Postpartum Inverted Takotsubo Cardiomyopathy After Intravenous Atropine Administration
Rienzi Diaz-Navarro,1,* and Petros Nihoyannopoulos2

1Professor of Cardiology, Departamento de Medicina Interna y Centro de Investigaciones Biomedicas, Escuela de Medicina, Universidad de Valparaiso, Chile
2Professor of Cardiology, Department of Cardiovascular Sciences, Hammersmith Hospital, Imperial College London, NHLI, Hammersmith Hospital, London W12 1NN, UK

*Corresponding author: Rienzi Diaz-Navarro, 4 Poniente 332, PO Box 252092, Vina del Mar, Chile. Tel: +56-322603071; +56-993427749, E-mail: diaz.rienzi@gmail.com

Abstract
Postpartum Takotsubo cardiomyopathy is mainly induced by drugs that enhance sympathetic nervous activity. We report a novel case of postpartum inverted Takotsubo cardiomyopathy triggered by intravenous atropine administration resulting in acute pulmonary edema. Cardiac troponin I and beta-type natriuretic peptide were elevated. Transthoracic color Doppler echocardiography demonstrated a nondilated left ventricle with mid-basal akinesis, a hyperdynamic apex, and moderate-to-severe mitral regurgitation likely linked to papillary muscle dysfunction. Coronary computed tomography angiography revealed normal coronary arteries. Atropine inhibits the parasympathetic nervous system, alters the autonomic system balance, and, thus, leads to increased sympathetic nervous activity, which seems to have been the cause of Takotsubo cardiomyopathy in this patient. Atropine should be listed among the drugs triggering Takotsubo cardiomyopathy.

Keywords: Takotsubo Cardiomyopathy, Cardiomyopathies, Echocardiography Doppler Color, Atropine

1. Introduction
Takotsubo cardiomyopathy (TCM) is an acute cardiac disease that most commonly presents with acute chest pain and/or dyspnea along with ECG abnormalities, increased levels of cardiac enzymes, and transient left ventricular dysfunction mimicking an acute myocardial infarction (1, 2) in the absence of epicardial coronary artery disease (3). Although the pathophysiological mechanism is still controversial, catecholaminergic stress appears to be the single causal pathway (4).

TCM is seen predominantly in postmenopausal women preceded by extreme emotional and/or physical stress (5). However, it can also occur in premenopausal women, mainly after Cesarean delivery. This is typically a stressful procedure that is commonly associated with the administration of drugs that can amplify sympathetic activity (6).

Here, we report a case of postpartum inverted TCM presenting with acute pulmonary edema after intravenous atropine administration.

2. Case Presentation
A 28-year-old primiparous woman was admitted for a scheduled Caesarean delivery at 36 weeks of gestation after an uncomplicated pregnancy. She gave birth to a healthy baby while under epidural anesthesia. There was no history of cardiac problems or risk factors. Fifteen minutes after her admission to the recovery room, 0.5 mg of atropine was administered intravenously to reverse asymptomatic bradycardia of 41 beats per minute, which was an indication that might be considered unnecessary in hindsight. Almost immediately, she complained of chest pain and progressive dyspnea. Her oxygen saturation dropped to 74%, and her blood pressure was 119/73 mm Hg. A chest X-ray revealed pulmonary alveolar/interstitial congestion, consistent with acute pulmonary edema (APO) (Figure 1A), and an ECG showed sinus rhythm with peaked symmetrical T waves, mostly in the precordial leads (Figure 1B). Transthoracic color Doppler echocardiography (TTDE) was used to demonstrate parasternal long-axis views of the left ventricle (LV) in diastole (Figure 1C) and systole (Figure 1D). The TTDE revealed a nondilated LV with basal akinesis that extended to the middle portion (yellow arrows, Figure 1D; Video 1 in Supplementary Materials), where the papillary muscle was located with a hyperdynamic apex, as well as an ejection fraction of 55% and moderate-to-severe mitral regurgitation (MR) (Figure 1E, white arrowheads; Video 2 in Supplementary Materials). The cardiac troponin I level reached 2.03 ng/mL (normal < 0.03 ng/mL), and the beta-type natriuretic peptide level reached 520.9 pg/mL (nor-
mal < 132 ng/mL). Coronary computed tomography angiography revealed normal epicardial coronary arteries. She responded well to intravenous furosemide and oxygen therapy with rapid clinical improvement. Additional treatment included therapy with bisoprolol, aspirin, and captopril. She was discharged home on day 7, free of symptoms. At a 9-month follow-up, she was asymptomatic with a normal TTDE (Figure 1F; Video 3 in Supplementary Materials).

Figure 1. The Case

3. Discussion

TCM is an acute syndrome characterized by transient LV dysfunction, presumably caused by sympathetic overstimulation. The most common variant of TCM is marked by the apical ballooning of the LV with basal hyperkinesis (5). We report what we think is the 1st case of inverted TCM induced by the administration of atropine in a woman after a Caesarean delivery. Most commonly, postpartum TCM is induced by the administration of drugs that enhance sympathetic nervous activity (6).

However, atropine as an anticholinergic agent can aggravate the signs and symptoms in cases of TCM because of the inhibition of the parasympathetic nervous system, leading to increased sympathetic activity, as was reported recently in a patient with TCM who received intravenous atropine to reverse symptomatic bradycardia (7). Our report to some extent reaffirms this finding because the intravenous injection of atropine was almost immediately followed by the onset of chest pain and dyspnea, which culminated in APO. An ultrasound evaluation performed immediately after the symptom started depicted an uncommon type of TCM known as inverted TCM, (5, 6, 8, 9) in which there was akinesis of the mid-basal segments of the LV with a hyperdynamic apex [Video 1 in Supplementary Materials]. The clear temporal link between atropine administration and the beginning of symptoms suggests that surgery itself cannot have precipitated this acute heart condition, and nor could it have been related to the noncardiac etiologies of pulmonary edema seen in the postpartum period such as amniotic fluid or air embolism, aspiration of gastric contents, or sepsis. The APO was most likely the result of the presence of significant MR (Figure 1F; Video 3 in Supplementary Materials) because the global LV function was preserved (ejection fraction = 55%). The most common causal mechanism of MR in TCM is the presence of the systolic anterior motion of the mitral valve, occurring in approximately one-third of patients (10), a feature not seen in this patient. MR in this case was most likely linked to the transient wall motion abnormalities of the LV with papillary muscle dysfunction caused by catecholamine-induced myocardial injury resulting in myocardial stunning (11).

3.1. Conclusions

This case illustrates that postpartum TCM can be induced by the use of intravenous atropine, a drug that should be listed among those triggering this acute form of cardiomyopathy. Therefore, atropine should be used cautiously in the postpartum period.

Supplementary Material

Supplementary material(s) is available here [To read supplementary materials, please refer to the journal website and open PDF/HTML].

Footnotes

Authors’ Contribution: Rienzi Diaz-Navarro: concept/design, data analysis/interpretation, drafting article, critical revision of article, approval of article, data collection. Petros Nihoyannopoulos: data analysis/interpretation, critical revision of article, approval of article.

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References


