CASE REPORT

Infectious causes of Addison's disease: 1 organ—2 organisms!

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Accepted 16 March 2018

To cite: Muhammed H, Nampoothiri RV, Gaspar BL, *et al*. *BMJ Case Rep* Published Online First: [*please include* Day Month Year]. doi:10.1136/bcr-2017-223633

BACKGROUND

Infectious aetiologies are the most common causes of primary adrenal failure (Addison's disease) in low/middle-income countries while in the western world autoimmune causes predominate. The infections attributed to cause Addison's include disseminated gonococcal infection, tuberculosis, histoplasmosis, cryptococcosis and cytomegalovirus (CMV) infection. Here, we describe two classical cases of Addison's due to infections of the adrenal gland.

CASE PRESENTATION

Case 1

A 55-year-old woman from North India was admitted with history of multiple episodes of vomiting. She had history of severe loss of appetite and loss of weight. There was also history of increased body pigmentation for the last 6 months. At admission, she was drowsy with tachycardia of 116 beats per minute and her blood pressure was 80/50 mm Hg in supine position associated with a significant postural drop. There was hyperpigmentation of fingers, face, oral mucosa and flexures. The rest of the system examination did not reveal any significant clinical finding. Initial biochemical parameters showed serum sodium of 116 meq/L and serum potassium of 6.1meq/L. Arterial blood gas analysis showed pH 7.2 and bicarbonate 14.9 mEq/L. A diagnosis of acute adrenal insufficiency was made, and after drawing blood sample for cortisol and adrenocorticotrpic hormone (ACTH), she was given a 100 mg bolus dose of intravenous hydrocortisone and 2 L of 0.9% saline. Investigations showed serum cortisol of 39.02 nmol/L (171–536) and elevated ACTH of 818.8 pg/mL (5–60) confirming diagnosis of primary adrenal insufficiency. With initial management, her blood pressure improved and steroids were tapered to oral hydrocortisone (25 mg/day) with oral fludrocortisone 0.1 mg once a day. Contrast-enhanced CT (CECT) of the abdomen revealed bilateral symmetrical enlarged adrenals with preservation of contour without any calcifications (figure 1A). The rest of her hormone profiles were within normal limits ruling out a polyendocrinopathy syndrome. Antinuclear antibody immunofluorescence (ANA-IF) and retroviral serology were negative. CT-guided biopsy from enlarged adrenal glands was performed which revealed dense lymphoplasmacytic cell infiltrates with collection of epithelioid cells forming granuloma (figure 1B–E). There were no giant cells and stain for acid-fast bacilli was negative. Chest X-ray was normal and Mantoux test showed induration of 20 mm (strongly positive).

CASE 2

A 52-year-old woman from north India presented to the medicine outpatient department with history of fever, fatigue and occasional episodes of vomiting of 6 months' duration. On examination, she was pale with a blood pressure of 96/60 mm Hg associated with a postural drop to 72/50 mm in erect posture. There were no other significant clinical signs. Investigations revealed anaemia with haemoglobin of 8.6 g/dL, elevated erythrocyte sedimentation rate (ESR (68 mm/hour), hyperkalaemia (serum potassium of 5.9 meq/L) and hyponatraemia (serum sodium of 122 meq/L). Abdominal ultrasound performed showed bilateral bulky adrenals. CECT abdomen confirmed bilateral symmetrical enlargement of adrenals with preserved adrenal contours (figure 2A). Her hormone profile showed decreased serum cortisol levels of 61.2 nmol/L (171–536) with elevated serum ACTH of 688 pg/mL (5–60). The rest of the hormones evaluated, that is, thyroid, oestrogen, progesterone, luteinising hormone, follicle-stimulating hormone and prolactin, were in normal range. ANA-IF and HIV serology were negative. She was started on oral hydrocortisone in divided doses (10 mg-5mg-5mg) for adrenal insufficiency. She was taken up for ultrasound-guided adrenal biopsy for aetiology of her adrenal failure. Biopsy showed tissues with predominant necrosis and inflammatory cells. There were scattered histiocytes along with intracellular with prominent extracellular fungal spores, suggestive of *Histoplasma* species (figure 2B–E). CECT of the head was normal.

OUTCOME AND FOLLOW-UP

Case 1

With a biopsy picture consistent with adrenal tuberculosis, the patient was started on antituberculous therapy of daily weight-based 4-drug regimen. The patient improved symptomatically and was discharged on oral hydrocortisone and fludrocortisone. Two months into the treatment, she is currently asymptomatic and is well compliant to antituberculous therapy with steroid supplementation and was started on continuation phase of antituberculous therapy with isoniazid and rifampin with normalisation of electrolytes and hyperpigmentation.