease and hence a hormonal profile was done which showed very low serum cortisol levels [0.5 mcg/dl (normal: 3.5 - 19)], highly elevated ACTH levels [> 2,000 pg/ml (normal: 7.2 - 63.3)], low aldosterone level [1.55 ng/dl (normal: 5 - 30)] and raised renin levels [> 500 mIU/ml (normal: 8 - 35)]. Other investigations like thyroid profile, parathyroid levels, urinary histoplasma antigen, HIV and vitamin B12 levels were normal. A diagnosis of primary adrenal insufficiency was made and CECT chest and abdomen was done for aetiology, which showed heterogeneously enhancing mass in the left adrenal with central non-enhancing necrotic area and another heterogeneous nodule in the right adrenal gland with surrounding fat stranding. There was no evidence of any enlarged mediastinal or abdominal lymphadenopathy (Fig. 4). A CT guided biopsy from left adrenal mass showed necrotising granulomas, confirming the diagnosis of primary adrenal tuberculosis. He was started on category I antituberculous therapy along with prednisolone (7.5 mg in morning and 2.5 mg in evening). He responded well to the treatment and biochemical parameters improved within few days.

Fig. 1: *Diffuse hyperpigmentation over face and lips.*

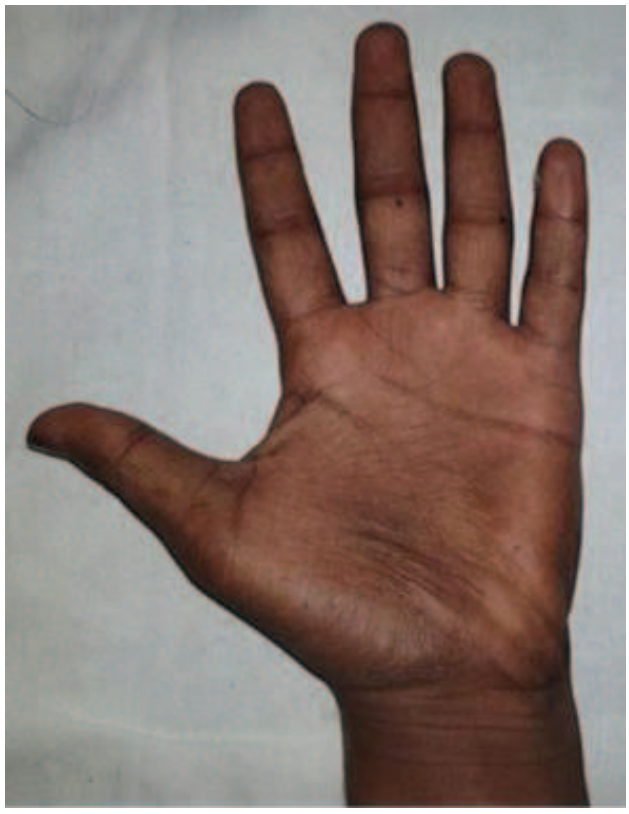

Fig. 2: *Palmar crease hyperpigmentation.*

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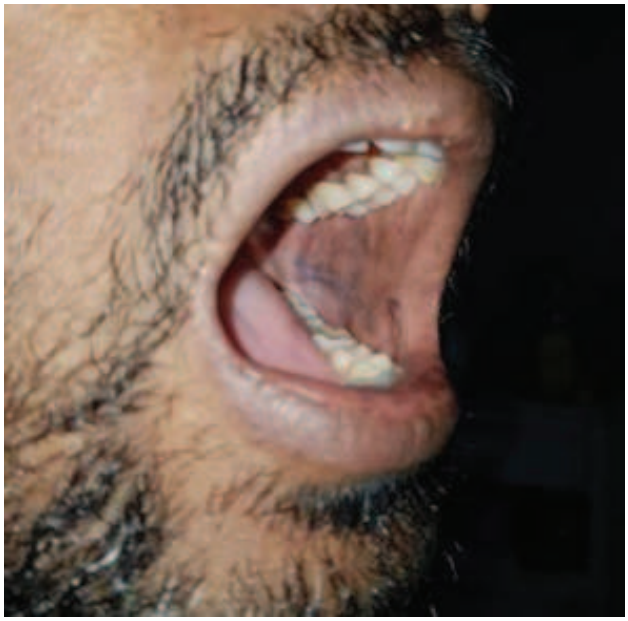

Fig. 3: *Oral mucosa hyperpigmentation.*

Addison's disease was first described by Thomas Addison in 1855, when he described adrenal insufficiency in eleven patients of adrenal tuberculosis. Adrenal tuberculosis is uncommon in developed countries, however, in developing world, adrenal tuberculosis still accounts for 20% of Addison's disease[1]. For adrenal insufficiency to appear more than 90% of the gland must be destroyed. The common symptoms being nausea, vomiting, fatigue and weight loss. Adrenal tuberculosis is generally associated with either tuberculosis of other site or history of tuberculosis in the past[2]. Only 12 per cent cases have isolated adrenal tuberculosis without active extra-adrenal tuberculosis[3]. CT imaging often shows bilateral enlargement of the adrenals in active tuberculosis and atrophy with calcification in remote infection[4]. Definite diagnosis is by biopsy of the adrenal gland which shows caseating granulomas. Antituberculous therapy does not restore adrenal function and these patients often need

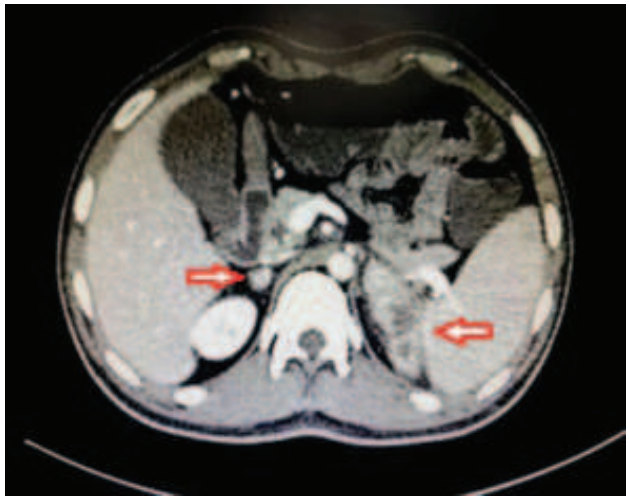

Fig. 4: *CECT abdomen showing bilateral adrenal enlargement (arrow).*

life long steroid supplementation. Also, dose of steroids should be increased if patient is on rifampicin, as it is a potent hepatic enzyme inducer. To conclude, isolated adrenal tuberculosis causing addison's disease is uncommon and physicians need to have high index of suspicion for this diagnosis.

References

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