



CASE REPORT

A rare case of persistent troponin elevation in a patient with chronic heart failure[☆]



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Received 21 February 2013; accepted 25 April 2013

Available online 17 December 2013

KEYWORDS

Hypertrophic cardiomyopathy;
Troponin;
Heart failure

Abstract We present the case of a woman diagnosed with hypertrophic cardiomyopathy who suffered a myocardial infarction when she was 28 years old, without coronary artery disease on coronary angiography. Two years later, she presented signs of heart failure and left ventricular systolic dysfunction with persistent troponin I elevation, followed by progressive worsening of ventricular dysfunction.

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PALAVRAS-CHAVE

Miocardopatia hipertrófica;
Troponina;
Insuficiência cardíaca

Um caso raro de elevação persistente da troponina em doente com insuficiência cardíaca crónica

Resumo Mulher com miocardopatia hipertrófica, que sofre enfarte do miocárdio aos 28 anos, sem doença coronária angiográfica. Dois anos mais tarde inicia manifestações de insuficiência cardíaca e instala-se disfunção sistólica ventricular esquerda com elevação persistente da troponina I cardíaca. A partir daí houve deterioração progressiva da função ventricular.

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Introduction

Troponin (Tn) is a laboratory marker of myocardial injury that has prognostic value in chronic heart failure (HF).^{1,2} Elevated troponin I (TnI) at a first HF consultation is associated with higher mortality, but the prognostic significance of persistent Tn elevation has yet to be fully determined.^{3,4} Assessment of Tn in advanced HF may be useful in risk stratification.^{1,5,6}

[☆] Please cite this article as: Cardoso G, Aguiar C, Ventosa A, et al. Um caso raro de elevação persistente da troponina em doente com insuficiência cardíaca crónica. Rev Port Cardiol. 2013;32:931-933.

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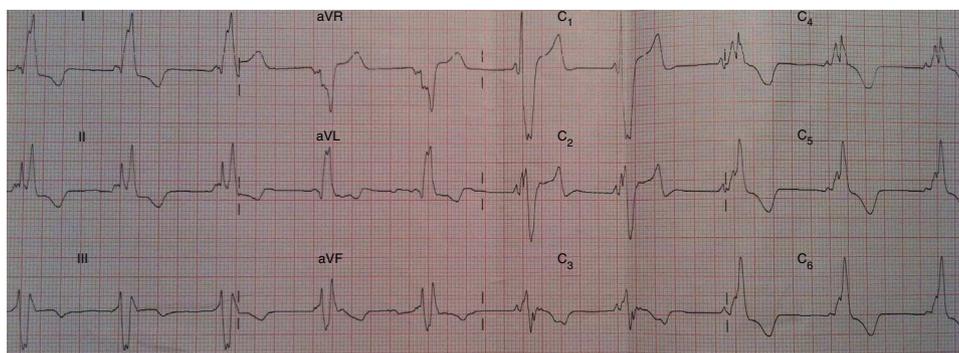


Figure 1 Electrocardiogram at first heart failure consultation.

Case report

A 35-year-old woman had been diagnosed with hypertrophic cardiomyopathy (HCM) at the age of six months. Her mother, with the same diagnosis and currently 52 years of age, underwent heart transplantation at age 44, and a maternal aunt suffered sudden death at age nine. There was no other relevant family history.

The patient was hospitalized at age 10 for lipothymia and was followed in adult cardiology consultations from age 16, when she was asymptomatic. Echocardiography revealed marked, asymmetric left ventricular (LV) wall hypertrophy with preserved LV ejection fraction (LVEF), right ventricular (RV) hypertrophy and mitral valve dysplasia.

She remained asymptomatic until the age of 28, when she was hospitalized (in November 2006) for non-ST-elevation myocardial infarction (peak Tnl 69 $\mu\text{g/l}$); coronary angiography excluded coronary artery disease. The pre-discharge echocardiogram showed mild LV dilatation, wall motion abnormalities of atypical distribution and LVEF of 55%. She was prescribed bisoprolol and ramipril and referred to the HF clinic.

Figure 1 shows the ECG at the first HF consultation. Genetic study identified mutations in the *TCAP* (c.C410T, p.Thr137Ile) and *MYH7* (c.C2167T, p.Arg723His) genes. Genetic study of her mother revealed three mutations, one in common with her daughter (*MYH7*).

A routine consultation in April 2009 for prognostic stratification showed LV systolic dysfunction (LVEF 42%) for the first time and Tn elevation (4.5 $\mu\text{g/l}$). Table 1 shows changes in levels of Tn and N-terminal pro-brain natriuretic peptide (NT-proBNP), with persistent Tn elevation in follow-up consultations.

The patient was asymptomatic until July 2009, when she presented NYHA class II HF. Due to progressively worsening symptoms, furosemide was begun in November 2009 and HF therapy was optimized. Echocardiography in the same month revealed worsening LV systolic function (LVEF <35%) compared to the previous echocardiogram, together with a reduction in dP/dt and increases in mitral regurgitation and in pulmonary artery systolic pressure (48 mmHg). Phenotypic alterations were also observed in the LV posterior and lateral walls, the endocardial border showing small protrusions similar to LV noncompaction. Cardiac magnetic resonance imaging revealed thrombi in the RV apex and left atrial appendage. Irregularities in LV trabeculation were observed, although without criteria for myocardial noncompaction.

Oral anticoagulation with warfarin was begun for a target INR of 2.0–3.0.

An implantable cardioverter-defibrillator was implanted in May 2010 for primary prevention of sudden cardiac death, after exclusion of thrombi by transesophageal echocardiography.

The patient was stable until May 2011, when clinical worsening was observed. Echocardiography in August 2011 showed severe biventricular systolic dysfunction (LVEF 26%) and she was hospitalized in September 2011 due to electrical storm. She was subsequently discharged medicated with amiodarone.

In the course of evaluation for heart transplantation, pulmonary artery catheterization (PAC) was performed, which showed marked pulmonary hypertension (mean pulmonary artery pressure 50 mmHg and pulmonary vascular resistance [PVR] 10.4 Wood units, without significant reversibility with high-flow oxygen). Pulmonary ventilation/perfusion scintigraphy excluded pulmonary thromboembolism. Sildenafil was prescribed.

PAC was repeated after six months of sildenafil therapy, which showed PVR had fallen from 15.6 to 3.6 Wood units, with inhaled nitric oxide.

Table 1 Changes in levels of troponin I and N-terminal pro-brain natriuretic peptide.

Date	Tnl ($\mu\text{g/l}$)	NT-proBNP (pg/ml)
March 2009	0.71	1450
April 2009	4.52	1370
June 2009	5.44	1520
November 2009	4.47	2090
January 2010	8.11	1930
March 2010	9.18	1430
June 2010	5.47	1160
November 2010	6.3	1130
January 2011	5.65	1380
May 2011	7.27	1200
September 2011	2.21	2170
October 2011	5.43	2370
December 2011	4.77	1880
January 2012	4.96	2040

NT-proBNP: N-terminal pro-brain natriuretic peptide; Tnl: troponin I.

In April 2012, the patient was in NYHA class III, and underwent heart transplantation the following month, which was uneventful and she was discharged clinically stable.

Sildenafil was electively discontinued three months after transplantation. The patient's clinical course has been favorable, with no episodes of acute rejection or infection, and she is currently in NYHA class I.

Discussion and conclusions

The case presented illustrates a rare clinical course of HCM. The prevalence of end-stage HCM varies between 2.4% and 15% depending on the series⁷; 5% of HCM patients have two or more mutations^{8,9} and the presence of multiple mutations is associated with more severe disease.¹⁰

This case also highlights the adverse prognostic significance of persistent Tn elevation in chronic HF. Tn elevation may be due to various factors and is associated with worse prognosis. The case presented suggests that it may have been due to progressive deterioration of LV systolic function, given that the latter began at the same time as persistently elevated Tn was observed. Only 10.4% of HF patients have detectable Tn (>0.01 ng/ml),² although more marked elevation is seen in acute HF^{11–13} but only 6.2% of these patients have elevation of >0.1 ng/ml.¹⁴

Finally, the case illustrates the value of pulmonary vasodilatory therapy as a bridge to heart transplantation in selected patients with HF and increased pulmonary vascular resistance.¹⁵

Ethical disclosures

Protection of human and animal subjects. The authors declare that no experiments were performed on humans or animals for this study.

Confidentiality of data. The authors declare that they have followed the protocols of their work center on the publication of patient data and that all the patients included in the study received sufficient information and gave their written informed consent to participate in the study.

Right to privacy and informed consent. The authors have obtained the written informed consent of the patients or subjects mentioned in the article. The corresponding author is in possession of this document.

Conflicts of interest

The authors have no conflicts of interest to declare.

Acknowledgements

The authors thank Dr. Isabel Gaspar, consultant in cardiac genetics at Centro Hospitalar Lisboa Ocidental, for

her invaluable contribution to the genetic study in this case.

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