

Case Report

Report of Takotsubo Cardiomyopathy Occurring During Cardiopulmonary Bypass

Hideshi Itoh, MS, MPhil, CCP; Yoichiro Miyake, MD, PhD; Iwao Hioki, MD, PhD; Satofumi Tanaka, MD; Manabu Okabe, MD, PhD

Department of Cardiovascular and Thoracic Surgery, Kochi Health Sciences Center, Ike, Kochi, Japan

Abstract: On weaning from cardiopulmonary bypass, a 59-year-old Japanese woman with mitral valve plasty suddenly showed a greatly increased heart rate, and an electrocardiogram revealed elevated ST-segments. There was also abnormal wall motion in the inferior region and apical ballooning of the left ventricle. We diagnosed the condition as takotsubo cardiomyopathy (acute left ventricle apical ballooning syndrome), possibly caused by catecholamine release and regional stress-induced ischemia. We believe this to be the first case report of takotsubo cardiomyopathy observed during heart surgery. We hypothesize that the condi-

tion was mediated by regional myocardial stunning and that it could be prevented by administration of angiotensin converting enzyme inhibitors before surgery and by the use of superior biocompatible cardiopulmonary bypass components. Once takotsubo cardiomyopathy occurs, we recommend mechanical circulatory assistance during weaning from the bypass. **Keywords:** takotsubo cardiomyopathy, regional myocardial stunning, catecholamine, angiotensin converting enzyme inhibitors, intra-aortic balloon pump. *JECT 2007;39:109–111*

Takotsubo cardiomyopathy (acute left ventricular apical ballooning syndrome) was first described by Dote et al. in 1991 (1). The condition is characterized by transient regional systolic dysfunction involving the left ventricular apex and mid-ventricle with hypokinesis of the basal left segments and no significant angiographic stenosis in the coronary artery (2). It is seen mostly in elderly women, and emotional or physical stress may be a triggering factor (3). In this paper, we hypothesize what caused the takotsubo cardiomyopathy we observed during open heart surgery, and we suggest a way to prevent and treat it. We believe this is the first case report of takotsubo cardiomyopathy occurring during cardiopulmonary bypass (CPB) surgery.

DESCRIPTION

A 59-year-old Japanese woman with a 10-year history of

mitral regurgitation came to us for treatment. Her height was 153 cm, her weight was 43 kg, and her body surface area 1.36 m². The CPB we used consisted of a hollow fiber membrane oxygenator (Oxia; JMS, Tokyo, Japan), open hard-shell reservoir (Oxia; JMS), arterial filter (LH-40AH; JMS), and roller pump for perfusion (Sarns 8000; Terumo, Tokyo, Japan). All components of CPB circuits were heparin-coated (COAFREE; JMS). The CPB circuit was primed with 500 mL acetate Ringer solution, 300 mL D-mannitol, 500 mL hydroxyethylated starch, 60 mL sodium bicarbonate, 1000 mg methylprednisolone sodium succinate, and 1 g flomoxef sodium to a total prime volume of 1360 mL. We used moderate hypothermic perfusion at 28°C and set the bypass flow rate at 2.4 L/min/m² to maintain venous saturation at >70%. We maintained perfusion pressure at 60 mmHg. Normal systemic vascular resistance was maintained by the addition of phenylephrine hydrochloride, chlorpromazine, and nicardipine hydrochloride. Blood gasses were regulated according to the alpha-stat regimen, and sodium bicarbonate was administered when the base excess dropped below -3.0 mmol/L. We used a dilutional ultrafiltration technique with a polyethersulfone membrane (AquastreamAS11; JMS) during CPB, and bispectral index monitoring (ASPECT; Aspect

Address correspondence to: Hideshi Itoh, MS, MPhil, CCP, Department of Cardiovascular and Thoracic Surgery, Cardiovascular Perfusion Division, Kochi Health Sciences Center, 2125-1, Ike, Kochi 781-0555, Japan. E-mail: Hideshi@perfusion.net

The senior author has stated that authors have reported no material, financial or other relationship with any healthcare-related business or other entity whose products or services are discussed in this paper.

Medical Systems, Boston, MA) was maintained at 40–60. Anticoagulation was achieved with an initial bolus of 400 IU/kg heparin sodium. Nafamostat mesylate was administered at 25 mg/h until the end of CPB, and protamine sulfate was administered at 3.5 mg/kg. Intermittent cold blood cardioplegia (20°C), which consists of CPB blood with 19 mmol/L potassium (miniplegia), was administered in antegrade and retrograde fashion every 20 minutes to induce arrest. We administered terminal warm blood cardioplegia (35°C) before clamping off the aorta, and dopamine HCl and dobutamine HCl, 3 µg/kg/min each, after clamping. Total CPB time was 242 minutes, arrest time was 62 minutes, and partial bypass for assisting circulation time was 101 minutes.

The heartbeat recovered spontaneously when the aorta clamp was removed, but suddenly, the ST-segment in the inferior region became elevated, and the heart rate jumped from 80 to 140 beats/min. We increased the dopamine HCl and dobutamine HCl to 7 µg/kg/min. A transthoracic echocardiogram revealed abnormal wall motion in the apex and hyperkinesis in the mid-basal left ventricle (Figure 1). A 12-lead electrocardiogram (ECG) showed ST-segment elevation in the inferior region (Figure 2). Suspecting that incomplete coronary air removal caused the abnormal wall motion, we continued circulatory assistance with left ventricular venting to remove the air. Transesophageal echocardiography, however, showed that no air was present (we had been pouring CO₂ on the surgical view during the procedure to reduce the air in the coronary artery and the heart). Hence, we diagnosed the abnormal wall motion as takotsubo cardiomyopathy. We inserted an 8-Fr 34-mL Fidelity intra-aortic balloon pump (IABP; Datascope 98; Edwards Lifesciences, Irvine, CA) and decreased the dopamine and dobutamine doses back

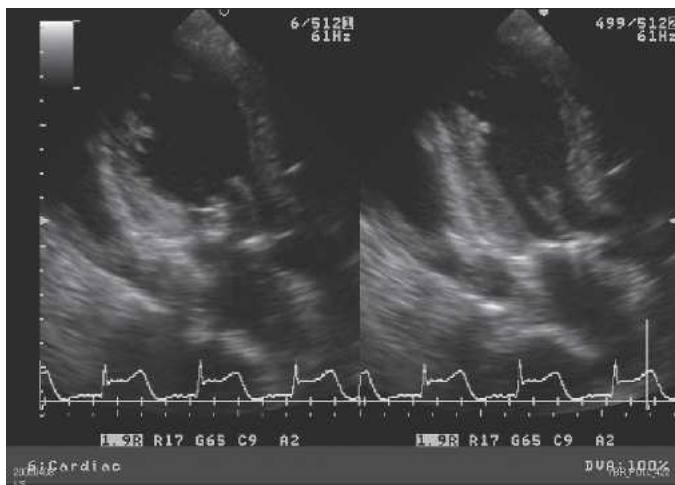


Figure 1. Post-surgical transthoracic echocardiogram showing features of takotsubo cardiomyopathy: ST elevation and abnormal wall motion in the apex of the left ventricle, akinesis of the apex and the mid-ventricle, and basal hyperkinesis.

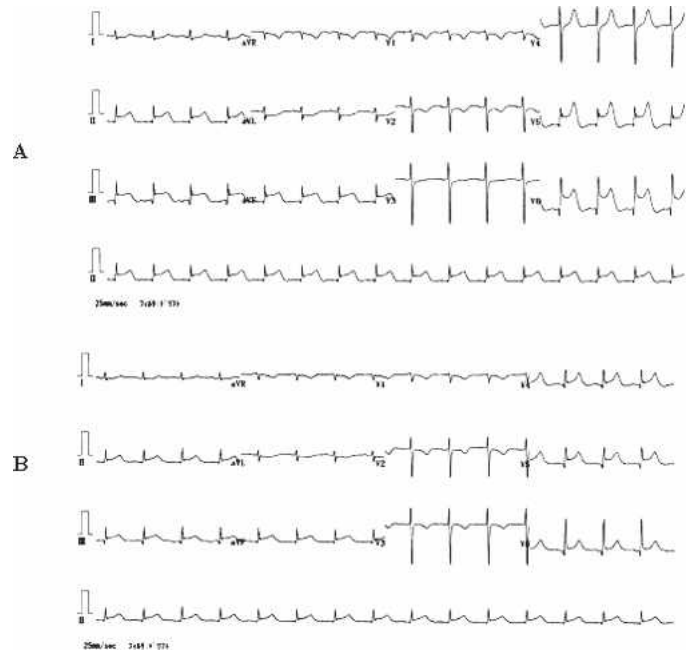


Figure 2. A, Twelve-lead ECG showing ST-segment elevation in lead II, III, and aVf immediately after surgery. B, Twelve-lead ECG showing ST-segment normalization 12 hours after surgery.

to 3 µg/kg/min. We were able to wean the patient from the bypass, and 18 hours later, we were able to wean her from the IABP.

COMMENT

Takotsubo cardiomyopathy is characterized by the acute onset of reversible left ventricle apical wall motion abnormalities (ballooning) with chest symptoms, ST elevation, minimal myocardial enzymatic release, and no significant stenosis evident in coronary angiography. We believe that this is the first report of takotsubo cardiomyopathy occurring during CPB. We hypothesize that the condition was mediated by regional myocardial stunning. Sato et al. (4) reported a case of takotsubo cardiomyopathy caused by microscopic polyangiitis, an active manifestation of the systemic inflammatory response syndrome. Microscopic polyangiitis is characterized by small-vessel vasculitis (5), and systemic inflammatory response syndrome can follow CPB (6). The exact mechanism that brings on takotsubo cardiomyopathy is unknown (2–4), but the combination of myocardial ischemia related to diffuse microvascular dysfunction, multivessel epicardial spasm, and metabolic injury may play a crucial role (6–8). Excessive catecholamine release stimulates the β-adrenergic receptors and depresses ventricular contraction (8). Ruck et al. (9) showed that, in stable angina pectoris patients, brain natriuretic peptide is over-expressed regionally in the myocardium under stress-induced ischemia. In the case we report here, local myocardial stunning caused

by microvascular dysfunction and excessive catecholamine release may have mediated the cardiomyopathy.

On weaning the patient from CPB, we reduced the inotropic dose—which we believe contributed to her recovery from the cardiomyopathy—and used IABP (2,3,5). Her pre-surgery serum brain natriuretic peptide level was 477 pg/mL. Because treatment with A angiotensin converting enzyme inhibitors before surgery reduces myocardial stress (7,10), it might also prevent takotsubo cardiomyopathy. As described in our recent review (6), the use of CPB components with superior biocompatibility can prevent microvascular dysfunction mediated by systemic inflammatory response syndrome; therefore, it might also prevent takotsubo cardiomyopathy. When takotsubo cardiomyopathy occurs during open heart surgery, we believe that a mechanical support such as IABP will help counter the excessive catecholamine.

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