Brief Communication

Evolving adrenal insufficiency

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ABSTRACT

Introduction: Tuberculosis is the most common cause of Addison's disease in India. The exact status of adrenal reserve in tuberculosis is still an enigma and recovery of adrenal function is unpredictable. **Objective:** We report a case with a pre-Addisonian state and unchanged adrenal size after 1 year treatment. **Materials and Methods:** A 31-year patient with adrenal tuberculosis was diagnosed and treated with anti tubercular drugs (ATDs) and steroid. **Results:** A 31-year male, presented with fever and weight loss for 3½ months with anorexia, nausea, hyperpigmentation of skin, and buccal mucosa and weakness with past h/o adequately treated pulmonary tuberculosis at 3 years of age. On examination, the patient was anemic. A non-tender, firm right (Rt.) submandibular lymphnode was palpable. Investigations revealed: High erythrocyte sedimentation rate (ESR), negative HIV, and sputum for acid fast bacilli (AFB). Initial cortisol was high but subsequently became low with negative short synacthin test (SST). Computed tomography showed bilateral (B/L) enlarged hypodense adrenal mass with inconclusive fine needle aspiration cytology (FNAC) and negative AFB culture. Rt. submandibular lymph node FNAC showed caseating granuloma. ATDs and steroids were started, the lymphadenopathy regressed and symptoms subsided. However, after 1 year of treatment steroid withdrawal failed and adrenal size remained the same. **Conclusion:** The adrenal has considerable capacity to regenerate during active infection and ultimately become normal or smaller in size. However, in the case reported here, they failed to regress. Reversal of adrenal function following ATD is a controversial issue. Some studies have shown normalization following therapy, while others have contradicted it similar to the finding in our case.

Key words: Evolving adrenal insufficiency anti tubercular drugs, Tuberculosis

Introduction

Tuberculosis is the most common cause of Addison's disease. The exact status of adrenal reserve in tuberculosis is still an enigma and recovery of adrenal function unpredictable.

A 31-year-old male presented with fever and weight loss for 3½ months with loss of appetite, nausea, progressive darkening of skin, and profound generalized weakness without any history of chronic cough or hemoptysis. There was no history suggestive of any meningeal involvement, any autoimmune endocrine involvement, or any history suggestive of connective tissue diseases (CTD) or vasculitis. There was a past h/o adequately treated pulmonary tuberculosis at the age of 3 years. There is no family history of TB, or high-risk behavior.

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On examination, patient was anemic with stable vitals without any postural drop of BP. A non-tender, firm lymphnode over the right submandibular region was palpable. There was hyperpigmentation of the skin and buccal mucosa. The patient was febrile and the systemic examination was normal [Figure 1].

Investigations revealed: Hemoglobin (Hb) = 10.4 g/dl, ESR = 70 mm, fasting blood sugar (FBS) = 86 mg/dl with normal renal function and electrolytes. Liver function tests (LFT) was normal except an increased alkaline phosphate (ALP) (495 U/l), chest X Ray (CXR): Normal. HIV: Negative, sputum for AFB: Negative. On day 10 (D10) after admission: Cortisol (8 am) = 14.9 μg/dl, Adrenocorticotrophic Hormone (ACTH) = 197.1 pg/ml; D22 cortisol (8 am) = 7.9 μg/dl. SST: 30 min cortisol

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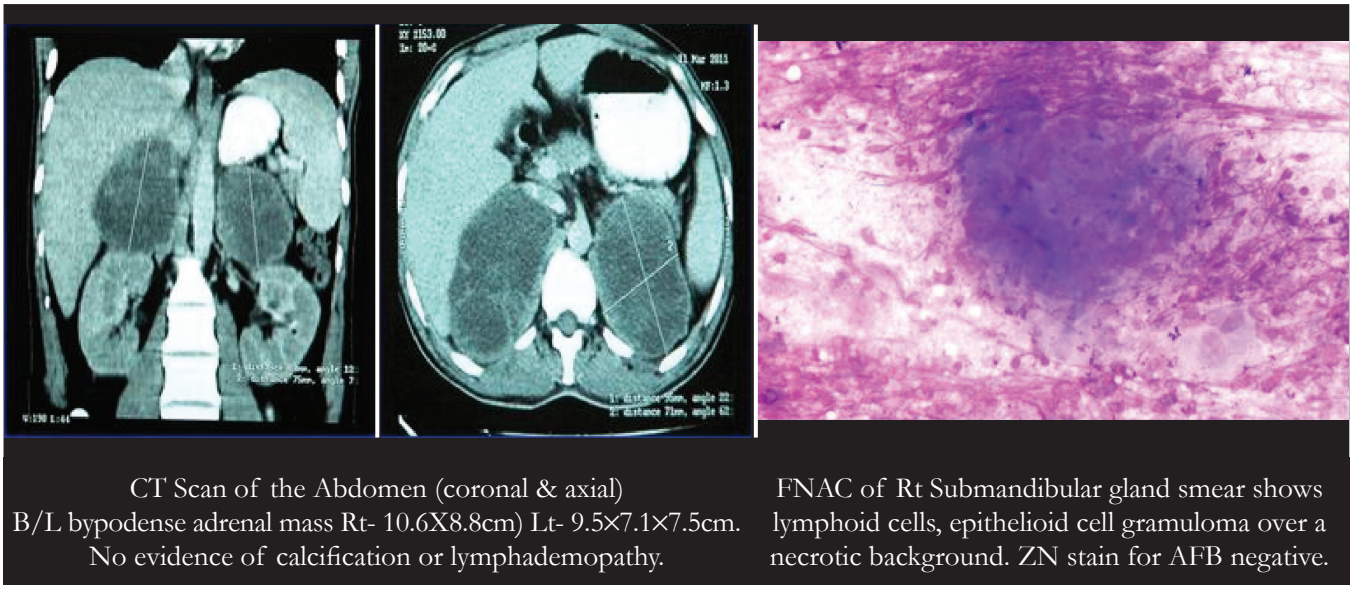

CT Scan of the Abdomen (coronal & axial) B/L bypodense adrenal mass Rt- 10.6X8.8cm) Lt- 9.5×7.1×7.5cm. No evidence of calcification or lymphademopathy.

FNAC of Rt Submandibular gland smear shows lymphoid cells, epithelioid cell gramuloma over a necrotic background. ZN stain for AFB negative.

Figure 1: FNAC of right submandibular gland: Lymphoid cells, epithelioid cell granuloma over a necrotic background with negative Ziehl Nelson (ZN) stain for AFB.

= 10.5 μg/dl; thyroid function test (TFT): within normal limits (WNL); computed tomography (CT) adrenal: B/L hypodense adrenal mass (Rt. 10.68 × 7.6 × 8.8 cm; left [Lt.] 9.5 × 7.1 × 7.5 cm). CT-guided FNAC: Inconclusive and culture and stain for AFB and histoplasma negative.

Thus, it was provisionally diagnosed as evolving adrenal insufficiency (tubercular etiology). Differential diagnosis of histoplasmosis and adrenal metastasis were considered, but ruled out by negative fungal culture and stain, and negative history of primary malignancy, and inconclusive FNAC.

The lymphadenopathy regressed and general well-being improved with a decrease in skin pigmentation following treatment with ATDs and glucocorticoid replacement. After 1 year of treatment, steroid withdrawal was attempted, but failed and adrenal size was almost the same (Rt. 9.7 × 9.2 cm; Lt. 10.4 × 8 cm).

Tuberculosis is known to affect adrenal glands directly. Rich vascularity and the high levels of local corticosteroids that suppress cell mediated immunity (CMI) make the adrenal gland an ideal nidus for *Mycobacterium tuberculosis*. Adrenal tuberculosis is seen in up to 6% of patients, with active tuberculosis and is usually bilateral. Adrenal destruction by tuberculosis may lead to overt or subclinical adrenal insufficiency, and it may present in an evolving state which was observed in this case.

The adrenal cortex has considerable capacity to regenerate with marked hyperplasia and hypertrophy of cortical cells, noted during the period of active infection. Ultimately, fibrosis ensues and the adrenals become normal or smaller in size with calcification evident in 50% of cases, after the disease becomes inactive.[1] CT scan of the abdomen in cases of tubercular adrenalitis, shows typical features of shrunken and calcified adrenals in chronic stage and enlargement in the active stage. However, in the present case, they failed to regress. Reversal of adrenal function following anti-tubercular therapy is a controversial issue.[2,3] Barnes *et al*.[3] reassessed adrenal function following therapy and showed that SST returned to normal in all, but one patient suggesting adrenal dysfunction to be uncommon and ATD having a favorable effect on adrenal function. While some of the studies have shown normalization of adrenal function following therapy in a large number of cases, others have contradicted it. In this case, because the adrenal size remained the same, it will be prudent to follow this patient for recovery of adrenal function and size.

References

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