Rev Port Cardiol. 2014;33(10):659

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

LETTER TO THE EDITOR

# Takotsubo cardiomyopathy and chronic obstructive pulmonary disease

## Miocardiopatia Takotsubo e doença pulmonar obstrutiva crónica

We read with great interest the recently published case report by Melão et al. (March 2014 issue of the *Portuguese Journal of Cardiology*) of a 56-year-old male with chronic obstructive pulmonary disease (COPD) who developed stress-induced cardiomyopathy associated with ipratropium bromide use, administered in the context of an acute exacerbation of COPD.

Although this timely and interesting case report provides important information, we would like to share some thoughts.

Acute COPD exacerbation and acute respiratory failure (hypoxemic and/or hypercapnic) have been described[1,2] as potential physical triggers of takotsubo cardiomyopathy (TCM). In a study by Sharkey et al.,[1] acute respiratory failure and COPD exacerbation were among the most prevalent physical triggers.

Hypoxemia and hypercapnia can induce sympathetic nerve stimulation; the effect of hypoxemia alone can produce a longer-lasting sympathetic activation than hypercapnia, however if both occur simultaneously the result may be a significant increase in sympathetic activity with concomitant increase in catecholamine release,[3–5] which has been widely implicated in the etiology of TCM.

Acidosis has been associated with high levels of catecholamine release and may have negative inotropic effects secondary to decreasing calcium release from the sarcoplasmic reticulum. Acidosis-induced cardiomyocyte injury has been reported.[6–8] Inhaled beta 2 agonists used in acute asthma and COPD exacerbation have also been implied as potential triggers of TCM.[9]

Rarely, paradoxical bronchospasm may occur in the setting of inhaled ipratropium; however, this should be carefully distinguished from inadequate response and from transient nocturnal desaturation in COPD.[10]

This patient had hypercapnia and respiratory acidosis and used inhaled salbutamol. We wonder why COPD exacerbation itself (especially in the context of a left paratracheal consolidation suggesting community-acquired pneumonia, which could potentially add hypoxia to this clinical scenario) was not considered the most likely trigger of TCM instead of a rare paradoxical reaction to an anticholinergic drug.

## Conflicts of interest

The authors have no conflicts of interest to declare.

## References

1. Sharkey SW, Windenburg DC, Lesser JR, et al. Natural history and expansive clinical profile of stress (tako tsubo) cardiomyopathy. J Am Coll Cardiol. 2010;55:333–41.
2. Madhavan M, Rihal CS, Lerman A, et al. Acute heart failure in apical ballooning syndrome (TakoTsubo/stress cardiomyopathy): clinical correlates and Mayo Clinic risk score. J Am Coll Cardiol. 2011;57:1400–1.
3. Xie A, Skatrud JB, Puleo DS, et al. Exposure to hypoxia produces long-lasting sympathetic activation in humans. J Appl Phys. 2001;91:1555–62.
4. Citro R, Patella MM, Bossone E, et al. Near-drowning syndrome: a possible trigger of tako-tsubo cardiomyopathy. J Cardiovasc Med. 2008;9:501–5.
5. Morgan BJ, Crabtree DC, Palta M, et al. Combined hypoxia and hypercapnia evokes long-lasting sympathetic activation in humans. J Appl Phys. 1995;79:205–13.
6. Nanda S, Longo S, Bhatt SP, et al. Stress cardiomyopathy – a unique presentation of diabetic ketoacidosis. Annal Clin Biochem. 2009;46 Pt 3:257–60.
7. Goldsmith SR, Iber C, McArthur CD, et al. Influence of acid-base status on plasma catecholamines during exercise in normal humans. Am J Physiol. 1990;258:R1411–6.
8. Thatte HS, Rhee JH, Zagarins SE, et al. Acidosis-induced apoptosis in human and porcine heart. Ann Thorac Surg. 2004;77:1376–83.
9. Salahuddin FF, Sloane P, Buescher P, et al. A case of apical ballooning syndrome in a male with status asthmaticus; highlighting the role of B2 agonists in the pathophysiology of a reversible cardiomyopathy. J Community Hosp Intern Med Perspect. 2013;3.
10. http://www.uptodate.com/contents/anticholinergic-agents-in-the-management-of-acute-exacerbations-of-asthma

André Dias[a,b,*], Emiliana Franco[a,b]

[a] *Danbury Hospital, Internal Medicine Department, Danbury, CT, United States*
[b] *Einstein Medical Center, Department of Cardiology, and Jefferson Medical College, Philadelphia, United States*

* Corresponding author.
*E-mail address:* andremacdias@gmail.com (A. Dias).