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Reversible impairment of myocardial function in hypoparathyroidism causing hypocalcaemia

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Abstract

A 25 year old woman presented with severe myocardial dysfunction. Laboratory tests showed hypocalcaemia caused by hypoparathyroidism. After the restoration of normal concentrations of serum total and free ionic calcium indices of left ventricular function returned to normal and her symptoms disappeared.

The important role of calcium ions in the contraction of cardiac muscle fibres is well known.[1] None the less, there are few reports of well documented cases in which hypocalcaemia caused reversible myocardial dysfunction.[2-4] We describe a patient in whom cardiac failure with a prolongation of the QT interval, cramps,[5] and convulsions was caused by hypocalcaemia. Hormone assay showed hypoparathyroidism.

Case report

A 25 year old woman was first admitted to our department with a history of pulmonary oedema and congestive heart failure, which had been treated successfully in a district hospital. When we first examined her in 1983 there was no sign of heart failure. The heart sounds were normal. Acquired and congenital heart diseases were excluded. Haemodynamic examination showed that the left ventricular end diastolic pressure was 10 mm Hg and the left ventricular end diastolic volume was 170 ml. The cardiac index was 4·1 l/m^2. The QT interval was within normal limits. The patient said that she drank about half a litre of spirits every day.

We diagnosed an alcoholic myocardial lesion; the patient's condition improved when she stopped drinking. The patient was monitored by echocardiography in the subsequent months and it seemed that she had been cured. She did not need any medical treatment.

In April 1985 she had generalised convulsions and was treated with antiepileptic drugs. But this did not stop the convulsions. Subsequently alopecia developed and her finger nails became ridged. She was admitted for a second time in November 1985. She was grey and pale. Both Chvostek's and Trousseau's signs were present and she had temporary episodes of unconsciousness with convulsions.

Her history did not include operations on the thyroid gland. The heart rate was 80 beats/minute and the systemic arterial blood pressure was 110/70 mm Hg. The liver was enlarged by 6 cm and there was mild oedema in the feet. The chest x ray showed a cardiac enlargement and evidence of severe pulmonary congestion. The QT interval was 0·64 s with negative T waves in leads I and V5–6 (fig 1). The echocardiogram showed left ventricular dilatation (end diastolic diameter 67 mm, end systolic diameter 58 mm). The ejection fraction was 17% by radionuclide ventriculography with technetium-99m. The carotid angiogram, isotopic brain scintigram, electroencephalogram, and skull and bone radiology were normal. Ophthalmological examination showed peripheral (mainly subcapsular) cataracts in the lenses which accorded with hypoparathyroidism. The erythrocyte sedimentation rate was 45 mm/hour and a complete blood count and urine analysis were normal. Serum concentrations of glucose, sodium, potassium, urea nitrogen, creatinine, haptoglobin, bilirubin, aspartate aminotransferase, alanine aminotransferase, glutamyltransferase, alkaline phosphatase, acid phosphatase, iron, total iron binding capacity, total protein, and albumin were normal. The Coombs, Kürten, latex, and lupus erythematosus tests were negative. The table shows the concentrations of serum total and free ionic calcium, magnesium, and inorganic phosphate on admission and before discharge.

Concentrations of hormone (with methods of measurements and normal ranges in brackets) were as follows: urine 17-hydroxycorticosteroids 3·9 mg/24 hour (Porter-Silber colorimetry, 1·8–6·0 mg/24 h); 17-kerosteroids 7·3 mg/24 h (Zimmermann colorimetry, 5·0–15·0 mg/24 h); serum cortisol 431 nmol/l (fluorimetry, 221–550 nmol/l); testosterone 1·88 nmol/l (radioimmunoassay (RIA), 0·86–

Data on admission and before discharge

Variable	*On admission*	*Before discharge*	*Range*
Total serum calcium (mmol/l)	0·53	1·98	1·90–2·50
Free ionic calcium (mmol/l)	0·32	1·24	0·95–1·25
Inorganic phosphate (mmol/l)	3·25	1·67	1·00–1·70
Tubular phosphate resorption (%)	95–99	71·8	65–90
Magnesium (mmol/l)	0·63	0·98	0·50–1·00

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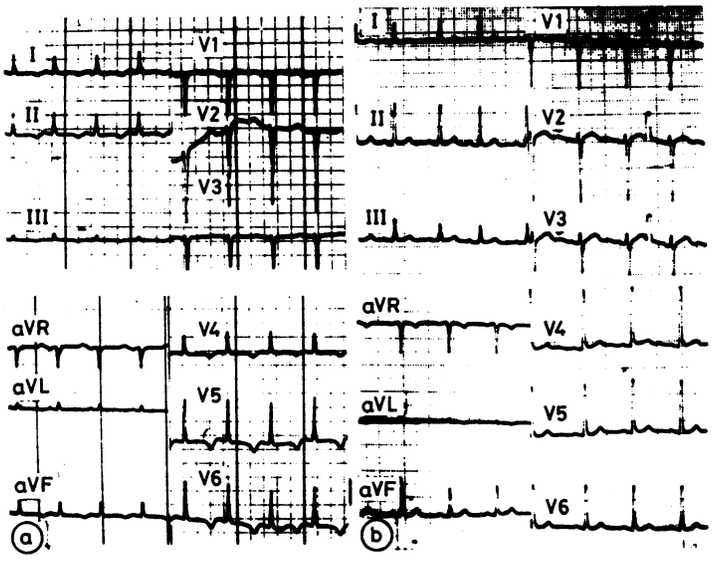

Figure 1 12 lead electrocardiogram on admission (a) and before discharge (b). The QT interval was initially 0·64 s. After restoration of the serum calcium it was 0·38 s. The heart rate was essentially unchanged.

2·77 nmol/l); calcitonin 8 pmol/l (RIA Mallinckrodt kit, 0–44 pmol/l); parathormone 21·06 pmol/l (RIA Mallinckrodt kit, 15–60 pmol/l). The parathormone was almost undetectable. No further decrease in serum parathormone concentration was detected after intravenous administration of calcium. The typical clinical signs, the long QT interval, the increased tubular phosphate resorption, and the low serum concentration of parathormone were consistent with extreme hypocalcaemia and led to a diagnosis of hypoparathyroidism caused by a parathormone deficiency. Alcohol induced hypomagnesaemia may also have contributed to the impairment of parathormone release. We treated the patient with intravenous calcium and dihydrotachysterol drops. Shortly afterwards the cramps disappeared; they have not recurred.

Figure 2 shows the changes in the serum total and free ionic calcium and inorganic phosphate, and also the left ventricular ejection fraction, fractional shortening, end diastolic diameter, and the effect on corrected QT interval. Serum concentrations of total and free ionic calcium correlated strongly with the left ventricular variables. These variables gradually increased during treatment, while the serum inorganic phosphate and corrected QT interval decreased to normal. Between days 50 and 60, a dihydrotachysterol and calcium overdose produced high concentrations of calcium and a low concentration of phosphate. Later, an optimum regimen of oral calcium and dihydrotachysterol was attained. Since then the patient has remained well. She takes 500 mg calcium three times a day and 12 drops of dihydrotachysterol. Tubular phosphate resorption and other variables have returned to normal (table).

Our patient had a sister who died of congestive heart failure at the age of 16. Congenital and acquired valve diseases were excluded. There was no satisfactory explanation for her death.

Discussion

Although free ionic calcium has a well defined role in myocardial contractility, there are few well documented reports of the association of hypocalcaemia and disturbances of myocardial contractility. Giles *et al* reviewed seven published case reports and presented an additional one of "reversible congestive cardiomyopathy": all of these patients had hypoparathyroidism and impaired myocardial function.[2] These cases suggested the role of hypo-

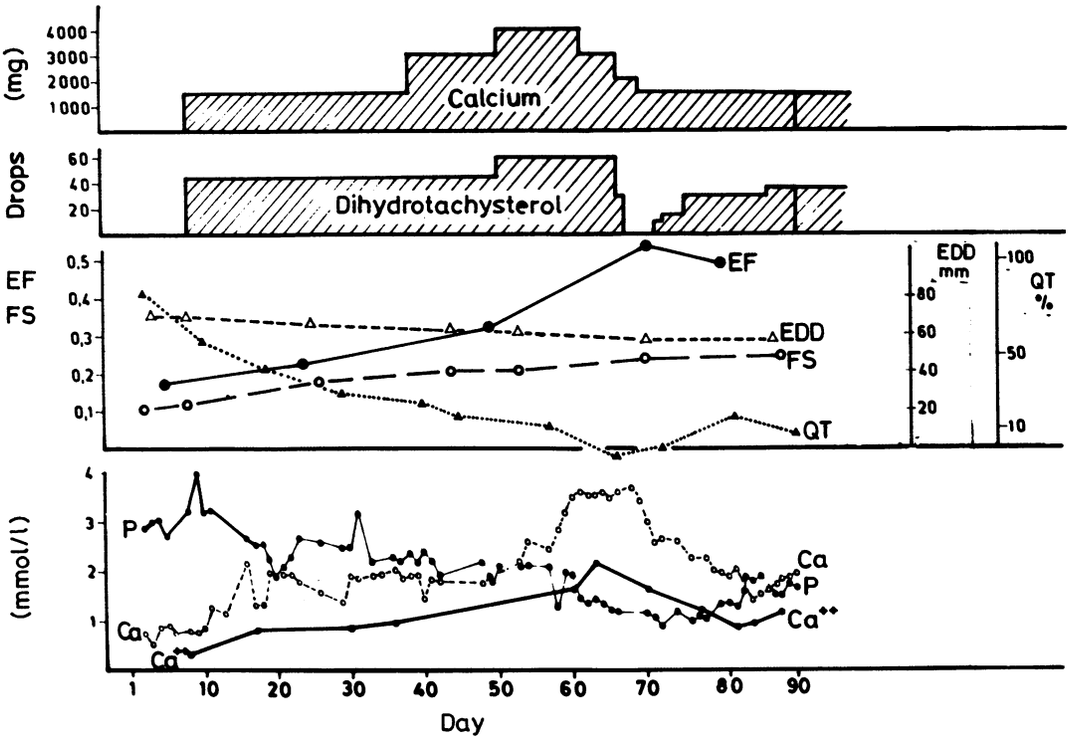

Figure 2 Treatment regimen, indices of left ventricular function, and the change in QT interval, serum concentrations of total calcium and free ionic calcium, and the serum concentration of inorganic phosphate. EF, ejection fraction; EDD, end diastolic diameter; FS, fractional shortening. The percentage difference between the measured and ideal QT intervals was calculated.

calcaemia and hypomagnesaemia in the development of congestive heart failure in hypoparathyroidism, but they did not establish causal relations. In 1983, Ginsburg *et al* reported the case of a boy with severe congestive heart failure who had a low serum concentration of free ionic calcium.[3] After this was restored to normal his myocardial contractility improved. They reported a direct relation between the serum concentration of free ionic calcium and myocardial performance. Connor *et al* reported a case in which hypocalcaemia precipitated congestive heart failure, and restoration of the normal serum concentration of calcium produced a clinical recovery but they did not monitor left ventricular function.[4] Our data confirmed that an increase in serum calcium (the total and free ionic calcium concentrations moved in parallel) caused an improvement in left ventricular function.

The unexpected death of a young sister of our patient owing to congestive heart failure suggests the possibility of the familial occurrence of congestive heart failure precipitated by hypocalcaemia.[6] Our case report shows that hypocalcaemia can cause reversible disturbances of myocardial contractility that resemble congestive cardiomyopathy.

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