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ADDISON'S DISEASE: A CASE REPORT

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Abstract
Addison's disease is a rare endocrine disease. This is a report of a case of Addison's disease as seen in a Nigerian female patient to highlight the occurrence in this environment as well as sensitize practitioners as to its possible aetiology, particularly in this era of tuberculosis and the pandemic of HIV/AIDS.

Key words: Addison's disease, tuberculosis

Introduction

Addison's disease is a rare endocrine disease[1] in which there is destruction of the adrenal cortex with resultant inadequate secretion of the adrenal cortical hormones-cortisol, aldosterone and androgens. Cortisol, the main hormone affected in the disorder is important in the body's ability to cope with stressful situation such as infection, hypotension, and surgical procedures. Also involved and with an overriding influence on the adrenals is the hypothalamic–pituitary–adrenal axis. Addison's disease is a term restricted to primary adrenocortical insufficiency. Other secondary or tertiary causes of adrenocortical insufficiency are not included in the term -'Addison's disease'. Primary adrenal insufficiency can be a life-threatening disorder particularly in stressful situation, since cortisol secretion cannot be increased on demand at all[1]. The prevalence of primary adrenal insufficiency (Addison's disease) has been reported to be 39 to 60 per million population. [2]

Case report

A 63-year-ol woman presented with increasing darkening of the skin, dizziness, and easy fatigability, nausea with occasional vomiting and progressive weight loss over eight months prior to presentation. There were no headaches, blurred vision, and neither loss of consciousness nor change in her bowel habit. The medical history and systemic review revealed no abnormality and were not significant as to the likely cause of her disease state.

Physical examination revealed an elderly lady, pale, asthenic with generalized hyperpigmentation especially on the face, oral mucosa, palmar creases and knuckles. No features of malnutrition or hypovitaminosis. There was no significant peripheral lymphadenopathy. Main findings in the systemic examination were a pulse of 106 bpm, regular and small; blood pressure 100/60 mmHg supine and 70/40mmHg sitting. She could not stand on account of severe postural dizziness. The apex beat was normal. Fundoscopy revealed a normal fundus. All other systems were essentially normal. A clinical assessment of Addison's disease to exclude paraneoplastic syndrome was made.

Laboratory investigations and results are shown in Table 1. Of note are the anaemia (haemoglobin-10gm/dl), with normal red cell morphology; ESR 58mm/hr (Westergreen method); fasting blood sugar was 76mg% and total serum protein of 7.8g/L (albumin-3.4g/L and globulin 4.4g/L). Plasma cortisol was undetectable at 0 and 30 minutes of cosyntropin administration (0.25 mg). Plasma rennin and aldosterone activity could not be estimated. HIV screening was negative (HIV 1 & II)

Radiological diagnostic tests included an abdominal ultrasound, which was reported as showing normal liver, spleen, pancreas and pelvic organs. However, the left kidney was not outlined. A computerised tomography scan (CT) of the abdomen showed a non-enhancing oval shaped left suprarenal mass with calcification and an ipsilateral hypoplastic but functional left kidney. There was neither ascites nor significant abdominal lymph nodes. The

conclusion was a suprarenal tumour-adenoma or adrenocortical carcinoma to exclude tuberculous adrenalitis. A CT brain scan was normal.

In view of the CT abdominal findings suggestive of adrenal tuberculosis, and ESR 58mm/hr, she was commenced on anti-tuberculous drugs.

Ten days after admission, she had nausea, vomiting, fever with chills and extreme lethargy. Cardiovascular examination showed a tachycardia, low volume pulse, BP 70/40 mmHg and blood film showed trophozoites of plasmodium falciparum. Blood cultures done thrice revealed no growth. She was managed with intravenous fluids (dextrose in saline), antimalarials, antibiotics and hydrocortisone. She made remarkable recovery and was maintained on oral prednisolone and fludrocortisone.

Table 1: Laboratory investigation results

Laboratory variable	Serum level
Na^+	136 mmo/L
Cl^-	100 mmo/L
K^+	3.4 mmo/L
HCO_3^-	22 mmo/L
Urea	2.8 mmo/L
Creatinine	17.7 Umol/L
Corticotrophin	550 pg per milliliter (upper limit of normal: 100 pg per milliliter)
Total bilirubin	8.7 Umol/L
Conjugated bilirubin	8.5 Umol/L
AST	7iu/L
ALT	8 iu/L
Total protein	7.8g/L
Albumin	3.4g/L
Fasting blood sugar	5.6 mmo/L
Packed cell volume	30%
White blood count	5,800/mm^3
Neutrophil	33
Lymphocyte	61
Eosinophil	6
Platelets	Adequate and Normal
Erythrocyte sedimentation rate	58mm/hr

She remained stable thereafter and made remarkable progress. After four weeks of admission, she was discharged to the medical outpatient clinic for further follow-up management. She had 3 episodes of Addisonian crisis after the initial hospital admission. These episodes were precipitated by acute malarial attacks, upper respiratory tract infection and gastroenteritis. Each episode, characterized by shock and altered consciousness, was managed with intravenous fluids (dextrose in saline), parenteral supplemental glucose, antibiotics, hydrocortisone and antimalarials where indicated. Each episode resulted in full recovery with no neurological deficit and was attended with rigorous counseling and health education of the patient and relatives as to the disease state and methods aimed at preventing this crisis. She has since remained in good health with lightening of the initial hyperpigmentation and also treatment of other clinical symptomatology. She is maintained on oral prednisolone, fludrocortisone and daily antituberculous medication.

Discussion

Thomas Addison (1855)[3] first described the clinical features of primary adrenal insufficiency, which may result from a variety of pathological processes. Hence the term Addison's disease. The characteristic form resulting from primary adreno-cortical insufficiency distinguishes Addison's disease from other forms of adrenal insufficiency which may result from pituitary or hypothalamic diseases, with decrease in adrenocorticotrophic hormone (ACTH) secretion and consequent adrenal cortex atrophy.

The commonest causes of Addison's disease are autoimmune and tuberculous adrenalitis. [1] Others are fungal infection, metastatic neoplasia, haemochromatosis and congenital adrenal hyperplasia-adrenoleukodystrophy. It is advised that tuberculosis should still be considered in the aetiology and diagnosis of adrenal insufficiency, particularly in areas where tuberculosis is endemic. [4] Between the mid-1980s and early 1990s, the synergistic combination of a deteriorating public health infrastructure worldwide, inadequate institutional control of infection, urban crowding and the epidemic of human immunodeficiency virus (HIV) infection resulted in a resurgence of tuberculosis, including infections with multidrug-resistant strains. [5] The development of active disease in persons with latent infection poses a continual threat of transmission, the control of which will be particularly difficult in regions where low case rates have resulted in decreased expertise in the identification and control of outbreaks of tuberculosis. [6] Currently, more than one third of the world's population is infected with *M. tuberculosis*; 8 million new cases and approximately 2 million deaths being reported each year. [7] Thus, it is expected that cases of extra-pulmonary manifestations of tuberculosis including the involvement of the endocrine system, should increase.

Addison's disease may manifest with diverse and

non-specific clinical and/or biochemical features. [1, 9] The most specific sign of primary adrenal insufficiency is hyperpigmentation of the skin and mucosal surfaces associated with fatigue and weight loss. [1] Soule reported that the presenting features among fifty patients seen over a 17-year period, as including hyperpigmentation (86%), weight loss (67%), abdominal pain (20%) and diarrhea (16%). [9] However, the disease may present atypically, requiring a high index of suspicion for diagnosis. [1,10]

In the South African experience, [9] thirty-nine patients (78%) were hyponatremic, 26(53%) were hyperkalaemic while only 9(18%) patients had hypoglycemia. Hyponatremia and hypokalemia are the usual findings in many cases of primary adrenal insufficiency. Although the serum levels of these electrolytes and plasma glucose were normal in our patient, it is still possible for patients to have normal electrolytes and plasma glucose level. [14]

Adrenal calcification and/or enlargement seen on abdominal CT are important signs of adrenal tuberculosis. [11] Calcification of the right adrenal gland was observed in our patient. It has been observed worldwide that CT signs of active tuberculous adrenalitis associated with Addison's disease were enlarged glands associated with necrotic areas, with or without calcification in many parts of the world. [14, 17, 18] A normal CT finding is also possible in a biochemically proven case of Addison's disease. [9] In the Soule study, adrenal CT scans were performed in 24 patients (48%) and were normal in 10[9]. Abnormalities were detected in 14 patients, reported as bilateral enlargement in 11, calcification in two and atrophic changes in one. Information obtained by CT scan is important in the etiological diagnosis of Addison's disease, hence it is advised to perform CT scan abdomen in all newly diagnosed cases. [14]

Although the destruction of the adrenal cortex by autoimmune adrenalitis has been documented as the commonest cause of primary adrenal insufficiency, [1, 9] this case is due to tuberculous adrenalitis. It is reported indicate that, Addison's disease, though rare, does occur our environment and to draw attention to the occurrence of tuberculous adrenalitis, especially with the pandemic of pulmonary tuberculosis and HIV/AIDS.

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