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CASE REPORT

Hypocalcemic cardiomyopathy☆

Patrício Aguiar[a,*], Diogo Cruz[a], Rita Ferro Rodrigues[b], Lígia Peixoto[a], Francisco Araújo[c], José Luís Ducla Soares[a]

[a] *Serviço de Medicina 1, Centro Hospitalar Lisboa Norte, EPE, Lisboa, Portugal*
[b] *Serviço de Doenças Infecciosas, Centro Hospitalar Lisboa Norte, EPE, Lisboa, Portugal*
[c] *Departamento de Medicina Interna, Hospital Beatriz Ângelo, Loures, Portugal*

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KEYWORDS
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Abstract The association between hypocalcemia and heart failure is rare. There are few reported cases in the literature of this association, which is termed hypocalcemic cardiomyopathy.

We report the case of a 61-year-old woman with no relevant medical history, admitted for progressively worsening exertional dyspnea, orthopnea and edema of the lower limbs over a period of one month. Physical examination showed diffuse muscle spasms, with no signs of latent tetany.

Further investigation revealed ionized calcium 0.54 mmol/l (normal 1.12–1.30), phosphorus 9.8 mg/dl, parathyroid hormone <2.5 pg/ml and CK >3000 U/l, with normal thyroid function. The electrocardiogram showed long QT interval and a pattern of left ventricular overload, and myocardial biomarkers were negative. The echocardiogram revealed regional wall motion abnormalities, coronary angiography was normal and a cranial CT scan detected calcification of basal ganglia and white matter.

She started diuretic and calcium replacement therapy which resulted in complete clinical recovery, with no need for heart failure therapy after normalization of serum calcium.

PALAVRAS-CHAVE
Insuficiência cardíaca;
Hipoparatiroidismo;
Hipocalcemia;
Miocardiopatia hipocalcémica

Miocardiopatia hipocalcémica

Resumo A associação entre hipocalcemia e insuficiência cardíaca é rara. Na literatura existem poucos casos descritos com esta associação, tendo-se estabelecido a entidade miocardiopatia hipocalcemica.

Relata-se o caso de uma mulher, 61 anos, sem antecedentes médicos relevantes. Internada por um quadro com um mês de evolução de dispneia de esforço, ortopneia e edema dos membros inferiores de agravamento progressivo. À observação apresentava espasmos musculares difusos, sem sinais de tetania latente.

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* Corresponding author.
E-mail address: patricio_aguiar@yahoo.com.br (P. Aguiar).

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Da investigação complementar destacavam-se cálcio ionizado 0,54 mmol/l (1,12-1,30), fósforo 9,8 mg/dl, hormona paratiroideia <2,5 pg/ml e CK total >3000 U/l, com função tiroideia normal. O electrocardiograma revelava prolongamento do intervalo QT e padrão de sobrecarga do ventrículo esquerdo e os marcadores de necrose miocárdica eram negativos. O ecocardiograma demonstrava alterações segmentares da contractilidade miocárdica, a coronariografia era normal e na TC-CE identificavam-se calcificações dos núcleos basais e substância branca.

Iniciou terapêutica diurética e de reposição do cálcio com remissão completa da insuficiência cardíaca, sem necessidade de terapêutica específica para a mesma após normalização da calcemia.

Introduction

The clinical syndrome of heart failure (HF) results from congenital or acquired alterations to cardiac structure and/or function that are manifested by an imbalance between cardiac output and tissue oxygen requirements.[1,2]

Among the different etiologies of HF are the cardiomyopathies, which are classified according to morphological type as dilated, restrictive and hypertrophic. Some forms of dilated cardiomyopathy, due to metabolic or toxic causes, are reversible (Table 1).[3]

Calcium plays an essential role in myocardial metabolism, and hypocalcemia reduces myocardial contractility. However, HF of this etiology is rare, with few cases reported in the literature,[4] but in most of these cases, correction of hypocalcemia led to resolution of HF.

Only one case has been reported in Portugal of dilated cardiomyopathy associated with post-surgical hypothyroidism, in which hypocalcemia, also in the context of post-surgical hypoparathyroidism, was the factor triggering decompensation of HF.[5]

Case report

We report the case of a 61-year-old woman from Brazil, where she had been a mathematics teacher, resident for around a year in Portugal, where she worked as a cleaner. She had a history of surgery for bilateral pseudophakia over 15 years previously; she reported no other previous conditions, relevant family history or cardiovascular risk factors, was taking no medication, and did not drink or smoke.

She had been asymptomatic until three months before admission, when she began to experience worsening exertional dyspnea, associated with orthopnea in the three days before admission. She reported no paroxysmal nocturnal dyspnea, chest pain, palpitations, syncope, cough, expectoration or fever throughout this period.

Due to symptoms on minimal exertion she went to the emergency department, where examination showed mental confusion, psychomotor slowing, depressed facial expression, blood pressure 97/59 mmHg, rhythmic heart rate 79 bpm, respiratory rate 28 cpm, thinning of the outer third of the eyebrows, limb tremor and muscle spasms, but no Chvostek or Trousseau sign. Crackling rales were audible in the lower half of both lung fields, as well as an S3 gallop and a grade I/VI systolic murmur more clearly audible in the mitral area. The rest of the physical exam was normal.

Further diagnostic tests revealed normal myocardial necrosis markers, elevated BNP, rhabdomyolysis, severe hypocalcemia and type 1 respiratory failure (Table 2). The electrocardiogram (ECG) (Figure 1) showed long QT interval (QTc 0.53 s) and T-wave inversion in V2–V4 and DI. The posteroanterior chest X-ray (Figure 2) revealed interstitial infiltration in the lower third of both lung fields, suggestive of edema.

The bedside echocardiogram in the emergency department showed left ventricular dilatation (60/44 mm) with

Table 1 Metabolic etiologies of reversible cardiomyopathy.

Congenital
Glycogenoses
Mucopolysaccharidoses
Fabry disease
Hemochromatosis
Danon disease
Friedreich ataxia
Acquired
Hypothyroidism
Hyperthyroidism
Hypocalcemia
Alcohol/drug toxicity
Diabetes
Pheochromocytoma
Acromegaly
Beriberi

Table 2 Initial laboratory assessment.

LDH cholesterol (U/l)	989
CK (U/l)	3784
NT-proBNP (pg/ml)	3994
TSH (U/ml)	1.32
Troponin (ng/ml)	<0.04
pH	7.46
PO_2 (mmHg)	67
PCO_2 (mmHg)	29
Ca^{2+} (mmol/l)	0.54 (1.13–1.32)

TSH: thyroid-stimulating hormone.