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

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D. Eleftheriadis, P Vrizidis, A. Mitsianis, N. Eleftheriadis

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 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), FT3: 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 (carbamazole 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/m² (normal: 20–25 kg/m²). 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.

Received: August 20th, 2002; accepted: November 2nd, 2004.
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Other Systems
Normal findings.

ECCG 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, urca 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: 0–220 IU/l), cholesterol 111 U/l, HDL cholesterol 50 mg/dl, uric acid 5.56 mg/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 IU/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.

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 (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.

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.

Thyroid Hormones Revealed Hyperthyroidism
hTSH < 0.005 IU/ml (normal: 0.27–4.2 IU/ml), T4 13.46 ng/dl (normal: 3.13–14.06 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).

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).
delayed onset of postoperative hypoparathyroidism [16].

that neck surgery, even long past, can be associated with a
moval of parathyroid glands. It is known from the literature
operative hypoparathyroidism, probably due to surgical re-
after the operation, made more likely the diagnosis of post-

the last 17 years

phosphataemia and low levels of parathormone, in combina-

present case, the combination of hypocalcaemia, hyper-
has not been previously reported in the literature. In the

vere hypocalcaemia in a normal young patient, as in this case,

However, a real acute myocardial infarction in relation to se-

hypocalcaemia in a normal young patient, as in this case, contrib-
ted to 0.2 % rate of acquired hyperparathyroidism. 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

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 as-
sumption 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 T orsades 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 remain-
der 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 hyperparathyroidism, 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 in-
advertent, long after the surgery.

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 hypo-
thesis [1–5]. Furthermore, clinical, biochemical, and electro-
cardiographic findings mimicking acute myocardial infarc-
tion have been also reported in a few case reports [2, 6].
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 se-
vere 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, hyper-
phosphataemia and low levels of parathormone, in combina-
tion 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 post-
operative hypoparathyroidism, probably due to surgical re-
moval 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].

Follow-Up

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

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

Figure 2. ECG after completion of thrombolysis showed serious reperfusion arrhythmias: 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 catheteri-
sation showed one-vessel disease (70 % stenosis of the prox-
imal section of left anterior descending coronary artery),
which was successfully treated with angioplasty and stenting.

The limited angiographic findings (one-vessel disease only),
only, not suitable with the extend of the infarction, in combina-
tion 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.

Acquired chronic hypoparathyroidism is usually the result of
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subtotal thyroidectomy, he had hypocalcaemia, probably in-
advertent, 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.

References:
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