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Case Report

Hemodynamics of cardiac tamponade during extracorporeal membrane oxygenation support in a patient with fulminant myocarditis

Toru Kondo (MD)[a], Ryota Morimoto (MD, PhD)[a,*], Tsuyoshi Yokoi (MD)[a], Shogo Yamaguchi (MD)[a], Tasuku Kuwayama (MD)[a], Tomoaki Haga (MD)[a], Hiroaki Hiraiwa (MD)[a], Yuki Sugiura (MD)[a], Naoki Watanabe (MD)[a], Naoaki Kano (MD)[a], Takeo Ichii (MD)[a], Kenji Fukaya (MD, PhD)[a], Akinori Sawamura (MD, PhD)[a], Takahiro Okumura (MD, PhD)[a], Tomo Yoshizumi (MD, PhD)[b], Masato Mutsuga (MD, PhD)[b], Kazuro Fujimoto (MD, PhD)[b], Naoyuki Matsuda (MD, PhD)[c], Akihiko Usui (MD, PhD)[b], Toyoaki Murohara (MD, PhD, FJCC)[a]

[a] Department of Cardiology, Nagoya University Graduate School of Medicine, Nagoya, Japan
[b] Department of Cardiac Surgery, Nagoya University Graduate School of Medicine, Nagoya, Japan
[c] Emergency and Critical Care Medicine, Nagoya University Graduate School of Medicine, Nagoya, Japan

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ABSTRACT

Fulminant myocarditis (FM) causes rapid onset severe heart failure requiring inotropes or mechanical circulatory support. Myocarditis is sometimes associated with pericardial effusion, however, how this effusion affects the hemodynamics in patients with FM under venoarterial extracorporeal membrane oxygenation (VA-ECMO) management has not been fully reported. We show a case of FM presenting with cardiac tamponade during VA-ECMO management. A 64-year-old female diagnosed as having FM showed a rapid hemodynamic collapse and that led to the application of VA-ECMO. Although her left ventricular ejection fraction did not improve despite proper hemodynamics management for several days, a pericardial effusion accumulated gradually. Apparent elevation of right atrial pressure and reduction of blood pressure were not observed, however, we performed pericardiocentesis because we were not able to wean off VA-ECMO. After the drainage of pericardial effusion, the blood pressure and cardiac output elevated as did the left ventricular ejection fraction. We successfully removed VA-ECMO and the patient was discharged without any complications. This is a case report in which a cardiac tamponade under VA-ECMO did not show typical signs and pericardiocentesis contributed to withdrawal of a VA-ECMO system.

<**Learning objective:** Typical findings of cardiac tamponade are less likely to appear in patients with fulminant myocarditis under venoarterial extracorporeal membrane oxygenation management (VA-ECMO). Drainage of pericardial effusion delivers dramatic improvement in blood pressure, cardiac output, and left ventricular ejection fraction. When VA-ECMO cannot be weaned off, pericardiocentesis should be considered in patients with fulminant myocarditis who showed gradual accumulation of pericardial effusion.>

Introduction

Fulminant myocarditis (FM) causes rapid progression to severe hemodynamic collapse and has a high mortality rate [1]. During its clinical course, FM may require peripheral venoarterial extracorporeal membrane oxygenation (VA-ECMO) to provide early temporary hemodynamic support. Pericardial effusion sometimes accompanies FM [2], however, the effect of pericardial effusion on the hemodynamic status in patients managed by VA-ECMO has not been fully described to date. We describe the case of cardiac tamponade in a patient with FM during VA-ECMO support and describe the resultant hemodynamics.

* Corresponding author at: Department of Cardiology, Nagoya University Graduate School of Medicine, 65 Tsurumai-cho, Showa-ku, Nagoya, Aichi 466-8550, Japan.
E-mail address: ryota.m0726@med.nagoya-u.ac.jp (R. Morimoto).

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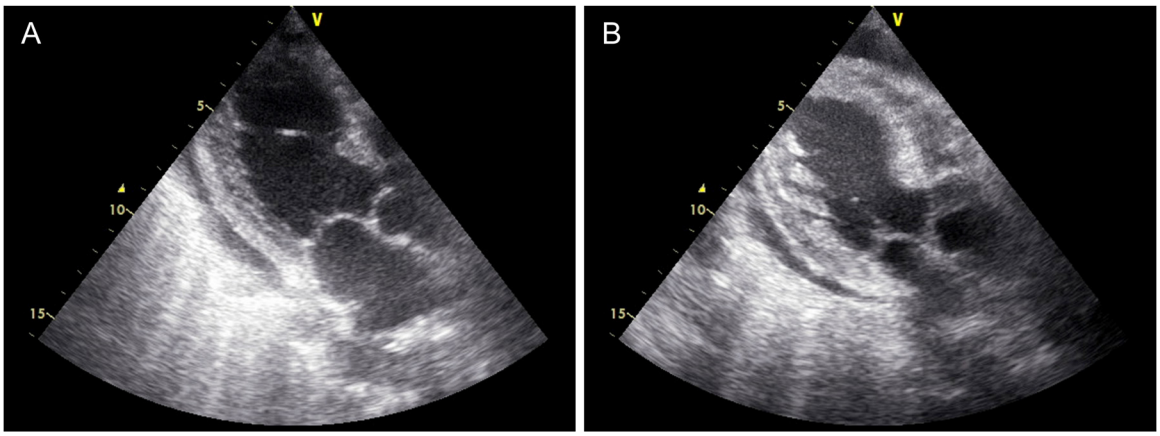

Fig. 1. Time course of the amount of pericardial effusion. (A) Slight pericardial effusion is observed behind the posterior wall of the left ventricle (7 mm) on admission. (B) Pericardial effusion increased in front of the right ventricle (16 mm) and behind the posterior wall of the left ventricle (8 mm) on day 5.

Case report

A 64-year-old female presented to the emergency department at another hospital with a 2-day history of fatigue and cough. Her blood pressure (BP) was 89/49 mmHg with a heart rate (HR) of 111 beats/min. Her body weight and body surface area were 54 kg and 1.55 m^2, respectively. An elevated cardiac enzyme (troponin T 1.84 ng/mL) was noted. The electrocardiogram revealed ST segment elevation in the II, III, aVF, and V4-6 precordial leads, and the echocardiography showed a generalized reduced left ventricular ejection fraction (LVEF) of 15%. Acute myocarditis was suspected as the cause of the reduced LVEF, and a cardiac catheter test and right ventricular endomyocardial biopsy were performed. Coronary angiography showed normal coronary arteries. The endomyocardial specimen revealed lymphocyte infiltration and myocardial necrosis without eosinophil degranulation or giant cell infiltration, and she was diagnosed as having acute lymphocytic myocarditis. Physicians initiated VA-ECMO because the patient's hemodynamics collapsed rapidly. The cannula sizes were 15 Fr for infusion and 19.5 Fr for drainage, via the right femoral vessels. She was transferred to our hospital for the intensive management of severe heart failure starting on the next day.

On arrival, her mean BP (mBP) was 59 mmHg under a blood flow rate of 2.5 L/min (1.6 L/min/m^2) with a rotation rate of 2500 rpm by Capiox® centrifugal pump (Terumo, Tokyo, Japan). Right atrial pressure was 15 mmHg, pulmonary capillary wedge pressure 20 mmHg, mean pulmonary artery pressure 22 mmHg, SvO_2 70%, respectively. Despite the continuous infusion of dobutamine (4 μg/kg/min) and dopamine (2 μg/kg/min), LVEF was severely reduced (10.3%) and a slight pericardial effusion was observed dominantly behind the posterior wall of the LV (7 mm) in the echocardiography (Fig. 1A , see Supplementary Videos 1 and 2). Intra-aortic balloon pumping was started for reducing cardiac afterload on day 1. Although proper hemodynamics was maintained (Fig. 2), her LVEF did not improve. Although pump flow was lower than expected considering the rotation speed of VA-ECMO, we decided not to change cannula position because the findings of apparent hemolysis were not observed and the total bilirubin level did not increase continuously. The pericardial effusion increased gradually and the swinging motion of the heart appeared in

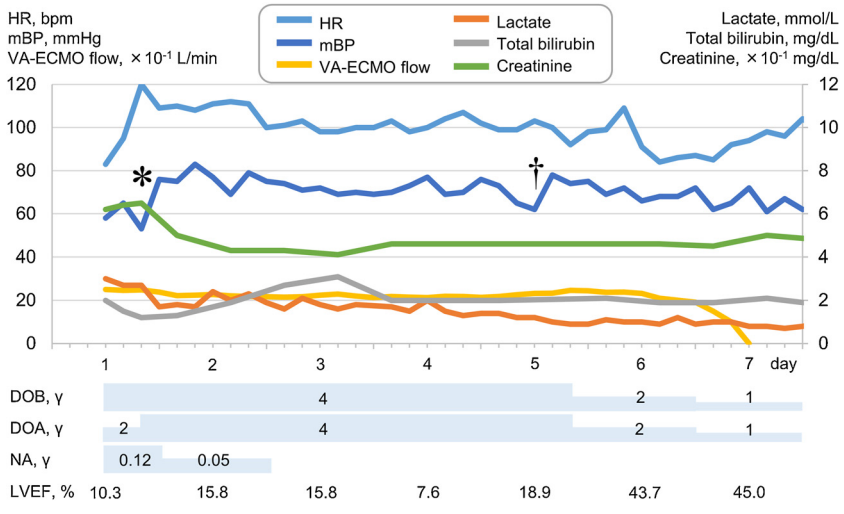

Fig. 2. Changes in hemodynamic findings, biomarkers, and inotrope usages during 1 week after transfer to our hospital.
*indicates the insertion of intra-aortic balloon pumping.
†indicates the pericardiocentesis.
HR, heart rate; mBP, mean blood pressure; VA-ECMO, venoarterial extracorporeal membrane oxygenation; DOB, dobutamine; DOA, dopamine; NA, noradrenaline; γ, μg/kg/min; LVEF, left ventricular ejection fraction.

Table 1 Hemodynamic parameters before and after drainage of pericardial effusion.

	Before drainage	After drainage
Blood pressure (mmHg)	62	78
Heart rate (beats/min)	103	100
Pulmonary capillary wedge pressure (mmHg)	12	14
Pulmonary artery pressure (mmHg)	14/11	26/18
Right atrial pressure (mmHg)	8	12
Left ventricular ejection fraction (%)	18.9	43.7
Left ventricular end-diastolic diameter (mm)	40.5	37.4
Cardiac output (L/min)	1.7	2.4

These parameters were recorded under a venoarterial extracorporeal membrane oxygenation (VA-ECMO) blood flow of 2.3 L/min (1.5 L/min/m^2) at a rotation rate of 2700 rpm. Cardiac output was evaluated under a blood flow of VA-ECMO less than 1.0 L/min.

echocardiography, which implied that this pericardial effusion affected her hemodynamic stability. On day 5, echocardiography was performed, and it showed a 16-mm echo-free space in front of the right ventricle and 8 mm behind the posterior wall of the LV (Fig. 1B, see Supplementary Videos 3 and 4). Despite accumulation of massive pericardial effusion, right atrial pressure was not elevated and her mBP (62 mmHg) was not severely reduced under a blood flow of 2.3 L/min (1.5 L/min/m^2) at a rotation rate of 2700 rpm. However, her mBP decreased to less than 50 mmHg and her own CO was 1.7 L/min (cardiac index: 1.1 L/min/m^2) when we reduced the blood flow of VA-ECMO to less than 1.0 L/min (0.7 L/min/m^2). Thus, we performed pericardiocentesis and drained 250 mL of blood-tinged pericardial fluid. Just after pericardiocentesis, mBP increased from 62 mmHg without pulsus paradoxus to 78 mmHg under a blood flow of 2.3 L/min (1.5 L/min/m^2) at a rotation rate of 2700 rpm (Table 1). Moreover, LVEF elevated from 18.9% to 43.7% (see Supplementary Video 5). Her own CO increased to 2.4 L/min (cardiac index: 1.6 L/min/m^2) under a blood flow of VA-ECMO less than 1.0 L/min (0.7 L/min/m^2). Pericardial effusion did not increase after the improvement in acute myocarditis and there were no abnormal findings in thyroid function and serum antinuclear antibody, therefore, pericardial effusion is thought to have been stored by pericarditis associated with myocarditis. We removed the VA-ECMO successfully and terminated the use of catecholamine on day 7. A further improvement in the LVEF was obtained (66.4%, on day 13), and we were able to discharge the patient from our hospital without any complications on day 25.

Discussion

The current case report demonstrates the hemodynamic change of cardiac tamponade in a patient with FM managed by VA-ECMO. This report suggests the possibility that an early detection and resolution of cardiac tamponade may contribute to improving the prognosis of FM which has a high mortality rate.

Cardiac tamponade is a life-threatening syndrome caused by slow or rapid compression of the heart due to the accumulation of fluid, pus, blood, clots, or gas [3]. The physical examination in cases with cardiac tamponade usually show tachycardia, hypotension with a narrow pulse pressure, and pulsus paradoxus. However, in patients with VA-ECMO, the blood flow rate and catecholamine administration significantly affect the BP and HR. Although the elevation of the right atrial pressure and pulmonary capillary wedge pressure are often observed in cardiac tamponade [4], blood drainage from the right ventricle by the VA-ECMO system leads to lower values for these parameters. In addition, sometimes right atrial and right ventricular chamber collapses occur because of hypovolemia and large blood drainage during VA-ECMO management, which attenuates the findings of cardiac tamponade. Thus, several mechanisms make it difficult to estimate whether pericardial effusion affects hemodynamics adversely in the patients with VA-ECMO. In fact, in our patient, we did not observe apparent reduction of BP and elevations in HR, right atrial pressure, and pulmonary capillary wedge pressure. If possible, hemodynamic and echocardiographic parameters should be assessed under the lowest VA-ECMO flow in order to reveal the signs of cardiac tamponade.

We observed marked improvements in BP and her own CO values just after the drainage of pericardial effusion. Surprisingly, LVEF was also elevated in this case, although LVEF has been shown to increase by the adrenergic stimulation due to compensation for the reduced stroke volume during cardiac tamponade in normal cases [5]. The reason for the LVEF improvement in our case just after pericardiocentesis was maybe that the cardiac tamponade did not induce adrenergic stimulation because of deep sedation and sufficient organ perfusion from the VA-ECMO support. In addition, according to Laplace's law, the relief of reduced left ventricular preload through the rise in the left ventricular radius and the left ventricular end-diastolic pressure by the drainage of pericardial effusion work to elevate the LVEF. Thus, cardiac tamponade under VA-ECMO management may lead to an underestimation of the underlying amelioration of cardiac function, causing prolonged VA-ECMO management and adverse outcomes.

When VA-ECMO cannot be weaned, pericardiocentesis should be considered in patients with FM who show gradual accumulation of pericardial effusion with swinging motion of the heart because its treatment delivers a dramatic hemodynamic improvement.

Acknowledgment

None.

Conflict of interest

None.

Appendix A. Supplementary data

Supplementary data associated with this article can be found, in the online version, at https://doi.org/10.1016/j.jccase.2018.08.009.

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