Acute adrenocortical crisis and an abnormal electrocardiogram

Arpandev Bhattacharyya, Subramaniam Jagadeesan, Roger J Wolstenholme, D James Tymms

Acute adrenocortical crisis is often the first presentation of Addison's disease. The combination of pigmentation, peripheral circulatory failure and the electrolyte abnormality usually lead to suspicion and early treatment with corticosteroids is the key to the success in such cases. Failure of response to the standard treatment should highlight the possibility of a coexisting condition. We report a case where the patient did not respond to the treatment until such a condition was recognized.

Dr Arpandev Bhattacharyya and **Dr Subramaniam Jagadeesan** are Specialist Registrars, and **Dr Roger J Wolstenholme** and **Dr D James Tymms** are Consultant Physicians in the Department of Medicine, Royal Albert Edward Infirmary, Wigan WN1 2NN

Correspondence to: Dr DJ Tymms

DISCUSSION

Autoimmune destruction of the adrenal cortex is the commonest cause of Addison's disease in developed countries (Orth et al, 1992). Infection is the commonest precipitating factor for acute crisis (Burke, 1985). Initial clinical features and investigations were characteristic of adrenocortical crisis in our patient. The main difficulty in the above patient was failure to improve with adequate therapy. The abnormal electrocardiogram helped in the management pointing towards pericardial pathology. The electrocardiogram can be abnormal in several ways in an acute adrenal crisis, i.e. hyperkalaemia (Nerup, 1974), and hypercalcaemia (Camblor et al, 1997). The association of cardiac tamponade and adrenocortical crisis has not been reported in the literature.

After detailed investigation, the aetiology of the exudative pericardial effusion remained obscure. It is possible that the patient had some sort of viral infection which caused the crisis. The patient had no risk factors for human immunodeficiency virus (HIV) infection, although a formal test was not performed. The patient stayed well on replacement therapy for the next 18 months, making HIV an unlikely cause.

In primary adrenal insufficiency, the haemodynamic consequences are primarily those of volume contraction, which would necessarily exacerbate the ill-effects of pericardial effusions. One would therefore expect the haemodynamic consequences of even small pericardial effusions to be much more severe in this context than in individuals with a normal circulatory volume.

CASE REPORT

An 18-year-old boy was admitted via the accident and emergency department with peripheral circulatory collapse. He complained of being generally unwell with intermittent vomiting for 1 year. On examination his blood pressure was 80/60 mmHg with a heart rate of 110/min. He was hyperpigmented and confused. He was apyrexial and the rest of the examination was unremarkable. An initial blood test revealed normal full blood count, hyponatraemia (123 mmol/litre, normal 135–145 mmol/litre), hyperkalaemia (5.9 mmol/litre, normal 3.5–5.3 mmol/litre), prerenal azotaemia (urea 19.7 mmol/litre, normal 2.7–8.1 mmol/litre), creatinine 184 μmol/litre (normal 78–120 μmol/litre), deranged coagulogram (prothrombin time 24 sec with a control of 13 sec and partial thromboplastin time 71 sec with a control of 35 sec), abnormal liver function tests (raised alanine aminotranferase 181 u/litre, normal 5–40 u/litre, gammaglutamyl transferase 49 u/litre, normal 7–45 u/litre with normal serum protein and bilirubin levels). Random plasma glucose was 3.6 mmol/litre. A blood sample was collected for serum cortisol measurement and he was treated with isotonic saline, intravenous hydrocortisone and antibiotics, with a presumptive diagnosis of acute adrenocortical crisis.

Over the next 4 hours his blood pressure failed to improve, in spite of a 3 litre saline infusion, his oxygen saturation dropped, confusion worsened and he was transferred to the intensive care unit in a critical condition. An electrocardiogram (ECG) pointed towards pericardial injury (*Figure 1*). Pulsus paradoxas was normal at 6 mmHg. Urgent echocardiogram showed pericardial effusion with early cardiac tamponade. Pericardiocentesis revealed 250 ml of straw-coloured fluid (protein 51 g/litre, glucose 7.0 mmol/litre with presence of inflammatory cells only). Pericardial fluid was negative for gram-, acid-fast and fungal staining and was subsequently sterile on bacteriological, viral and tubercular culture. Blood pressure, oxygen saturation and confusion improved following pericardiocentesis. His ECG showed remarkable improvement when taken 6 hours later (*Figure 2*).

Over the next few days he improved slowly but steadily. Subsequently, the serum cortisol was reported to be 45 nmol/litre with ACTH 112 ng/litre (normal 5–50 ng/litre), from a sample taken at admission before giving hydrocortisone. The hydrocortisone dose was reduced gradually to 30 mg daily and fludrocortisone was started at a dose of 100 mg daily. His liver function, coagulogram and renal function returned to normal. Antiadrenal antibody was positive, blood for autoantibody screening was negative and thyroid function was normal. Infective screening failed to isolate any organism (blood, urine, throat and nasal swab). Paired sera (acute and convalescent) were negative for influenza A and B, adenovirus, respiratory syncitial virus, *Mycoplasma pneumoniae*, Q2 fever, *Chlamydia* species and Coxsackie virus. Computed tomography of the abdomen showed bilateral adrenal atrophy without calcification, the rest of the examination, including the liver, was normal. He went home 12 days after admission on daily 30 mg hydrocortisone and 100 mg fludrocortisone. Repeat echocardiogram 4 weeks after discharge showed resolution of the pericardial effusion and the ECG was completely normal (*Figure 3*).

CONCLUSIONS

In cases of acute adrenocortical crisis (possibly in all cases of peripheral circulatory failure), failure of improvement with initial management should highlight the possibility of coexistent cardiac pathology, irrespective of the clinical examination. This case also illustrates that in acute circulatory problems, echocardiography has a place in the assessment of the patient. HM

Burke CW (1985) Adrenocortical insuffiency. *Clin Endocrinol Metab* **14:** 947–76

Camblor AM, Compes DLC, Breton LI, Pinilla LB (1997) Severe hypercalcaemia secondary to an Addisonian crisis and hyperthyroidism. *Rev Clin Esp* **197(6):** 465–6

Nerup J (1974) Addison's disease. Clinical studies. A report of 108 cases. *Acta Endocrinol* **76:** 127–41

Orth DN, Kovacs WJ, DeBold CR (1992) The adrenal cortex. In: Wilson JD, Foster DW, eds. *Williams Textbook of Endocrinology.* WB Saunders, Philadelphia: 489–619

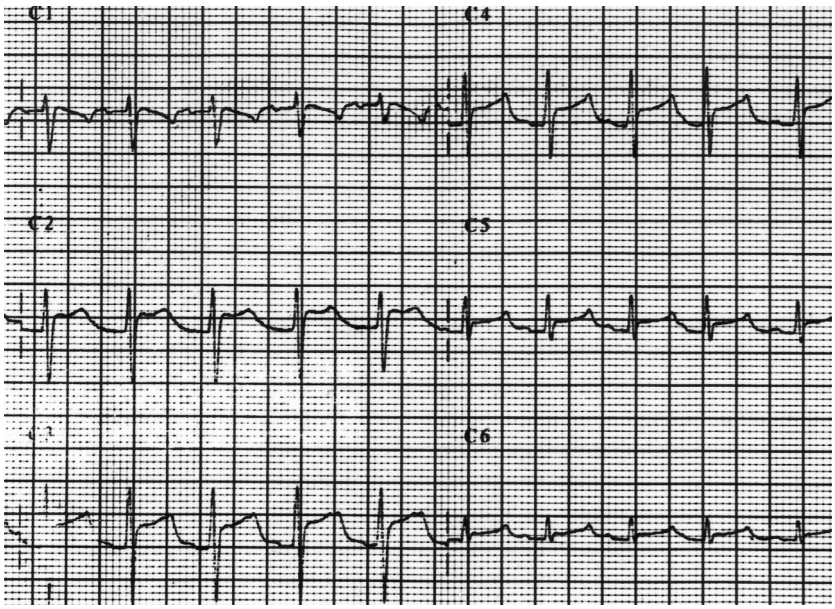

Figure 1. Electrocardiogram at diagnosis.

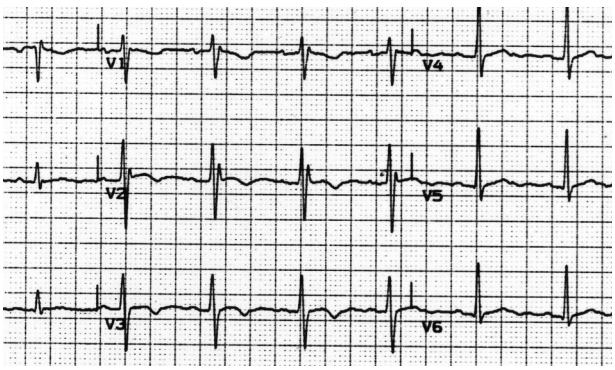

Figure 2. Electrocardiogram 6 hours after pericardiocentasis.

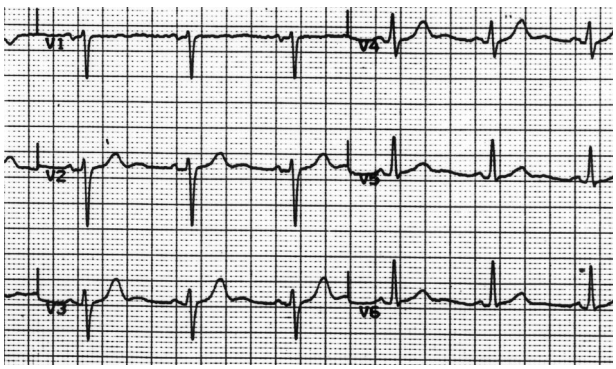

Figure 3. Electrocardiogram 4 weeks after discharge.