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(Accepted 2 December 1988)

Mineralocorticoid deficiency in HIV infection

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Br Med J 1989;**298**:496-7

Infection with the human immunodeficiency virus (HIV) may result in adrenal necrosis and hence a deficiency of cortisol.[1-3] We report on a patient who developed severe sodium and water depletion because of a deficiency in production predominantly of mineralocorticoids. This important, easily treatable complication has probably been overlooked in patients infected with HIV.

Case report

A homosexual man aged 41 was admitted in April 1988 with a diagnosis of pneumocystis pneumonia. He was positive for HIV antibody. On admission his serum sodium concentration was 129 mmol/l, potassium 4·7 mmol/l, and urea 5·1 mmol/l. Treatment with high doses of co-trimoxazole was started. His symptoms improved, and he was discharged 11 days later. His serum sodium concentration, however, had fallen during his admission and at discharge was 114 mmol/l; serum potassium and urea concentrations were 5·8 mmol/l and 9·7 mmol/l, respectively. His serum cortisol concentration was measured, but the result was not available at discharge.

He was readmitted the next day complaining of feeling faint on walking, leg weakness, and breathlessness. Examination showed profound postural hypotension (blood pressure lying 120/80 mm Hg, unrecordable on standing); hyponatraemia (sodium concentration 110 mmol/l) and hyperkalaemia (potassium concentration 6·7 mmol/l) were more pronounced. Treatment with intravenous physiological saline relieved the symptoms, and Addison's disease was diagnosed provisionally. At this stage, however, the serum cortisol concentration in the sample obtained during the first admission was found to be 992 nmol/l (normal 250-550 nmol/l). He was given oral dexamethasone (0·5 mg twice daily) and sustained release sodium 600 mg three times daily, and an intravenous tetracosactrin test was performed (table). Plasma renin activity (after 10 minutes' sitting) was appreciably raised at 55 nmol/l/h (normal 0·4-1·9 nmol/l/h), and plasma aldosterone concentration was 149 pmol/l, which was low considering his high plasma renin activity. His 24 hour urinary excretion of sodium was 240 mmol. Treatment was changed to hydrocortisone 20 mg in the morning and 10 mg at night with fludrocortisone 0·1 mg daily, which was increased to 0·2 mg daily. Within 10 days this had corrected his postural hypotension (blood pressure lying 110/70 mm Hg, standing 130/80 mm Hg) and restored his serum electrolyte concentrations to normal (sodium 136 mmol/l, potassium 3·6 mmol/l, and urea 3·3 mmol/l). Plasma renin activity fell to 5·8 nmol/l/h and plasma aldosterone concentration remained low at 59 pmol/l. He was negative for autoantibodies, and computed tomography of his adrenal glands showed a normal size and structure.

Comment

The patient's high plasma renin activity and low plasma aldosterone concentration with profound hyponatraemia indicated a severe mineralocorticoid

Results of intravenous tetracosactrin test (500 mg in 500% 5% dextrose) performed when patient was taking dexamethasone 0·5 mg twice daily

Time (hours)	Plasma cortisol concentration (nmol/l)*
0	75
1	346
2	412
3	421
4	470
5	479

*Expected maximum >600 nmol/l.

deficiency. The high cortisol concentration during his first admission suggested that there was no deficiency in production of glucocorticoids, only in mineralocorticoids, although the suboptimal rise during infusion of tetracosactrin indicated some loss of reserve cortisol.

Postmortem studies have shown a high prevalence of adrenal disease in patients with AIDS. In one study of 41 subjects at necropsy 51% had cytomegalovirus adrenalitis. Hyponatraemia was the most common finding (in 75%), although only two subjects had been suspected during life of having adrenal insufficiency.[1] Stimulation with tetracosactrin may indicate impairment of adrenal function despite normal or high cortisol concentrations measured at random.[1-3]

The cause of the adrenal dysfunction in our case is unknown; the dysfunction would probably have been overlooked if reliance had been placed on the raised cortisol concentration. We suggest that in patients with AIDS with lethargy, hypotension, hyponatraemia, or hyperkalaemia, deficiencies of mineralocorticoids and glucocorticoids should be investigated by measurement of aldosterone concentration and plasma renin activity; prolonged stimulation with tetracosactrin (intravenously or intramuscularly); and, perhaps, assay of serum adrenocorticotrophic hormone concentration when the serum cortisol concentration is normal or low.

Our findings suggest that some of the morbidity associated with AIDS is a result of mineralocortical deficiency. Adrenal complications caused by HIV are easily treated and should be considered in all patients with infections related to AIDS.

We thank Dr Louise Sugden for measuring plasma renin activity; Mrs Michelle Miller for measuring plasma aldosterone concentration; and Professor A Guz for permission to report this case.

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(Accepted 14 November 1988)

Assessing glycaemic control in non-insulin dependent diabetes: acceptability of blood sampling at home

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Br Med J 1989;**298**:497-8

Measurement of glycosylated haemoglobin concentration is an accurate and convenient method of assessing overall glycaemic control in diabetes but is expensive and labour intensive.[1] In non-insulin dependent diabetes fasting blood glucose concentration correlates well with glycosylated haemoglobin concentration and is much cheaper to measure.[2] In our hospital patients are therefore asked to have their fasting blood glucose concentration estimated before their visit to the clinic. This requires patients to make a visit to the laboratory while fasting.

We studied the acceptability and relative costs of three methods of obtaining samples for measuring fasting glucose concentration: by venepuncture in the laboratory and by finger pricking at home to produce blood spots on filter paper and reagent strips (BM 1-44; Boehringer Mannheim). Blood spots on filter paper have been used in previous studies,[3,4] but their acceptability to patients is not known.

Patients, methods, and results

Sixty eight non-insulin dependent diabetics who did not regularly monitor their blood glucose concentrations at home agreed to produce blood spots and use reagent strips at home after fasting. All received oral and written instructions on performing the tests from one investigator. Samples were enclosed in plastic bags and returned in envelopes to the laboratory.

We analysed blood spots, which are stable at room temperature for eight days, by the method described by Gamlen *et al.*[5] The results correlated well with venous blood glucose concentrations measured with a Yellow Springs analyser ($r=0·977$; $p<0·001$; $n=56$). Values obtained with reagent strips read with a reflectance meter at the time of sampling ($r=0·909$; $p<0·001$; $n=45$) and after postal delivery to the clinic ($r=0·900$; $p<0·001$; $n=45$) also correlated well with venous concentrations.

Fifty patients returned both blood tests, and one patient returned only the blood spot. There were no significant differences in age, sex, or duration of diabetes between respondents and non-respondents. Four of the 10 patients who had used reagent strips before produced inadequate strips (Fisher's exact test $p=0·043$), although all produced adequate blood spots. Patients with no experience of finger pricking produced significantly better spots than strips (table).

*Adequacy of reagent strips and blood spots on filter paper produced by 40 patients with no experience of finger pricking**

Reagent strips	Spots		
	Adequate	Inadequate	Total
Adequate	14		14
Inadequate	18	8	26
Total	32	8	40

McNemar's test statistic (with Yates's correction) = 16·05, $p<0·0005$.
*Reagent strips were considered inadequate if blood was smeared or did not cover the whole colour block, or if they could not be read by a reflectance meter. Filter paper spots were considered inadequate if blood had not soaked through the filter paper to a diameter of 6 mm.

Patients who produced adequate strips were on average younger (mean (SD) age 57·4 (14·1) years) than those who produced inadequate strips (65·6 (9·2)) ($p=0·033$). This was also true for spots (61·3 (12·4) *v* 67·3 (7·2) years, $p=0·033$).

Fifty one patients, 47 of whom had returned samples, later returned a questionnaire. Eighteen found visits for laboratory sampling inconvenient. Of the 45 who stated preferences, 31 preferred home sampling to venepuncture at the laboratory or the general practitioner's surgery. Preference was not significantly related to age, sex, or duration of diabetes.

With information from the questionnaire and the laboratory we estimated costs of materials and technicians' time and costs to patients of the three methods of sampling; blood spots had to be posted to the laboratory, whereas patients took reagent strips to the clinic to be read by a reflectance meter. Sampling at the hospital was most expensive for both the NHS