



CASE REPORT

Hypocalcemia as a reversible cause of heart failure[☆]

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Abstract Calcium plays a key role in heart muscle contraction and relaxation. Hypocalcemic heart failure is a rare and potentially reversible disturbance, which reflects this intrinsic relationship.

The authors present the case of a 35-year-old woman who developed acute heart failure during the early postoperative period following total thyroidectomy. The echocardiogram showed severe global left ventricular dysfunction. Laboratory tests showed severe hypocalcemia and new-onset hypoparathyroidism. Cardiac catheterization showed angiographically normal coronary arteries. After clinical, hemodynamic and metabolic stabilization, a repeat echocardiogram revealed recovery of left ventricular function. Subsequently, cardiac magnetic resonance imaging was performed, which also showed no alterations. The patient was discharged asymptomatic, medicated with calcium carbonate, calcitriol and levothyroxine.

This case highlights the importance of considering hypocalcemia as a cause of reversible myocardial dysfunction.

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Hipocalcemia como uma causa reversível de insuficiência cardíaca

Resumo O cálcio assume um papel fundamental na contração e relaxamento do músculo cardíaco. A insuficiência cardíaca hipocalcémica é um distúrbio raro e potencialmente reversível, que traduz esta relação intrínseca.

Os autores apresentam o caso de uma mulher de 35 anos que desenvolveu um quadro de insuficiência cardíaca aguda, durante o pós-operatório precoce de tiroidectomia total. O ecocardiograma mostrou disfunção global grave do ventrículo esquerdo. Analiticamente apresentava hipocalcemia grave e hipoparatiroidismo de novo. O cateterismo cardíaco mostrou artérias coronárias angiograficamente normais. Após estabilização clínica, hemodinâmica e metabólica, repetiu ecocardiograma que revelou recuperação da função ventricular esquerda. Posteriormente, realizou ressonância magnética cardíaca que também não mostrou alterações. A doente

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teve alta assintomática, medicada com carbonato de cálcio, calcitriol e levotiroxina. Este caso salienta a importância de considerar a hipocalcemia como uma causa reversível de disfunção miocárdica.

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Introduction

The role of calcium in heart muscle contraction and relaxation is well established.¹ Hypocalcemia is a rare cause of heart failure; there are few reports in the literature documenting a causal relation between the two.

The authors present a case of acute heart failure induced by hypocalcemia in a setting of iatrogenic hypoparathyroidism, in which myocardial function normalized following resolution of the metabolic disturbance.

Case report

A 35-year-old Caucasian woman with no known cardiovascular risk factors and a history of a large multinodular euthyroid goiter diagnosed five years previously, recently causing deviation of the trachea and dysphagia, was hospitalized in January 2010 for elective total thyroidectomy.

Preoperative laboratory tests showed parathyroid hormone (PTH) and total calcium within normal limits and slightly reduced ionized calcium (1.10 mmol/l).

The surgery was uneventful, a markedly multinodular goiter being resected, the parathyroid glands being preserved.

Two hours after the operation, the patient developed acute pulmonary edema. On physical examination she was apyretic, with tachycardia, low blood pressure, signs of marked pulmonary congestion and type 1 respiratory failure.

The electrocardiogram showed sinus tachycardia with a slightly increased corrected QT interval (QTc) (481 ms). The chest X-ray also showed clear signs of pulmonary congestion. Troponin I was elevated (1.43 ng/ml) but BNP was within normal limits.

The echocardiogram revealed cardiac chambers of normal dimensions, severe left ventricular systolic dysfunction with no evidence of wall motion abnormalities, transmitral flow with a restrictive pattern (type III diastolic dysfunction) and preserved right ventricular systolic function.

The patient was admitted to the cardiac care unit with cardiogenic shock and required inotropic support with dobutamine for the first 24 h. The clinical course was then favorable with rapid clinical and hemodynamic stabilization.

During the initial stage of stabilization she had several episodes of tetany which resolved with calcium and calcitriol supplementation.

Laboratory tests 12 h after symptom onset showed continued euthyroidism and severe hypocalcemia (ionized calcium: 0.88 mmol/l), together with low PTH (4.0 pg/ml) and elevated phosphate levels (Table 1). At this stage BNP was significantly elevated and troponin I was falling, with a peak value similar to the initial measurement.

A wide-ranging immunological and viral study revealed no abnormalities.

On the fourth day of hospitalization, by which time the patient had regained normal calcium levels and QTc, a repeat echocardiogram revealed recovery of globally preserved left ventricular function, with no wall motion abnormalities and with normal transmitral flow. Cardiac catheterization showed angiographically normal coronary arteries.

In order to further clarify the clinical picture, cardiac magnetic resonance imaging was performed, which also showed no morphological or functional alterations or contrast uptake in delayed enhancement sequences.

The patient was discharged asymptomatic, medicated with calcium carbonate, calcitriol and levothyroxine. Out-patient assessment showed her to be clinically and echocardiographically stable.

Discussion

Hypoparathyroidism affects both sexes and all ages, and can have different causes, particularly idiopathic and surgical.

The main clinical manifestations of hypoparathyroidism are due to its effects on calcium–phosphorus metabolism.

Hypocalcemia following thyroidectomy can be caused by the accidental removal of one or more parathyroid glands, ischemia caused by damage to their delicate blood supply, or simply manipulating the glands.^{2,3} It is now known that hypocalcemia is a relatively common complication of surgery that in most cases is transient and asymptomatic.³

The role of serum calcium in excitation and contraction of heart muscle fibers is well established. Inflow of extracellular calcium together with that from the stimulated sarcoplasmic reticulum raises the levels of cytosolic calcium available to bind to troponin C. This in turn interacts with the troponin–tropomyosin complex to form crossbridges between actin and myosin, resulting in muscle contraction.¹ Although the kinetics of intracellular calcium is clearly related to muscle contraction and relaxation, the mechanism of myocardial dysfunction secondary to hypocalcemia is not fully understood.^{1,5–7}

Cardiac performance is demonstrably reduced by hypocalcemia, with decreased myocardial contractility and hence decreased left ventricular stroke work index, ejection fraction and cardiac index.⁴ However, in the few cases in the literature describing heart failure as a complication of hypocalcemia, all patients recovered myocardial function when calcemia was corrected, which supports the hypothesis that it is a reversible cause of heart failure.^{5–7}

This relationship was also observed in our patient, because recovery of myocardial function was seen once the metabolic disturbance was resolved.

Table 1 Changes in laboratory parameters in the early stages of disease presentation.

	Preop. ^a	Postop. ^b	Postop. ^c	Reference value
Total calcium (mEq/l)	4.7		3.6	4.2–5.1
Ionized calcium (mmol/l)	1.10		0.88	1.13–1.32
PTH (pg/ml)	29.7		4.0	10.0–65.0
Phosphorus (mg/l)			50.2	27–45
Myoglobin (ng/ml)		57.3	34.8	<146.9
Tnl (ng/ml)		1.43	1.24	<0.012
CK-MB (ng/ml)		14.9	18.60	<6.4
BNP (pg/ml)		88.9	743.5	<100

BNP, brain natriuretic peptide; PTH, parathyroid hormone; Tnl, troponin I.

^a Preoperative.

^b Postoperative: 2 h after symptom onset.

^c Postoperative: 12 h after symptom onset.

The case presented describes a case of acute heart failure secondary to hypocalcemia in the context of iatrogenic hypoparathyroidism following thyroidectomy. Although symptoms of hypocalcemia do not usually begin until 24–48 h after surgery, in this case the clinical setting developed significantly earlier.

The short half-life of PTH has prompted several authors to investigate the importance of perioperative PTH measurement as a predictor of symptomatic hypocalcemia.^{3,8} The best timing for measurement has not been defined; different authors have suggested between 10 min and 24 h after thyroidectomy.^{2,9,10}

Early measurement has the advantage of enabling prompt treatment, including calcium and calcitriol supplementation and, in extreme cases, intravenous parathyroid hormone.

Conclusions

Hypocalcemic heart failure is a possible complication of iatrogenic hypoparathyroidism following thyroidectomy.

The case presented highlights the importance of considering hypocalcemia as a cause of acute heart failure, since myocardial dysfunction can be fatal but is rapidly reversible after correction of the metabolic disturbance.

Conflicts of interest

The authors have no conflicts of interest to declare.

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