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Acute Myocardial Infarction as a Consequence of Hypocalcaemia and Hyperthyroidism in a Young Patient Long After Subtotal Thyroidectomy

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Aim: Long-standing hypocalcaemia can be complicated by congestive heart failure. However, acute myocardial infarction as a first complication of hyperthyroidism and severe hypocalcaemia due to delayed onset of postoperative hypoparathyroidism, in a previously normal young patient, long after subtotal thyroidectomy, has been rarely reported in the literature.

Patient and Methods: We report the case of a 37 year old male urgently admitted to our department because of chest pain, ECG changes, and laboratory findings of acute myocardial infarction. A previous history of subtotal thyroidectomy 17 years ago is reported. Since then he was totally asymptomatic, reported no drug or alcohol abuse and except for mild smoking he had no other risk factors for coronary artery disease. Moreover, clinical examination revealed a positive Trousseau's sign, while ECG also showed prolonged QT interval. Chest x-ray was normal. Laboratory analyses revealed severe hypocalcaemia in repeated measurements (Ca^{++}: 5.86 mg/dl; normal: 8.2–10.7 mg/dl), hyperphosphataemia (Phos: 6.06 mg/dl; normal: 2.4–4.9 mg/dl), low serum levels of PTH (5 pg/ml; normal: 10–55 pg/ml), while thyroid function tests revealed hyperthyroidism (hTSH: 0.005 IU/ml; normal: 0.27–4.2 IU/ml), FT_3: 7 pg/ml (normal: 1.82–4.62 pg/ml). All the other haematological and blood chemistry results were normal (normal renal function tests, normal albumin levels).

Results: Except for thrombolysis and treatment of the acute myocardial infarction in the intensive care unit, supplementation with calcium and vitamin D3, as well as anti-thyroid drugs (carbimazole 15 mg 3 times daily) were initiated. Cardiac catheterisation, which followed, showed one-vessel disease (70 % stenosis of the proximal section of left anterior descending coronary artery), which was treated with angioplasty and stenting. Thereafter, the patient remained asymptomatic and is under continuous calcium and vitamin D3 supplementation and anti-thyroid drugs.

Conclusions: According to this case, we considered the acute myocardial infarction of this patient being a consequence of hyperthyroidism and severe hypocalcaemia, due to delayed onset of postoperative hypoparathyroidism, in a previous asymptomatic patient, with minimal angiographic lesions and absence of other risk factors. ***J Clin Basic Cardiol 2005; 8: 69–72.***

Key words: *acute myocardial infarction, hypocalcaemia, hyperthyroidism, subtotal thyroidectomy*

Calcium ions play a vital role in the sequence of excitation-contraction of the cardiac muscle fibers and they are essential in both the cardiac and systemic vasculature [1, 2]. Furthermore, hypocalcaemia impairs myocardial contractility and there are several reports of congestive heart failure caused by severe hypocalcaemia, while long-standing hypocalcaemia has been implicated in the pathogenesis of cardiomyopathy [3–5]. Moreover, coronary spasm due to hypocalcaemia has been reported as the most likely mechanism of chest pain in young patients mimicking acute myocardial infarction [2, 6].

On the other hand, myocardial ischaemia is a rare but severe and possibly life threatening manifestation of hyperthyroidism, since it is known that thyroid hormones increase oxygen demand [7–9]. What is more, a number of well-documented cases of myocardial infarction in patients with thyroid hormone excess and normal coronary arteries in angiography have been reported in the literature [10, 11].

However, to our knowledge, overt acute myocardial infarction in relation to undiagnosed hyperthyroidism and severe hypocalcaemia, due to delayed onset of postoperative hypoparathyroidism, in a normal young patient, long after subtotal thyroidectomy, has not been previously reported in the literature. The aim of this study was to report on a 37 year old male, admitted to cardiology department with clinico-laboratory findings of acute myocardial infarction and found to have severe hypocalcaemia and hyperthyroidism, remained asymptomatic 17 years after subtotal thyroidectomy.

Case Report

A 37 year old male presented to the emergency department because of severe chest pain, more than one-hour duration and haemodynamic instability. ECG showed ST segment elevation in leads I, aVL, and V1–V6 and concurrent ST segment depression in leads II, III, and aVF, while laboratory findings confirmed extended anterior acute myocardial infarction. Subsequently the patient was admitted to the coronary care unit.

Personal History

Except for subtotal thyroidectomy 17 years ago, his previous history was totally negative. Since then he was entirely asymptomatic. He reported no drug or alcohol abuse, no hypertension and no other risk factors for coronary artery disease, except for mild smoking.

Physical Examination

The patient was a thin, tachycardic, severe ill patient, with systolic blood pressure upon admission of 95 mmHg, pulse rate of 94 beats/min. and body mass index (BMI) of 17 kg/m^2 (normal: 20–25 kg/m^2). The interesting point of physical examination was the positive Trousseau's sign, during blood pressure measurement. No previous signs of hypocalcaemia (no cramps, no hand numbness) were reported.

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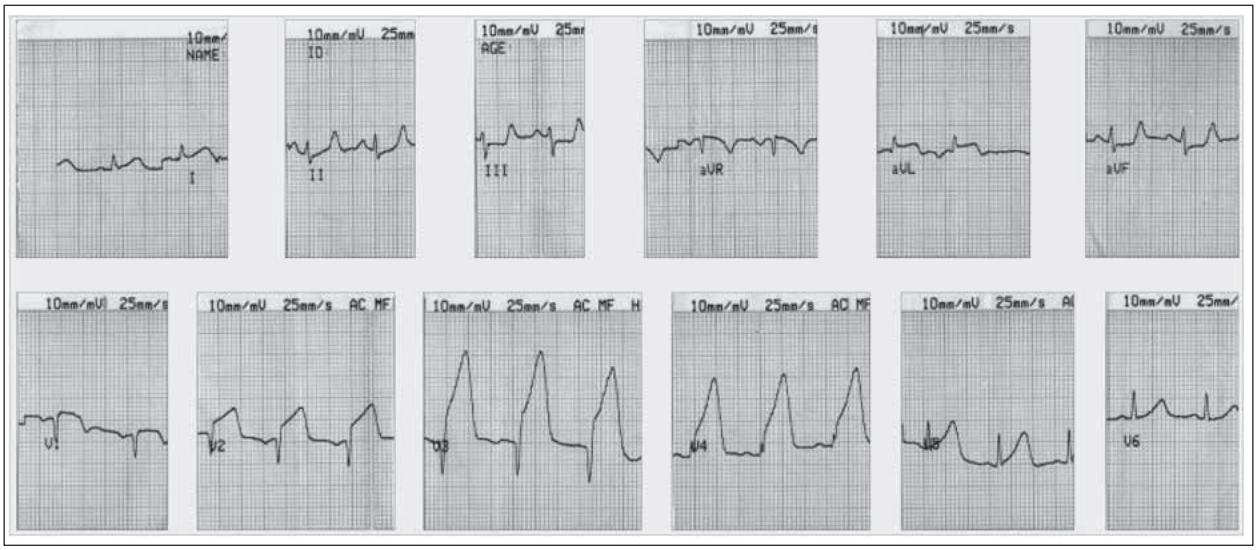

Figure 1. ECG on admission shows ST segment elevation in leads I, aVL, and V1–V6, concurrent ST segment depression in leads II, III, and aVF and prolonged QTc interval (QTc: 0.49 s; normal: < 0.39 s).

Other Systems
Normal findings.

ECG on Admission
Sinus tachycardia, ST segment elevation in leads I, aVL, and V1–V6 and concurrent ST segment depression in leads II, III, and aVF. In addition the QTc interval, as calculated using Bazett's correction (in which the raw interval from beginning of QRS complex to the apex of the T wave is divided by the square root of the R-R interval) was prolonged (QTc: 0.49 s; normal values for men: < 0.39 s) [12–13] (Fig. 1).

Laboratory Examination on Admission
Ht 40 %, Hb 13 g/dl, WBC 14,500/ml with neutrophilia (polymorphonuclear: 83 %; lymphocytes: 12 %; monocytes: 4 %; other: 1 %), PLT 250,000/ml, ESR 4 mm/1 h, glucose 147 mg/dl, urea 45 mg/dl, creatinine 1.21 mg/dl, K^+ 3.9 mEq/dl, Na^+ 141 mEq/dl, AST 454 IU/l (normal: 2–40 IU/l), ALT 138 IU/l (normal: 20–65 IU/l), CK 5418 IU/l (normal: 10–190 IU/l), CK-MB 272 IU/l (normal: 0–6 IU/l), LDH 970 IU/l (normal: 90–220 IU/l), cholesterol 159 mg/dl, triglycerides 111 U/l, HDL cholesterol 50 mg/dl, uric acid 5.56 mg/dl (normal: 2.6–7.2 mg/dl), Ca^{++} 5.86 mg/dl (normal: 8.2–10.7 mg/dl), P 6.23 mg/dl (2.4–4.9 mg/dl), total proteins 6.98 g/dl (albumin 3.48 g/dl, globulins 3.5 g/dl), total bilirubin 0.82 mg/dl, alkaline phosphatase 82 IU/l (normal: 30–140 IU/l), γ-GT 30 IU/l (normal: 7–32 UI/l), CRP: 1.54 g/dl (normal: < 0.8 g/dl), iron 107 mg/dl, INR 1.2, Prothrombin Time: control 12 s/patient 12.4 s, fibrinogen 177 mg/dl, B12 428.8 pg/ml (normal: 157–1059 pg/ml), folate 5.4 ng/ml (normal: 5.3–14.4 ng/ml). All the other blood chemistry results were totally normal.

Thyroid Hormones Revealed Hyperthyroidism
hTSH < 0.005 IU/ml (normal: 0.27–4.2 IU/ml), T4 13.46 ng/dl (normal: 5.13–14.06 ng/dl), T3 2.14 ng/ml (normal: 0.846–2.02 ng/ml), FT4 2.4 ng/dl (normal: 0.932–1.71 ng/dl), FT3 7 pg/ml (normal: 1.82–4.62 pg/ml).

Low Serum Parathormone Levels
PTH 8 pg/ml (normal: 10–55 pg/ml). Myocardial enzymes on admission and the days thereafter followed the expected serial changes following an acute myocardial infarction, while serial serum Ca^{++} and phosphorus measurements revealed severe hypocalcaemia (Ca^{++}: 5.86/6.17/5.86/6.11 mg/dl; normal: 8.2–10.7 mg/dl) and hyperphosphataemia (Phos: 6.06/5.5/6.23 mg/dl; normal: 2.4–4.9 mg/dl). Furthermore, except for slight leucocytosis, all the other haematological and blood chemistry results were totally normal, particularly the patient had a normal renal function test and normal serum albumin, cholesterol and triglyceride levels.

Radiological Examination
Chest x-ray was normal.

Management
The patient was initially treated in the coronary care unit and received thrombolysis with accelerated infusion of 100 mg alteplase infused over a period of 90 minutes in combination with small molecular weight heparin (SMWH), aspirin, β-blocker, and captopril (12.5 mg twice daily). Nitroglycerine was not initially used due to low blood pressure, but was initiated after haemodynamic stabilisation. The chest pain was totally subsided soon after the completion of thrombolysis, however serious reperfusion arrhythmias (bigeminy, multifocal, pair or couplet and ventricular tachycardias) (Fig. 2) appeared during the first 24 h after the myocardial infarction, which persisted despite the use of antiarrhythmic drugs (at first instance xylocaine and thereafter amiodarone intravenously).

Moreover, after having the laboratory results of severe hypocalcaemia, which was confirmed in repeated measurements, supplementation with calcium (at first instance intravenous infusion of calcium gluconate and two days later continuous oral calcium bicarbonate) and vitamin D3 were initiated. The interesting point of this case was that arrhythmias subsided soon after the intravenous administration of calcium gluconate.

Furthermore, on basis of thyroid hormones, which revealed hyperthyroidism, anti-thyroid drugs (carbimazole 15 mg 3 times daily) were also administered.

The patient's clinical condition improved impressively during the following days and he was discharged from the cardiology department in a good condition seven days later. ECG at discharge from the hospital showed Q waves in leads I,

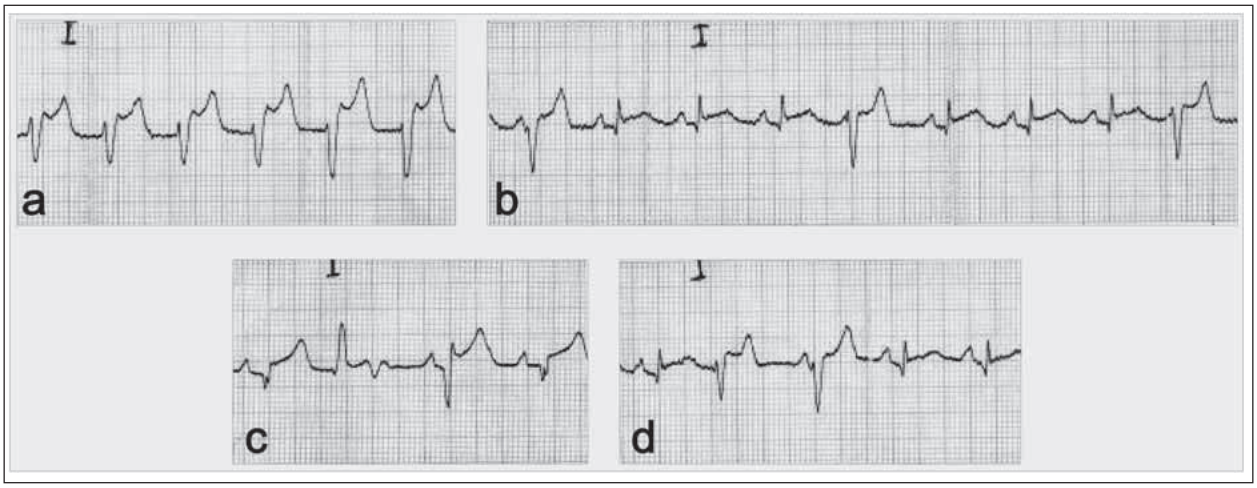

Figure 2. ECG after completion of thrombolysis showed serious reperfusion arrythmias: **a)** ventricular tachycardias, **b)** trigeminy, **c)** multifocal, **d)** pair

aVL, and V1–V4. There was subsequent improvement in laboratory findings (normal calcium levels Ca^{++}: 7.8 mg/dl) as well as clinical performance. Subsequent cardiac catheterisation showed one-vessel disease (70 % stenosis of the proximal section of left anterior descending coronary artery), which was successfully treated with angioplasty and stenting.

Follow-Up

Two months after angioplasty the patient was totally asymptomatic, living a normal life and is under continuous antianginic therapy (β-blocker, ACE inhibitor and aspirine), calcium and vitamin D3 supplementation and anti-thyroid drugs.

Discussion

There are several case reports in the literature of congestive heart failure [1–3] and cardiomyopathy [4, 5] associated with hypocalcaemia caused by hypoparathyroidism. Although this does not necessarily establish a causal relationship between heart dysfunction and hypocalcaemia, the resolution of heart failure, as well as the improvement of cardiomyopathy after the correction of hypocalcaemia strongly supports this hypothesis [1–5]. Furthermore, clinical, biochemical, and electrocardiographic findings mimicking acute myocardial infarction have been also reported in a few case reports [2, 6]. Reddy et al. [14] in 1974 and Khardori et al. [15] in 1985 each described a single patient with an acute anteroseptal injury pattern on the ECG with no proven subsequent infarction, which they associated with hypocalcaemia. Coronary spasm in the clinical setting of hypocalcaemia appears the most likely cause of the chest pain mimicking acute myocardial infarction, in the above-mentioned cases [2–4].

However, a real acute myocardial infarction in relation to severe hypocalcaemia in a normal young patient, as in this case, has not been previously reported in the literature. In the present case, the combination of hypocalcaemia, hyperphosphataemia and low levels of parathormone, in combination with the previous history of subtotal thyroidectomy, although the patient remained asymptomatic the last 17 years after the operation, made more likely the diagnosis of postoperative hypoparathyroidism, probably due to surgical removal of parathyroid glands. It is known from the literature that neck surgery, even long past, can be associated with a delayed onset of postoperative hypoparathyroidism [16].

On the other hand, there are many reports in the literature about the relevant effects of thyroid hormone excess on the heart, mainly tachycardia, atrial fibrillation, myocardial hypertrophy, and dilated cardiomyopathy associated with Graves' disease, mitral valve prolapse and coronary artery disease [7–11]. Myocardial ischaemia is a rare but severe and potentially life threatening manifestation of hyperthyroidism and seems to be a consequence of the increase in consumption of oxygen in the presence of an unchanged oxygen supply rather than of obstruction of coronary circulation [8]. Well-documented cases of myocardial infarction in patients with thyroid hormone excess and normal coronary arteries in angiography substantiate this theory [8, 10].

The limited angiographic findings (one-vessel disease only), not suitable with the extend of the infarction, in combination with the absence of other serious risk factors for coronary artery disease in this patient, lead us to hypothesise that the acute myocardial infarction of the patient was a rare consequence of hyperthyroidism and hypocalcaemia induced vasospasm in a coronary artery with minimal atherosclerotic lesions.

Another interesting finding contributing to the above assumption was that the serious reperfusion arrhythmias of the patient subsided soon after the intravenous administration of calcium gluconate and restoration of hypocalcaemia, although they did not respond to intravenous antiarrhythmic therapy with xylocaine at first instance and amiodarone thereafter. Cardiac arrhythmias in relation to hypocalcaemia are rarely reported in the literature [2, 13], including Torsades de pointes, while the ECG hallmark of hypocalcaemia remains the prolongation of the QTc interval, which was also found in our patient (QTc interval 0.49 s; normal: < 0.39 s, according to Bazett's correction) [13].

Acquired chronic hypoparathyroidism is usually the result of inadvertent surgical removal of all the parathyroid glands, in some instances, not all the tissue is removed, but the remainder undergoes compromise of vascular supply secondary to fibrotic changes in the neck after surgery. In the past the most frequent cause of acquired hypoparathyroidism was surgery for hyperthyroidism, as in this case, contributed to 0.2 % rate after thyroid surgery in some reports, mainly after total thyroidectomy [17]. In our case, although the patient had subtotal thyroidectomy, he had hypocalcaemia, probably inadvertent, long after the surgery.

Furthermore, hyperthyroidism is known to increase the levels of serum calcium and in this case the real levels of serum calcium after correction of hyperthyroidism would be even lower, making the contribution of hypocalcaemia in the manifestation of the acute myocardial infarction of the patient more likely [18].

Conclusively, we considered the acute myocardial infarction of our patient being a rare complication of hyperthyroidism and severe hypocalcaemia, due to delayed onset of postoperative hypoparathyroidism, in a previous asymptomatic patient, with minimal angiographic lesions and absence of other risk factors.

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