Journal of Artificial Organs (2019) 22:330–333
https://doi.org/10.1007/s10047-019-01112-8

CASE REPORT

Artificial Heart (Clinical)

A case of Takotsubo cardiomyopathy with cardiogenic shock after influenza infection successfully recovered by IMPELLA support

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Received: 1 May 2019 / Accepted: 15 June 2019 / Published online: 21 June 2019

Abstract
We recently experienced a case of cardiogenic shock due to influenza-related Takotsubo cardiomyopathy with atrial tachycardia and respiratory distress syndrome. Temporary mechanical circulatory support by IMPELLA 2.5 improved end-organ failure and resulted in cardiac recovery with sinus rhythm conversion.

Keywords Ventricular assist device · Supraventricular tachycardia · Stress cardiomyopathy · Mechanical circulatory support

Introduction

Takotusbo cardiomyopathy is characterized by transient systolic and diastolic left ventricular dysfunction with a variety of wall motion abnormalities and often preceded by emotional or physical trigger. In the acute phase of Takotsubo cardiomyopathy, patients often represent an acute heart-failure syndrome [1] and in-hospital mortality is about 4–5%, a figure comparable to that of ST elevation myocardial infarction [2]. Among the patients with Takotsubo cardiomyopathy, concentrations of plasma catecholamine and stress-related circulating neuropeptides were several times higher than those in patients with myocardial infarction and remained elevated even a week after the onset of symptoms [3]. Such catecholamine surge may lead to myocardial damage [4]. Here, we present a case of Takotsubo cardiomyopathy with left ventricular dysfunction and atrial tachycardia after influenza who had compromised hemodynamics despite intravenous inotropes and eventually required mechanical support (IMPELLA 2.5). After 4 days' support of IMPELLA, his left ventricular systolic function recovered to normal range with sinus rhythm conversion. Hemodynamic support and left ventricular unloading by IMPELLA were helpful for cardiac recovery of Takotsubo cardiomyopathy.

Case report

The patient was a Japanese 82-year-old gentleman who was admitted to our hospital for disturbance of consciousness with slight fever (37.4 °C). Although head MRI did not show typical findings of encephalitis, such as cortical high signal of the brain, he was suspected of encephalopathy due to influenza A infection, and was given peramivir hydrate (300 mg) and methylprednisolone pulse therapy (1 g daily for 3 days) on emergency department. On admission, he was hypertensive (220/125 mmHg) and EKG showed sinus tachycardia with ST changes in multiple leads (Fig. 1a). Cardiac troponin I was elevated, but cardiac enzymes were within normal limit. On the 2nd day, his heart rate (HR) was elevated to 160–180 bpm and 2 μg/kg/min of landiolol was administered. On the 4th day, he became hypoxic, and chest X-ray showed bilateral pulmonary infiltration, and he was suspected of pneumonia and acute respiratory distress syndrome. On the 5th day, he became hypotensive (87/45 mmHg) with atrial tachycardia, and fluid replacement was administered. EKG showed inverted T wave in broad leads (Fig. 1b). Transthoracic echocardiography revealed

Electronic supplementary material The online version of this article (https://doi.org/10.1007/s10047-019-01112-8) contains supplementary material, which is available to authorized users.

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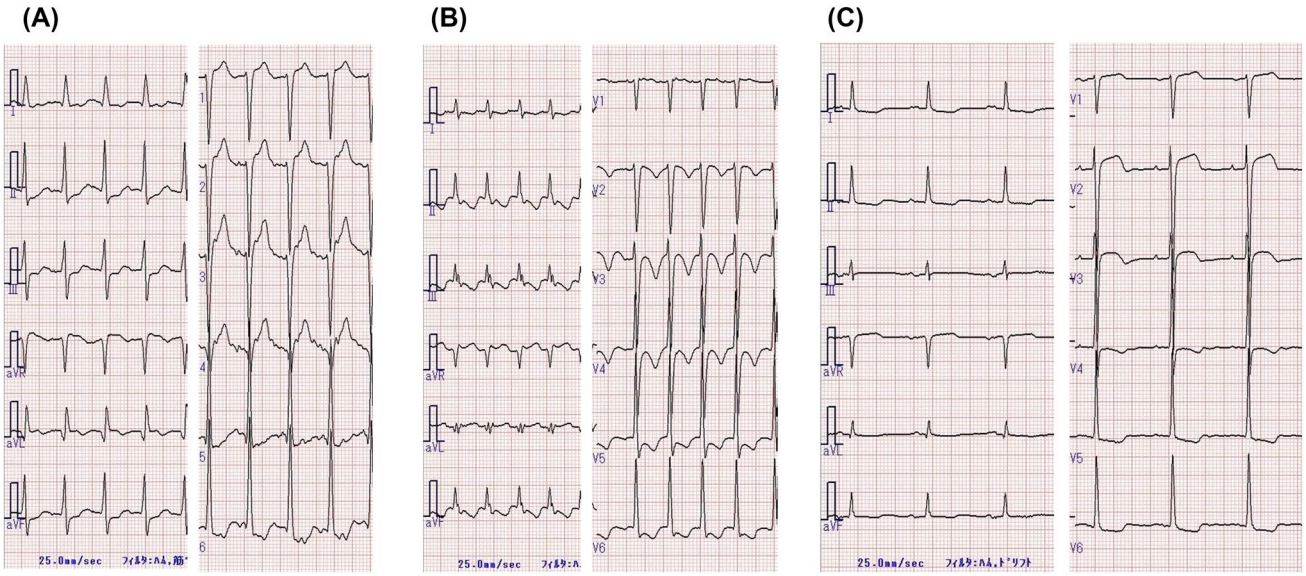

Fig. 1 Changes of electrocardiogram during the hospitalization. **a** Was on admission, **b** was on the 8th day, and **c** was on the 13th day

reduced left ventricular ejection fraction of 34% with severe hypokinesis in the antero-apical wall in contrast to hyperkinetic region in the base (Fig. 2a). No outflow obstruction or no significant valvulopathy was observed, and left ventricular diastolic dimension was 44 mm. BNP level was high as 634 pg/mL on the 6th day and intravenous dobutamine infusion of 2 μg/kg/min and furosemide 10–20 mg daily were administered. Intravenous landiolol was increased to 5 μg/kg/min for supraventricular tachycardia. On the 7th day, intravenous dobutamine was increased to 2.5 μg/kg/min and intravenous amiodarone infusion (150 mg per day) was also administered. On the 8th day, his consciousness improved and he was transferred from the department of neurology to our ward for intensive care of heart failure. Cardiac catheterization showed mean right atrial pressure of 8 mmHg, pulmonary artery pressure of 31/23, and mean pulmonary artery wedge pressure of 15 mmHg, respectively. Cardiac output (CO) and cardiac index (CI) were 3.18 L/min and 1.95 L/min/m^2, respectively, and mixed venous oxygen saturation (SvO_2) was low as 49.5%. Left

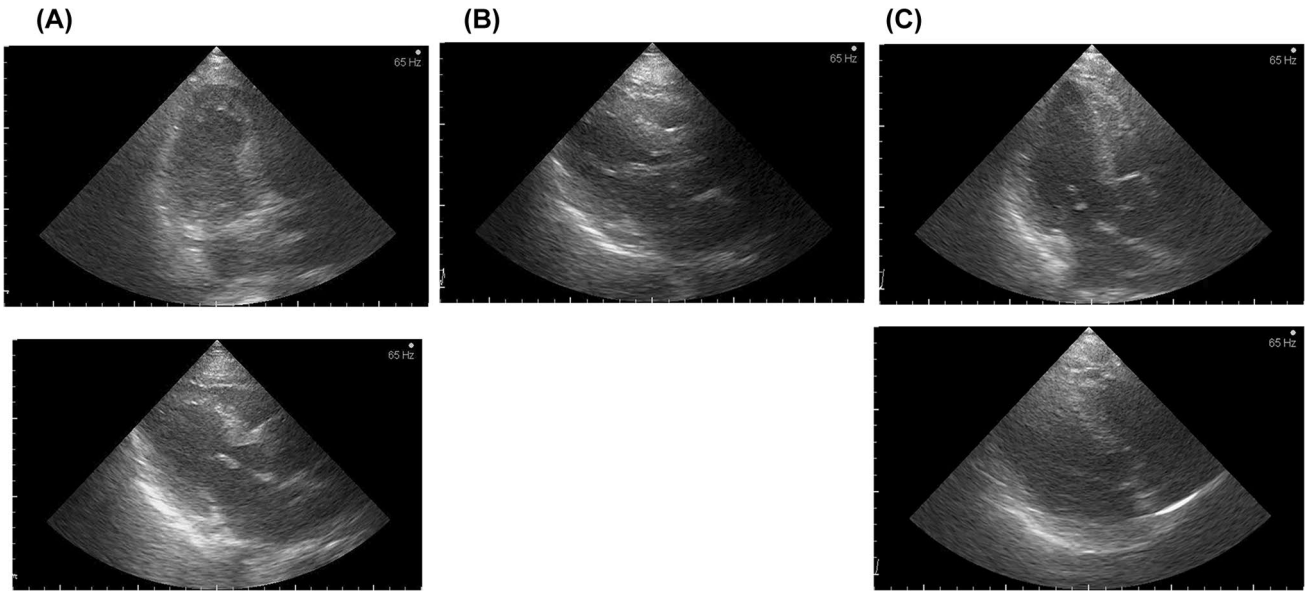

Fig. 2 Apical three-chamber view on transthoracic echocardiography. **a** Was on the 6th day, **b** was on the 8th day during IMPELLA support, and **c** was on the 29th day

ventricular end-diastolic pressure (LVEDP) was elevated to 23 mmHg and systolic blood pressure was 120–130 mmHg when HR was 160–170 bpm, but was low as 70–80 mmHg when HR reached to 190–200 bpm. Coronary angiogram showed no organic stenosis, and myocardial biopsy showed no inflammatory cell infiltration nor eosinophils or giant cells. Accordingly, we diagnosed him as influenza-related Takotsubo cardiomyopathy. In spite of intensive medical treatment, his hemodynamics was still compromised with elevated lactate level of 3.2 mmol/L. Atrial tachycardia did not resolve even by DC cardioversion. Then, we decided to insert IMPELLA 2.5 from his right femoral artery as a temporary mechanical circulatory support on the 8th day (Fig. 2b). After insertion of IMPELLA, pulmonary artery diastolic pressure was decreased to 6 mmHg, and CO and SvO_2 were increased to 4.0 L/min and 52%, respectively. Lung congestion and infiltrations were gradually improved. Left ventricle wall motion improvement was also found by echocardiography on the 11th day, and IMPELLA was eventually removed on the 12th day (Fig. 3). During IMPELLA support, there was no device-related adverse event. He spontaneously restored sinus rhythm on the 13th day (Fig. 1c) and left ventricular ejection fraction improved to 55% with no asynergy (Fig. 2c). He was discharged with BNP of 29.2 pg/mL on the 35th day.

Discussion

In this report, we described a case of 82-year-old Takotsubo cardiomyopathy after influenza infection. Though he was complicated with impaired consciousness, congestive heart failure, refractory tachyarrhythmia, and acute respiratory distress syndrome, IMPELLA support markedly improved his hemodynamics and he was rescued with complete cardiac recovery.

On admission, his EKG resembled to acute coronary syndrome with elevated cardiac troponin I, but we did not perform coronary angiogram considering his age and possible brain damage. Over the next couple of days, he became hypotensive and hypoxic with emergence of atrial tachycardia. He was initially suspected of influenza-related myocarditis, but myocardial biopsy revealed no signs of myocarditis. In consideration of echocardiographic findings, we then diagnosed as Takotsubo cardiomyopathy. Different emotional and psychological stressors have been identified to precede of the onset of Takotsubo syndrome. Acute stressors induce brain activation, increasing bioavailability of cortisol and catecholamine. This catecholamine surge leads direct catecholamine toxicity, adrenoceptor-mediated damage, epicardial and

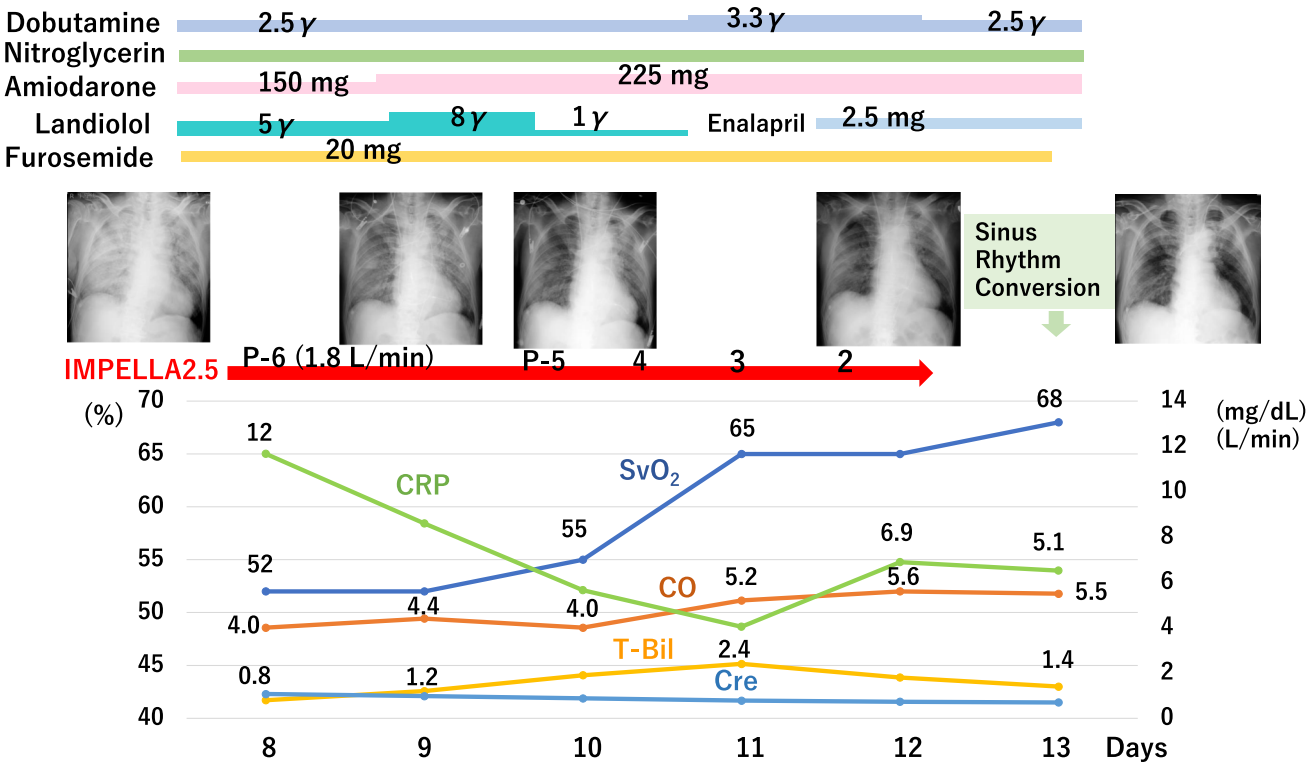

Fig. 3 Clinical course after IMPELLA 2.5 insertion. The dose of administered drugs was also shown. *CRP* C-reactive protein, *T-Bil* total bilirubin, *Cre* serum creatinine, *SvO_2* mixed venous oxygen saturation, *CO* cardiac output

microvascular coronary vasoconstriction and/or spasm, and increased cardiac workload, to myocardial damage [4]. In this case, the physical stress of influenza infection was highly attributable to the development of Takotsubo syndrome. The plasma concentration of noradrenaline was high as 1047 pg/mL (100–450 pg/mL) on the 8th day, and slightly decreased to 732 pg/mL on the 12th day. Myocardial involvement during influenza virus infection has been described in 10% of cases [5] and the cases of Takotsubo cardiomyopathy triggered by Influenza A or B have also been known [6, 6]. Once he was diagnosed as Takotsubo cardiomyopathy, we had to avoid increasing the dose of intravenous inotropes because of the nature of this disease. Therefore, we decided to use temporary mechanical support. IABP may be one of the options, but his heart rate was too fast to be followed by IABP. Considering his wet lung, V-A ECMO may not be a wise option either. On the other hand, IMPELLA ameliorates pulmonary congestion by reducing LV preload and increases cardiac output regardless of HR [8]. Therefore, IMPELLA was considered as the best choice in this setting. A case of cardiogenic shock secondary to Takotsubo cardiomyopathy rescued by IMPELLA 2.5 was previously reported [9]. Beneduce A. et al. also reported the usefulness of IMPELLA for Takotsubo syndrome complicated with cardiogenic shock and left ventricular outflow obstruction [10]. However, we believe that this is the first IMPELLA case of Takotsubo cardiomyopathy in a Japanese patient.

In summary, an 82-year-old patient with acute heart failure due to influenza-related Takotsubo cardiomyopathy was safely treated with IMPELLA 2.5 and successfully obtained cardiac recovery.

Compliance with ethical standards

Conflict of interest All authors have no conflict of interest.

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