

Ventricular fibrillation in hypercalcaemic crisis due to primary hyperparathyroidism

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ABSTRACT

We report the case of a 64-year-old man who presented with severe hypercalcaemia secondary to primary hyperparathyroidism. Soon after admission he developed ventricular fibrillation with no other cause than this severe hypercalcaemia. Although the occurrence of cardiac arrhythmias in hypercalcaemia is widely known, ventricular fibrillation has never been described before.

INTRODUCTION

Hypercalcaemic crisis, a serum calcium concentration greater than 3.50 mmol/l, is infrequent in primary hyperparathyroidism.^{1,2} Nevertheless, it is a life-threatening condition requiring emergency treatment.

Although the effects of hypercalcaemia on cardiac conduction are well established, reports of severe arrhythmias caused by primary hyperparathyroidism are rare. We present a case of severe hypercalcaemia, due to primary hyperparathyroidism, complicated by ventricular fibrillation.

CASE REPORT

A 64-year-old man was admitted to the emergency department for drowsiness and dehydration. His condition used to be good, despite hypertension, pulmonary embolism and a stroke. Yet, progressive forgetfulness, polyuria and polydipsia had been present for several months, but his condition had deteriorated rapidly three days before admission.

Physical examination showed a drowsy man who was disoriented in time and place. His heart rate was 55 beats/min. Blood pressure was 110/55 mmHg despite pre-existent hypertension treated with a calcium antagonist (amlodipine). He was dehydrated.

There were no abnormalities of the neck, chest and abdomen. A 12-lead electrocardiogram (ECG) showed non-specific ST and T wave abnormality and a QTc of 425 ms (*figure 1*). A chest X-ray was normal. Laboratory evaluation showed a total serum calcium of 4.95 mmol/l (reference values: 2.2-2.6 mmol/l), a serum albumin level of 28 g/l (35-50 g/l), and substantial renal insufficiency (creatinine 312 µmol/l (75-110 µmol/l)). Potassium level was 3.3 mmol/l (3.5-5.0 mmol/l).

The patient was transferred to the ICU. Amlodipine was stopped. A combination therapy of forced saline diuresis (350 ml/h NaCl 0.9% and furosemide 7 mg/h with potassium chloride 10 g/24 h) and pamidronate 60 mg was started but the serum calcium concentration declined only slightly: within four hours from 4.95 mmol/l to 4.75 mmol/l.

Several hours after admission cardiac monitoring showed a transition from sinus rhythm to ventricular fibrillation after a very short episode of torsades des points (*figure 2*). Sinus rhythm was restored on direct current cardioversion with 360 J after two unsuccessful attempts with 200 J. Soon afterwards a new episode of ventricular fibrillation occurred (*figure 2*), which again was terminated by cardioversion. Calcium concentration was 4.92 mmol/l, potassium concentration 3.5 mmol/l and magnesium concentration 0.79 mmol/l (0.7-1.0 mmol/l).

Because of this inadequate response, we decided to start

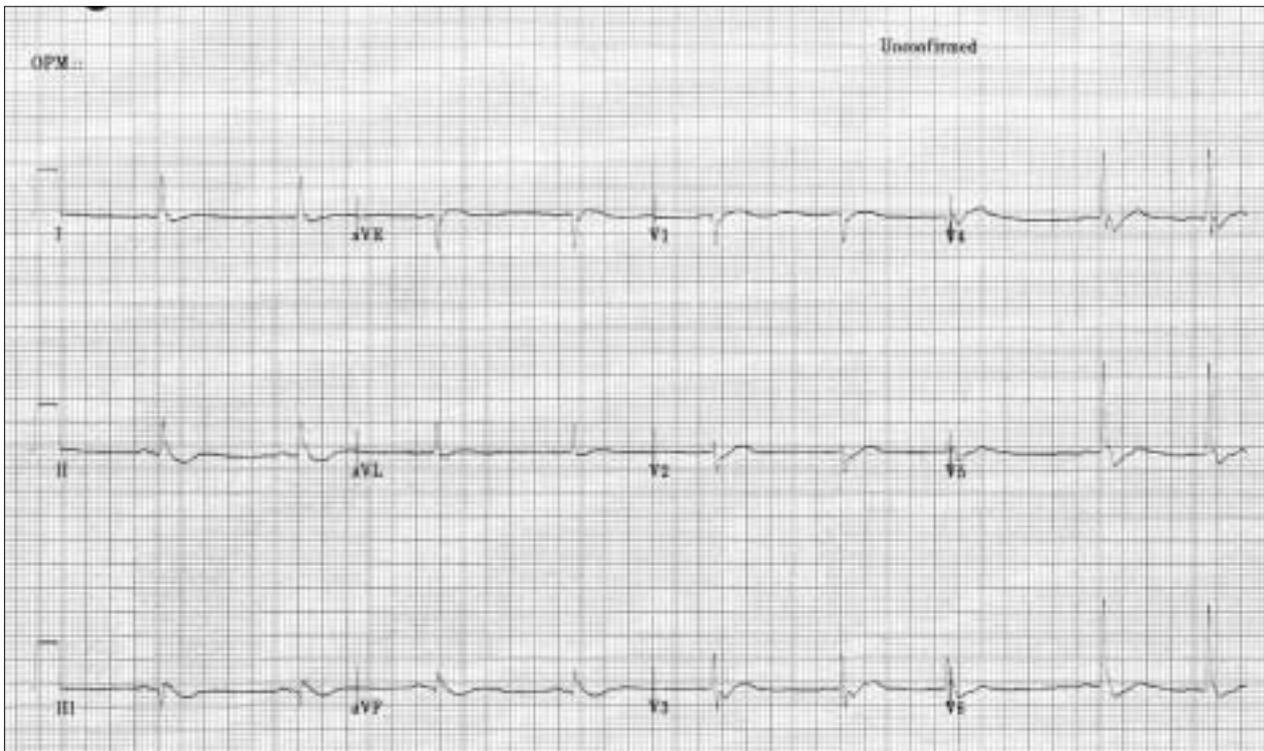


Figure 1
12-lead electrocardiogram on admission



Figure 2
ECG tracing of the start of the first (upper tracing) and the second (lower tracing) episode of ventricular fibrillation

calcium free haemodialysis. After four hours the serum calcium concentration had fallen from 4.92 mmol/l to 3.56 mmol/l. However, after ending haemodialysis a rebound effect occurred with a calcium level rising to 4.11 mmol/l

after five hours. This rebound phenomenon reoccurred after the next episode of haemodialysis: a serum calcium concentration decreasing from 3.90 mmol/l to 2.81 mmol/l but rising again to 3.20 mmol/l.

In the meantime, extended laboratory testing revealed a parathyroid hormone level of 172 pmol/l (<6 pmol/l). 25-OH vitamin D and FT₄ levels were normal (56 nmol/l and 10 pmol/l, respectively). A neck ultrasound showed a 2.5 x 1.5 cm mass located dorsally of the right lobe of the thyroid.

Because of the two episodes of ventricular fibrillation the patient had preoperative cardiac screening including echocardiography and a dipyridamole scan. Both were normal.

After normalisation of the serum calcium level (total serum calcium 2.23 mmol/l, serum albumin 30 g/l), the patient underwent a successful parathyroidectomy and recovered fully.

DISCUSSION

Since calcium plays a major part in cardiac conduction, it is generally accepted that disorders of calcium concentration can cause arrhythmia. Hypercalcaemia decreases ventricular conduction velocity and shortens the effective refractory period. The main ECG manifestation is an alternation in the QT interval, sometimes associated with prolongation of PR interval and QRS duration.^{3,4} In the second place, apart from hypercalcaemia, parathormone itself has a positive inotropic and chronotropic effect on heart cells, partly mediated by direct enhancement of calcium influx.⁵ The combination of decreased ventricular conduction velocity and shortened refractory period makes the occurrence of re-entry and thereby ventricular fibrillation very likely. However, case reports on this complication of hypercalcaemia are rare.

A wide variety of rhythm disorders, such as sinus arrest, atrial fibrillation and supraventricular tachycardia in hypercalcaemia due to primary hyperparathyroidism, have been described.⁶ Moreover, single case reports of sudden death have been reported.^{1,7} This is generally attributed to ventricular fibrillation, despite absence of electrocardiographic monitoring.

To our knowledge, documented evidence of ventricular arrhythmias in hypercalcaemic crisis due to primary hyperparathyroidism has been reported in only four cases, namely bigeminal arrhythmia,⁸ ventricular tachycardia⁹ and runs of multiform ventricular tachycardia degenerating into ventricular fibrillation.^{10,11}

Before attributing ventricular fibrillation in the presence of hypercalcaemia solely to this electrolyte disorder, other possible causes should be excluded. Two important other causes are serum potassium level and underlying heart disease.

Firstly, hypokalaemia and hypercalcaemia together facilitate ventricular fibrillation,^{3,4} and hence it is important to know the serum potassium level in arrhythmias attributed to hypercalcaemia. In many case reports information on potassium concentration has been lacking^{1,8} or described as below normal.^{7,10}

Secondly, an underlying heart disease could contribute to the propensity of a patient to develop arrhythmia in hypercalcaemia, although a clear relationship remains to be established. Both myocardial scarring facilitating re-entry and vigorous treatment with forced saline diuresis might play a role. Enhanced Na⁺/Ca²⁺ exchanger activity predisposes to ventricular tachyarrhythmias due to hypercalcaemia in patients with dilated cardiomyopathy.¹¹

In our patient, hypokalaemia on admission was treated by intravenous administration of potassium chloride and at the moment of occurrence of ventricular fibrillation, the serum potassium concentration was within the normal range. In the second place, neither his medical history nor cardiac screening showed any underlying cardiac disease. Hence, it is very likely that in this case, ventricular fibrillation was caused by hypercalcaemia due to primary hyperparathyroidism, which makes cardiac monitoring in patients with severe hypercalcaemia absolutely necessary.

REFERENCES

1. Hewson JS. Parathyroid crisis. A cause of sudden death. *Arch Intern Med* 1958;102:199-203.
2. Sarfati E, Desportes C, Gossot D, Dubost C. Acute primary hyperparathyroidism: experience of 59 cases. *Br J Surg* 1989;76:979-81.
3. Surawicz B. Role of electrolyte in etiology and management of cardiac arrhythmia. *Prog Cardiovasc Dis* 1966;8:364-86.
4. Surawicz B. Relationship between electrocardiogram and electrolytes. *Am Heart J* 1967;73:814-34.
5. Bogin E, Massry SG, Harary I. Effect of parathyroid hormone on rat heart cells. *J Clin Invest* 1981;67:1215-27.
6. Voss DM, Drake EH. Cardiac manifestations of hyperparathyroidism, with presentation of a previously unreported arrhythmia. *Am Heart J* 1967;72:235-9.
7. Naik BK, Sarma RN, Gopalrao V, Pargaonker PS, Gopal P. Acute hyperparathyroidism. *Arch Intern Med* 1963;111:729-33.
8. Chaieb Chadli M, Chaieb L, Jemni L, et al. Bigeminal arrhythmia associated with hyperparathyroid crisis. *Can Med Assoc J* 1988;138:1115-6.
9. Chang CJ, Chen SA, Tai CT, et al. Ventricular tachycardia in a patient with primary hyperparathyroidism. *PACE* 2000;23:534-7.
10. Kearney P, Reardon M, O'Hare J. Primary hyperparathyroidism presenting as torsades de points. *Br Heart J* 1993;70:473.
11. Kolb C, Lehmann G, Schreieck J, Ndrepepa G, Schmitt C. Storms of ventricular tachyarrhythmias associated with primary hyperparathyroidism in a patient with dilated cardiomyopathy. *Int J Cardiol* 2003;87:115-6.