Psychosis, central hyperventilation and inappropriate secretion of antidiuretic hormone in systemic lupus erythematosus

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Summary

A case of systemic lupus erythematosus with reversible psychosis and hyperventilation related to hyponatraemia secondary to a syndrome of inappropriate secretion of antidiuretic hormone (SIADH) is presented. The SIADH was treated successfully by oral urea for more than 8 months.

Introduction

Psychiatric manifestations are frequent in systemic lupus erythematosus (SLE) (Dubois, 1974). A case is reported in which acute psychosis and central hyperventilation were related to low serum sodium secondary to a syndrome of inappropriate secretion of antidiuretic hormone (SIADH). This is believed to be the first report of SIADH as the only clinical manifestation of brain lupus.

Case report

A 16-year-old boy was admitted with psychosis. When he was 12 years old, a nephrotic syndrome secondary to diffuse membranoproliferative glomerulonephritis associated with SLE had been diagnosed. For 4 years the disease was well controlled by variable doses of steroids and azathioprine. Before admission, he was on 20 mg prednisone every other day, a salt-free diet and 100 mg azathioprine daily. One month before admission, he became logorrheic, hyperactive, and he suffered from insomnia and megalomania. Physical examination was normal except for mania and high blood pressure (160/100 mmHg). There was no oedema. Blood urea was 5.5 mmol/l, creatinine 61 µmol/l, creatinine clearance 1.07 ml/s, uric acid 0.12 mmol/l, blood glucose 5.3 mmol/l, cholesterol 6.97 mmol/l, serum sodium 113 mmol/l, potassium 3.5 mmol/l, chloride 86 mmol/l, carbon dioxide 18 mmol/l. The

arterial pH was 7.51. Arterial blood gases were P_{CO_2} 3.33 kPa and P_{O_2} 17.02 kPa when breathing room air. Urine pH was 7. Total protein was 47 g/l with 63% albumin. Blood osmolality was 240 mmol/kg H_2O with a concomitant urine osmolality of 460 mmol/kg H_2O and a urine sodium of 105 mmol/l. A 24-hr urine collection contained 2.5 g protein. Urinalysis was normal. Thyroid function was normal. Plasma renin activity and serum/urinary aldosterone while the patient was on a normal salt diet were normal. Electroencephalogram showed diffuse slow theta waves. Chest X-ray, CT scans of the head and lumbar puncture were normal. Anti-nuclear factor titre was 1/128; total haemolytic complement, C_3 and C_4 were normal.

The patient received atenolol 100 mg and urea 30 g daily (Fig. 1). Urea therapy (Decaux *et al.*, 1980) induced a normalization of serum sodium and simultaneously his behaviour improved. The hyperventilation disappeared and after 12 days' treatment arterial blood gases were P_{O_2} 13.03 kPa, P_{CO_2} 4.66 kPa, pH 7.45, and carbon dioxide 24 mmol/l. Electroencephalogram was normalized. The next day urea was discontinued and severe water restriction was ordered but not applied by the patient. Two days later, he was acutely psychotic and responded poorly to neuroleptics. Serum sodium was then 118 mmol/l, and respiratory alkalosis was present again. Urea was reinstituted and the mental state rapidly improved, together with normalization of serum sodium and carbon dioxide. A standard urine acidification test (oral ammonium chloride 0.1 g/kg body weight) was then performed and was normal. The patient was discharged with 30 g urea daily and he was allowed to drink 1.5 litre water/day. After 8 months of this regimen, the patient is doing well and the electrolytes remain normal despite the persistence of SIADH.

FIG. 1. Evolution of blood and weight parameters during urea therapy.

Discussion

The main features of this case were that the SIADH was the only evidence for brain lupus, and that psychosis and central hyperventilation were related to hyponatraemia. The hyponatraemia was due to SIADH since urine sodium was low (10 mmol/l) when the patient was on a salt-free diet and high during normal salt intake (105 mmol/l), while serum sodium was low. These data excluded a salt-losing nephritis. Drugs, tumour and endocrine failure were not present as causes of SIADH, which was therefore attributed to brain lupus. Such an association has been described in one previous case (Kaplan, Curl and Decker, 1970) but it had more extensive brain lesions. Psychosis in the present patient was clearly related to low serum sodium concentrations. Psychiatric symptoms in SLE are frequent but the possible role of the serum sodium concentration has not yet been mentioned. Central hyperventilation in SLE has been reported (Kaplan *et al.*, 1970; Elkington, 1966) and was related to lesions of the hypothalamus and in the

pons (Kaplan *et al.*, 1970); in the present case however, hyperventilation disappeared when hyponatraemia and psychosis were cured.

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References

- BARTTER, F.C. & SCHWARTZ, W.B. (1967) The syndrome of inappropriate secretion of antidiuretic hormone. *American Journal of Medicine*, **42**, 790.
- DECAUX, G., BRIMIOULLE, S., GENETTE, F. & MOCKEL, J. (1980) Treatment of the syndrome of inappropriate secretion of antidiuretic hormone by urea. *American Journal of Medicine*, **69**, 99.
- DUBOIS, EL. (Ed) (1974) *Lupus Erythematosus*, 2nd edn. University of Southern California Press, Los Angeles.
- ELKINGTON, J.R. (1966) Clinical disorders of acid-base regulation. *Medical Clinics of North America*, **50**, 1325.
- KAPLAN, A.P., CURL, F.D. & DECKER, J.L. (1970) Central hyperventilation and inappropriate antidiuretic hormone secretion in systemic lupus erythematosus. *American Journal of Medicine*, **48**, 661.