Case Report

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Adrenal Insufficiency Mimicking Gastrointestinal Disorder: A Case Report

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Abstract

Adrenal insufficiency is a rare disease with varied and non-specific clinical manifestations. Tuberculosis is one of the major causes of adrenal insufficiency in many developing countries. Effect of tuberculosis may be evident even after several years of affection of the gland. Acute tuberculosis of the adrenal gland may result in enlargement of the gland, but chronic tuberculosis may shrink and calcify the gland. The diagnosis may be delayed if the clinical presentation mimics a gastrointestinal disorder or personality changes resembling a psychiatric illness. The disease carries a high degree of morbidity and mortality if not recognized and treated in time.

Key words: Adrenal insufficiency, Crisis, Gastrointestinal, Shock, Tuberculosis

INTRODUCTION

Adrenal insufficiency (Addison's disease) is a rare disease with an incidence of 0.8/100,000 cases.[1] The clinical presentation of the disease is varied and non-specific[2] and hence a diagnostic and therapeutic challenge for the consulting physician. The tuberculous affection of the adrenal gland is one important cause of adrenal insufficiency, especially in the developing countries. Tuberculosis may involve many endocrine glands including the hypothalamus, pituitary, and thyroid.

Adrenal gland may be involved by hematogenous spread of infection or rarely by primary involvement of adrenal gland.[3] The rich blood supply and suppressed immune response locally due to high concentration of corticosteroids make adrenal gland an easy target for Mycobacteria.[4] Post-tuberculous adrenal insufficiency may be diagnosed in acute, chronic phase, or during an episode of adrenal crisis. Sub clinical adrenal insufficiency observed during active pulmonary tuberculosis can reverse with treatment.[5] Addison's disease presents with gastrointestinal (GI) complaints in 20% of cases. The GI symptoms of adrenal insufficiency may be anorexia, nausea, vomiting, weight loss, constipation, diarrhea, and abdominal pain, especially during adrenal crisis mimicking an acute abdomen.

In addition to estimation of the adrenal reserve by cortisol estimation, imaging modalities such as computed tomography (CT) scan and magnetic resonance imaging of adrenals may aid in the assessment of the gland structure. A high index of clinical suspicion is a mainstay for the diagnosis of the disease and failure to recognize early carries a high rate of mortality if not treated in time. We report a case of a young boy who presented to us in a state of shock with preceding GI symptoms.

CASE REPORT

A young man aged 24 years was admitted with a history of fever, altered sensorium, abdominal pain, and vomiting of 2 days duration. The patient was having vomiting and episodes of diarrhea on and off, dyspepsia, and failure to gain weight for the past 6 months for which he had undergone various investigations including upper GI endoscopy, which was normal. He was treated on various

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occasions symptomatically with no lasting improvement. He was treated for pulmonary tuberculosis 1½ years back and was declared cured.

At the time of admission, the patient was febrile (101°F), conscious but drowsy, minimally responding to commands. There was no neck stiffness. He appeared dehydrated. His supine blood pressure was 60/30 mmHg, pulse 100/min regular. The boy was of dark complexion with hyperpigmentation evident over both palms (Figure 1a), tongue (Figure 1b) and a hyper pigmented scar (Figure 2). The fundoscopic examination was normal.

Investigations revealed the following findings: Hemoglobin - 14.3 g/dl, total leucocyte count-16500 cells/cu.mm, differential count-polymorphs - 68, leucocytes – 30, and eosinophils - 2% platelet count - 2, 52, 000 cells/cu.mm.

Random blood glucose - 62mg/dl, blood urea - 56 mg/dl, serum creatinine - 2 mg/dl, serum sodium - 122 meq/L, serum potassium - 5.5 meq/L, serum chloride - 89 meq/dl, and HCO_3 - 22 meq/dl.

Liver Function Tests

Total bilirubin - 0.7 mg/dl, direct bilirubin - 0.2 mg/dl, serum glutamic oxaloacetic transaminase - 43 IU/L, serum glutamic-pyruvic transaminase - 28 IU/L, alkaline phosphatase - 89 IU/L, total protein - 6.1 g/dl, albumin - 3.4 g/dl, HIV 1, and 2 (enzyme-linked immunosorbent assay) negative. Blood and urine culture - sterile, electrocardiography - sinus tachycardia.

X-ray chest posterior-anterior view-normal, CT brain (plain) normal study, ultrasonography abdomen-normal.

Random serum cortisol (chemiluminiscence immuno assay) - 0.19 mcg/dl (6.2-19.4 mcg/dl).

CT Abdomen

Bilateral atrophic and calcified adrenals due to tuberculous granulomatous lesions (Figure 3).

This patient was managed with normal saline dextrose infusion, inotropes, and antibiotics initially. The patient continued to be in hypotension for 4 h. In view of past history of tuberculosis with hyper pigmented lesions, adrenal insufficiency was suspected and after collecting serum sample for cortisol, the patient was started on hydrocortisone 100 mg IV Q6H following which the patient improved dramatically.

A final diagnosis of Addison's disease due to the post-tuberculous destruction of adrenal gland was made. The patient was discharged with oral prednisolone

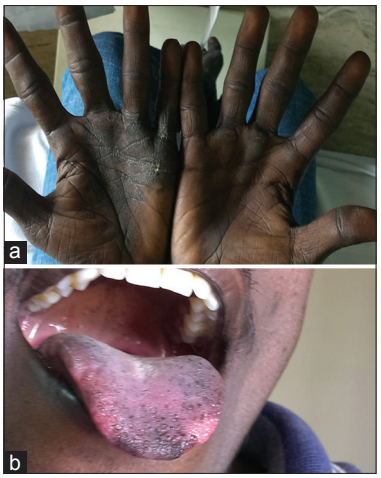

Figure 1: (a) Hyperpigmentation of palms, (b) shows hyperpigmentation of tongue

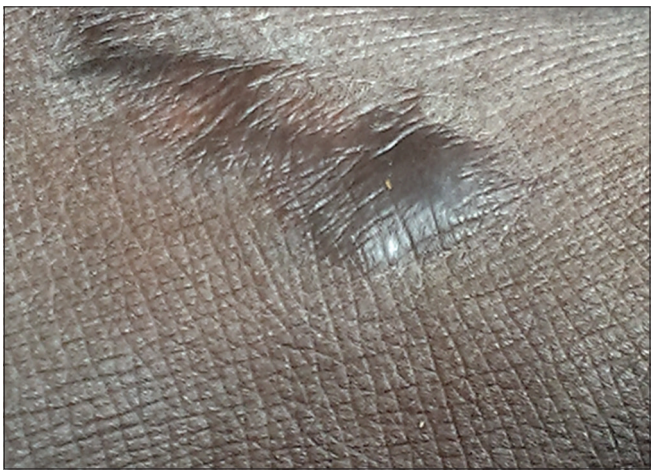

Figure 2: Hyperpigmented scar

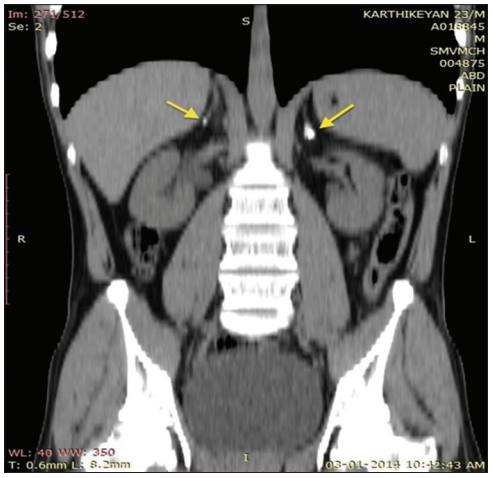

Figure 3: Bilateral adrenal calcification (left > right)

40 mg/day and reduced to 10 mg/day over 4 weeks with mineralocorticoid supplementation, fludrocortisone 0.05 mg/day. He was also educated about stress dose of steroids. He is doing well and is on regular follow-up.

DISCUSSION

Adrenal insufficiency (Addison's disease) is a rare disease with varied, non-specific manifestations.[2,3] The common manifestations are fatigue, anorexia, weight loss, vomiting, skin hyperpigmentation (80%), diarrhea (20%), craving for salt, personality changes, and fulminant shock. 90% of the adrenal gland must be destroyed before insufficiency develops.[6] If not recognized early and treated in time can lead to considerable mortality.[7]

Adrenal gland maybe involved in tuberculous, fungal, viral, or parasitic infections. Though autoimmune involvement of the adrenal gland is common in developed countries,[8] tuberculosis still remains an important cause of adrenal insufficiency in India and other developing countries.[9] The presence of vitiligo may point towards an autoimmune etiology of adrenal insufficiency.

The adrenal gland affected commonly by hematogenous spread of tuberculous infection may initially enlarge and finally get fibrosed and shrink in size and become calcified.[10-12]

The adrenal gland affected by tuberculosis may recover its function following treatment with anti tuberculous drugs, but adrenal insufficiency maybe evident even years after treatment.[13,14] Adrenal crisis is precipitated by infection, dehydration, withdrawal of steroids, surgery, or trauma. Adrenal crisis is the cause of death in 5% of the patients.

Our patient who was treated for pulmonary tuberculosis earlier presented with adrenalcrisis 1½ years following completion of treatment. Since the disease presents with various non-specific symptoms, the diagnosis was delayed until the patient presented with adrenal crisis.[15] Our patient had GI symptoms predominantly and was getting empirical treatment with no permanent relief. Studies have shown that patients with predominant GI symptoms presented most often with the crisis.[16] Diagnosis may be delayed for a variable period ranging from 6 months to more than 5 years when the patient may present with adrenal insufficiency or crisis.[17] Past history of tuberculosis, hyperpigmentation of the palms, palate and the resistant shock, and electrolyte abnormalities guided us toward the correct diagnosis. It is ideal to dorapid adrenocorticotropin stimulation test to confirm the diagnosis. A serum cortisol level of >20 μg in 60 min following administration of 250 μg of cosyntropin rules out primary adrenal insufficiency. In emergency situations, if random cortisol is <3 mcg/dl, hypo function of the adrenals is confirmed[17] as was the case in our patient.

CONCLUSION

Adrenal insufficiency should be suspected and investigated in any patient with unexplained weight loss and GI symptoms since failure to diagnose the disease in time may lead to considerable morbidity and mortality. Primary care physicians should be aware of this disorder, and a thorough general examination would help in early detection of the cases and institution of appropriate treatment.

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