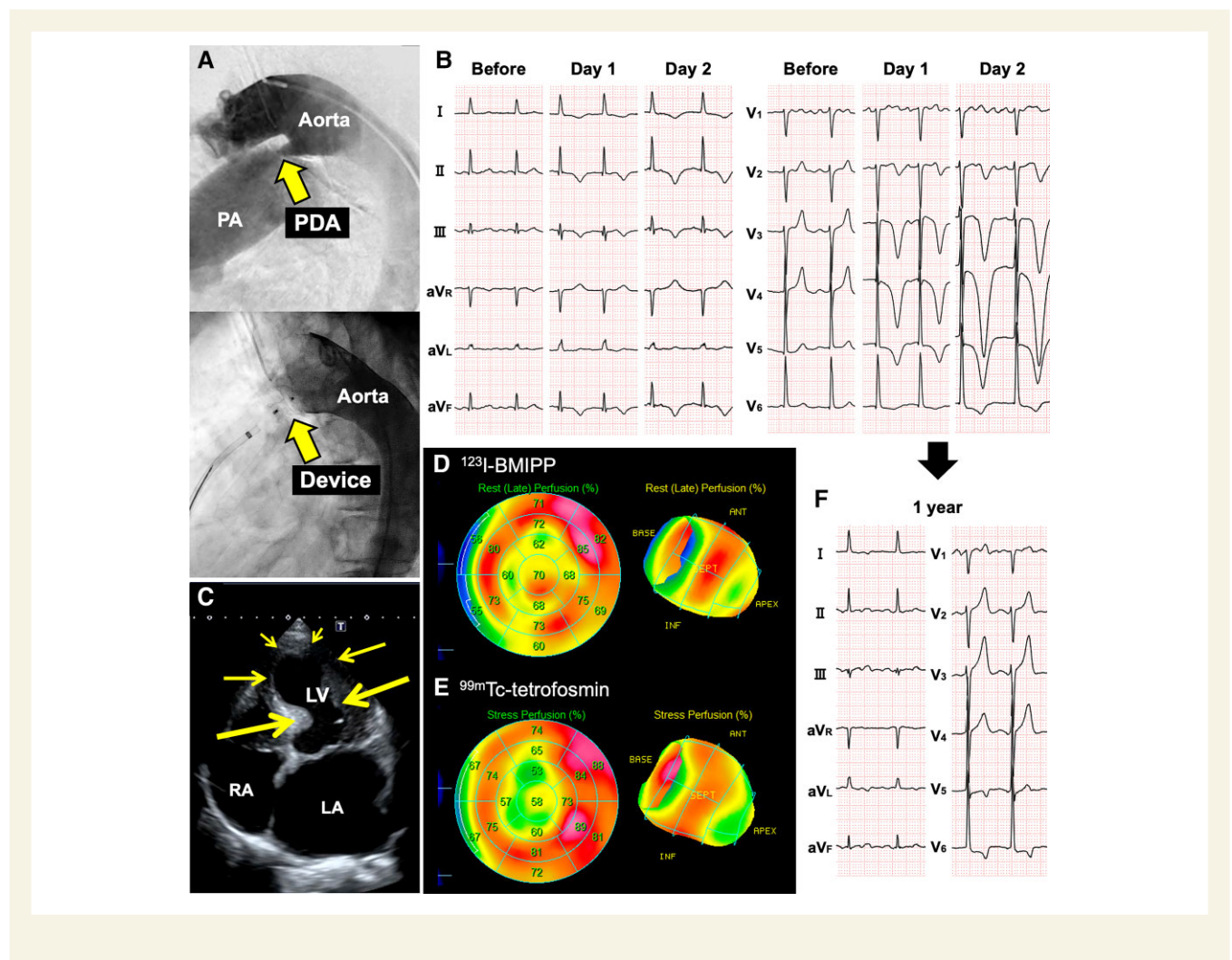


Takotsubo syndrome following patent ductus arteriosus device closure

Yusuke Akazawa ^{1,2,3}, Takashi Higaki ^{2,3*}, Haruhiko Higashi ¹, and Osamu Yamaguchi ¹

¹Department of Cardiology, Pulmonology, Hypertension and Nephrology, Ehime University Graduate School of Medicine, Toon, Ehime, 791-0295, Japan; ²Department of Regional Pediatrics and Perinatology, Ehime University Graduate School of Medicine, Toon, Ehime, 791-0295, Japan; and ³Department of Pediatrics, Ehime University Graduate School of Medicine, Toon, Ehime, 791-0295, Japan

Received 17 February 2022; first decision 28 February 2022; accepted 24 May 2022; online publish-ahead-of-print 10 June 2022



* Corresponding author. Tel: +81 89 960 5068, Fax: +81 89 960 5071, Email: higaki@m.ehime-u.ac.jp

Handling Editor: Pierpaolo Pellicori

© The Author(s) 2022. Published by Oxford University Press on behalf of the European Society of Cardiology.

This is an Open Access article distributed under the terms of the Creative Commons Attribution-NonCommercial License (<https://creativecommons.org/licenses/by-nc/4.0/>), which permits non-commercial re-use, distribution, and reproduction in any medium, provided the original work is properly cited. For commercial re-use, please contact journals.permissions@oup.com

An 84-year-old woman with chest discomfort on exertion was referred to our hospital for patent ductus arteriosus (PDA) device closure. She had a history of atrial fibrillation and transthoracic echocardiography (TTE) showed left ventricular (LV) enlargement with evidence of LV volume overload. Based on these findings, although the patient was elderly woman, we performed closure for PDA. Cardiac catheterization revealed a PDA defect measuring 4.4 mm in diameter at the pulmonary end, with a pulmonary-to-systemic blood flow ratio of 2.1. The PDA closure was successfully performed with a 12/10 mm-Amplatzer Duct Occluder (St. Jude Medical, St. Paul, MN, USA) (Panel A). On the day after the procedure, the 12-lead electrocardiogram showed a giant negative T wave in I–III, aVF, V2–5 (Panel B). Although her creatinine kinase level was within the normal range (<226 U/L), her high-sensitivity serum troponin I level was elevated at 311.2 pg/mL (normal range, <26.2 pg/mL). Transthoracic echocardiography revealed LV apical ballooning with hypercontractile basal segments (Panel C, yellow arrows, [Video 1](#)). Preprocedural cardiac computed tomography (CT) angiography showed no significant atherosclerotic plaque with zero-calcium score. Based on these findings, Takotsubo syndrome was suspected rather than acute coronary syndrome.¹²³I-β-methyl-p-iodo phenyl-pentadecanoic acid (¹²³I-BMIPP) (Panel D) and ^{99m}Tc-tetrofo-

sm dual myocardial single-photon emission CT (Panel E) confirmed the diagnosis of Takotsubo syndrome, with a decrease in both fatty acid metabolism and perfusion at the LV apex. In addition to the previously administered beta-blocker for rate control in atrial fibrillation, the patient was treated with angiotensin-converting enzyme inhibitor expecting LV recovery. She did not receive any diuretics during the perioperative period. Within 1 week, her LV function improved significantly, and electrocardiography revealed the gradual resolution of the negative T wave, which normalized after 1 year (Panel F). She was stable and showed no symptoms of heart failure at the 2-year follow-up.

It is known that PDA device closure produces marked hemodynamic changes which is decreasing LV preload and increasing LV afterload immediately after the procedure. The haemodynamic LV stress might have triggered the Takotsubo syndrome.

Consent: The authors confirm that written consent for submission and publication of this case report including images and associated text has been obtained from the patient in line with COPE guidance.

Conflict of interest: None declared.

Funding: None declared.