A CASE OF DISSEMINATED TUBERCULOSIS PRESENTING AS ADDISON'S DISEASE

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Abstract: The term Addison's disease is now used to refer to all forms of chronic primary adrenocortical insufficiency. Autoimmune adrenalitis is the most common cause of adrenal insufficiency in the developed countries. Tuberculosis is still a common cause in the developing world. We report a case of a young male who presented with low grade fever, generalised hyperpigmentation, fatigue, headache, giddiness, postural hypotension, papilledema and proximal myopathy. A clinical diagnosis of Addison's disease was made which was confirmed by ACTH stimulation test. MRI brain revealed multiple tuberculomata and CT abdomen revealed lesions in the adrenal gland. The patient was put on steroids and category 1 ATT. The patient responded to treatment.

Keyword: Addison's disease, disseminated tuberculosis

CASE REPORT:

A 17 year old male hailing from Thiruvanamalai presented with complaints of increased pigmentation all over the body including the palms, soles and oral cavity with generalized fatigue for past 6 months. Patient complained of giddiness which increased on standing. The patient had history of low grade fever and diffuse dull aching headache. Patient suffered from loss of appetite and loss of weight. The patient had been bed-ridden for past one month due to the above complaints. No h/o contact with tuberculosis patients. The patient was on mixed diet, bowel and bladder habits were normal and he had no addictions. On examination the patient was conscious, oriented, afebrile and pale. He had no icterus, cyanosis, clubbing, pedal edema or significant lymphadenopathy. There was diffuse hyperpigmentation including hyperpigmented palms (fig.1), soles and oral cavity. Pubic and axillary hair was absent (fig.2). His pulse rate: 102/min, feeble, felt in all peripheral pulses; Blood pressure recorded in supine position - 96/60 mm of Hg, right upper limb. On standing: 60/36 mm of Hg right upper limb. Neurological examination revealed generalized wasting and a power of 4/5 in all 4 limbs more of proximal weakness than distal weakness. Deep tendon reflexes were just elicitable. Plantar response was flexor bilaterally. Fundus revealed bilateral papilledema. No neck stiffness was made out. Examination of cardiovascular system, respiratory system and abdomen did not reveal any abnormality.

Laboratory investigations revealed hemoglobin 7.6g/dl, TC 4300cells/cu mm, DC P50% L50%, platelet 1.4 lakh cells/cu mm, ESR-85mm/hour. Renal and liver function tests were within normal limits. ECG : rate -100/min, normal sinus rhythm. USG ABDOMEN : Normal. Thyroid profile was normal. HIV: Non reactive. MANTOUX: No reaction. Chest x-ray was normal.

ACTH STIMULATION TEST: SERUM CORTISOL AFTER ONE HOUR-0.18 µg/dl [Reference range: 18 – 20 µg/dl]
A provisional diagnosis of disseminated tuberculosis with multiple tuberculomata brain and tuberculosis of adrenal gland (Addison’s disease) was made. The patient was treated with category 1 ATT and hydrocortisone and the patient improved.

**DISCUSSION:**

**INTRODUCTION**

Overt clinical features of adrenal insufficiency appear only after the destruction of more than 80 to 90% of both the adrenal glands. However subclinical adrenal insufficiency may exist in stressful situations such as acute and chronic infections. The rich vascularity and the high local levels of corticosteroids which suppress cell mediated immune response, make the adrenal glands an ideal nidus for organisms, such as mycobacteria and histoplasma. Involvement of the adrenal gland frequently occurs following hematogenous dissemination in tuberculosis. Acute infections are usually associated with increased release of adrenal steroids. However adrenal reserve in chronic infections especially in TB has been a subject of great controversy. Estimation of adrenal reserve assumes greater importance in view of the appearance of reports of clinical deterioration and sudden death in patients with TB on commencement of ATT, especially rifampicin. Rifampicin, a potent inducer of hepatic microsomal enzymes, is known to reduce the half-life and tissue availability of corticosteroids. Hence it may unmask subclinical adrenal insufficiency which may lead to clinical Addisonian crisis.

**TUBERCULOSIS AND ADDISON’S DISEASE:**

In 1855, Thomas Addison first described the chronic type of hypoadrenalinism. Presently the term Addison’s disease is used to refer to all forms of chronic primary adrenocortical insufficiency. Several studies have revealed that autoimmune adrenalitis is the most common cause of adrenal insufficiency in developed countries and TB is still a common cause in the developing world. Activated hypothalamo-pituitary-adrenal axis in TB causes increased cortisol secretion which results in a shift in the Th1/Th2 T-cell dysfunction due to high cortisol and low dehydroepiandrosterone levels. This may be responsible for immunologically-mediated tissue damage in TB. Nearly half of the patients with active TB have a subclinical adrenal insufficiency indicated by an impaired response to ACTH stimulation test. The duration of symptoms was inversely proportional to basal cortisol levels, while a direct co-relation existed with parameters like sputum positivity, extent of disease, fever and raised ESR.

**CLINICAL MANIFESTATIONS:**

Patients with acute adrenal insufficiency present with severe hypotension or hypovolemic shock, acute abdominal pain, vomiting and fever. Patients with chronic adrenal insufficiency complain of fatigue, lack of stamina and energy, reduced muscle strength, irritability, nausea, anorexia, weight loss, arthralgia and hyperpigmentation.

**DIAGNOSIS: SHORT COSYNTROPIN TEST:**

Plasma cortisol is measured 30-60 minutes after 250µg Cosyntropin IM or IV. The cut off for failure is usually defined as cortisol levels of <18-20µg/dl. IMAGING APPEARANCES: In patients with Addison’s disease due to TB with recurrent or active infection, the adrenal glands may appear enlarged. Non-homogenous appearance and presence of non-enhancing areas may indicate foci of caseous necrosis. In patients with remote infection, the gland may be atrophied or calcified.
TREATMENT:
Acute- Rehydration with saline infusion – 1 litre and hydrocortisone 100mg Chronic- 15-25 mg oral hydrocortisone Category-1 ATT for 6 months.

CONCLUSION:
The incidence of adrenal involvement is around five percentage in patients with active tuberculosis at the same time more than fifty percentage of patients with active tuberculosis can have subclinical adrenal insufficiency which can worsen as the patient is started on ATT, so a high index of clinical suspicion and clinical monitoring is needed to identify Addisonian crisis.

REFERENCES: