plasma ACTH together with radiological visualization of the adrenal glands will have to be relied upon to place the lesion primarily in the hypothalamic-pituitary axis or in the adrenal.

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Acute renal failure complicating McArdle's syndrome

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Summary

A case of McArdle's syndrome is described in which an epileptiform seizure was followed by acute reversible renal failure with hypercalcaemia in the diuretic phase.

Introduction

McArdle's syndrome is a rare disorder of muscle metabolism which presents in early life with pains and stiffness in the muscles during exercise. The disorder is known to be due to muscle phosphorylase deficiency (Schmid & Mahler, 1959) and behaves as

an autosomal recessive. Myoglobinuria is known to occur during attacks, but acute oliguric renal failure has been reported in only two patients (Grünfeld *et al.*, 1972). We report a further case of renal failure associated with McArdle's syndrome with the added complication of hypercalcaemia occurring during the diuretic phase.

Case report

The patient, R.G., previously reported in a family study of McArdle's syndrome (Salter,

Adamson & Pearce, 1967) first presented in 1961 at the age of 16 years with stiffness and weakness of the legs on exertion. These symptoms continued and during the next 3 years the patient had three episodes of loss of consciousness associated with the passage of dark urine. In 1964 the urinary pigment was identified as myoglobin and subsequent muscle biopsy showed the changes characteristic of McArdle's syndrome (Pearce, 1965). The attacks of unconsciousness were considered to be epileptiform seizures, and phenobarbitone was prescribed. Several further fits again associated with pigmenturia were noted over the next few years.

In August 1972, R.G. was admitted for observation after losing consciousness and falling 10 feet from a ladder. He was noted at the time to be passing dark urine and spectroscopic examination suggested that the pigment was methaemoglobin. He was discharged apparently well after 36 hr. One week later he was re-admitted complaining of nausea, malaise, oliguria and swelling of the ankles and face. Physical examination revealed mild facial swelling, slight sacral oedema, but no ankle oedema. Pulse rate was 80/min, and normal in character, blood pressure 130/90 mmHg, there was no jugular venous congestion and heart sounds were normal. Crepitations were heard in the lower lobes of both lungs. Other systems appeared normal.

Investigations

Plasma sodium 135 mEq/l, potassium 5.8 mEq/l, chloride 86 mEq/l, bicarbonate 19 mEq/l. Blood urea 510 mg/100 ml, blood sugar 208 mg/100 ml, serum calcium 8.4 mg/100 ml, serum phosphate 11.1 mg/100 ml, alkaline phosphatase 6 King-Armstrong units, serum urate 17.7 mg/100 ml and serum creatinine 16.8 mg/100 ml. Haemoglobin was 11.1 g/100 ml, white cell count 9800/mm³, with normal film.

Initially the patient was completely anuric and no urine was therefore available for examination.

Progress

Peritoneal dialysis was required for 5 days after which diuresis ensued and the blood urea slowly fell. The only medication at the time was phenytoin, and the patient received a diet containing 20 g protein/day. On the tenth day following admission bilateral episcleritis was noted and the serum calcium was found to be 12.3 mg/100 ml and phosphate 7.7 mg/100 ml. Slit lamp examination of the eyes was, however, normal. A week later the calcium had fallen to 9.7 mg/100 ml and the eye signs had resolved. Three weeks after admission the patient was discharged with a blood urea of 41 mg/100 ml and a creatinine clearance of 40 ml/min. Blood urate, calcium, phosphate and alkaline phosphatase were within normal limits.

Discussion

Acute renal failure complicating McArdle's syndrome would appear to be rare, only two previous cases having been reported (Grünfeld *et al.*, 1972). Renal failure associated with myoglobinuria, however, is a well recognized entity and has been described in association with crush syndrome, exercise induced myoglobinuria and primary paroxysmal myoglobinuria. Presumably the severe muscle exertion of a grand mal seizure which is a recognized association of McArdle's syndrome (Salmon & Turner, 1965) in our patient caused a degree of muscle breakdown sufficient to release a toxic quantity of myoglobin. The reason why the urinary pigment was not identified as myoglobin may be due to the fact that myoglobin readily converts to the met form which is difficult to distinguish from methaemoglobin spectroscopically (Muehrcke, 1969). The occurrence of hypercalcaemia during the diuretic phase was a feature of one of the cases of Grünfeld *et al.*, and has in addition been reported in seven cases of acute renal failure associated with severe muscle or soft tissue injury, where it also occurred in the diuretic phase (Leonard & Nelms, 1970). Although the authors were unable to explain this phenomenon they did note an elevation of parathormone in one of the two patients in whom it was assayed. It is, however, known that in the experimental animal acute renal failure may cause the deposition of large amounts of calcium in areas of necrotic muscle (Meroney *et al.*, 1957). Bütikofer & Molleyres (1968) suggest that the hypercalcaemia may be due to subsequent reabsorption of calcium from such areas. No specific treatment would appear to be indicated for the hypercalcaemia as it appears to resolve spontaneously.

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Acute onset diabetes due to an ACTH secreting oat cell carcinoma of the bronchus

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Summary

Diabetes of acute onset led to this patient's referral to hospital. Following investigation, it became apparent that the diabetes was only part of the wider clinical picture produced by an ACTH secreting oat cell carcinoma of the bronchus.

DIABETES, which is secondary to other diseases, is quite commonly seen. However, diabetes of acute onset due to ACTH production by an oat cell carcinoma of the bronchus is a distinctly uncommon presentation. Cushing's disease occurs in between 0.5% and 2% of bronchial carcinomas (Azzopardi, Freeman & Poole, 1970; Ross, 1966) and diabetes may be one of the features of its variable clinical effects (Bayliss, 1971). In the present case, clinical evidence of adrenal cortical over-activity appeared before the underlying malignant tumour had been diagnosed and the patient was referred to hospital with a diagnosis of diabetes.

Case report

A 66-year-old man had an appointment at the diabetic clinic, but due to the urgency of his symptoms, the general practitioner decided to ask for an emergency admission to hospital. His main complaints were thirst and polyuria for the last 7 days and he had heavy glycosuria. There was also a history of increasing shortness of breath for the past 2 years. Although this symptom had been investigated 18 months previously with an X-ray chest and an ECG, a diagnosis had not been reached. He had smoked 20 cigarettes a day all his life and shortness of breath did not alter his habits.

Clinical examination showed an ill, drowsy,

dehydrated man with a pigmented facial appearance. He had bilateral ankle oedema, but no finger clubbing or enlarged lymph glands. His pulse was feeble and irregular and his blood pressure was 120/60 mmHg. There were no murmurs over the praecordium and the breath sounds were normal with no accompaniments. In the abdomen a firm liver was palpable two fingers below the costal margin.

The initial investigations confirmed a diagnosis of diabetes mellitus as his random blood sugar result was 250 mg/100 ml. Other investigations on admission to hospital were serum Na⁺ 144 mEq/l, serum K⁺ 2.5 mEq/l, blood urea 40 mg/100 ml, pH 7.53, pCO₂ 44, and standard bicarbonate 35 mEq/l. Total proteins 5.3 g, albumin 3.1 g, globulin 2.2 g, serum Ca 8.4 mg/100 ml, alkaline phosphatase 19 units, bilirubin 1.7 mg/100 ml and aspartate aminotransferase (SGOT) 160. Repeated electrocardiograms showed supraventricular arrhythmias with varying atrioventricular conduction. A portable X-ray of his chest indicated bilateral hilar gland enlargement.

Within a couple of days, despite a restricted carbohydrate intake and oral hypoglycaemic agents, his blood sugars had risen to between 300 and 400 mg/100 ml and treatment with insulin became necessary. Although potassium supplements were being given, the serum potassium remained below 2.6 mEq/l and 24 hr urine collections showed that he was still losing between 90 and 96 mEq of potassium per day. He was, therefore, given an infusion of 270 mEq of potassium in 0.45% N saline over 36 hr and in this way the serum potassium rose to 4.2 mEq/l. At this stage the ECG reverted to sinus rhythm. Serum cortisol levels were determined on venous bloods