Rev Port Cardiol. 2014;33(3):175.e1–175.e4

Revista Portuguesa de Cardiologia
Portuguese Journal of Cardiology
www.revportcardiol.org

CASE REPORT

Stress-induced cardiomyopathy associated with ipratropium bromide therapy in a patient with chronic obstructive pulmonary disease

Filipa Melão[a,*], José P. L. Nunes[a,b], Mariana Vasconcelos[a], Paula Dias[a], Pedro B. Almeida[a], Rui Rodrigues[a], Teresa Pinho[a], António Madureira[c], Maria J. Maciel[a,b]

[a] *Serviço de Cardiologia, Centro Hospitalar São João, Porto, Portugal*
[b] *Faculdade de Medicina da Universidade do Porto, Portugal*
[c] *Serviço de Radiologia, Centro Hospitalar São João, Porto, Portugal*

Received 24 April 2013; accepted 29 October 2013
Available online 27 March 2014

KEYWORDS
Stress cardiomyopathy;
Takotsubo cardiomyopathy;
Ipratropium bromide

Abstract Stress-induced cardiomyopathy, also known as 'broken heart syndrome' or Takotsubo cardiomyopathy, is characterized by transient systolic dysfunction of the apical and/or mid segments of the left ventricle, in the absence of significant coronary artery disease. We report the case of a 56-year-old male patient with chronic obstructive pulmonary disease (COPD), with stress-induced cardiomyopathy associated with the use of ipratropium bromide, administered in the context of an acute exacerbation of COPD.

PALAVRAS-CHAVE
Cardiomiopatia de *stress*;
Cardiomiopatia de Takotsubo;
Brometo de ipratrópio

Cardiomiopatia associada ao *stress* após terapêutica com brometo de ipratrópio em doente com doença pulmonar obstrutiva crónica

Resumo A cardiomiopatia associada ao *stress*, também conhecida por cardiomiopatia de Takotsubo, é caracterizada por uma disfunção sistólica transitória dos segmentos apicais e/ou médios do ventrículo esquerdo, na ausência de doença coronária significativa. Apresentamos um caso clínico de um doente do sexo masculino, com 56 anos de idade, com doença pulmonar obstrutiva crónica (DPOC), com cardiomiopatia associada ao *stress* que surgiu após uso de brometo de ipratrópio, administrado no contexto de uma exacerbação de DPOC.

* Corresponding author.
E-mail address: filipamelao@gmail.com (F. Melão).

Introduction

Stress-induced cardiomyopathy, also known as 'broken heart syndrome' or Takotsubo cardiomyopathy (TC), is characterized by transient systolic dysfunction of the apical and/or mid segments of the left ventricle that mimics myocardial infarction, in the absence of significant obstructive coronary artery disease.[1] There is often a history of recent emotional or physical stress or severe acute illness. Stress cardiomyopathy has been described in patients with chronic obstructive pulmonary disease (COPD).[2–7]

Case report

A 56-year-old Caucasian patient with COPD (irregularly treated with tiotropium bromide, acetylcysteine and mometasone) was admitted to the emergency room (ER) of our hospital with dyspnea and cough with sputum for two weeks. On admission to the ER, the patient was hemodynamically stable (systolic/diastolic blood pressure: 126/65 mmHg; heart rate: 98 bpm), eupneic at rest and with fever (39.1 °C). Pulmonary auscultation revealed rumbles in the left hemithorax. The chest radiograph showed left paratracheal consolidation. There was no evidence of respiratory failure on arterial blood gas analysis. Laboratory tests revealed elevated leukocytes. After treatment with inhaled ipratropium bromide, the patient suffered severe bronchospasm and retrosternal chest pain accompanied by sweating and pallor. An electrocardiogram (Figure 1A), with pain, showed sinus tachycardia (heart rate 110 bpm), incomplete right bundle branch block, 2-mm ST-segment elevation in leads V1–V3 and Q waves in leads V3–V6. Given the possibility of an acute coronary syndrome, emergency coronary angiography was carried out, which showed normal coronary arteries (Figure 2A and B), severe systolic dysfunction with mid-apical akinesia and basal hypercontraction (Figure 2C). The echocardiogram confirmed severe compromise of left ventricular systolic function (LVSF), with akinesia of the mid-apical segments and an aneurysm-like dilatation (Figure 3). During hospitalization the patient remained hemodynamically and electrically stable, but the electrocardiographic pattern evolved with T-wave inversion in the left anterior precordial leads (Figure 1B) and plasma troponin I was elevated (peak value 2.28 ng/ml). After further use of ipratropium bromide, on the second day of hospitalization, the patient developed a new episode of marked bronchospasm and respiratory acidosis (pH 7.29; pCO_2 53 mmol/l). He was started on non-invasive ventilation and was treated with hydrocortisone, inhaled salbutamol and furosemide, with progressive clinical improvement. He was also started on antibiotics (azithromycin plus ceftriaxone), but microbiological screening (bacteriological study of sputum and search for urinary antigens of *Streptococcus pneumoniae*

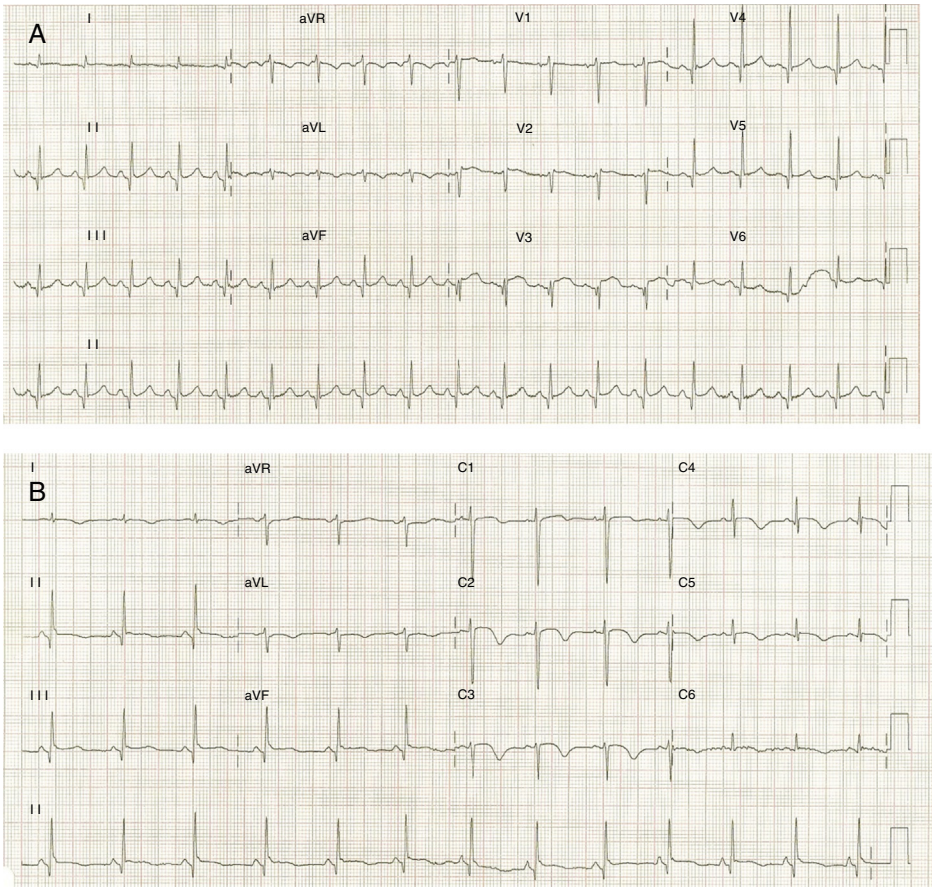

Figure 1 (A) ECG on admission showing ST-segment elevation in precordial leads; (B) ECG on the third day of hospitalization demonstrating T-wave inversion in precordial leads.